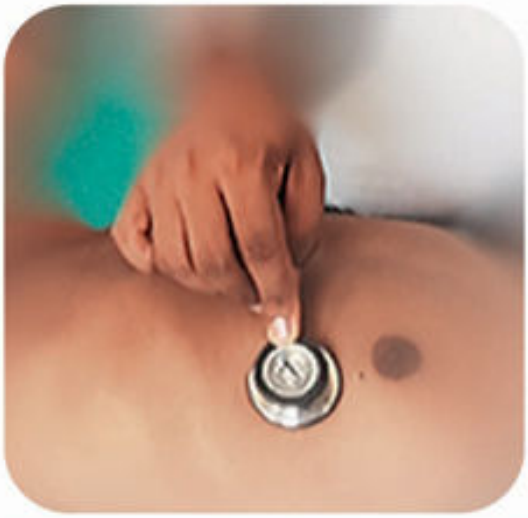

Archith Boloor
Anudeep Padakanti

An Insider's Guide to CLINICAL MEDICINE

- Comprehensive collection of all aspects of practical examination:
  - Long cases, short cases and semi-long cases
  - ECGs and X-rays
  - Instruments and spotters
  - Laboratory data interpretation

- Exclusive sections on case sheet format, diagnosis format and system-wise summary of findings
- Separate annexures on definitions and grading systems
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Foreword
Chakrapani M

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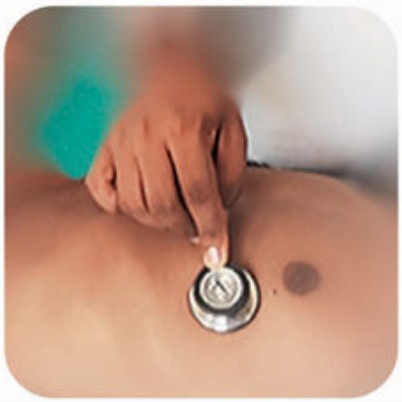

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Dr Sitamahalakshmi

Foreword

Medicine is a science and an art. Clinical examination is fast becoming a forgotten art in the face of technological onslaught. This book is an important step in bringing the students back to the basics of clinical medicine. This book will be valuable for examination preparations. It is a comprehensive compilation of clinical signs for students of internal medicine—both undergraduates and postgraduates. Illustrations are self-explanatory and help in understanding difficult concepts.

Dr Archith has been actively and extensively involved in the clinical teaching of undergraduate and postgraduate students for many years. He has been a popular teacher among medical students and has received “best teacher award” many times at Kasturba medical College, Mangaluru, Karnataka, India. He has understood the limitations of the present clinical examination books and also identified the knowledge gap that needs to be cleared for undergraduate and postgraduate students. His student Dr Anudeep, an enthusiastic learner and teacher has initiated the process of compiling this wonderful book.

Many common concepts which are very pertinent and relevant for university clinical examinations are discussed in detail in this book. Coverage of the topics are comprehensive, contemporary, and clear.

The authors have done extensive research while compiling the details in the book and has presented it in a very convenient to understand format by giving the details of many of these concepts in the form of tables and bullet notes. This will help the student in remembering the important points. They have explained the basic concepts, and this will help the student in understanding and then performing the clinical examinations.

Information compiled in the book is evidence-based and experience enhanced by an eminent teacher. They have taken the feedback from all the stakeholders including teachers and students before finalizing the final version of this book. This book can be strongly recommended for students, teachers and practising physicians.

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Preface

The clock had struck a solid 1:30 PM. The examiner was hungry, the last student was jittery and in between them lay a central nervous system (CNS) case that was going to determine whether a four-and-half-year ripe child of medicine would be prefixed with a "Dr" or not.

The examiner was more bored than he could care to admit. Lakshman, aged 32, hailing from Shivamogga, Karnataka with chief complaints of bilateral lower limb weakness was being presented for the 14th time that day. The same boring questions had been asked in the same uninspired fashion.

"List the causes of neck pain", the examiner asked.

A little taken aback but the student realized that the question was within the realm of a CNS case. After gathering his thoughts for a

moment, he began listing out, “Meningitis causing neck muscle spasm, cervical spondylosis, cervical spondylolisthesis...” his voice trailing off in response to the examiner’s unimpressed face.

”Go ahead, what else?”

Not to lose face in front of the examiner, the student once again reset his thoughts, and a few umms and ahhs later continues:

”Sir, other cervical causes like cervical intraepithelial neoplasia, cervical cancer, etc. can also cause neck pain”.

Jokes apart, getting psyched for an exam is an absolutely normal and foreseeable predicament. We often notice the most brilliant students fumbling to show off years’ worth of hard work simply because the psyche overpowers their preparation. As the saying goes “For most diagnoses, all that is needed is an ounce of knowledge, an ounce of intelligence, and a pound of thoroughness.” With that very thought in mind, it is our pleasure to present to you a simple, comprehensive and exam-oriented clinical manual: A compass to guide you through the art of clinical medicine.

The practical examinations pose a real challenge to the medical student: He has to finish writing an entire case sheet, elicit the expected clinical findings and finally arrive at a proper diagnosis. All this to be done before the examiner has even made eye-contact with the student. The catch here being the limited availability of what we all take for granted: Time. One asks the wrong questions, examines the wrong systems, latches on to the wrong points and before we realize, we are knee-deep in heaps of unorganized information that has no head or tail. Having been in the same shoes at some point in the past, this book was made to solve those problems: complete case sheets on all organ systems, with added emphasis on the common examination cases have been incorporated. We hope it will teach the reader to anticipate questions that are asked in different contexts. The book is as visually charged as we could possibly make it because we believe that seeing is learning. We have dealt with spot and short cases which are meant to test a student’s take on the bigger picture of diseases. The diagnostic clues given in this book

will help the student to arrive at a definitive decision sooner. X-rays, spotters and instruments are dealt with extensively and in exquisite detail.

We have read several clinical books in an attempt to make this one different. In doing so, we have found that this is one single guide which can be safely relied upon to deal with the practicals of Final MBBS Part II. We hope that the fruit of our labor becomes as close to your bookshelf as it is to our hearts. Any suggestions and/or constructive criticism is always welcome, and we hope you enjoy reading *An Insider's Guide to Clinical Medicine*.

Archith Boloor

Anudeep Padakanti

Remembering the Father of Modern Medicine

Medicine is a science of uncertainty and an art of probability.

The best preparation for tomorrow is to do today's work superbly well.

Every patient you see is a lesson in much more than the malady from which he suffers. Listen to your patient. He is telling you the diagnosis.

He who studies medicine without books sails an uncharted sea, but he who studies medicine without patients does not go to sea at all.

The good physician treats the disease; the great physician treats the patient who has the disease.

We are here to add what we can to life. Not to get what we can from life. Too many men slip early out of the habit of studious reading and yet that is essential.

One of the duties of the physician is to educate the masses not to take medicine.

The practice of medicine is an art. Not a trade; a calling. Not a business: A calling in which your heart will be exercised equally with your head.

Happiness lies in the absorption in some vocation which satisfies the soul. To have striven. To have made the effort. To have been true to certain ideals------ this alone is worth the struggle.

Acquire the art of detachment, the virtue of method and the quality of thoroughness but above all the grace of humility.

Sir William Osler,
(July 12, 1849 – December 29, 1919)

Acknowledgments

It was our long-standing dream to write a clinical book that would encompass all the relevant matter needed for a student with due emphasis on clinical methods. Incorporating many years of clinical teaching and an astute understanding of the actual needs of a medical student, this book has been compiled to cater to their unmet needs. It has been a Herculean task of reading, writing, rewriting and editing this vast amount of information into this concise textbook.

When we began this work, almost a year ago, little did we anticipate the shape our ideas would finally take in the form of this *"An Insider's Guide to Clinical Medicine"*. This endeavor of ours would have been impossible without the constant support and encouragement of our well wishers.

Firstly, we thank all our students : undergraduates, postgraduates for having kindled in us this idea, for compiling our notes and most importantly, for asking the questions whose answers have taken the form of this book.

This book would not have seen the light of day without the constant persuasion of Dr Vivek Koushik, Dr Abu Thajudeen and Dr Nikhil Kenny Thomas. They are and will continue to be the pillars of strength on whom our life and this book would gain sustenance… Thank you.

We profusely thank Dr Chakrapani M, for writing the foreword for this edition. Sir is the embodiment of a true teacher of clinical medicine and we thank him for his constant support and inputs during this process.

We thank Dr Sheetal Raj for the chapter on Comprehensive Geriatric assessment. We thank Dr Sriraksha Nayak and Dr Vaddi

Rohit, for compiling the chapter Approach to Psychiatric illness.

We thank Dr Kaushiki Kirty, Dr Vishnu B Chandran, Dr Rama Kishore Yalampati and Dr Navyashree HC, for helping us with inputs and proofreading.

Also, we convey our sincere thanks to Shri Jitendar P Vij (Group Chairman), Mr Ankit Vij (Managing Director), Mr MS Mani (Group President), Dr Madhu Choudhary (Publishing Head–Education), Ms Pooja Bhandari (Production Head), Ms Sunita Katla (Executive Assistant to Group Chairman and Publishing Manager), Dr Aakanksha Shukla Sirohi (Development Editor), Mr Rajesh Sharma (Production Coordinator), Ms Seema Dogra (Cover Visualizer), Mr Laxmidhar Padhiary (Proofreader), Mr Kapil Dev Sharma (Typesetter), Mr Manoj Pahuja (Graphic Designer) and their team members, for publishing the book in the same format as wanted, well in time.

Special thanks to Dr Ashwini MV, Dr G Suresh Reddy, Dr Lakshmi Nivedana B Dr Sriram M, Dr Pranjal Sharma, Dr Tejaswini Lakshmikeshava, Dr Nagendra C, Dr Thejus Bhaskar, Dr Mohammed Shaheen, Dr Jane Mendonca and Dr Madhav Hande, for helping us with the clinical images, editing, proofreading and designing of this book. They have lived our dream with us.

We are especially grateful for the ongoing encouragement from the management and administration of our university, the Manipal Academy of Higher Education (MAHE), Manipal, Karnataka, India.

We are grateful to our family members, colleagues and friends who have supported us all along the way.

A very special gratitude goes out to all our teachers, who are solely responsible for what we are today and for having ignited the passion of teaching in us.

Lastly, we thank God Almighty, for making us what we are, guiding us through our life, and helping us in bringing this book to you all.

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Case Sheet Format
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Discussion

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Case Sheet Format
Diagnosis Format
Discussion on Examination
Discussion on Diseases and Diagnostic Criteria

Abbreviations

°C : Degree celcius
°F : Degree Farenheit
ALL : Acute lymphoblastic leukemia
ABPA : Allergic bronchopulmonary aspergillosis
ACS : Acute coronary syndrome
ACR : American college of Rheumatology
ARF : Acute renal failure
ADHD : Attention deficit hyperactivity disorder
ADR : Adverse drug reaction
ARDS : Acute respiratory distress syndrome.
AGN : Acute glomerulonephritis
AION : Anterior ischemic optic neuritis
AKI : Acute kidney injury
ALL : Acute lymphoblastic leukemia
ASCVD : Atherosclerotic cardiovascular disease
ACD : Anaemia of chronic disease
ADC : Apparent diffusion coefficient
ACA : Anterior cerebral artery
ACE : Addenbrooke’s cognitive examination
ACEI : Angiotensin converting enzyme inhibitor
ARB : Angiotensin receptor blocker
ACTH : Adrenocorticotropic hormone
ADHF : Acute decompensated heart failure
AEM : Ambulatory electrocardiogram monitoring
AI : Aortic insufficiency
AIDP : Acute inflammatory demyelinating polyneuropathy
AF : Atrial fibrillation
AICA : Anterior inferior cerebellar artery
AICD : Automated implantable cardioverter defibrillator
AML : Acute myeloid leukemia
ANS : Autonomic nervous system

ARVD : Arrhythmogenic right ventricular dysplasia
ASD : Atrial septal defect
AVF : Arteriovenous fistula
AVM : Arteriovenous malformation
AVR : Aortic valve replacement
AVRT : Atrioventricular re-entrant tachycardia
AVNRT : AV nodal re-etrant tachycardia
APB : Atrial premature beat
ALS : Amyotropic lateral sclerosis
ADL : Activities of daily living
ACPA : Anticitrullinated protein antibody
APLA : Antiphospholipid antibody syndrome
AP : Anteroposterior
Bx : Biopsy
BAL : Bronchoalveolar concentration
B/L : Bilateral
BIH : Benign intracranial hypertension
BAV : Bicuspid aortic valve
BBB : Bundle branch block
BC : Bone conduction/blood culture
BCAT : Brief cognitive assessment tool
BER : Benign early repolarization
BLS : Basic life support
BSA : Body surface area
BP : Blood pressure
BT : Bleeding time
BUN : Blood urea nitrogen
BM : Bone marrow
BMI : Body mass index
BMV : Bag and mask ventilation/balloon mitral valvotomy
BVP : Biventricular pacing
B-ALL : B-cell acute lymphoblastic leukemia
BADL : Basic activities of daily living
CRP : C-reactive protein
CXR : Chest X-ray
CCA : Common carotid artery
C/L : Contralateral
CMT : Charcot Marie tooth Disease
CN : Cranial nerve
C/O : Complaints of
CT : Computed tomography

CAMCOG : Cambridge cognitive examination
COST : Cognitive state test
CPR : Cardiopulmonary resuscitation
CCF : Congestive cardiac failure
CHF : Congestive heart failure
CBC : Complete blood count
CBD : Common bile duct
CHB : Complete heart block
CKD : Chronic kidney disease
CIDP : Chronic inflammatory demyelinating polyneuropathy
CLD : Chronic liver disease
CLL : Chronic lymphoid leukemia
CML : Chronic myeloid leukemia
CMV : Cytomegalovirus
CNS : Central nervous system
CVA : Cerebrovascular accident
CNS : Central nervous system
CABG : Coronary artery bypass graft
CAD : Coronary artery disease
CAUTI : Catheter associated UTI
CBE : Clinical breast examination
CRF : Chronic renal failure
COPD : Chronic obstructive pulmonary disease
CCCU : Critical coronary care unit
CCS : Canadian cardiovascular society
CVS : Cardiovascular system
CVP : Central venous pressure
CP angle : Cerebellopontine angle
CPB : Cardiopulmonary bypass
CDC : Centers for disease control and prevention
CDAI : Clinical disease activity index
CGA : Comprehensive geriatric assessment
CSF : Cerebrospinal fluid
DDx or D/D : Differential diagnosis
DPI : Dry powder inhaler
DIC : Disseminated intravascular coagulation
DIP joint : Distal interphalangeal joint
DKA : Diabetic ketoacidosis
DLCO : Diffusion lung capacity for carbon monoxide
DM : Diabetes mellitus
DR : Diabetic retinopathy

DNR : Do not resuscitate
DTR : Deep tendon reflex
DTA : Descending thoracic aorta
DSM : Diagnostic and statistical manual of mental disorders
DVT : Deep venous thrombosis
DLE : Disseminated lupus erythematosus
DAS : Disease activity score
DWI : Diffusion weighted imaging
ECA : External carotid artery
EAT : Ectopic atrial tachycardia
ECG : Electrocardiogram
ECF : Extracellular fluid
ECHO : Echocardiogram
ECMO : Extracorporeal membrane oxygenation
EPS : Extrapyramidal system
EF : Ejection fraction
EM : Erythema multiforme
ECD : Endocardial cushion defects
EDH : Extradural hematoma
EOM : Extraocular muscles/movement
EPO : Erythropoietin
EDM : Early diastolic murmur
ESM : Ejection systolic murmur
ESRD : End-stage renal disease
ET : Endotracheal tube
ESV : End-systolic volume
EULAR : European league against rheumatism
FMS : Fibromyalgia syndome
FBS : Fasting blood sugar
FEV1 : Forced expiratory volume in first second
FTT : Failure to thrive
FVC : Forced vital capacity
GI : Gastrointestinal
GBS : Guillain–Barré syndrome
GCS : Glasgow coma scale
GERD : Gastroesophageal reflux disease
GH : Growth hormone
Hb : Hemoglobin
HMF : Higher mental functions
HOCM : Hypertrophic obstructive cardiomyopathy
HBV : Hepatitis B virus

HL : Hodgkin lymphoma
HUS : Hemolytic uremic syndrome
HAI : Hospital acquired infection
HE : Hepatic encephalopathy
HDS : Hemodynamically stable
HIT : Heparin induced thrombocytopenia
HCC : Hepatocellular carcinoma
HTN : Hypertension
HIV/AIDS : Human immunodeficiency virus/acquired immunodeficiency syndrome
HDL : C-High density lipoprotein cholesterol
HD : Huntington's disease
IADL : Instrumental activities of daily living
IP joint : Interphalangeal joint
IGF : Insulin-like growth factor 1
ICA : Internal carotid artery
ICD : Intercostal drain
ICS : Intercostal space/inhaled corticosteroid
ICH : Intracerebral hemorrhage
IVH : Intraventricular hemorrhage
INO : internuclear ophthalmoplegia
INR : International normalized ratio
ICP : Intracranial pressure
IBD : Inflammatory bowel disease
IBS : Irritable bowel syndrome
IDDM : Insulin-dependent diabetes mellitus—Type 1 diabetes
ICSOL : Intracranial space occupying lesion
IHD : Ischemic heart disease
IJV : Internal jugular vein
ILD : Interstitial lung disease
IMN : Infectious mononucleosis
IVC : Inferior vena cava
INH : Isoniazid
IPPV : Intermittent positive pressure ventilation
ITP : Immune thrombocytopenic purpura
IV : Intravenous
JME : Juvenile myoclonic epilepsy
JRA : Juvenile rheumatoid arthritis
JVP : Jugular venous pressure
KUB : Kidney, ureters and bladder
KDIGO : Kidney disease improving global outcomes
KF Ring : Kayser fleischer ring

LSM : Late systolic murmur
LV : Left ventricle
LVH : Left ventricular hypertrophy
L\A : Local anesthetic
LDL : C-Low density lipoprotein cholesterol
LP : Lumbar puncture
LMN : Lower motor neuron
LVE : Left ventricular enlargement
LVF : Left ventricular failure
LOC : Loss of consciousness
LQTS : Long QT syndrome
LGIB : Upper gastrointestinal bleed
MAP : Mean arterial pressure
MAT : Multifocal atrial tachycardia
MoCA : Montral cognitive assessment
MMSE : Mini-mental state examination
MCA : Middle cerebral artery
MCP joint : Metacarpophalangeal joint
MDS : Myelodysplastic syndrome
MDM : Mid-diastolic murmur
MLF : Medial longitudinal fasciculus
MND : Motor neuron disease
MS : Mitral stenosis/multiple sclerosis
MVP : Mitral valve prolapse
MVR : Mitral valve replacement
MSA : C-Multisystem atrophy—cerebellar
MSA : P-Multisystem atrophy—Parkinson’s
MCTD : Mixed connective tissue disease
MI : Myocardial infarction
MRC : Medical research council
mMRC : Modified medical research council
MRI : Magnetic resonance imaging
MDI : Metered dose inhaler
MODS : multiorgan dysfunction syndrome
NHL : Non-Hodgkin lymphoma
NASH : Non-alcoholic steatohepatitis
NCV : Nerve conduction velocity
NMJ : Neuromuscular junction
NPPV : Noninvasive positive pressure ventilation
NPH : Normal pressure hydrocephalus
NTS : Nucleus Tractus solitarius

REM : Rapid eye movement
NREM : Non rapid eye movement
NST : Non-stress test
NSTEMI : Non-ST-Elevation myocardial infarction
NSAIDs : Nonsteroidal anti-inflammatory drugs
NYHA : New York heart association
NG Tube : Nasogastric tube
O/E : On examination
OSA : Obstructive sleep apnea
OA : Osteoarthritis
OP : Organophosphorus
PA : Posteroanterior
PAN : Polyarteritis nodosa
PDA : Patent ductus arteriosus
PAH : Pulmonary Artery Hypertension
PCI : Percutaneous Coronary Intervention
PCA : Posterior cerebral artery
PCV : Packed Cell Volume
PCWP : Pulmonary Capillary Wedge Pressure
PD : Parkinson's Disease
PE : Pulmonary Embolism
PEEP : Positive End Expiratory Pressure
PEFR : Peak Expiratory Flow Rate
PAH : Pulmonary Artery Hypertension
PIP Joint : Proximal interphalangeal joint
PICA : Posterior inferior cerebellar artery
PLS : Progressive Lateral Sclerosis
PND : Paroxysmal Nocturnal Dyspnea
PUO/FUO : Pyrexia (fever) of Unknown Origin
PVC : Premature Ventricular Contractions
pO_2/paO_2 : Partial pressure of oxygen
$paCO_2$: Partial pressure of carbon dioxide
PMI : Point of maximal impulse
PPBS : Post-prandial Blood Sugars
qSOFA : Quick sequential organ failure assessment
QSART : Quantitative sudomotor axon reflex test
RA : Rheumatoid Arthritis
RF : Rheumatoid factor
RAI scan : Radioactive iodine scan
RAS : Reticular activating system
RAPD : Relative apparent pupillary defect

RCM : Restrictive cardiomyopathy
RCC : Renal cell carcinoma
RDW : Red cell distribution width
RS : Respiratory system
RSOV : Ruptured sinus of valsalva
RHD : Rheumatic heart disease
RLN : Recurrent laryngeal nerve
RR : Respiratory rate
RV : Right ventricle
RVH : Right ventricular hypertrophy
RVF : Right ventricular failure
REMS : Regional examination of musculoskeletal system
SAAG : Serum–ascites albumin gradient
SAH : Subarachnoid hemorrhage
SACD : Subacute combined degeneration of cord
SANRT : Sinoatrial node re-entrant tachycardia
SLRT : Straight leg raise test
SOFA : Sequential organ failure assessment
SIRS : Systemic inflammatory response syndrome
SSPE : Subacute sclerosing pan-encephalitis
SDAI : Simplified disease activity index
STMS : Short test of Mental status
SV : Stroke volume
SVT : Supraventricular tachycardia
SMA : Spinal muscular atrophy
SDH : Subdural haematoma
SCM : sternocleidomastoid
SLE : Systemic lupus erythematosus
STEMI : ST-Elevation myocardial infarction
SVC : Superior vena cava
SSR : Sympathetic skin response
SLICC : Systemic lupus international collaborating clinics
TAPVC : Total anomalous pulmonary venous connection
TIA : Transient ischemic attack
TB : Tuberculosis
TBI : Traumatic brain injury
TIN : Tubulointerstitial nephritis
TG : Triglycerides
TST : Thermoregulatory sweat test
TMJ : Temporomandibular joint
TSH : Thyroid stimulating hormone

U/L : Unilateral
UA : Unstable angina
UMN : Upper motor Neuron
UIP : Usual interstitial pneumonitis
UGI : Upper gastrointestinal
UGIB : Upper gastrointestinal bleed
URTI : Upper respiratory tract infection
UTI : Urinary tract infection
US/USG : Ultrasonogram
VA : Visual acuity
VAP : Ventilator acquired pneumonia
VC : Vital capacity
VDRL : Venereal disease research laboratory
VPC : Ventricular premature contractions
VSD : Ventricular septal defect
VT : Ventricular tachycardia
V/Q scan/ratio : Ventilation/perfusion
VUR : Vesicouretreric reflux
WHO : World health organisation
WPW : Wolff–Parkinson–White syndrome
ZES : Zollinger ellison syndrome

Prerequisites for Practical Examination and Common Examination Cases

CHAPTER 1

PREREQUISITES FOR PRACTICAL EXAMINATION

Clinical skills, such as the physical examination remain an important instrument in the physician's armamentarium and assessment of these skills form the basis of the final clinical examination. Every student appearing for the examination will be under a lot of stress, which even though justifiable becomes detrimental for the performance of the student. Here are some suggestions:

1. The first and foremost is preparation. Try to have a timetable and cover all important cases well in advance. You have a set of cases that are usually kept for the examination and most of the questions asked are also predictable. Do not keep any important things pending to read on the day prior to examination.
2. Sleep is of utmost importance on the day prior to the exam. You need to sleep for a **minimum 4–5 hours on the day prior to the exam**. The curriculum being vast, compromising a few hours of sleep would do more harm than good.
3. Have a **light breakfast**. Hypoglycemia hampers your thought process, delays your reaction time and severely impairs the performance. Agreed that the feel of exam maybe like undergoing a surgery, but NIL PER ORAL status is not needed.

4. Attire is important. Be neatly groomed and dressed. Wear a clean apron with a number badge.
5. Carry all your instruments.
6. Write a detailed case sheet. Examine each case thoroughly. Never rely on expert's diagnosis. Make your own diagnosis. Always justify it with your own views.
7. Stick to the set time limits. Do not waste time.
8. Be gentle to the patient when you examine. The more cooperative the patient is, the better will be your performance. Always take the permission of the patient and explain before examining and do not forget to thank them at the end.
9. Never forget to wish the examiner good morning/evening. If you do not know an answer, say sorry! (Most of the examiners will change the question or give you a clue). Always finish with a thank you!
10. Confidence is of paramount importance. Practice presenting cases without referring to the case sheet. Be clear in the order of presentation, both history and examination. Stress on relevant important findings. To be expressive is important, but not over expressive. Eye-contact is essential. Answer clearly and to the point. Do not speak about rare causes. When demonstrating signs, do it clearly.
11. Most importantly, have faith in yourself and your preparation. You shall succeed.

CHECKLIST FOR PRACTICAL EXAMINATION

1. Clean apron with roll number tag
2. Hall ticket
3. Stationery
4. Stethoscope with a bell
5. Knee hammer
6. Key (to test plantar reflex, stereognosis)
7. Wristwatch with seconds needle

8. Measuring tape
9. Two scales
10. Pins
11. Glass slides
12. Two small boxes for testing smell (soap and coffee)
13. Four boxes for testing taste (sugar, salt, bitter and sour)
14. Four cards with the words “sweet”, “sour”, “bitter” and “salt” written on them.
15. Snellen’s chart
16. Ishihara’s chart
17. Cotton
18. Tuning forks (128 Hz and 512 Hz)
19. Divider
20. Ophthalmoscope with full batteries
21. Torch with full batteries
22. Thermometer
23. Tongue depressor
24. Cotton wick/throat swab stick—gag reflex
25. Two test tubes preferably aluminum for temperature testing (glass test tubes may be used if aluminium test tubes are not available)
26. Pulse oximeter (not mandatory)
27. Gloves
28. Mask
29. Hand rub

FORMAT OF CLINICAL EXAMINATION

The general format of cases in the examination is as follows:

Type of case	Time given for examination of patient	Time for clinical viva	Marks
Long	45–60 min	15–20 min	50/40

	Detailed case sheet needed		marks
Short	15 min	7–10 min	20 marks
Semilong	15 min	7–10 min	20 marks
Spotters	1 min	2–3 min	5 marks each
Charts (laboratory data, clinical)	1 min	2–3 min	5 marks each
OSCE (any clinical sign)	5 min	5 min—observed	5–10 marks each
Viva voce	4 table vivas, each carrying 5 marks, each timed for 5 minutes Topic—X-rays, ECG, instruments, drugs, charts, general viva		

COMMON EXAMINATION CASES

Respiratory system	
Long case	***Short case***
• Bronchial asthma • Emphysema • Chronic bronchitis • Bronchiectasis • Pleural effusion/empyema • Lung abscess • Bronchial carcinoma • Consolidation • Pneumothorax • Hydropneumothorax • Collapse of the lung • Diffuse parenchymal lung disease/Interstitial lung disease • Fibrosis/fibrocavity • Fibrothorax	• Bronchial asthma • Emphysema • Chronic bronchitis • Bronchiectasis • Pleural effusion/empyema • Lung abscess • Bronchial carcinoma • Consolidation • Pneumothorax • Hydropneumothorax • Collapse of the lung • Diffuse parenchymal lung disease/Interstitial lung disease • Fibrosis/fibrocavity • Fibrothorax

Cardiovascular system	
Long case	***Short case***
• Mitral stenosis • Mitral regurgitation • Mixed mitral stenosis with mitral regurgitation • Aortic stenosis • Aortic regurgitation • Mixed aortic stenosis and regurgitation • Multivalvular heart diseases • Subacute bacterial endocarditis • Eisenmenger's syndrome • Tetralogy of Fallot • Ventricular septal defect • Atrial septal defect • Patent ductus arteriosus • Hypertrophic cardiomyopathy • Dilated cardiomyopathy • Congestive cardiac failure	• Mitral stenosis • Mitral regurgitation • Mixed mitral stenosis with mitral regurgitation • Aortic stenosis • Aortic regurgitation • Mixed aortic stenosis and regurgitation • Hypertension • Subacute bacterial endocarditis • Rheumatic fever • Eisenmenger's syndrome • Tetralogy of Fallot • Ventricular septal defect • Atrial septal defect • Patent ductus arteriosus • Coarctation of aorta • Hypertrophic cardiomyopathy • Dilated cardiomyopathy • Congestive cardiac failure
Gastrointestinal system	
Long case	***Short case***
• Jaundice • Acute/chronic hepatitis • Chronic liver disease (cirrhosis of liver) • Liver abscess • Ascites • Hepatomegaly • Splenomegaly • Hepatosplenomegaly • Polycystic kidney disease	• Jaundice • Acute/chronic hepatitis • Chronic liver disease (cirrhosis of liver) • Liver abscess • Ascites • Hepatomegaly • Splenomegaly • Hepatosplenomegaly • Polycystic kidney disease
Nervous system	
Long case	***Short case***
• Cerebrovascular disease	• Motor system examination

• Ataxia • Peripheral neuropathy • Guillain–Barré syndrome • Chronic inflammatory demyelinating polyneuropathy • Myasthenia gravis • Spastic paraplegia (cord compression) • Transverse myelitis • Myopathy • Parkinsonism • Motor neuron disease • Multiple sclerosis	• Facial nerve palsy • Foot drop • Claw hand • Examination of cranial nerves • Cerebellar signs • Involuntary movements • Sensory system examination

Semilong cases/therapeutic cases	
Renal	• Nephrotic syndrome • Glomerulonephritis • Chronic kidney disease
Rheumatology	• Systemic lupus erythematosus • Rheumatoid arthritis • Ankylosing spondylitis • Systemic sclerosis
Endocrine	• Diabetes mellitus • Hypothyroidism • Graves' disease (with thyrotoxicosis) • Cushing's syndrome • Addison's disease • Hypopituitarism • Acromegaly • Obesity • Short stature
Hematology	• Anemia • Bleeding disorders • Hepatosplenomegaly • Lymphadenopathy
General	• Pyrexia of unknown origin • Hypertension • Edema

	• Heart failure • Dyspnea • Comprehensive geriatric assessment

General Examination

CHAPTER 2

A. CASE SHEET FORMAT

PATIENT

- Conscious
- Oriented
- Cooperative
- Obeying commands.

BODY MASS INDEX (BMI)

- Weight (kg)/Height (m^2)
- Grading according to World Health Organization (WHO) for Southeast Asian countries.

VITALS EXAMINATION

- Pulse
  - Rate
  - Rhythm
  - Volume
  - Character
  - Vessel wall thickening
  - Radio-radial delay and radio-femoral delay
  - Peripheral pulses
- Blood pressure
  - Right arm
  - Left arm
  - Both legs
- Respiration
  - Rate
  - Abdominothoracic (male) or thoracoabdominal (female)
  - Usage of accessory muscles
- Jugular venous pulse
  - Waveform
- Jugular venous pressure
  - _____ cm of blood/water above sternal angle (+ 5 cm water from right atrium)
- Temperature ____ degree of °C or °F measured at _____ site
- Pulse oximetry
- Pain

PHYSICAL EXAMINATION

- Pallor
- Icterus
- Cyanosis

- Clubbing
- Lymphadenopathy
- Edema

OTHERS

Note: General physical examination findings relevant to each system shall be discussed in the respective chapters.

B. VITALS EXAMINATION

PULSE

Definition

Pulse is defined as a pressure distension wave produced by the contraction of the left ventricle against a partially filled aorta which is transmitted to peripheries and is felt on a peripheral artery against a bony prominence.

Assessment of arterial pulse	
Characteristics	***Best assessed by palpating***
Rate	Radial artery
Rhythm	
Volume	Carotid artery
Character or quality	Carotid artery **Exceptions:** • Collapsing pulse, pulsus alternans and pulsus paradoxus are appreciated at the radial artery • Pulsus bisferiens best appreciated in brachial artery
Radio-radial and radio-femoral delay	
Whether all peripheral pulses are felt	
Condition of vessel wall	

Example: 72 beats per minute, regular rhythm, normal volume and character, all peripheral pulses are well felt, no radio radial or radiofemoral delay, no vessel wall thickening

Method of Palpation of Radial Artery (Fig. 2B.1)

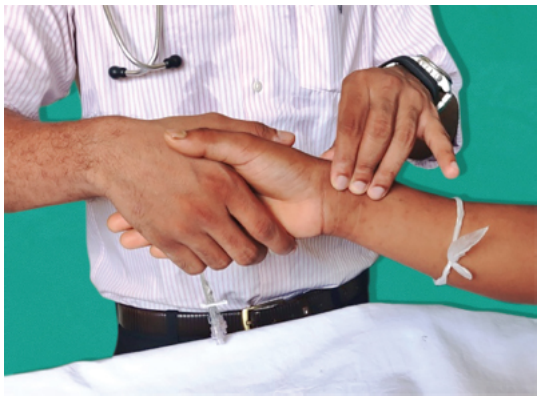


Fig. 2B.1: Method of palpation of radial artery.

The radial pulse is felt using 3 fingers. The distal finger is to prevent the backflow, proximal finger is to stabilize artery on the bone and middle finger is used to feel and count the pulse (3-finger method).

Another accepted method of palpating the pulse is by using two fingers.

Pulse Rate

Calculate the rate by counting the radial pulse for **one full minute**. Normal heart rate is 60–100 beats per minute.

<60 (bradycardia)	>100 (tachycardia)
Physiological: Athletes, sleep **Pathological:** • Severe hypoxia • Hypothyroidism/myxedema • Obstructive jaundice • Hypothermia • Sick sinus syndrome • Drugs—β-blockers, verapamil, and digoxin • Heart block • Raised intracranial tension (Cushing's reflex)	**Physiological:** Infants, children, emotion, exertion, anxiety and pregnancy **Pathological:** • Tachyarrhythmias • High output states: Severe anemia, thyrotoxicosis, beri-beri, Paget's disease of the bone, cirrhosis of liver, AV fistula • Cardiac failure • Cardiogenic shock • Drugs (e.g. atropine, nifedipine, salbutamol, terbutaline, nicotine, and caffeine)

Relationship between pulse to temperature	
For every degree F rise in temperature, the pulse rate increases by 10	
Relative tachycardia	***Relative bradycardia***
• Acute rheumatic carditis • Diphtheric myocarditis • Tuberculosis	• Yellow fever (Faget's sign) • Dengue fever • First week of enteric fever • Pyogenic meningitis/intracerebral abscess • Brucellosis • Legionella • Psittacosis • Typhus • Q fever • Leptospirosis • Noninfectious: – Patients on β-blockers – Lymphomas – Factitious fever – Drug fever

Rhythm

Rhythm is assessed by palpating the radial pulse. The normal rhythm is regular.

Causes of irregular rhythm
Regularly irregular • Atrial tachyarrhythmias with fixed AV blocks, sinus arrhythmia, partial/second degree atrioventricular (AV) blocks • Ventricular bigeminy and trigeminy
Irregularly irregular • Ventricular ectopics/ventricular premature complexes (VPCs) • Atrial fibrillation (AF) • Atrial tachyarrhythmia with varying AV blocks
Regular with occasional irregularity • Extrasystoles

Arrhythmias with Regular Rhythm

1. Atrial flutter
2. Ventricular tachycardia
3. First degree heart block
4. Second degree heart block

Pulse deficit (Apex-pulse deficit) (Fig. 2B.2) is the difference between the heart rate (counted by auscultation) and pulse rate when counted simultaneously for one full minute by two individuals.

Causes

Pulse deficit of more than 10/minute occurs in atrial fibrillation (AF) and less than 10/minute may be found with ventricular premature beats or slow/controlled AF.

Differences Between Atrial Fibrillation and Ventricular Premature complexes (VPCs)

	Atrial fibrillation	VPCs
Apex pulse deficit	Usually >10	Usually <10
JVP 'a' wave	Absent	Normal
S_1	Variable intensity	Normal
Effect of exercise/hand grip	Irregularity persists	Pulse becomes regular

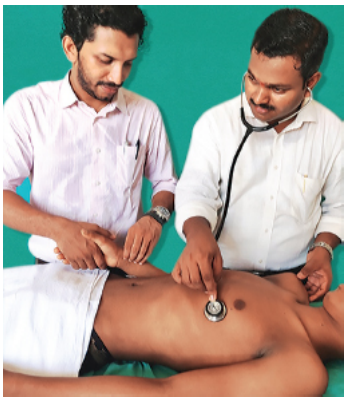


Fig. 2B.2: Demonstration of apex pulse deficit.

Volume of the Pulse

Volume of the pulse is a measure of the pulse pressure. The pulse pressure is the difference between systolic and diastolic blood pressure.

Normal pulse pressure is 30–60 mm Hg	
<30 mm Hg (low volume) **Hypokinetic pulse**	***>60 mm Hg (high volume)*** **Hyperkinetic pulse**
• Congestive cardiac failure • Hypovolemia • Shock • Mitral stenosis • Aortic stenosis **(pulsus minimus)** • Constrictive pericarditis	**Physiological:** Fever, pregnancy, alcoholism, and exercise **Pathological:** • **High output states:** Anemia, beriberi, hypercarbia • Cirrhosis liver (hypoproteinemia) thyrotoxicosis, • Arterio-venous fistula (AV) fistula

	• Paget's disease of the bone **Cardiac causes (pulsus magnus):** • Aortic regurgitation • Severe mitral regurgitation • Complete heart block • Patent ductus arteriosus (PDA) • Rupture of sinus of Valsalva and aortopulmonary window
Varying volume: Seen in atrial fibrillation **Anisosphygmia:** Varying volume of pulses in bilateral brachial/radial vessels. Seen in Takayasu's arteritis **Coanda effect:** In supravalvular aortic stenosis, pulse volume is better in the right upper limb compared to left due to the selective jet of the blood directed to the right subclavian vessel. *Note:* Pulsus alternans, pulsus bigeminus, and pulsus paradoxus are also abnormalities in volume (described under the section of character of pulse).	

Grading of Pulse

The examination of the arterial pulses is tabulated using a scale as follows:

Grade	Description
0	Complete absence of pulsation
1	Small or feeble/reduced pulsation
2	Palpable but diminished as compared to other side
3	Normal pulsation
4	Large or high volume/bounding pulsation

Character of Pulse

Best assessed in the carotids.

Exceptions:

- Collapsing pulse which is appreciated better at radial artery
- Pulsus bisferiens best appreciated in brachial artery.

Trisection Method

Varying degrees of pressure are applied with the finger pads of the thumb or first two fingers to assess upstroke, systolic peak and diastolic slope of the **pulse**.

Components of pulse wave **(Figs. 2B.3A and B):**

Individual components of pulse waveform	
Wave	***Description***
Percussion wave	It is due to arrival of the impulse generated by LV ejection
Tidal wave	It is due to the reflected waves from the upper part of the body
Dicrotic wave	It is due to the reflected waves from the lower part of the body
Dicrotic notch or incisura	This corresponds to S_2 (closure of aortic and pulmonary valves)

Speed of Pulse Wave and Time Taken to Reach the Peripheral Arteries

Speed of pulse wave	5 m/sec
Speed of blood flow	0.5 m/sec
Time taken for transmission of pulse to	

Carotid	30 ms
Brachial	60 ms
Femoral	75 ms
Radial	80 ms

- Normally radial pulse is felt 5–10 msec later than femoral pulse.

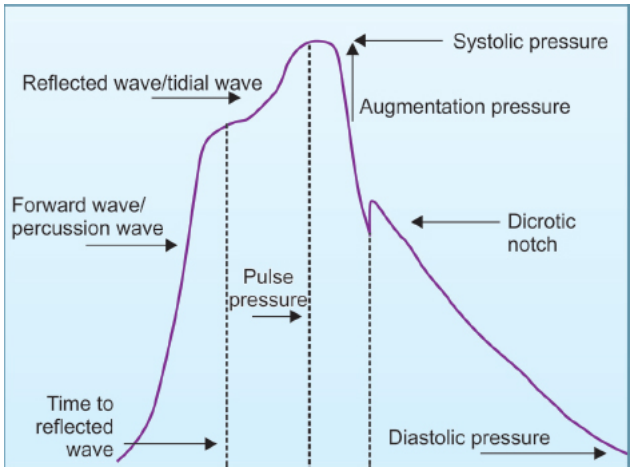


Fig. 2B.3A: Arterial pulse tracing.

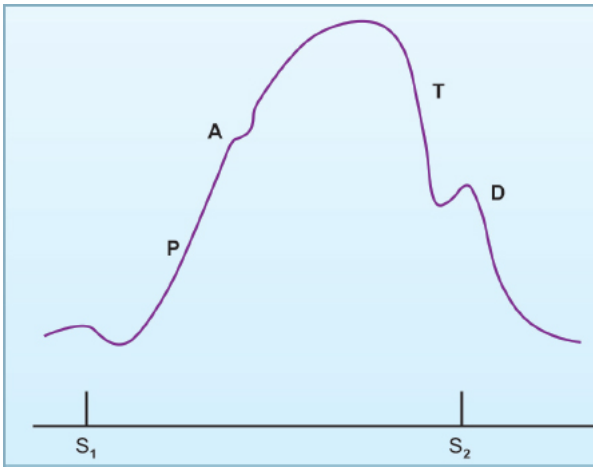


Fig. 2B.3B: Waveform showing different components of pulse wave.

Characters of pulse (Fig. 2B.4)		
Character	***Description***	***Condition seen***
Catacrotic pulse	It is the normal character of the pulse	
Pulsus parvus et tardus	A low amplitude pulse (parvus) with a slow rising and late peak (tardus)	Severe aortic stenosis (AS)
Pulsus anacroticus	Single peak low volume	Severe aortic stenosis
Spike and Dome pulse	Seen in HOCM	
Water hammer pulse or collapsing pulse or Watsons pulse or pulsus celer	• High (large) volume pulse • Sharp rise (systolic pressure is high) • Ill-sustained, sharp fall (diastolic pressure is low) • Pulse pressure is at least 60 mm Hg	Aortic regurgitation, patent ductus arteriosus (PDA), aortopulmonary window, rupture of sinus of valsalva, arteriovenous fistula, severe mitral regurgitation
Twin beating pulse		
Pulsus bisferiens	Two peaks in systole	• Severe aortic regurgitation (AR) • Moderate AR +AS • Hypertrophic obstructive cardiomyopathy (HOCM)
Pulsus dicroticus	One peak in systole, other peak in diastole. Seen when pulse rate and diastolic pressure is low	• Typhoid fever • Severe left ventricular failure (LVF) • Dehydration • Dilated cardiomyopathy endotoxic shock
Alternating volume pulses		
Pulsus alternans	• Alternating high volume and low volume pulse	Left ventricular failure

	• Regular rhythm • Korotkoff sounds double on lowering cuff pressures	
Pulsus bigeminus	Pulse wave with normal beat followed by a premature beat and a compensatory pause, occurring in rapid succession, resulting in alteration of the strength of pulse	Digoxin toxicity
Pulsus paradoxus		
Pulsus paradoxus	Systolic blood pressure falls more than 10 mm Hg during inspiration (exaggeration of normal phenomenon)	• Constrictive pericarditis • Acute severe asthma/chronic obstructive pulmonary disease (COPD) • Cardiac tamponade, tension pneumothorax, and massive pulmonary embolism • Others—anaphylactic shock, and obesity
Reverse pulsus paradoxus (inspiratory rise in pulse volume and pressure): seen in intermittent positive-pressure ventilation in the presence of left ventricular failure, hypertrophic obstructive cardiomyopathy (HOCM) and isorhythmic AV dissociation		
Absent pulsus paradoxus in constrictive pericarditis: If associated with large atrial septal defect/ventricular septal defect/aortic regurgitation (ASD/VSD/AR)/pericardial adhesions		

Method of Eliciting Pulsus Paradoxus (Fig. 2B.5)

- Pulsus paradoxus refers to an exaggerated fall in a patient's blood pressure during inspiration by greater than 10 mm Hg
- Patient is placed in a semirecumbent position; respirations should be normal. *Do not instruct them to change their breathing pattern as the depth of respiration influences the magnitude of pulsus paradoxus and will be amplified in patients with pulmonary disease*
- The blood pressure cuff is inflated to at least 20 mm Hg above the systolic pressure and slowly deflated until the first Korotkoff sounds are heard
- Initially sounds will be heard only during expiration. Note the level
- As the cuff is further deflated, the first korotkoff sound will be heard during both inspiration and expiration. Note this level.
- If difference between the two is more than 10 mm Hg, then it is pulsus paradoxus

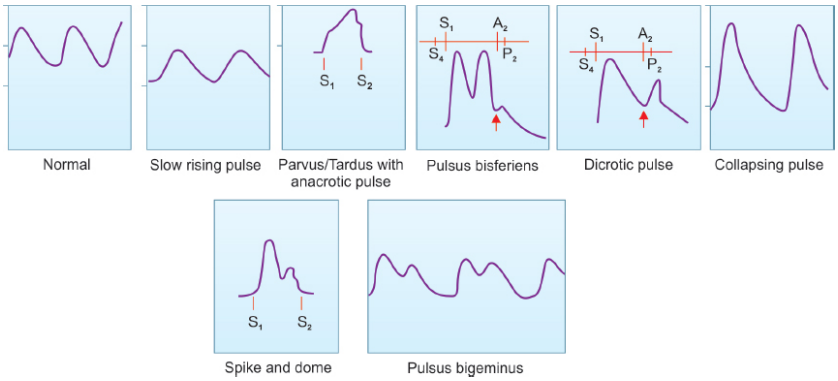


Fig. 2B.4: Image showing different pulse waveforms.

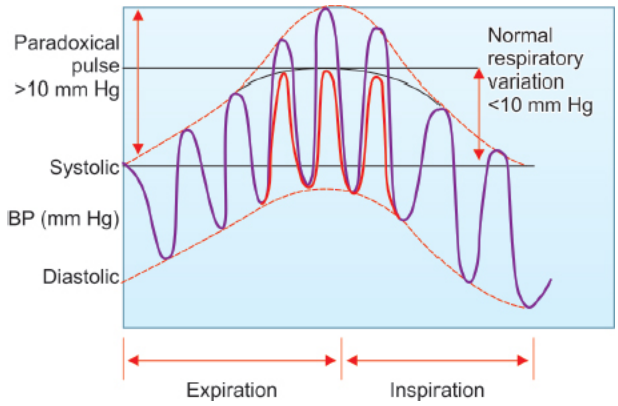


Fig. 2B.5: Pulsus paradoxus.

- This is not a true paradox as it is an exaggeration of normal phenomenon of fall of BP during inspiration.

Then, What is the Paradox?

The paradox is that, in patients in patients with constrictive pericarditis, during inspiration the blood pressure might drop significantly enough that the peripheral pulses will be absent; however, the heart sounds will still be heard.

Other Paradoxes in Medicine

French paradox: The observation that the French suffer a relatively low incidence of coronary heart disease, despite having a diet relatively rich in saturated fats.

"Thrombotic paradox" of hypertension (or) "Birmingham paradox": Hypertension is a prothrombotic state, hence paradoxially thrombotic strokes are more common than hemorrhagic.

Venous paradox—Kussmaul sign is a **paradoxical** rise in jugular **venous** pressure (JVP) on inspiration, or a failure in the appropriate fall of the JVP with inspiration.

Ulnar paradox: Higher the lesion minimal is the deficit.

Paradoxical respiration: It causes the chest to contract while inhaling and to expand during exhaling, the opposite of how it should move. The causes of paradoxical breathing include chest trauma and diaphragmatic paralysis. Neurological problems that can paralyze the diaphragm.

Kinesia paradoxa: Seen in parkinsonism, patients who generally cannot move but under certain circumstances exhibit a sudden, brief period of mobility (walking or even running).

Method of Elicitation of Pulsus Alternans (Fig. 2B.6)

- Pulsus alternans refers to alternating high and low volume pulses.
- Patient is placed in a semirecumbent position.
- The blood pressure cuff is inflated to at least 20 mm Hg above the systolic pressure and slowly deflated until the first Korotkoff sounds are heard.
- Initially, the Korotkoff's sounds due to the high volume pulses will be heard.
- On lowering the blood pressure, Korotkoff sounds will be heard due to both high volume and low volume pulses.
- This will produce doubling of Korotkoff's sounds.

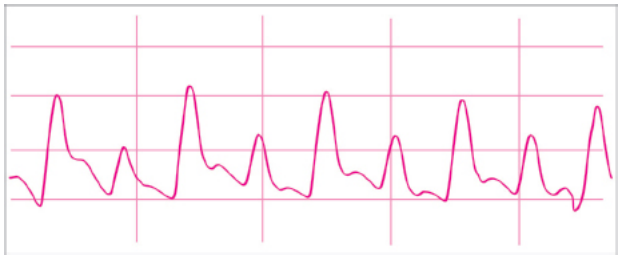


Fig. 2B.6: Pulsus alternans.

Method of Eliciting Collapsing Pulse (Fig. 2B.7)

- Palpate the radial artery and trace the artery proximally to a point where it is just felt
- At this point, wrap your wrist around the patient's forearm, so as to place the heads of the metacarpals over the artery.
- Simultaneously, palpate the radial and ulnar arteries by encircling the patient's wrist with your other hand.
- Now, abruptly raise the patients hand above the shoulder (artery becomes in line with the central aorta, allowing direct systolic ejection and diastolic backflow).
- In collapsing pulse, both radial and ulnar arteries are felt distinctly and, there is an abrupt thrust/knock and collapse under the metacarpal heads on elevation.
- Thrust produced is similar to the one produced by tilting of water hammer toy.
- It is due to diastolic run-off in aortic regurgitation.

Collapsing pulse is characterized by rapid upstroke (percussion wave) followed by rapid descent (collapse) of the pulse wave without dicrotic notch, which reflects low systemic vascular resistance.

- Rapid upstroke is due to the rapid ejection of greatly increased stroke volume.
- The rapid descent or collapsing character is due to:
  a. Diastolic "run-off" (backflow) into the left ventricle
  b. Reflex vasodilation mediated by carotid baroreceptors secondary to large stroke volume
  c. The rapid run-off to the periphery due to decreased systemic vascular resistance.

Corrigan's pulse/sign is largely used to describe the abrupt distension and quick collapse of carotid pulse in aortic regurgitation, whereas the term **Watson's water hammer pulse** is used for the characteristic pulse seen in peripheral arteries like the radial artery

Note: Make sure the patient does not have shoulder pain before doing this.

Method of Eliciting Pulsus Bisferiens

- Best felt in brachial and carotid arteries
- Felt by applying graded pressure
- With fingers press and occlude the brachial artery
- On slowly releasing the pressure, the double peaking of the pulse is appreciated.

Condition of Vessel Wall

Vessel wall thickening is assessed by using Osler's sign (described under the pseudohypertension in chapter blood pressure in chapter 2, page no 13).

Peripheral Pulses

Refer **Figure 2B.8.**

Palpation of Carotid Pulse (Figs. 2B.9 and 2B.10)

- Ask the patient to relax the neck.
- Palpate the right carotid artery by placing your left thumb near the upper neck between the sternomastoid and trachea roughly at the level of cricoid cartilage.
- Note the character of the pulse.
- Now, repeat the procedure on other side by placing your right thumb over the patients left carotid.
- *Note:* Make sure not to compress the carotid sinus.
- It is advisable to auscultate for carotid bruit prior to palpation, to prevent possible dislodgement of the atherosclerotic plaque (if present).

Fig. 2B.7: Demonstration of collapsing pulse.

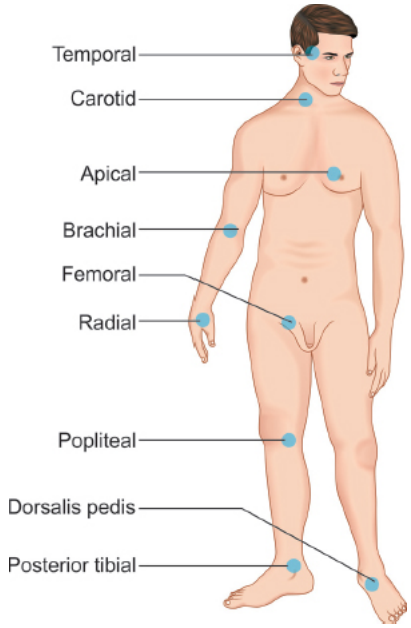


Fig. 2B.8: Image showing site of different peripheral pulses.

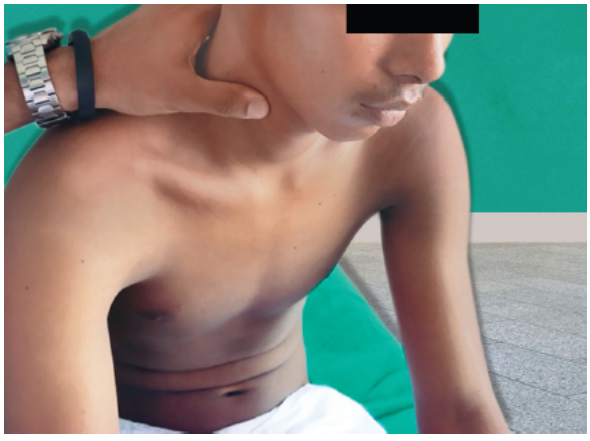


Fig. 2B.9: Demonstration of palpation of right carotid pulse.

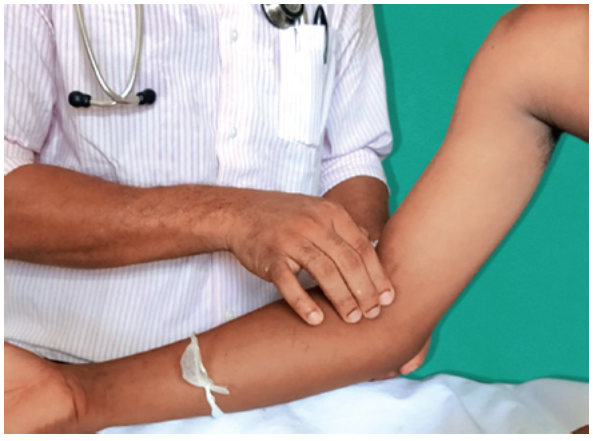


Fig. 2B.11: Demonstration of palpation of brachial pulse.

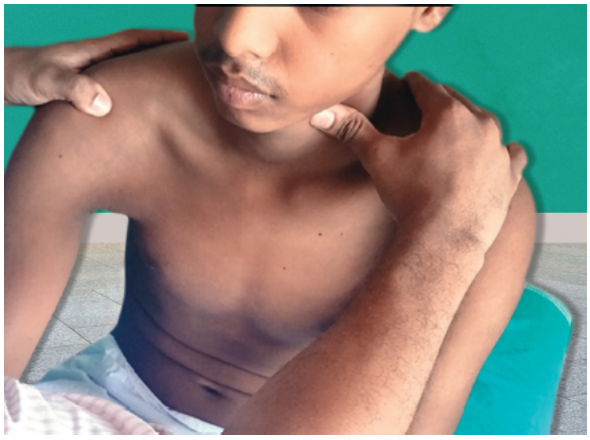


Fig. 2B.10: Demonstration of palpation of left carotid pulse.

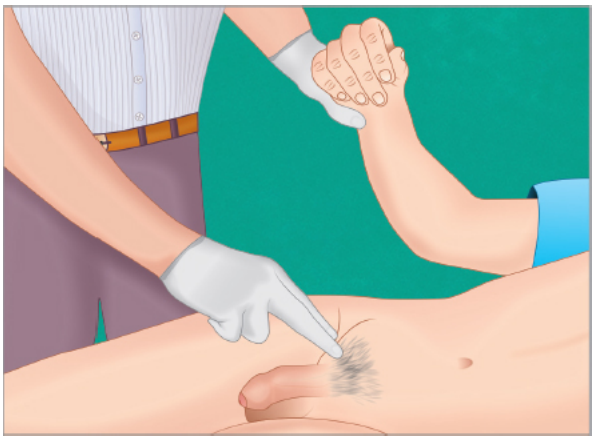


Fig. 2B.12: Site of examination of femoral pulse.

Palpation of Brachial Pulse (Fig. 2B.11)

- To examine the *brachial artery* in the right arm, the examiner supports the patient's forearm in his left hand.
- Patient's upper arm abducted, the elbow slightly flexed, and the forearm externally rotated.
- The examiner's right hand is then curled over the anterior aspect of the elbow to palpate along the course of the artery just medial to the biceps tendon and lateral to the medial epicondyle of the humerus.
- The position of the hands should be switched when examining the opposite limb.

Palpation of Abdominal Aorta

- The *abdominal aorta* is best palpated by applying firm pressure with the flattened fingers of both hands to indent the epigastrium toward the vertebral column.
- For this examination, it is essential that the subject's abdominal muscles be completely relaxed; such relaxation can be encouraged by having the subject flex the hips and by providing a pillow to support the head.
- In extremely obese individuals or in those with massive abdominal musculature, it may be impossible to detect aortic pulsation.

Palpation of Common Femoral Artery (Fig. 2B.12)

- The *common femoral artery* emerges into the upper thigh from beneath the inguinal ligament one-third of the distance from the pubis to the anterior superior iliac spine.
- It is best palpated with the examiner standing on the ipsilateral side of the patient and the fingertips of the examining hand pressed firmly into the groin.

Palpation of Popliteal Artery (Fig. 2B.13)

- *The popliteal artery* passes vertically through the deep portion of the popliteal space just lateral to the midplane.
- Generally, this pulse is felt most conveniently with the patient in the supine position and the examiner's hands encircling and supporting the knee from each side.
- The pulse is detected by pressing deeply into the popliteal space with the supporting fingertips.

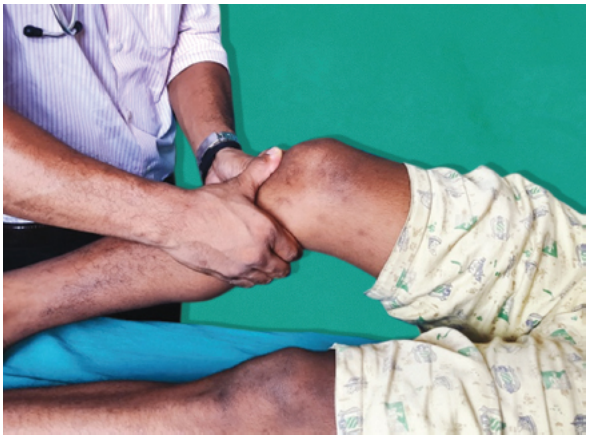


Fig. 2B.13: Demonstration of palpation of popliteal artery.

- Since complete relaxation of the muscles is essential to this examination, the patient should be instructed to let the leg "go limp" and to allow the examiner to provide all the support needed.

Palpation of Posterior Tibial Artery (Fig. 2B.14)

- The *posterior tibial artery* lies just posterior to the medial malleolus.
- It can be felt most readily by curling the fingers of the examining hand anteriorly around the ankle, indenting the soft tissues in the space between the medial malleolus and the Achilles tendon, above the calcaneus.
- The thumb is applied to the opposite side of the ankle in a grasping fashion to provide stability.

Palpation of Dorsalis Pedis Artery (Fig. 2B.15)

- The *dorsalis pedis artery* is examined with the patient in the recumbent position and the ankle relaxed.
- The examiner stands at the foot of the examining table and places the fingertips across the dorsum of the forefoot near the ankle.
- The artery is palpated lateral to the extensor hallucis tendon, against the navicular bone.
- This pulse is congenitally absent in approximately 10% of individuals.

Radio-Radial Delay

Proceed to palpate both radial pulses simultaneously to detect any inequality in timing. This is known as radio-radial delay. Causes include:

- Presubclavian coarctation
- Thoracic inlet syndrome: Cervical rib
- Takayasu's disease
- Aortic arch aneurysm.

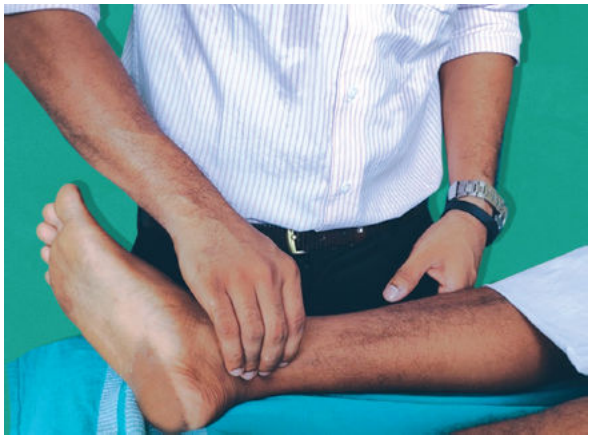


Fig. 2B.14: Demonstration of palpation of posterior tibial pulse.

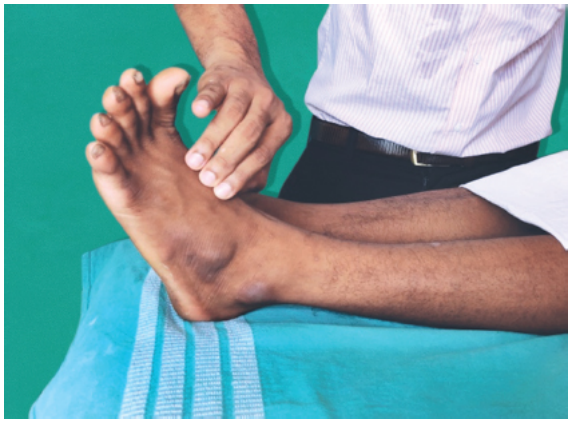


Fig. 2B.15: Demonstration of palpation of dorsalis pedis artery.

Radio-Femoral Delay (Fig. 2B.16)

If the femoral pulse is appreciated at the same time as the radial pulse, the patient is said to have radio-femoral delay. This is a sign of coarctation of aorta. This can rarely be seen with aortoarteritis.

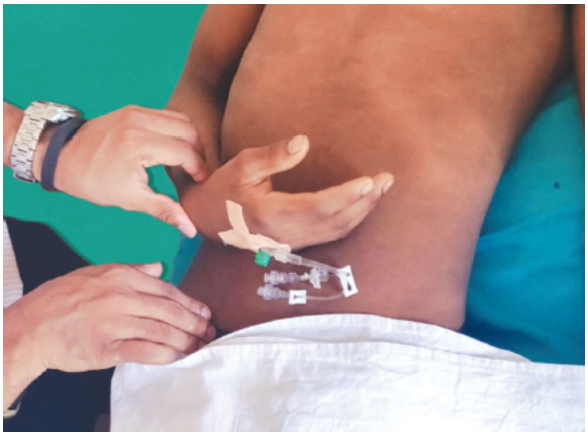


Fig. 2B.16: Demonstration of radio-femoral delay.

RESPIRATION

Respiratory Rate

Counted by placing the examiner's palm over the patient's abdomen, noting the rise and fall of the abdomen. Simultaneously divert the patient's attention by measuring the patient's pulse with your other hand **(Fig. 2B.17)**.

Normal pulse rate : respiratory rate = 4:1

Normal (16–20)	
Tachypnea	***Bradypnea***
>20	<10
Physiological: • Anxiety • Exertion **Pathological:** • Emphysema • Pneumothorax • Acute respiratory distress from infections • Pleurisy • Pulmonary embolism • Metabolic acidosis • Cardiac insufficiency • Anemia • Hyperthyroidism • Weakness of respiratory muscles • Obesity • Restrictive chest wall disease	• CNS-depressant drugs (e.g. opiates, benzodiazepines, barbiturates, alcohol) • Uremia • Increased intracranial pressure • Hypothermia • Hypothyroidism

Muscles of Respiration

Inspiration	Expiration
Main: • External intercostal muscle • Diaphragm	Predominantly passive process
Accessory muscles: • Serratus anterior • Sternocleidomastoid (SCM) • Scalenus anterior • Pectoralis • Trapezius	**Accessory muscles (used in forceful expiration):** • Internal intercostals • Abdominal muscles • Quadratus lumborum • Latissimus dorsi

Type of Respiration

Keep two hands flat, one on the chest and other on the abdomen and watch for movements of hand (Fig. 2B.18).

In abdominothoracic—movements of hand over the abdomen are more prominent.

In thoracoabdominal—movements of hand over the thorax are more prominent.

Abdominothoracic	Thoracoabdominal
Due to well-developed abdominal muscles	Well-formed internal intercostal muscles
Seen in males	Seen in females

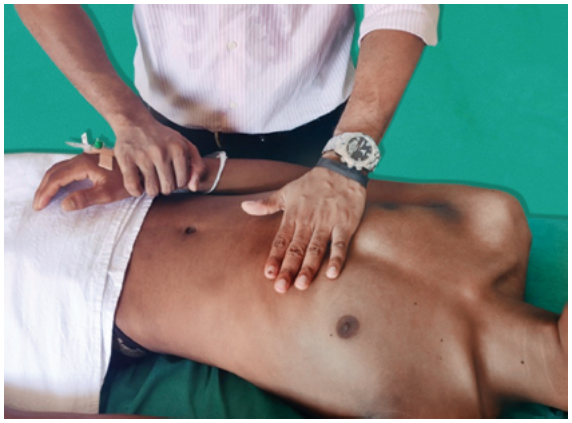


Fig. 2B.17: Method of calculating respiratory rate.

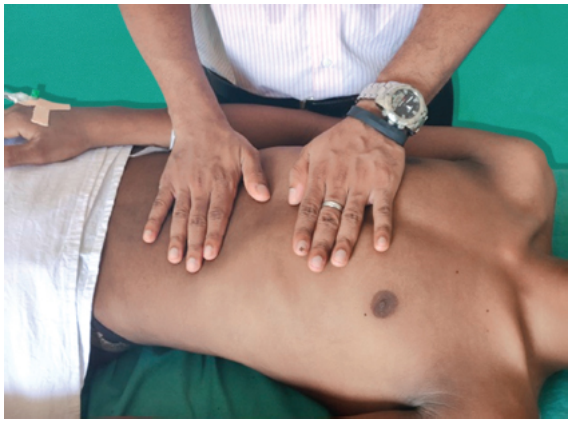


Fig. 2B.18: Method of assessing type of respiration.

Variants

Purely thoracic	Purely abdominal
Abdominal movement during respirations is absent	**Thoracic movement during respiration is absent**
• Peritonitis • Pregnancy • Ascites/ovarian cyst	• Pleuritic chest pain • Defective chest wall • Respiratory muscle paralysis [neurogenic, neuromuscular junction (NMJ), and muscular]

Abnormal Patterns of Breathing (Fig. 2B.19)

Regular	Irregular
Cheyne–Stokes (periods of apnea alternating with hyperapnea) • Cardiac failure (LVF)—most common cause • Raised intracranial pressure (ICP) • Brainstem lesions	**Biot breathing** (an uncommon variant of Cheyne-Stokes respiration. Periods of apnea alternate irregularly with a series of breaths of equal depth that terminates abruptly) • Meningitis
Kussmaul's (rapid deep breathing)	**Ataxic** • Brainstem disorders

• Metabolic acidosis [diabetic ketoacidosis (DKA) and renal failure]	
	Apneustic • Pontine lesions

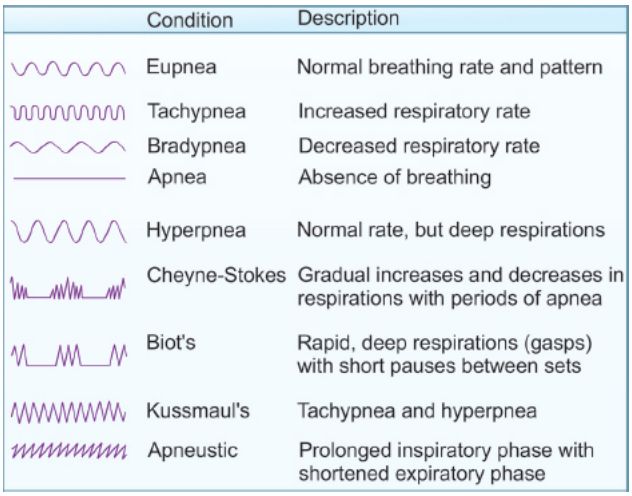


Fig. 2B.19: Different type of breathing patterns.

Pursed Lip Breathing

- Seen with chronic obstructive pulmonary disease (COPD)
- Mechanism of auto-positive end-expiratory pressure (PEEP)
- The purpose of this breathing is to slow down the air flow during the exhalation to build up back pressure in the airway to avoid a sudden drop in intrapulmonary pressure resulting in alveolar and airway collapse.

Airway Obstruction

- Upper airway obstruction—prolonged inspiration
- Lower airway obstruction—prolonged expiration.

BLOOD PRESSURE

Definition

Arterial blood pressure (BP) can be defined as the lateral pressure exerted by the moving column of blood on the walls of the arteries.

BP = Cardiac output × Peripheral resistance

Systolic blood pressure (SBP) • Defined as the maximum BP in the arteries attainable during systole • Normal: 120 + 20 mm Hg	**Diastolic blood pressure (DBP)** • Defined as the minimum pressure that is obtained at the end of the ventricular diastole • Normal range: 60–90 mm Hg
Pulse pressure (PP) • Denotes the difference between systolic and diastolic pressure • PP = SBP − DBP = 40 mm Hg	**Mean arterial pressure (MAP)** • DBP + one-third pulse pressure • Normal = 95 mm Hg

Korotkoff Sounds

KOROTKOFF SOUNDS	**Systolic blood pressure (SBP)**	**120 mm Hg**	Phase 1: A thud
		110 mm Hg	Phase 2: a blowing noise
		100 mm Hg	Phase 3: a softer thud
		90 mm Hg	Phase 4: a disappearing blowing noise (muffling)
	Diastolic blood pressure (DBP)	**80 mm Hg**	Phase 5: No korotkoff sounds

Types and Character of Korotkoff Sounds

AHA 2017 classification			
Blood pressure (BP) category	***Systolic BP***		***Diastolic BP***
Normal	<120 mm Hg	And	<80 mm Hg
Elevated	120–129 mm Hg	And	<80 mm Hg
Stage 1 hypertension	130–139 mm Hg	Or	80–89 mm Hg
Stage 2 hypertension	≥140 mm Hg	Or	≥90 mm Hg

Note: ESC guidelines 2018 and comparison table of JNC 7 and AHA 2017 are discussed in page 493 in annexures.

Steps of examination blood pressure	
Key steps	***Specific instructions***
Step 1: Properly prepare the patient	• The patient should rest comfortably for 5 minutes prior to the measurement in the seated position with their back supported. The patient's legs should be uncrossed with feet flat on the floor **(Fig. 2B.20)**. • The patient should avoid caffeine, exercise, and smoking for at least 30 minutes before measurement • Ensure that the patient has emptied his/her bladder • Neither the patient nor the observer should talk before or during the measurement • Measurements made while the patient is sitting or lying on an examining table do not fulfill these criteria
Step 2: Use proper technique for BP measurements	• Use a BP measurement device that has been validated, and ensure that the device is calibrated periodically. • The arm should be bare, supported and kept at heart level • Position the middle of the cuff on the patient's upper arm at the level of the right atrium (the midpoint of the sternum) **(Fig. 2B.21)**. • Use a cuff with an appropriate bladder size: Bladder width should be close to 40% of the arm circumference and length should cover 80-100% of the arm circumference. The lower edge of the cuff should sit 3 cm above the elbow crease with the bladder centered over the brachial artery • Either the stethoscope diaphragm or bell may be used for auscultatory readings
Step 3: Take the proper measurements needed for diagnosis and treatment of elevated BP/hypertension	• At the first visit, record BP in both arms. Use the arm that gives the higher reading for subsequent readings • Repeat blood pressure measurements should be taken 1–2 minutes apart • Increase the pressure to 30 mm Hg above the level at which the radial pulse is extinguished • Place the bell or diaphragm of the stethoscope over the brachial artery • Open the control valve so that the rate of deflation of the cuff is 2 mm Hg per heart beat • Systolic blood pressure is the appearance of the first Korotkoff sound • The diastolic blood pressure is the point at which the sound disappears (phase 5 Korotkoff) • If Korotkoff sounds continue as the level approaches 0 mm Hg, listen for when the sound becomes muffled to indicate the diastolic blood pressure
Step 4: Properly document accurate BP readings	• Record BP to the closest 2 mm Hg on the sphygmomanometer, as well as the arm used and the position of the patient (supine, sitting or standing)

	• Note the time of most recent BP medication taken before measurements
Step 5: Average the readings	• Use an average of ≥2 readings obtained on ≥2 occasions to estimate the individual's level of BP. • In presence of atrial fibrillation, minimum of 3 BP readings have to be estimated
Step 6: Provide BP readings to patient	• Provide patients the SBP/DBP readings both verbally and in writing

Fig. 2B.20: Demonstration of BP measurement.

Fig. 2B.21: Demonstration of placement of BP cuff.

Selection Criteria for BP Cuff Size for Measurement of BP in Adults

Arm circumference	Usual cuff size
22–26 cm	Small adult
27–34 cm	Adult
35–44 cm	Large adult
45–52 cm	Adult thigh

White Coat Hypertension

Normal blood pressure at home or on ambulatory blood pressure monitoring but elevated office blood pressure.

Masked Hypertension

Elevated blood pressure at home or on ambulatory blood pressure monitoring but normal office blood pressure.

Paroxysmal Hypertension

Episodic elevated BP.

- Pheochromocytoma
- Panic disorders
- Labile hypertension
- Carcinoid
- Clonidine withdrawal
- Hyperthyroidism
- Coronary insufficiency
- Cluster or migraine headaches
- Seizure disorder
- CNS lesions (such as stroke, tumor, hemorrhage)

- Renovascular hypertension
- Hypoglycemia
- Cheese reaction
- Anxiety
- Drugs—cocaine, lysergic acid diethylamide, amphetamine
- Baroreflex failure
- Factitious hypertension

Pseudohypertension

Defined as cuff diastolic blood pressure ≥15 mm Hg higher than simultaneously measured intra-arterial blood pressure. A palpable although pulseless, radial artery while the BP cuff is inflated above systolic pressure, is a positive **Osler sign**. Osler sign occurs due to Monckeberg's sclerosis of arteries.

Paradoxical Hypertension

On starting treatment with antihypertensives, the BP rises instead of falling in the following conditions.

1. Angiotensin-converting enzyme (ACE) inhibitors or angiotensin receptor blockers (ARBs) for a patient with renal artery stenosis
2. Beta-blockers given to a patient with pheochromocytoma
3. Beta-blockers in a patient with diabetic autonomic neuropathy.

HYPOTENSION

Hypotension is defined as blood pressure that is lower than 90/60 mm Hg.
Reference: NIH

Causes (Fig. 2B.22)

Younger adult	Any adult age group	Older adult
• Pregnancy • Vasovagal syncope • Situational syncope • Primary amyloidosis • Primary autonomic failure	• Chronic liver disease • Diabetic autonomic neuropathy • Secondary amyloidosis • Addison's disease • Hypopituitarism • Severe hypothyroidism	• Parkinson's disease • Dysrrhythmia • Micturition syncope • Carotid sinus syndrome • Vitamin B_{12} deficiency

Fig. 2B.22: Cause of hypotension according to age group.

Postural Hypotension/Orthostatic Hypotension

- A drop in blood pressure (hypotension) due to a change in body position (posture) when a person moves to a more vertical position, i.e. from sitting to standing or from lying down to sitting or standing.
- Postural (orthostatic) hypotension is diagnosed when, within 2–5 minutes of quiet standing (after a 5-minute period of supine rest), one or both of the following is present:
  - At least a 20 mm Hg fall in systolic pressure
  - At least a 10 mm Hg fall in diastolic pressure.
- Many disorders can cause orthostatic hypotension, with the two major mechanisms being autonomic failure, which can be caused by multiple disorders, and severe volume depletion.

Autonomic failure	Volume depletion
• Diabetic neuropathy • Parkinson disease • Dementia with Lewy bodies • MSA (Shy-Drager syndrome)	• Acute or subacute volume depletion (due to diuretics, hyperglycemia, hemorrhage, or vomiting) • Chronic hypovolemia, a frequent feature of autonomic failure, exacerbates orthostatic symptoms

• Spinal cord transection • Chronic kidney disease • Amyloidosis • Guillain-Barré syndrome • Paraneoplastic autonomic neuropathy • Familial dysautonomia (Riley-Day syndrome) • Primary autonomic failure (Bradbury-Eggleston syndrome)	

Postprandial Hypotension

In postprandial hypotension, blood pressure falls occur within one to two hours after a meal.

JUGULAR VENOUS SYSTEM

Jugular Venous Pulse

It is defined as undulating top of oscillating column of blood in right internal jugular vein that faithfully represents the pressure and volumetric changes in the right side of heart which changes with various stages of cardiac cycle and respiration.

Why is the Right IJV Preferred?

- Right side internal jugular vein (IJV) is in direct connection and in straight line.
- Veins in the left side of the neck reach the heart by crossing the mediastinum, where they may be compressed by the normal aorta; causing the left jugular venous pressure to appear elevated even when the CVP and right atrial pressures are normal.

Why internal jugular vein preferred over external jugular vein for JVP assessment?	
Internal jugular	***External jugular***
Straight communication with right atrium	Not in straight communication with right atrium
Less valves	More valves
Less influenced by fascial planes	More kinked by fascial planes
Less affected by sympathetic system	More affected by sympathetic system
	Vasoconstriction secondary to hypotension (in CCF) can make EJV small and barely visible

Differences between carotid and JVP	
Carotid pulse	***Jugular venous pulse***
1. Better felt	1. Better seen
2. Cannot be obliterated	2. Can be obliterated (by pressure at root of neck)
3. One positive wave	3. Two positive and two negative waves
4. Medially seen	4. Laterally seen
5. Seen in lower part	5. Seen in upper part
6. Definite upper level absent	6. Definite upper level present
7. Expansile impulse (outward)	7. Retractile impulse (inward)
8. Does not change with position	8. Changes with position
9. Does not change with respiration	9. Changes with respiration
10. Does not change with abdominal compression	10. Changes with abdominal compression

Steps of Examination of JVP (Figs. 2B.24 and 2B.25)

- Patient comfortably lying in semi reclined position (45° position).
- The patient's neck should be slightly turned towards the left side.
- Shine a torch light onto the neck tangentially from the left side.
- Observe for pulsation between two heads of sternocleidomastoid
- Trace the pulsation and locate the upper level
- Take two scales. Place one scale at the upper level of the JVP, parallel to the ground.
- Now place the second scale at the level of the sternal angle, perpendicular to the first scale.
- Measure the vertical height on the second scale.
- Express as ___ cm of water above sternal angle. Add 5 cm to this value to determine the right atrial pressure.
- Conversion: 1.36 cm of H_2O or blood = 1 mm Hg
- The normal JVP is **less than 4 cm** above the sternal angle; or is just visible above the clavicle in 45° position.
- Normal CVP is <7 mm of Hg or 9 cm H_2O.

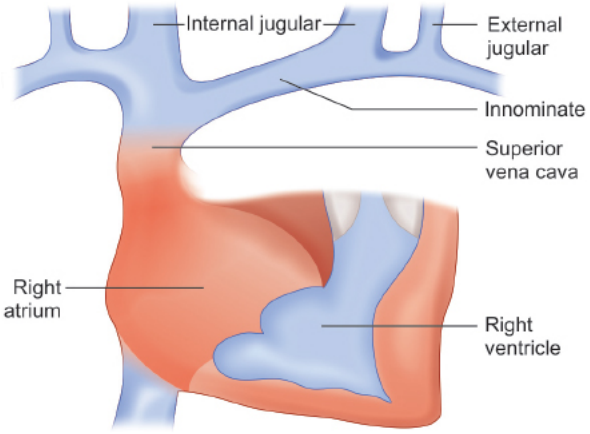


Fig. 2B.23: Anatomy of the right IJV.

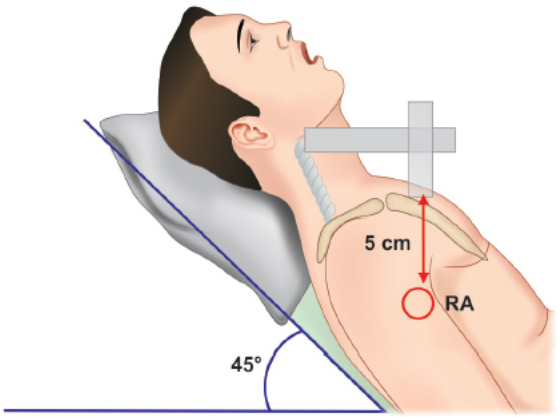


Fig. 2B.24: Method of measuring the JVP.

Fig. 2B.25: Examination of height of JVP.

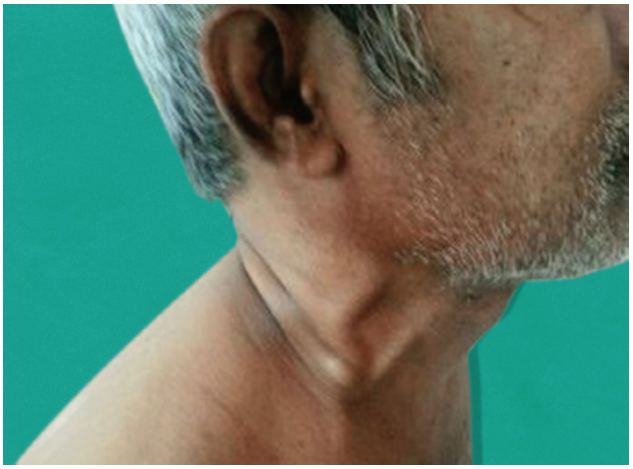


Fig. 2B.26: Image showing engorged neck veins.

Causes of Raised JVP

Engorged (Fig. 2B.26) and pulsatile neck vein	Engorged and nonpulsatile neck vein
Cardiac causes	• Superior mediastinal syndrome • Valsalva maneuver • Chronic constrictive pericarditis (advanced stage)
• Right heart failure • Congestive cardiac failure • Chronic constrictive pericarditis • Cardiac tamponade • Complete heart block • Restrictive cardiomyopathy • Superior vena cava (SVC) obstruction • Tricuspid stenosis	
Noncardiac causes	
• Pulmonary thromboembolism • Pulmonary hypertension • Acute nephritis • Pregnancy • Fluid overload status	

Waveforms of JVP:

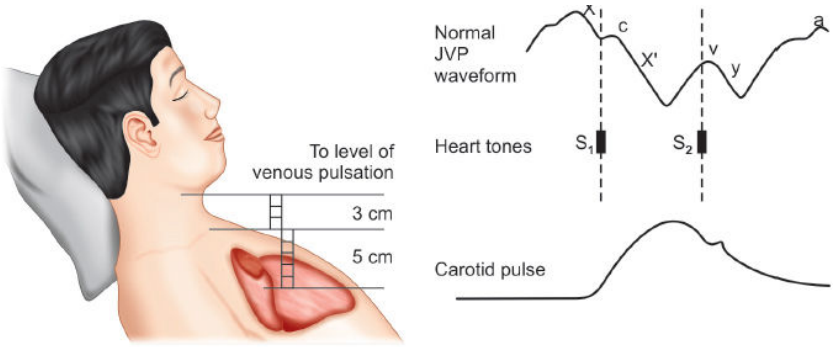
Component	Cardiac event responsible
A wave	Atrial contraction/systole
X wave (initial x descent)	Atrial relaxation
C wave	Closure of the tricuspid valve (some consider c wave is due to the impact of carotid pulsation)
X' wave (X descent following "C" wave)	Downward movement of the floor of the right atrium while the right ventricle contracts (called the 'descent of the base')
V wave	Atrial filling during ventricular systole
Y wave	RA emptying during ventricular diastole
H wave (Hirschfelder wave)	Seen in diastasis

"a" wave (most prominent of JVP)			
Absent	Atrial fibrillation		
Large/giant "a" wave	Tricuspid stenosis (TS) Tricuspid atresia (TA) Right atrium (RA) myxomas	Right ventricular (RV) infarct RV cardiomyopathy	Pulmonary hypertension (PH) Pulmonary stenosis (PS) Pulmonary embolism (PE)
	Aortic stenosis (AS)* Hypertrophic cardiomyopathy (HCM)* (**Bernheim effect***)		
Cannon "A" waves	Regular	Junctional rhythm Ventricular tachycardia (VT) (1:1 retrograde conduction)	
	Irregular	Complete heart block (CHB) Atrioventricular (AV) dissociation Ventricular ectopics Ventricular tachycardia V pacing	

Bernheim effect: Left-sided diseases causing prominent a wave, (ie) severe LVH with septal thickening interfere with RV filling resulting in prominent a wave.

"v" wave	
Diminished	Cause of diminished v wave is hypovolemia
Prominent	• Tricuspid regurgitation (TR)* • Atrial septal defect (ASD) • Ventricular septal defect (VSD), Gerbode defect—abnormal shunting between the left ventricle and the right atrium due to either a congenital defect or prior cardiac insults • Congestive heart failure (CHF) • Atrial fibrillation • Cor pulmonale

*In TR due to absent X and prominent V wave merging with C wave, it results in large positive systolic and regurgitant waves (CV wave) followed by a rapid deep 'y' descent. This may cause subtle motion of earlobe with each heart beat (The LANCISI's sign)



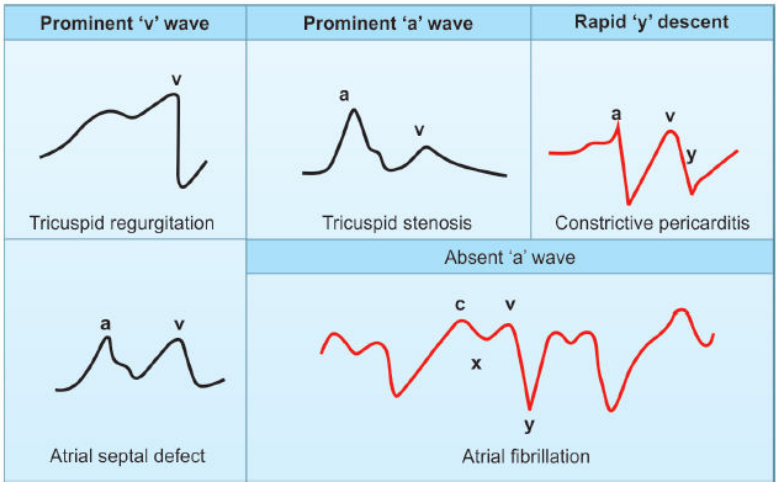


Fig. 2B.27: Jugular venous pulse demonstration.

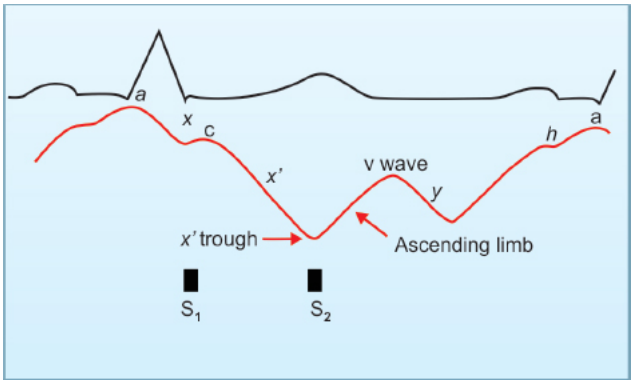


Fig. 2B.28: Jugular venous wave pattern JVP components and waveforms **(Fig. 2B.27)**.

'X' descent (systolic collapse)	
Absent	Tricuspid regurgitation
Prominent	Tamponade Atrial septal defect (ASD) Pericarditis—constrictive

'Y' descent (diastolic collapse)	
Slow descent	Tamponade Tricuspid stenosis (TS) Right atrial (RA) myxoma
Rapid descent	Constrictive pericarditis Severe tricuspid regurgitation (TR) Severe right ventricular (RV) failure

Differences between Constrictive Pericarditis and Cardiac Tamponade (Fig. 2B.29)

	X wave	Y wave
Pericarditis—constrictive	+	++ **(prominent Y)**
Tamponade	++ **(prominent X)**	--
TR	--	++

(Mnemonic: Prominent Y and X waves can be remembered with mnemonic **PaY TaX**)

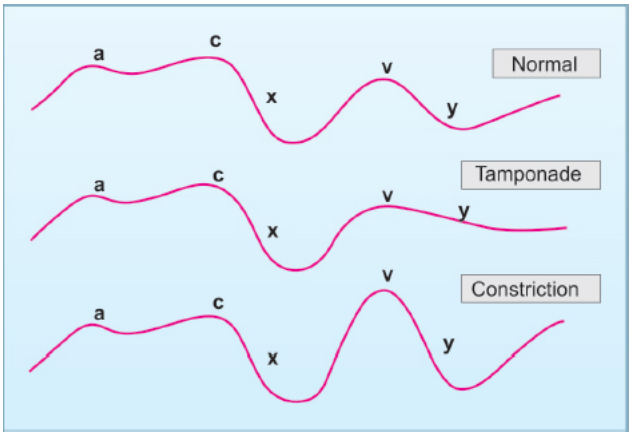


Fig. 2B.29: Waveforms of JVP in tamponade versus constrictive pericarditis.

OTHER SITES OF JVP ESTIMATION

Gaertner's Method

Normally, the superficial veins of dorsum of hand collapse when raised above the sternal angle. Persistent prominence is suggestive of raised central venous pressure (**anthem sign**—when the same is tested by asking the patient to make a fist and raise the arm like an anthem pledge).

May's Sign

Visible engorged vein on the undersurface of tongue in sitting posture.

ABDOMINOJUGULAR (AJR) REFLUX OF RUNDOTT (PREVIOUSLY KNOWN AS HEPATOJUGULAR REFLUX)

Demonstration (Fig. 2B.30)

- The patient is placed in a 45° semirecumbent position and firm, consistent abdominal pressure 40 mm Hg is applied, preferably over the right hypochondrium (an inflated BP cuff may be used).

- Historically pressure was applied for 15 seconds; however, recent studies suggest 10 seconds is adequate

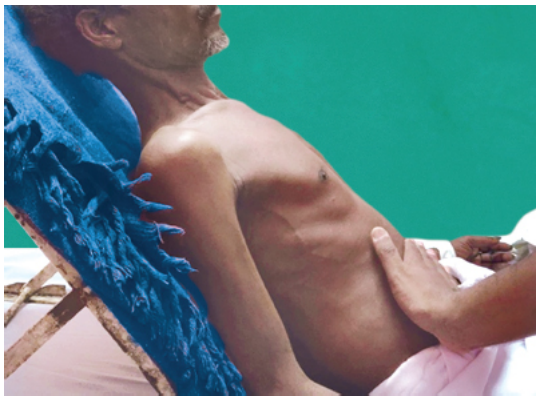


Fig. 2B.30: Demonstration of abdominojugular reflux.

- **Normal response**:
  - Transient rise of around 4 cm for about 4–5 cardiac cycles (approximately 5 sec)
- **Sustained response/positive response**:
  - Earliest sign of right heart failure (RHF), also seen in tricuspid regurgitation (TR)
- **Absent response/negative response**:
  - Obstruction/thrombosis of inferior vena cava (IVC) or hepatic veins as seen in Budd-Chiari syndrome.

Friederick's Sign of Constrictive Pericarditis

Friederick's sign describes a rapid fall and rise in the JVP. It occurs when stiff ventricles are unable to accommodate the rapid ventricular filling that should follow opening of the tricuspid valve in the presence of elevated atrial pressure.

Square Root Sign of JVP

Dip and plateau pattern of JVP seen in constrictive pericarditis.

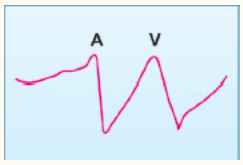
Kussmaul Sign of JVP

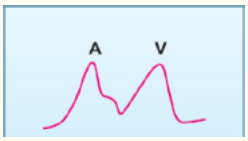
Normally when the patient inspires there is fall in the height of JVP due to increased negative intrathoracic pressure.

Kussmaul sign is the paradoxical elevation of JVP during inspiration.

Seen in:

- Constrictive pericarditis
- Severe heart failure
- Right ventricular infarction
- Restrictive cardiomyopathy.

M pattern in JVP		
Constrictive pericarditis	Due to prominent x and y waves	

ASD	Due to prominent A and V waves	

Raised jugular venous pressure with shock

- Congestive heart failure
- Cardiac tamponade
- Right ventricular infarction
- Tension pneumothorax
- Massive pulmonary embolism

BODY TEMPERATURE

Core Body Temperature

It usually refers to the temperature of the internal body core, measured under the tongue, in the ear canal or in the rectum.

Normal range (oral): 36.8 ± 0.4°C (98.2 ± 0.7°F)

Regulation of temperature: Under the control of neurons of preoptic anterior hypothalamus and posterior hypothalamus.

Site of Examination of Temperature

Oral temperature	• Probe placed under the tongue into the sublingual pockets and the lips closed around the instrument • The patient should not have recently smoked or ingested cold or hot food or drink • Usually tested for about 3 minutes Oral temperature reflects changes in core body temperature through the branch of the external carotid artery which perfuses the posterior sublingual pockets
Rectal readings are 0.4–0.6°C higher than oral recordings	• Measured with a lubricated blunt-tipped glass thermometer inserted 4– 5 cm (2.5 cm in children) into the anal canal at an angle 20° from the horizontal with the patient lying prone • Usually tested for about 3 minute • Lags behind changes at other core sites as it is located far from the central nervous system as well as from the pulmonary artery Indicates the deep visceral temperature. Can be affected by the temperature of the skin of the buttocks, the iliac artery and iliac vein
Tympanic temperature	• The scanning tip should be gently placed in the ear canal and then slowly inserted against the tympanic membrane snugly • Measures the infrared heat waves from the tympanic membrane • Close to hypothalamus and rapid measurement of core body temperature
Axillary readings lag behind oral temperature by 0.1–0.2°C	• Thermometer placed in the axilla and shoulder adducted • Convenient for patient • Core temperature cannot be assessed directly • Lags behind the changes in core body temperature
Temporal (forehead) measurement	• Placed on the skin of the forehead • An electronic thermometer that is fast and accurate • Less invasive than the tympanic thermometer and more reliable when used correctly

Thermometers (Fig. 2B.31)

- Glass thermometer and electric digital thermometer
- Glass thermometer bulbs contain an alloy called galinstan.

Electric digital thermometers are more convenient than glass instruments because the probe cover is disposable, response time is quicker (allowing accurate measurements within 10–20 seconds), and there is a signal when the rate of change in temperature becomes insignificant.

The most common methods of temperature assessment that carry the least amount of risk for patient injury are the use of glass or electronic digital thermometers to measure oral, rectal, axillary, or vaginal temperatures; basal thermometers; temporal artery thermometers; tympanic thermometers; and liquid crystal forehead temperature strips. These methods can be utilized in healthcare settings and also within the patient's home.

Although the more invasive methods are more accurate, they carry a higher risk of potential complications, so they are not routinely utilized in areas outside of a critical care or surgical setting. Examples of invasive methods of temperature assessment are esophageal and rectal temperature probes, temperature-sensing indwelling urinary catheters, temperature-sensing pulmonary artery (PA) catheters, a cardiopulmonary bypass (CPB) machine, and extracorporeal membrane oxygenation (ECMO).

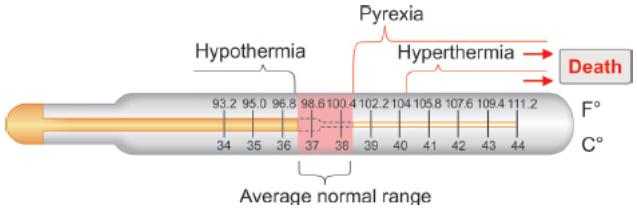


Fig. 2B.31: Thermometer showing marking in both Celsius and Fahrenheit.

Circadian Variation of Temperature

- Circadian rhythm is governed by suprachiasmatic nuclei in anterior hypothalamus.
- Normal variation is 0.5–1.0°C over the day
- Lowest temperature is noted at 6:00 am and peaks at 4:00–6:00 pm.

Variation of Temperature during Menstrual Cycles

An abrupt increase of 0.3–0.5 °C accompanies ovulation and may be useful as a fertility guide.

Fever

Fever is an elevation of body temperature that exceeds the normal daily variation and occurs in conjunction with an **increase in the hypothalamic set point**.

It can be defined as temperature of >37.2°C (98.9°F) at 6 am or >37.7°C (99.9°F) at 4–6 pm.

When the hypothalamic set point is raised, the body is perceived to be cooler than the new set point. Shivering is initiated to generate heat. Blood is shunted from the periphery to the core to conserve heat and sweating is diminished. The generated heat will raise the body temperature to match the elevated set point. When the hypothalamic set point is lowered, either as part of the normal diurnal fluctuations that occur during an infection or in response to antipyretic agents, heat is lost by evaporation (sweating) and radiation (cutaneous vasodilation).

Types of fever based on duration		
Acute fevers	<7 days	Infectious diseases such as malaria and viral-related upper respiratory tract infections
Subacute fevers	Usually not more than 2 weeks in duration	Typhoid fever and intra-abdominal abscess

Chronic or persistent fevers	>2 weeks duration	Chronic bacterial infections such as tuberculosis, viral infections like human immunodeficiency virus (HIV), cancers and connective tissue diseases

Grading of Fever based on Body Temperature

Body temperature	°C	°F
Normal	37–38	98.6–100.4
Mild/low grade fever	38.1–39	100.5–102.2
Moderate grade fever	39.1–40	102.2–104.0
High grade fever	40.1–41.1	104.1–106.0
Hyperpyrexia	>41.1	>106.0

The conversion formula is:

1. T°F = 9/5 (T°C) + 32
2. T°C = 5/9 (T°F) – 32

Patterns of fever (Fig. 2B.32)		
Type of fever	***Description***	***Seen in***
Continuous or sustained fever	Defined as fever that does not fluctuate more than about 1°C (1.5°F) during 24 hours, but does not touch the baseline	Lobar and gram-negative pneumonia, typhoid, and acute bacterial meningitis
Remittent fever	Defined as fever with daily fluctuations exceeding 2°C but does not touch the baseline	Remittent fevers are often associated with infectious diseases such as infective endocarditis, rickettsia infections, and brucellosis
Intermittent fever	Defined as fever present only for several hours during the day	Malaria, pyogenic infections, tuberculosis (TB), schistosomiasis, lymphomas, leptospira, *Borrelia*, Kala-azar, or septicemia
	Double quotidian fever (12 hours periodicity)	Kala-azar, gonococcal endocarditis. Adult-onset Still's disease
	Quotidian fever (periodicity of 24 hours)	Mixed falciparum and vivax
	Tertian fever (periodicity of 48 hours)	*Plasmodium falciparum*, ovale and vivax
	Quartan fever (periodicity of 72 hours)	*Plasmodium malariae*
	Pel-Ebstein's fever (intermittent low-grade fever characterized by 3–10 days of fever with subsequent afebrile periods of 3–10 days)	It is thought to be a typical but rare manifestation of Hodgkin's lymphoma
Relapsing fevers	Refer to those that are recurring and separated by periods with low-grade fever or no fever	Seen in malaria, lymphoma, *Borrelia*, cyclic neutropenia, and rat-bite fever

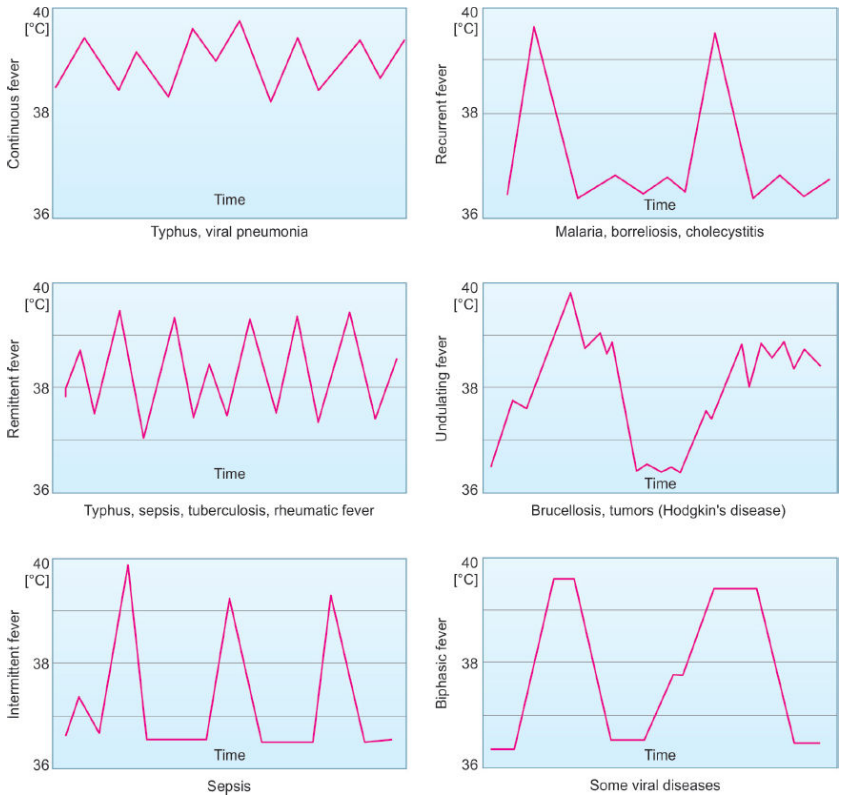


Fig. 2B.32: Clinical pattern of fevers.

Fever with Night Sweats

It has been described in infectious diseases such as TB, *Nocardia*, brucellosis, liver or lung abscess, and subacute infective endocarditis, as well as in noninfectious diseases such as polyarteritis nodosa and cancers such as lymphomas.

Fever with Bradycardia

It is a feature of untreated typhoid, leishmaniasis, brucellosis, Legionnaire's disease and psittacosis, and yellow fever.

Fever with Unknown Origin

In 1961, pyrexia of unknown origin (PUO) was originally defined by Petersdorf and Beeson as an illness of more than 3 weeks duration, fever higher than 38.3°C (101°F) on several occasions and diagnosis uncertain after 1 week of study in hospital.

This definition has been modified, removing the requirement that the evaluation must take place in the hospital and refined to include four different subgroups, each requiring different investigative strategies: classical, nosocomial, neutropenic, and human immunodeficiency virus (HIV)-related.

Hyperpyrexia

(Body temperature >105°F)

Causes Include:

- Pontine hemorrhage
- Rheumatic fever
- Meningococcal meningitis
- Cerebral malaria

- Septicemia
- Encephalitis
- Serotonin syndrome
- Thyroid storm
- Neuroleptic malignant syndrome.

Aseptic Fever

- Malignancies
- Acute myocardial infarction
- Sarcoidosis
- Chronic renal failure
- Collagen vascular diseases
- Drug fever
- Radiation sickness
- Postsurgical patients.

Drug Fever

It is a prolonged fever with relative bradycardia and hypotension. It persists 2–3 days even after drug is withdrawn and is associated with rash and eosinophilia. For example, penicillin, procainamide, propylthiouracil, sulfonamides, anticonvulsant, etc.

Note: All drugs except digitalis can cause drug induced fever.

Nature of Defervescence

The **nature of fever defervescence** may also provide some diagnostic clues.

Defervescence by crisis (Fig. 2B.33)	Defervescence by lysis (Fig. 2B.34)
Within hours	Gradually over days
Example: Effective antimalarial therapy leads to fever defervescence by crisis	Example: Typhoid fevers resolution occurs by lysis following effective antibiotics

Disorders of increased body temperature	
Hyperpyrexia	The body's temperature regulation mechanism sets the body temperature above the normal temperature, then generates heat to achieve this temperature
Hyperthermia	Unchanged (normothermic) setting of the thermoregulatory center in conjunction with an uncontrolled increase in body temperature that exceeds the body's ability to lose heat
Heat stroke	Acute condition of hyperthermia that is caused by prolonged exposure to excessive heat/± humidity. The heat-regulating mechanisms of the body eventually become overwhelmed and unable to effectively deal with the heat, causing the body temperature to climb uncontrollably
Malignant hyperthermia	Occurs in individuals with an inherited abnormality of skeletal-muscle sarcoplasmic reticulum that causes a rapid increase in intracellular calcium levels in response to halothane and other inhalational anesthetics or to succinylcholine
Neuroleptic malignant syndrome (NMS)	Seen with neuroleptic use (antipsychotic phenothiazines, haloperidol, prochlorperazine, and metoclopramide) or the withdrawal of dopaminergic drugs. Characterized by "lead-pipe" muscle rigidity, extrapyramidal side effects, autonomic dysregulation, and hyperthermia

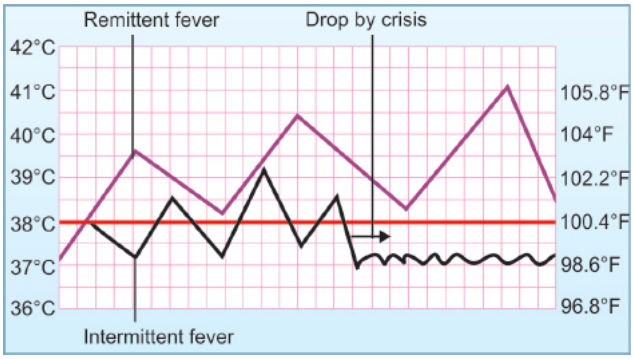


Fig. 2B.33: Defervescence by crisis.

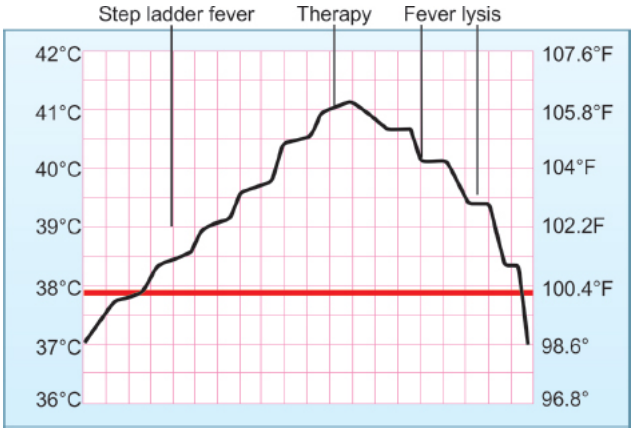


Fig. 2B.34: Defervescence by lysis in typhoid fever.

Hypothermia	
Hypothermia is defined as a core temperature below 35°C (95°F).	
Mild hypothermia	Core temperature 32– 35°C (90–95°F)
Moderate hypothermia	Core temperature 28–32°C (82–90°F)
Severe hypothermia	Core temperature below 28°C (82°F)
Profound hypothermia	Core temperature <24°C (75°F) or <20°C (68°F)

Causes of Hypothermia

Decreased heat production • Hypopituitarism • Hypoadrenalism • Hypothyroidism	**Increased heat loss** • Burns • Cold immersion injuries • Vasodilatation from pharmacologic or toxicologic agents • Cold infusions • Overenthusiastic treatment of heatstroke
Impaired thermoregulation • Central nervous system (CNS) trauma • Strokes • Toxicologic and metabolic derangements • Intracranial bleeding • Parkinson disease • CNS tumors	**Miscellaneous causes** • Sepsis • Multiple trauma • Pancreatitis • Prolonged cardiac arrest • Uremia

• Wernicke disease • Multiple sclerosis	

Named fevers	Disease/organism
Glandular fever	Infectious mononucleosis (EBV)
Pappataci, 3 days, sandfly fever	Phlebotomus fever
Goal fever	*Rickettsia prowazekii*
Malta, undulating fever	Brucellosis
Relapsing fever	*Borrelia recurrentis (louse)* *B. duttoni* (Tick)
Rat bite fever	*Spirillum minus* *Streptobacillus moniliformis*
Trench or 5 day fever	Bartonella quintana
Oroya fever	*Bartonella bacilliformis*
Q fever	*Coxiella burnetti*
7 day fever	*Leptospira hebdomadis*
Pretibial fever	*L. atumnale*
Haverhill fever	*Streptobacillus moniliformis*
Pontiac fever	*Legionella*
Monkey fever	Kyasanur forest disease
Biphasic fever	• Dengue • Kala-azar • Chikungunya • Polio
Valley fever	Coccidioidomycosis
Dumdum/burdwan fever	Kala-azar
Brazilian purpuric fever	*H. aegyptius*

PAIN: THE FIFTH VITAL SIGN

Pain is recognized as the fifth vital sign.

Assessment should include:

- Location
- Intensity
- Character/quality
- Frequency
- Duration
- Pattern.

Location—determine as precisely as possible where the pain is felt. Indicate if the pain radiates or moves.

Intensity—a grade of how severe the pain is, using a pain assessment tool the resident finds easy to use, e.g. a numerical, verbal descriptor, faces, or behavioral.

Frequency:

- The occurrence of the pain.
- How often the pain occurs?
- Is it breakthrough pain?

Quality—aching, annoying, cramping, exhausting, nauseating, pounding, sharp, throbbing, stabbing, agonizing, blowing, dull, fearful, nagging, penetrating, quivering, shooting, suffocating, numbness, tingling, weakness, spasm, burning, gnawing, pressure, squeezing, radiating, tingling, touch sensitive, etc.

- Pain behaviors—facial (wrinkled forehead, tightly closed eyes, grimacing, and frowning), nonverbal behavior (bracing, rubbing, and guarding), and vocalizations (crying, yelling, groaning, and moaning).

Nonverbal indicators of discomfort—aggressive, crying, fearful, noisy respirations, pacing, repetitive, restless, rocking, confusion, irritability, increased activity, withdrawal, tense, calling out, grunting, knees pulled up, other change in usual activities, or behavior patterns/routine.

Duration:

- How long does the pain last (minutes or hours)?
- Sudden or gradual onset.
- Is it consistent or persistent?
- Does it change over time or come and go (intermittent)? If intermittent—frequency, duration, and circumstances in which it occurs.

Pattern:

- How does the pain start?
- What was being done when it started?
- What makes it better?
- What makes it worse?

Types of Pain

- Somatic pain (bone and muscle) is:
  - Relatively well localized, worse on movement
  - Tender to pressure over the area
  - Often accompanied by a dull background aching pain.
- Visceral pain is:
  - Often poorly localized, deep, and aching
  - Usually constant
  - Often referred (e.g. diaphragmatic irritation may be referred to the right shoulder; pelvic visceral pain is often referred to the sacral or perineal area).

Pain assessment model

S	Site	Where exactly is the pain?
O	Onset	What were they doing when the pain started?
C	Character	What does the pain feel like?
R	Radiates	Does the pain go anywhere eles?
A	Associated symptoms	Nausea/vomiting
T	Time/duration	How long have they had the pain?
E	Exacerbating/relieving factors	Does anything make the pain better or worse?
S	Severity	Obtain an initial pain score

Fig. 2B.35: Pain assessment model.

- Neuropathic pain is:
  - A constant, superficial burning sensation, or a deeply aching quality that may be accompanied by some sudden, sharp, shooting, and lancinating (stabbing) pain.
  - In a relatively constant area of the body surface (dermatome), if caused by actual damage to a specific peripheral nerve, plexus, root, or spinal cord.

C. PHYSICAL EXAMINATION

PALLOR

Definition

Paleness of skin and mucous membranes.

Sites of Examination

1. Conjunctiva **(Fig. 2C.1)**
2. Tongue
3. Oral mucosa
4. Palmar crease **(Fig. 2C.2)**
5. Nail bed (Hb <8 g/dL).

Fig. 2C.1: Method of demonstration of pallor over conjunctiva.

Grading of Pallor

Mild	Moderate	Severe
Cannot be detected clinically	Clinically visible	Clinically visible plus one of the following features 1. Palmar crease disappearance 2. Cervical venous hum (suggestive of chronic compensation)

Method of Elicitation of Cervical Venous Hum (Fig. 2C.3)

- Auscultate the root of the neck on the right side with bell of stethoscope, with patient in standing or sitting position.
- A continuous murmur will be heard.
- The cervical venous hum was first described by Pontain and hence called **Pontain's murmur.**
- The presence of a cervical venous hum indicates chronic compensated severe anemia.

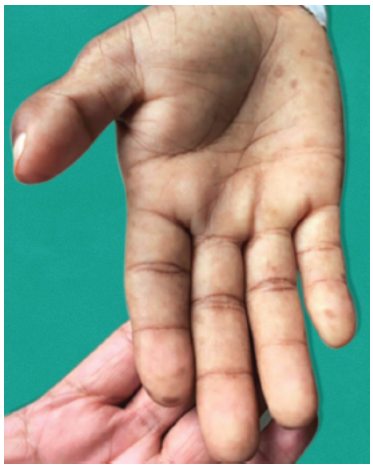


Fig. 2C.2: Demonstration of pallor in hands.

Fig. 2C.3: Demonstration of cervical venous hum.

Conditions Causing Pallor without Anemia

- Hypopituitarism
- Hypothyroidism
- Hypogonadism
- Shock
- Left heart failure.

Definition of Anemia

Anemia is defined as decrease in circulating red blood cell (RBC) mass. It is characterized by decrease of hemoglobin concentration (Hb)/RBC count/hematocrit [packed-cell volume (PCV)] below normal for the patient's age, sex, and altitude of residence.

Normal adult hemoglobin level is in the range of 13–17 g/dL in males and 12–15 g/dL in females.

Clues for Etiology of Anemia

Iron deficiency anemia	
Specific symptoms	Pica, dysphagia, restless leg syndrome, and melena

Specific signs	Bald tongue **(Fig. 2C.4)** Koilonychia **(Fig. 2C.5)** Blue sclera **(Fig. 2C.6)**
Peripheral smear	Microcytic hypochromic red cells
Other specific investigation	Iron studies, BM staining for iron, stool/urine for occult blood, and endoscopy
Megaloblastic anemia	
Specific symptoms	Tingling and numbness Sensory ataxia
Specific signs	Glossitis, knuckle pigmentation **(Fig. 2C.7)**, absent deep tendon reflexes (DTRs), sensory loss, and positive Romberg's test
Peripheral smear	Macrocytic RBC's, hypersegmented neutrophils, and pancytopenia
Other specific investigation	Serum vitamin B_{12} levels, red cell folate levels, bone marrow examination, and schillings test
Anemia of chronic disease	
Specific symptoms	Symptoms of chronic kidney, liver disease, and connective tissue disorders
Specific sign	• Hypertension, arteriovenous (AV) fistula—chronic kidney disease (CKD) • Signs of liver cell failure—chronic liver disease (CLD) • Signs of rheumatoid arthritis, systemic lupus erythematosus (SLE), etc.
Peripheral smear	Normocytic normochromic anemia ± pancytopenia
Other specific investigation	Renal function test, liver function tests, autoantibodies, and raised serum ferritin
Hemolytic anemia	
Specific symptoms	History of associated jaundice, developmental delay, family history positivity, recurrent blood transfusions, and gallstones
Specific signs	• Triad of anemia + jaundice + splenomegaly • Hemolytic (Chipmunk) facies **(Fig. 2C.8)** • Hyperpigmentation **(Fig. 2C.9)**, short stature, and leg ulcers.
Peripheral smear	• Microcytic hypochromic (thalassemia) • Microspherocytes (hereditary spherocytosis) • Sickle cells • Reticulocytosis
Other specific investigation	Hemoglobin electrophoresis, Coombs test, sickling test, and osmotic fragility
Aplastic anemia	
Specific symptoms	Recurrent infections Bleeding manifestations
Specific signs	Signs of pancytopenia No organomegaly
Peripheral smear	Pancytopenia
Other specific investigation	• Bone marrow examination • Cytogenetics

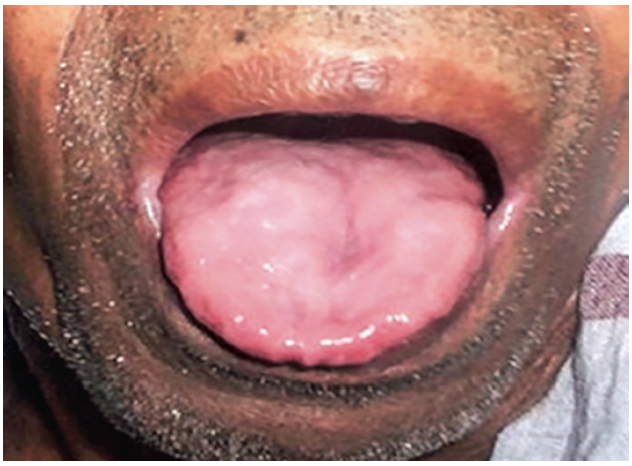


Fig. 2C.4: Bald tongue.

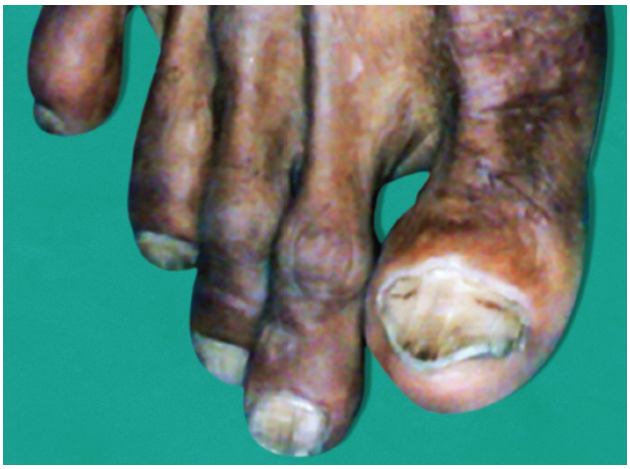


Fig. 2C.5: Koilonychia

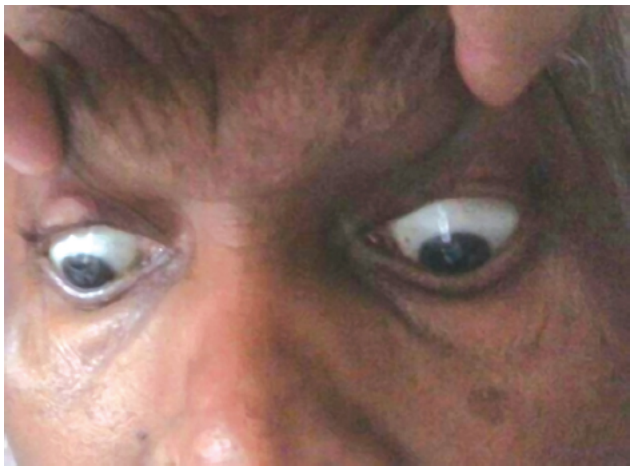


Fig. 2C.6: Blue sclera.

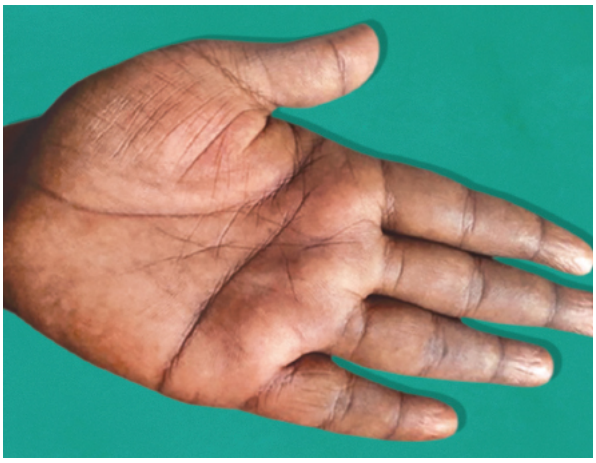


Fig 2C.9: Hyperpigmentation of palm.

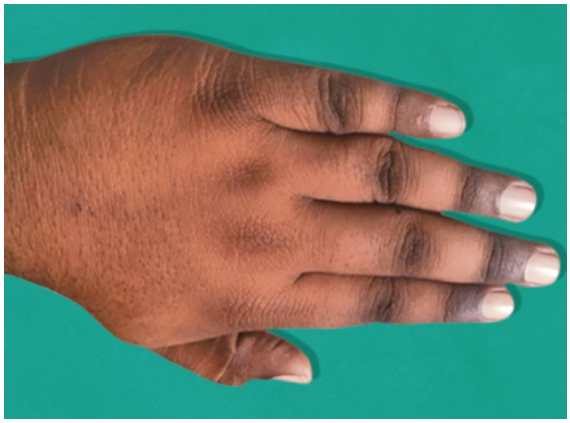


Fig. 2C.7: Knuckle pigmentation.

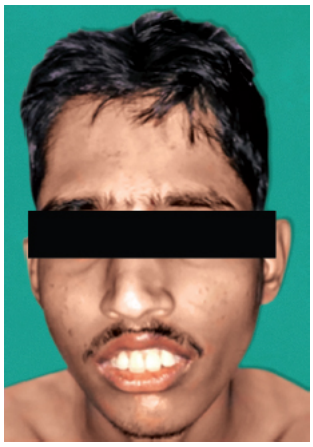


Fig. 2C.8: Chipmunk facies.

ICTERUS

Definition

Yellowish discoloration of skin, mucous membranes, sclera, and blood vessels secondary to increased bilirubin (bile pigments have affinity for elastin tissue).

Sites to Look for Jaundice

1. Sclera **(Fig. 2C.10)**
2. Sublingual mucosa
3. Oral cavity
4. Palms and soles
5. Skin.

Scleral icterus is a term commonly used but from a histopathologic perspective, it is a misnomer. Bilirubin has a high affinity for elastin, which is an abundant protein in the conjunctivae as well as the superficial, fibrovascular episclerae, but not the sclerae proper. One actually is observing icterus of the bulbar conjunctiva against the white background provided by sclera. Conjunctival icterus is often the first sign of hyperbilirubinemia. Hence we recommended the use of term "conjunctival icterus" instead of "scleral icterus".

Why unexposed sclera/conjunctiva seen

- When the sclera/conjunctiva is exposed to sunlight, bilirubin gets converted to its soluble form and hence exposed part of conjunctiva may not reveal mild jaundice.
- Yellowish discoloration can be normally seen in the exposed parts of sclera/conjunctiva which is called as muddy sclera/conjunctiva.

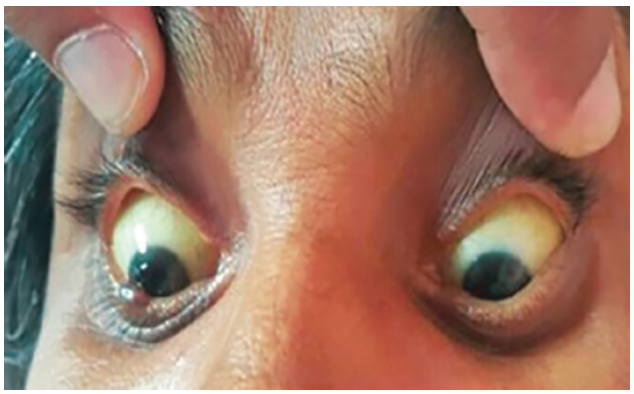


Fig. 2C.10: Demonstration of icterus.

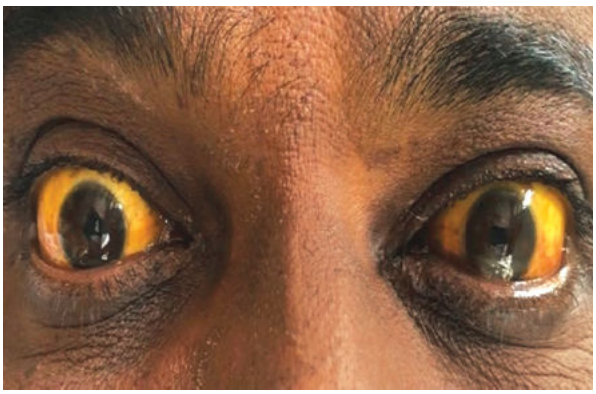


Fig. 2C.11: Dark yellow icterus.

Serum Bilirubin Levels and Jaundice

0.3–1.2 mg/dL	Normal
1.2–2.5 mg/dL	Latent jaundice (generally not appreciated on clinical examination)
>2.5 mg/dL	Clinically appreciated

Yellowish discoloration without jaundice:

1. Hypercarotenemia (here sclera is not affected)
2. Hypothyroidism (due to decreased metabolism of carotene)
3. Excessive exposure to phenols/nitric acid
4. Quinacrine intake.

Grading

No standard grading system is available; however, few examiners prefer the following:

Mild jaundice	Only sclera becomes yellow
Moderate jaundice	Skin also becomes yellow

Differentiating Type of Jaundice Based on Scleral Color

Lemon yellow	Most likely hemolytic jaundice
Dark yellow (Fig. 2C.11)	Obstructive jaundice
Greenish dark yellow	Longstanding obstructive jaundice due to oxidation of bilirubin to biliverdin

Differentiating Jaundice Based on Clinical and Laboratory Findings

	Prehepatic (hemolytic)	Hepatic	Posthepatic (obstructive/surgical)

History			
Urine	Normal	Yellow	Yellow
Stools	Normal	Normal	Pale clay like
Pruritis	-	±	++
Examination			
Bradycardia	-	-	+
Pallor	Present	Absent	Absent
Jaundice	Mild	Moderate	Severe
Splenomegaly	Present	Variable	Absent
Palpable gallbladder	±	-	++
Features of liver cell failure	Absent	+ (early feature)	± (late feature)
Laboratory data			
Serum bilirubin	UCB↑	UCB↑ + CB↑	CB↑
Serum enzymes	LDH ↑	AST ↑ ALT ↑	ALP ↑
Urine bilirubin	-	+	+
Urine urobilinogen	+	+	-
Examples			
Examples	Thalassemia Sickle cell anemia Spherocytosis Malaria Immune hemolytic anemias	Hepatitis (viral/alcoholic/drug induced) Infiltrative disorders Ischemic hepatitis	CBD stones Helminths in the CBD Carcinoma—head of pancreas Primary biliary cirrhosis Primary sclerosing cholangitis

(AST: aspartate aminotransferase; ALP: alkaline phosphatase; CB; conjugated bilirubin; CBD: common bile duct; LDH: lactate dehydrogenase; UCB: unconjugated bilirubin)

CYANOSIS

Definition

Bluish color of skin and mucous membranes resulting from an increased quantity of reduced hemoglobin (deoxygenated) or hemoglobin derivatives (methemoglobin or sulfhemoglobin) in the small vessels of those tissues.

Criteria

Deoxy Hb >5 g% or abnormal Hb (metHb or sulf Hb) ± SaO_2 <85%.

Classification

1. **True cyanosis:**
   a. Central cyanosis
   b. Peripheral cyanosis
   c. Mixed cyanosis.
2. **Pseudocyanosis.**

Etiology of Cyanosis

1. True cyanosis	
a. Central cyanosis	
Cardiac T T T T T E E	**Cyanotic heart diseases** • Truncus arteriosus • Transposition of great arteries • Total anomalous pulmonary venous connection (TAPVC) • Tetralogy of Fallot • Tricuspid atresia • Ebstein's anomaly • Eisenmengerization **(tardive cyanosis)**
Pulmonary	• Asthma • Chronic obstructive pulmonary disease (COPD) • Cor pulmonale • Respiratory failure of any cause like pneumonia, tension pneumothorax, massive pleural effusion, and acute pulmonary edema
Others	• High altitude • Polycythemia • **Enterogenous or pigment cyanosis (replacement cyanosis)** – Methemoglobinemia (>1.5 g/dL) – Sulfhemoglobinemia (>0.5 g/dL) • Carboxyhemoglobin (produces cherry red discoloration)
b. Peripheral cyanosis	
• Low cardiac output • Local vasoconstriction (cold, frostbite, and Raynaud's phenomenon) • Arterial obstruction • Venous obstruction • Hyperviscosity conditions (multiple myeloma and polycythemia) • Cryoglobulinemia	
c. Mixed cyanosis	
Left ventricular failure (has both central and peripheral cyanosis)	
2. Pseudocyanosis	
• Metals: – Gold – Silver – Mercury – Arsenic. • Drugs: • Minocycline • Chloroquine • Amiodarone.	

Atypical presentation of cyanosis		
	Description	***Example***
Differential cyanosis	Cyanosis is seen in only lower limbs	PDA with eisenmengerization
Reverse differential cyanosis	Cyanosis is seen in only upper limbs	PDA with eisenmengerization and transposition of great arteries
Three by four cyanosis	In addition to lower limbs, the left upper limb may also be cyanosed	When the patent ductus opens proximal to the origin of left subclavian artery
Intermittent cyanosis		Seen in Ebstein's anomaly

Cyclical cyanosis		Bilateral choanal atresia
Orthocyanosis	Development of cyanosis only in upright position due to hypoxia occurring in erect posture	Seen in pulmonary arteriovenous malformation
Cyanosis absent despite of sufficient reduced hemoglobin		In severe anemia, carbon monoxide poisoning

Differences between Central and Peripheral Cyanosis

Central cyanosis	Peripheral cyanosis
Due to inadequate oxygenation of systemic circulation	Due to sluggish peripheral circulation
It is a hypoxic hypoxia	It is a stagnant hypoxia
Site of examination: Tongue **(Fig. 2C.12)** Oral mucosa **(Fig. 2C.13)**	**Site of examination:** • Tip of nose • Ear lobule • Outer lips • Finger tips • Nail bed • Extremities
Extremities are warm	Extremities are cold
Do not improve on rewarming	Improves on rewarming
PaO_2 <85%	PaO_2 >85
Improves on oxygenation	Does not improve with oxygenation
Dyspnea and high volume pulse seen	Usually absent
Exercise may worsen	Exercise may improve
May be associated with clubbing and polycythemia	

Note: Cyanosis is best appreciated in areas of the body, where the overlying epidermis is thin and the blood vessel supply abundant, such as the lips, malar prominences (nose and cheeks), ears, and oral mucous membranes (buccal and sublingual); it is better appreciated in fluorescent lighting.

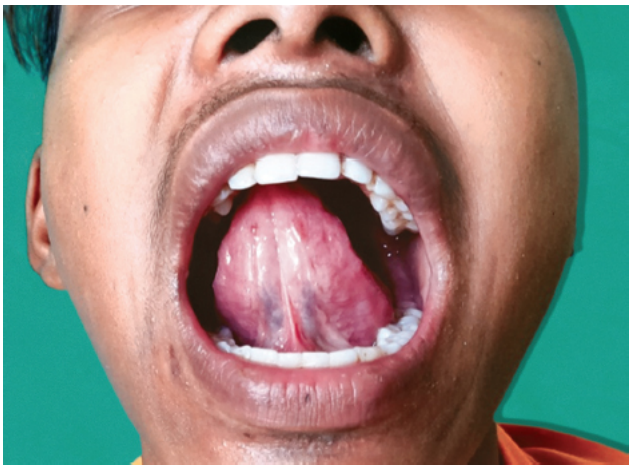


Fig. 2C.12: Demonstration of central cyanosis. (In this patient mucosa is pink and lingual veins can be clearly demarcated, which is normal).

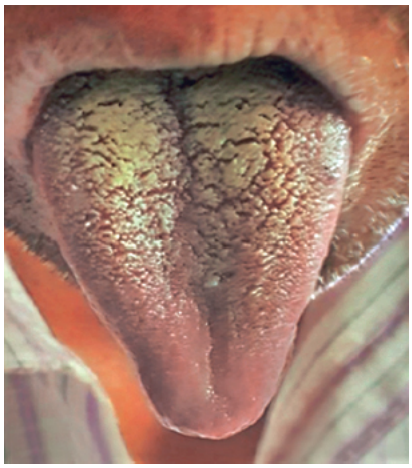


Fig. 2C.13: Bluish discoloration of tongue and oral mucosa suggestive of central cyanosis.

Hyperoxia Test (Cardiac vs Pulmonary Cyanosis)

After giving 100% oxygen for 10 minutes, a repeat arterial blood gas (ABG) is done and if PaO_2 is <150 mm Hg then the cause is cardiac and if the PaO_2 improves to >200 mm Hg, the cause is respiratory.

Iron Replete Cyanosis Versus Iron Deplete Cyanosis

Iron replete cyanosis	Iron deplete cyanosis
It is compensated erythrocytosis which establishes equilibrium with hematocrit	It is decompensated erythrocytosis which fails to establish equilibrium with unstable, rising hematocrit
Iron replete cells are deformable	Iron deplete cells are less deformable
Hyperviscosity symptoms are rare	Hyperviscosity symptoms are frequent

Theories of Cyanosis

Admixture cyanosis	Secondary to shunts
Tardive cyanosis	Due to reversal of shunt (eisenmengerization)
Hypoxic cyanosis	Due to type 1 respiratory failure
Replacement cyanosis	Due to abnormal hemoglobins
Distributive cyanosis	Venous pooling of blood

CLUBBING (HIPPOCRATES FINGERS)

Definition

Selective bulbous enlargement of distal segment of digits with subsequent loss of normal angle between the nail and nail bed.

Theories of Clubbing

PDGF (role of platelet)	The megakaryocytes preferably lodge in the tips of the digits and locally release platelet derived growth factor (PDGF) and vascular endothelial growth factor (VEGF). These growth factors along with other mediators increase endothelial permeability and activate and cause proliferation of connective tissue cells (e.g. fibroblasts)
Neurogenic	Persistent vagal stimulation causes vasodilation and clubbing (e.g. lung carcinoma)

Hypoxic	Causes opening of deep arteriovenous fistula in fingers (e.g. tetralogy of Fallot)
• **Ferritin** • **Prostaglandins** • **Bradykinin** • **Adenine nucleotides** • **5-hydroxytryptamine**	Circulating vasodilators, which are usually inactivated as blood passes through the lungs, bypass the inactivation process in the patients with right to left shunts

Grades of clubbing (Figs. 2C.14 to 2C.19)	
Grade 1	Increased fluctuation of nail bed
Grade 2	• Loss of Lovibond angle/onychonychial angle (normal is <180°) • Profile sign • Schamroth sign
Grade 3	• Parrot beaking • Drumstick fingers (seen in severe cyanotic heart disease, bronchiectasis, and empyema)
Grade 4	• Pain along the distal ends of long bone due to subperiosteal new bone formation • Condition seen generally seen with bronchogenic carcinoma
Grade 5	Glossy changes in nails and adjacent skin with longitudinal striations(as proposed by Lung India)

Causes of clubbing	
Respiratory causes	
Malignancies	Bronchogenic carcinoma Mesothelioma
Suppurative diseases	Bronchiectasis Lung abscess Empyema
Interstitial lung disease (ILD)	
Tuberculosis	Seen in 30% cases as a sequelae to complications
Sarcoidosis	Can be seen
Cardiac causes	
• Subacute bacterial endocarditis • Atrial myxoma • Cyanotic heart disease • Acyanotic heart disease with Eisenmengerization	
Gastrointestinal causes	
Inflammatory bowel disease • Ulcerative colitis • Crohn's disease Primary biliary cirrhosis Hepatocellular carcinoma	
Neurological causes	
• Syringomyelia • Median nerve injury • Hemiplegia	
Miscellaneous	
Pachydermoperiostosis (pan digital hereditary clubbing)	

Touraine-Solente-Gole syndrome

Note: Chronic obstructive pulmonary disease (COPD) never causes clubbing.

Fig. 2C.14: Demonstration of grade 1 clubbing.

Fig. 2C.15: Demonstration of profile sign.

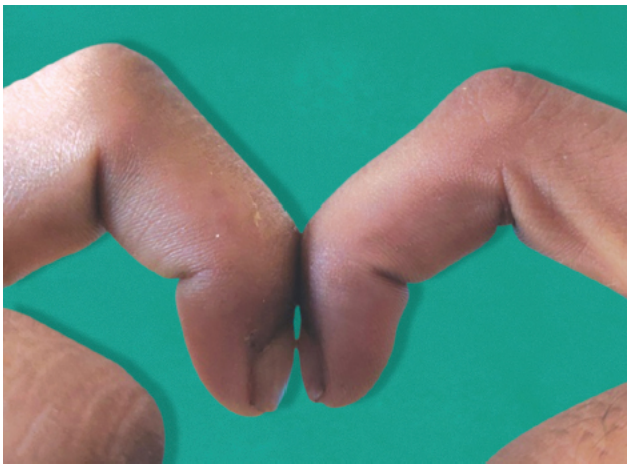


Fig. 2C.16: Demonstration of Schamroth's sign.

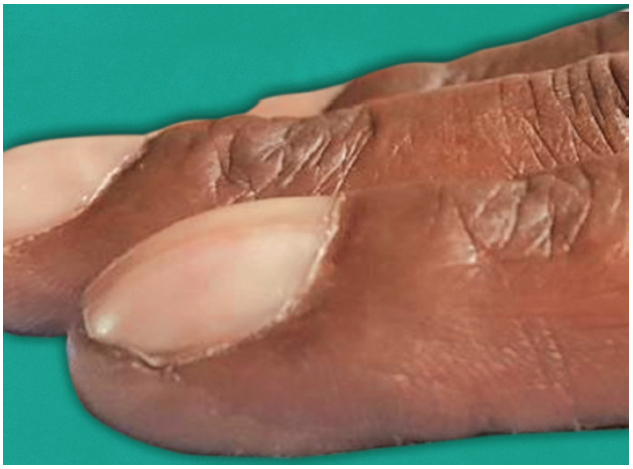


Fig. 2C.17: Demonstration of grade 3 clubbing.

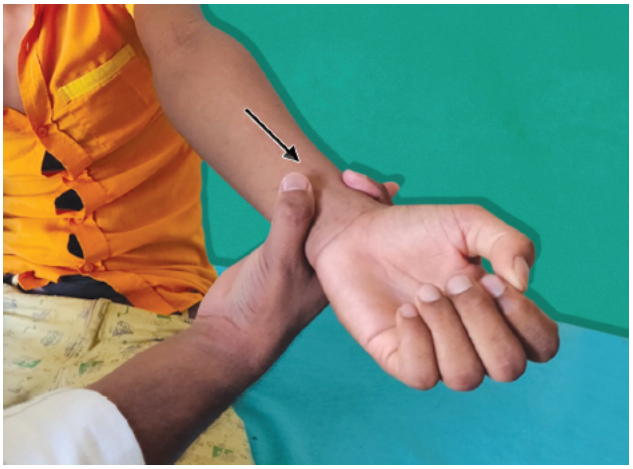


Fig. 2C.18: Demonstration of grade 4 clubbing.

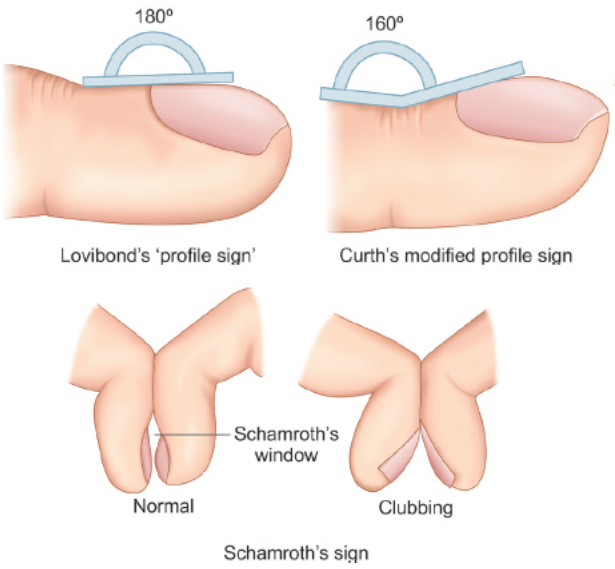


Fig. 2C.19: Image depicting profile sign and Schamroth's sign.

Atypical presentation of clubbing	
Acute clubbing	• Subacute bacterial endocarditis • Lung abscess • Empyema
Unilateral clubbing	• Hemiplegia • Aneurysm of subclavian artery • Pancoast tumor
Pseudoclubbing	• Leprosy • Leukemic infiltration • Hyperparathyroidism • Thyroid acropachy • Sclerodactyly • Exposure to vinyl chloride • Subungual tumors or cysts
Painful clubbing	• Bronchogenic carcinoma • Subacute bacterial endocarditis • Lung abscess
Reversible clubbing	• Lung abscess • Empyema
Unidigital clubbing	• Median nerve injury • Trauma
Clubbing with cyanosis	• Cyanotic congenital heart diseases • ILD
Differential clubbing: Upper limb (N) Lower limb (clubbing)	Patent ductus arteriosus (PDA) with reversal of shunt
Reverse differential clubbing: Upper limb (clubbing) Lower limb (N)	PDA + transposition of the great arteries (TGA) + reversal of shunt

Phalangeal Depth Ratio (Fig. 2C.20)

- Ratio of distal phalangeal depth (DPD) with interphalangeal depth (IPD).
- <1 is normal, >1 is suggestive of clubbing.

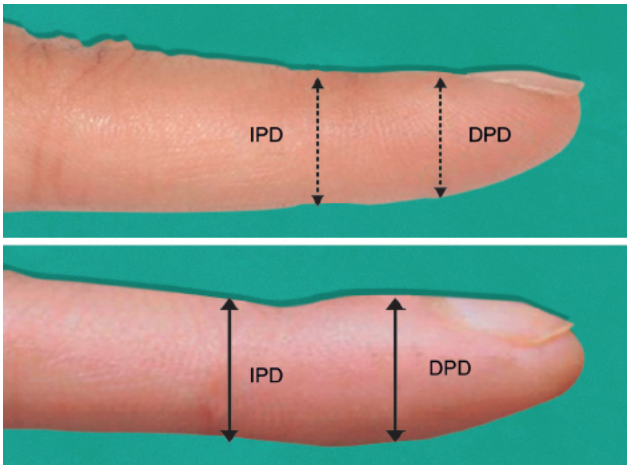


Fig. 2C.20: Picture depicting the phalangeal depth at proximal and distal interphalangeal joints.

Digital Index

- Sum of phalangeal depth ratios of 10 fingers
- A digital index of 10.2 or higher is indicative of clubbing. Although, a phalangeal depth ratio of 1.0 or greater in any finger is suggestive of clubbing, digital index is more specific for clubbing.

Other Nail Changes

Nail changes	Causes
Koilonychia	• Iron deficiency anemia (IDA) • Hemochromatosis
Beaus lines	• Measles • Pneumonia • Pulmonary infarction
Plummer nails	Seen in hyperthyroidism
Red nails	Congestive cardiac failure (CCF)
Blue nails	Copper or silver deposit
Black nails	• Peutz-Jegher's syndrome • Cushing's disease • Addison's disease
White nails	• Anemia • Hypoalbuminemia • Diabetes mellitus (DM) • CCF • Rheumatoid arthritis

EDEMA

Definition

Abnormal accumulation of fluid in interstitium.

Sites of Examination of Edema

In mobile patient	• Legs 2–3 cm above the medial malleolus
In bed ridden supine patient	• Sacrum • Back over the scapula
To check for abdominal wall edema	• Pinch the skin over the abdomen

Technique (Fig. 2C.21)

Press the skin and subcutaneous tissue for at least 15–20 seconds against a bony prominence (except for abdominal wall edema where we pinch the skin and subcutaneous tissue).

Grading of Pitting Edema (Fig. 2C.22)

1+	2-mm depression, immediate rebound
2+	4-mm deep pit, a few seconds to rebound
3+	6-mm deep pit, 10–12 seconds to rebound
4+	8-mm deep pit, >20 seconds to rebound

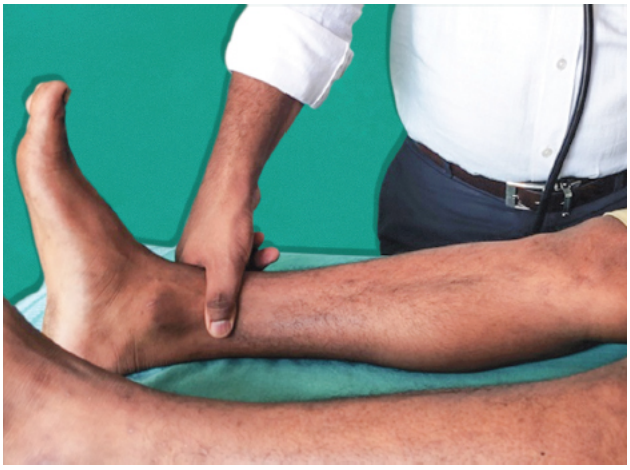


Fig. 2C.21: Method of eliciting pedal edema.

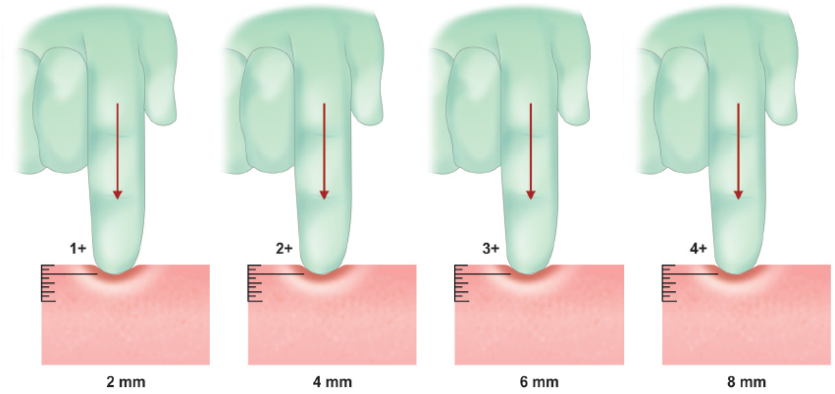


Fig. 2C.22: Grading of pitting edema.

Pitting		*Nonpitting (Brawny edema)*
Rapid recovery	**Slow recovery**	
Recovers in <40 seconds	Recovery takes >40 seconds	• Does not pit or recover in few seconds • Nontender • Skin shows hyperkeratosis
Mechanism: ↓oncotic pressure	**Mechanism:** ↑hydrostatic pressure	**Mechanism:** Lymphedema
Low serum protein	(N) serum protein	Lymphatic obstruction
Causes: **Increased protein loss** • Burns • Nephrotic syndrome • Bowel disease **Decreased intake or synthesis** • Kwashiorkor • Malabsorption • Liver disease	**Causes:** Systemic venous hypertension (HTN) • Congestive heart failure (CHF) **(Fig. 2C.23)** • Pericarditis • Tricuspid valve diseases **Local venous HTN** • Deep venous thrombosis (DVT) • Inferior vena cava syndrome	**Causes:** **Myxedema (Fig. 2C.24)—**hypothyroidism **Pretibial myxedema—**Graves's disease **Upper limb** • Breast cancer • Radiation induced **Lower limb** • Aplasia cutis • Congenital (praecox, tarda, milroy's disease, and Meigs disease) • Filariasis **(Fig. 2C.25)** • Recurred streptococcal infection • Malignancies
Facial edema: Trichinosis, hypothyroidism, allergies, nephrotic syndrome, and angioedema (Quincke's edema) ***Neurogenic edema:*** Secondary to autonomic dysfunction ***Drug-induced edema***: Nifedipine, corticosteroids, estrogen, nonsteroidal anti-inflammatory drugs (NSAIDs), and insulin • **May-Thurner syndrome**—chronic, unilateral, pitting edema due to compression of the left iliac vein by the right common iliac artery against the lumbar spine • **Idiopathic edema**—chronic, bilateral, and pitting In females <50 age, more during menstrual cycles.		

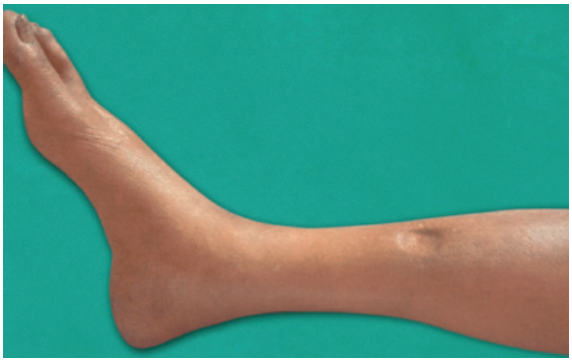


Fig. 2C.23: Pitting type of pedal edema seen in congestive cardiac failure.

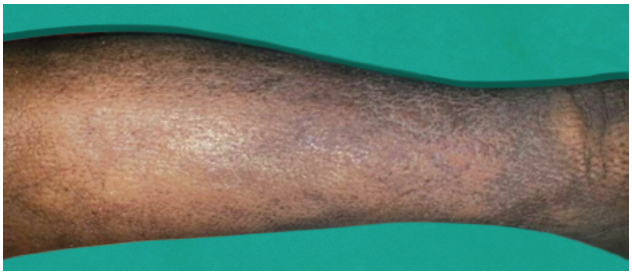


Fig. 2C.24: Nonpitting type of pedal edema seen in myxedema.

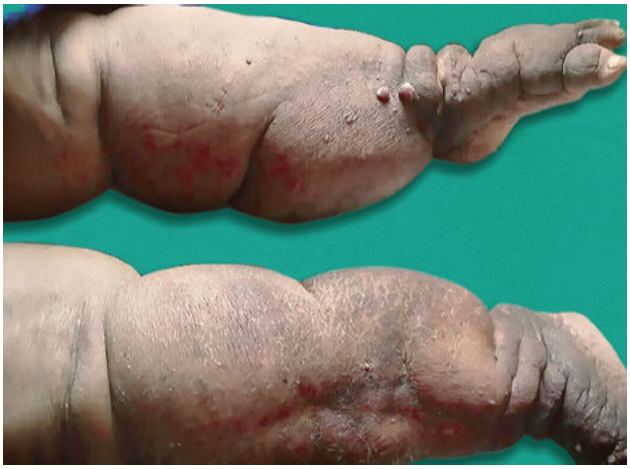


Fig. 2C.25: Nonpitting type of pedal edema seen in filariasis.

LYMPHADENOPATHY

Definitions

Generalized Lymphadenopathy

Generalized lymphadenopathy is defined as involvement of ≥2 noncontiguous lymph node groups and is typically indicative of systemic disease.

Significant Lymphadenopathy (based on Size, Fixity and Consistency)

Size >2 cm in	Inguinal region
Size >1 cm in	Extrainguinal region
Any size	• Supraclavicular • Epitrochlear • Popliteal • Any lymph node with a lesion in the draining area
Based on fixity	• Fixed to each other (matting) • Fixed to underlying tissues • Fixed to skin
Based on consistency	• Hard/firm lymph nodes

Persistent Generalized Lymphadenopathy

It is defined as lymph nodes of more than **1** cm in size, in **2** or more areas persisting for **3** or more months (mnemonic **1-2-3**). Seen in human immunodeficiency virus/acquired immune deficiency syndrome (HIV/AIDS).

Causes of generalized lymphadenopathy		
Infections	**Bacterial**	• Disseminated TB • Secondary syphilis
	Viral	• HIV • Infectious mononucleosis
	Parasitic	• Toxoplasmosis
	Fungal	• Histoplasmosis • Coccidioidomycosis • Paracoccidioidomycosis
Malignancy	• Lymphomas • Acute leukemias • Chronic lymphocytic leukemia (CLL) • Chronic myeloid leukemia (CML) (in blast crisis)	
Immunological	• Systemic lupus erythematosus (SLE) • Adult-onset Still's disease • Juvenile rheumatoid arthritis (JRA) • Sjogren's syndrome • Kawasaki disease • Serum sickness (postzone phenomenon—excess of antibody)	
Granulomatous	• Sarcoidosis • Amyloidosis • Histiocytosis X	
Endocrine	Hyperthyroidism	
Drugs	• Phenytoin (pseudolymphoma) • Primidone • Carbamazepine • Allopurinol • Captopril • Cotrimoxazole • Sulindac (NSAIDs) • Hydralazine • Beta-blockers	
Syndromic lymphadenopathy	• Kikuchi-Fujimoto disease • Castleman's disease • Kimura disease • Rosai–Dorfman syndrome • Familial Mediterranean fever	
Miscellaneous	Niemann-pick disease	

Describing a Lymph Node

1. Size (significant or not)
2. Site
3. Number
4. Consistency
5. Overlying skin
6. Mobility
7. Tenderness

8. Draining area.

Consistency

Soft	Normal consistency
Hard	Malignancy
Indian rubber	Hodgkin's lymphoma
Shotty lymph node	Syphilis
Bubo (large node with central necrosis)	Lymphogranuloma venereum
Matted	Tuberculosis **(due to periadenitis)**
Hard lymph nodes in tuberculosis	Hyperplastic tuberculosis lymphadenopathy

Different Group of Lymph Nodes (Fig. 2C.26)

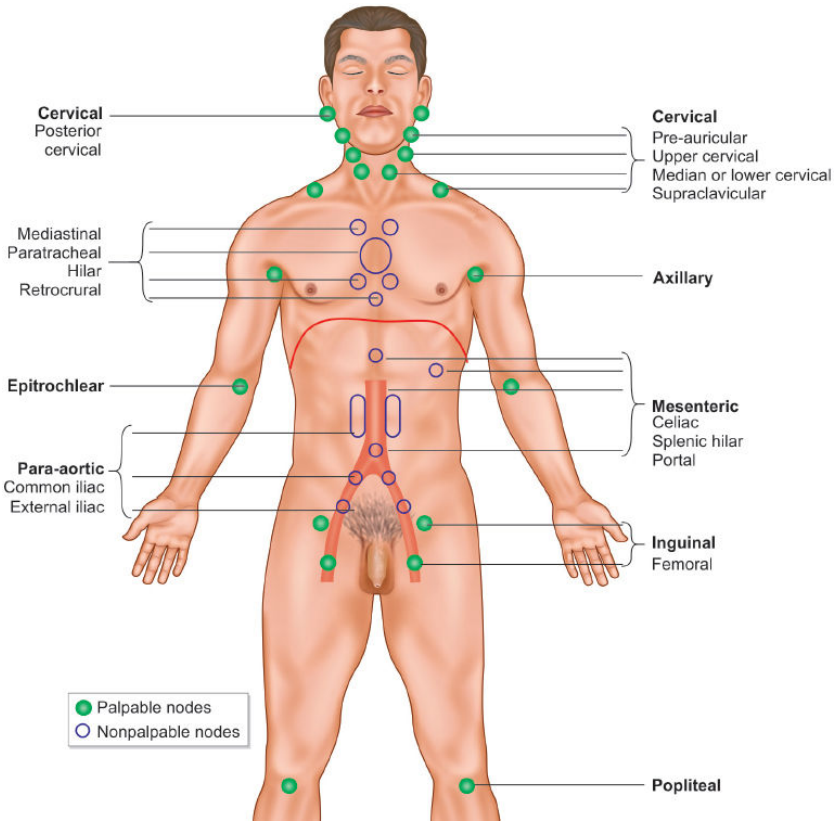


Fig. 2C.26: Image showing different groups of lymph nodes.

Cervical Lymph Nodes

Divided into:

- Superficial or deep (based on whether above or below deep cervical fascia)
- Vertical or horizontal

Superficial Cervical Lymph Nodes

- They are superficial to deep cervical fascia
- They include:

- **External Waldeyer ring**
  - » Submental
  - » Submandibular bilateral
  - » Preauricular bilateral
  - » Postauricular bilateral
  - » Occipital lymph nodes.
- Pretracheal
- Paratracheal
- Posterior triangle lymph nodes.

Deep Cervical Lymph Nodes

- Horizontal: Supraclavicular lymph nodes
- Vertical: Jugulodigastric and jugulo-omohyoid lymph nodes.

Examination of Cervical Lymph Nodes

- **Examination of anterior group** of lymph nodes is done by standing behind the patient→flex the neck to relax the fascia→first feel for the submental group (using a single finger) **(Fig. 2C.27)** and then→bilateral submandibular **(Fig. 2C.28)** → bilateral preauricular **(Fig. 2C.29)** → jugulodigastric **(Fig. 2C.30)** → juguloomohyoid **(Fig. 2C.31)** →supraclavicular groups **(Fig. 2C.32)** (± pre- and paratracheal).
- **Examination of posterior group** of lymph nodes is done by standing in front of the patient→feel for the post auricular **(Fig. 2C.33)** → occipital **(Fig. 2C.34)** → posterior triangle group of lymph nodes **(Fig. 2C.35)**.

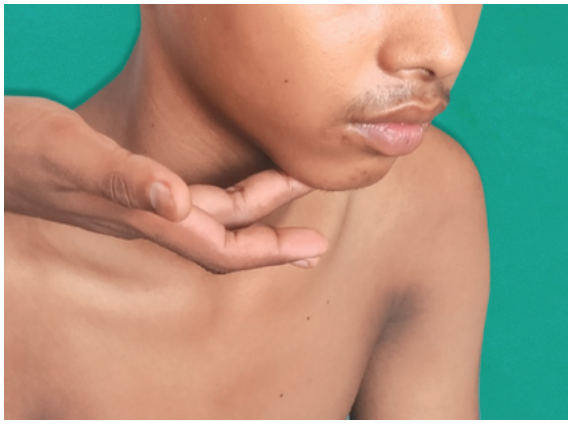


Fig. 2C.27: Method of examining submental group of lymph node.

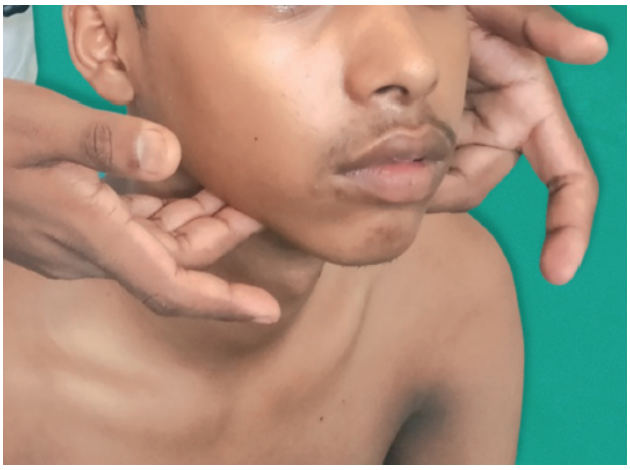


Fig. 2C.28: Method of examining submandibular lymph nodes.

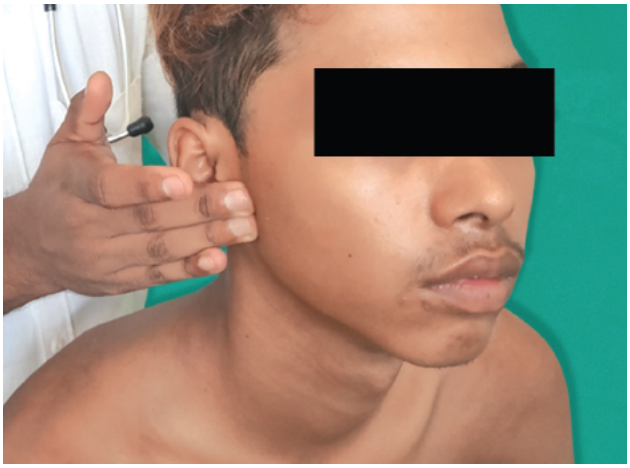


Fig. 2C.29: Method of examining preauricular lymph nodes.

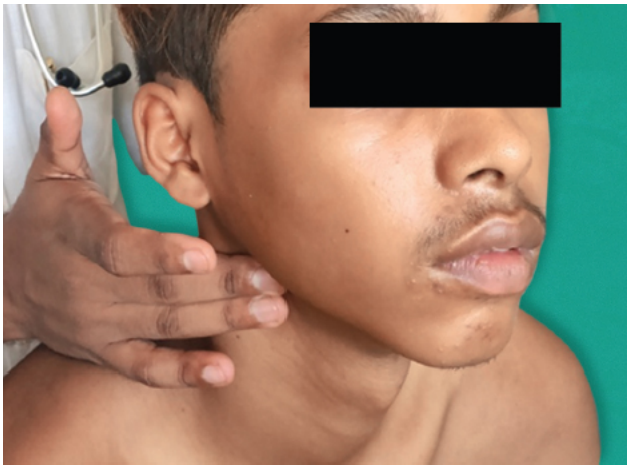


Fig. 2C.30: Method of examining jugulodigastric lymph nodes.

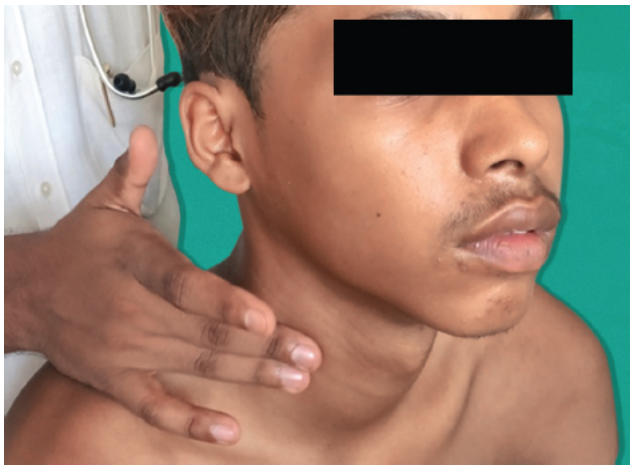


Fig. 2C.31: Method of examining jugulo-omohyoid lymph nodes.

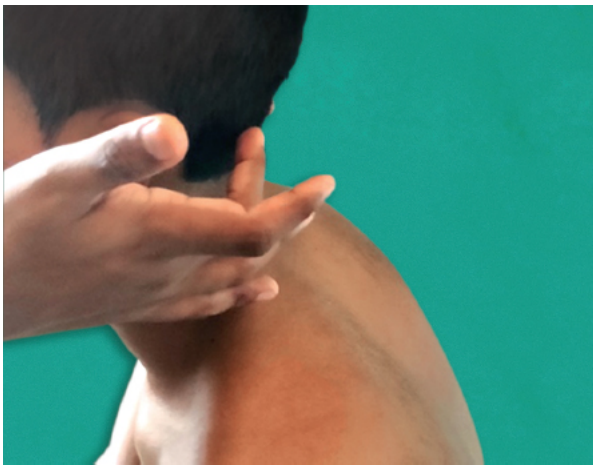


Fig. 2C.34: Method of examining occipital lymph nodes.

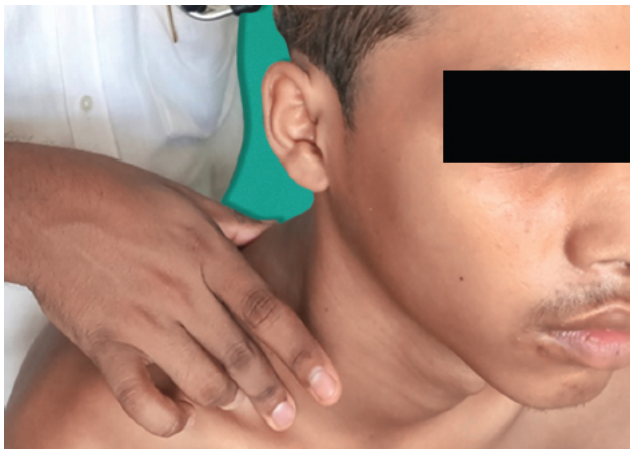


Fig. 2C.32: Method of examining supraclavicular lymph nodes.

Fig. 2C.35: Method of examining posterior triangle lymph node

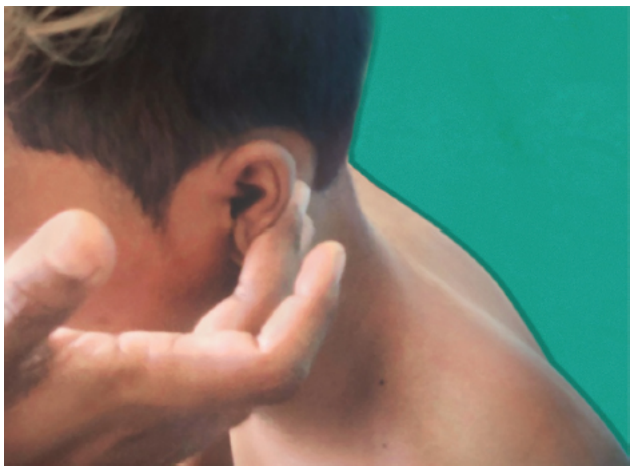


Fig. 2C.33: Method of examining postauricular lymph nodes.

Supraclavicular Lymph Nodes and Drainage

Right supraclavicular	Left supraclavicular
Right lung (all three lobes) Left lung lower lobe	Left lung upper lobe
	4 B's and Gonads: 1. Breast 2. Bronchus 3. Bowel 4. Bladder, and Gonads (testis/ovaries)
Note: **mechanism of left supraclavicular lymphadenopathy** in GI and other malignancies—reflux of tumor cells from the thoracic duct into left supraclavicular node at the junction of thoracic duct and left subclavian	
Trousseau sign of tetany: Carpopedal spasms ***Trousseaus syndrome:*** Migratory thrombophlebitis in malignancy ***Troisier's sign:*** Enlarged hard left supraclavicular lymphnode (Virchow's node).	

Other named lymph nodes	
Virchow node	Left supraclavicular node
Scalene node **(Fig. 2C.36)**	• Sentinel node of bronchogenic carcinoma • Relax neck • Palpate (deep) between the two heads of SCM
Winterbottom sign	• Posterior triangle lymph node enlargement • Seen in early phase of African trypanosomiasis
Causes of posterior triangle lymph node enlargement	• Scalp infection • Measles • Rubella • Infectious mononucleosis • Trypanosomiasis.
Node of Woods	Jugulodigastric lymph node enlargement seen in TB when spread via tonsils
Delphian node	Pretracheal node
External Waldeyer ring	Commonly seen to be enlarged in non-Hodgkin's lymphoma
Berry's node	Jugulo-omohyoid lymph nodes seen in thyroid malignancy

Axillary Group of Lymph Nodes

- There are five axillary lymph node groups
- Lymph nodes include:
  - Lateral (humeral),
  - Anterior (pectoral),
  - Posterior (subscapular),
  - Central and
  - Apical nodes.

  The apical nodes are the final common pathway for all of the axillary lymph nodes.

Note: Examine the right axillary lymph nodes with the left hand except for humeral (lateral) group (which is examined with right hand).

Examination of Right Axillary Lymph Nodes (Figs. 2C.37 to 2C.46)

Hyperabduct the right arm of patient

↓
Place the right forearm of patient on your left forearm
↓
Insinuate your left hand fingertips deep in axilla of patient
↓
Using your right hand to apply pressure over the patient's shoulder, feel for the apical lymph nodes using your left hand
↓
Central group can be felt just below the apical group
↓
Anterior group can be felt on the anterior axillary fold
↓
Posterior group can be felt on the posterior axillary fold
↓
Lateral group is felt with examiner's right hand by palpating over the patient's humerus

Drainage areas of axillary lymph nodes:

1. Chest wall with breast
2. Parietal pleura
3. Upper limb.

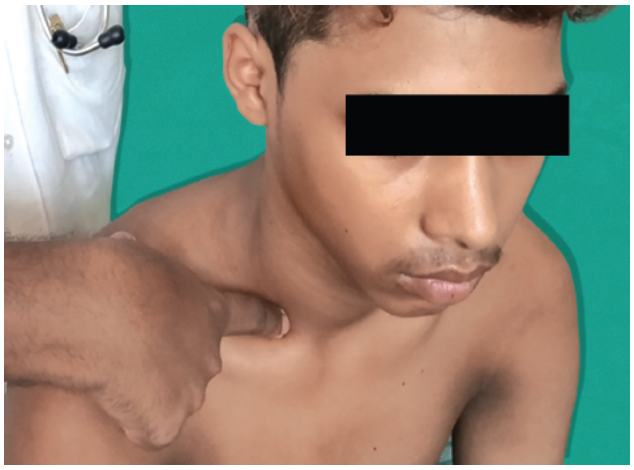


Fig. 2C.36: Method of examining scalene lymph nodes.

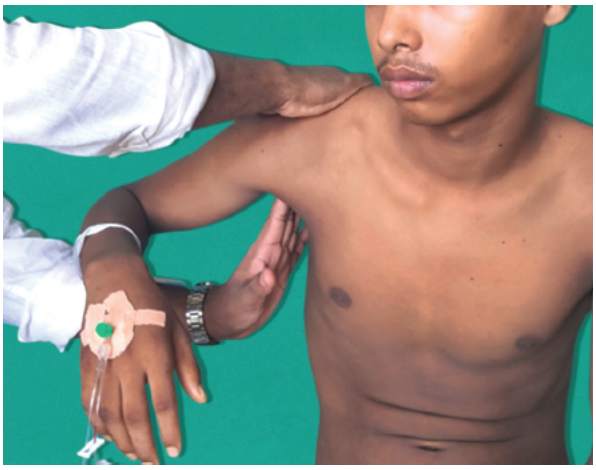


Fig. 2C.37: Method of examining right apical group (axillary) lym nodes.

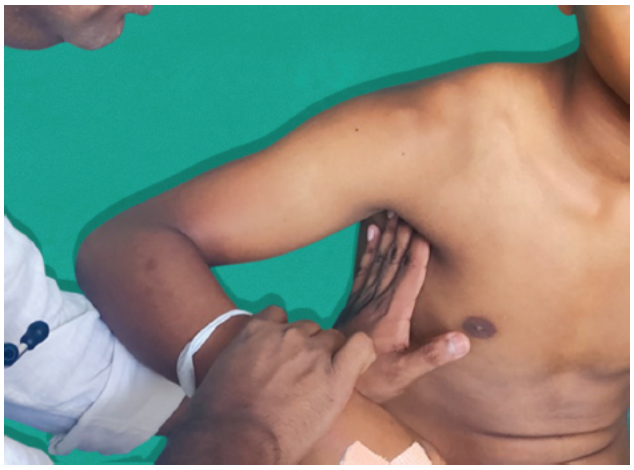


Fig. 2C.38: Method of examining right central group (axillary) lymph nodes.

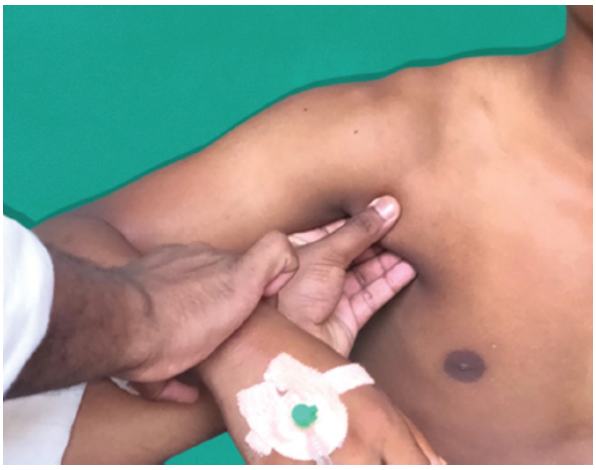


Fig. 2C.39: Method of examining right anterior group (axillary lymph nodes.

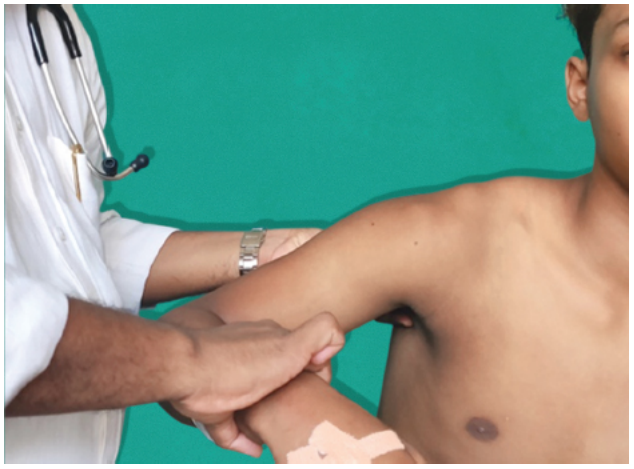


Fig. 2C.40: Methods of examining right posterior group (axillary) lymph nodes.

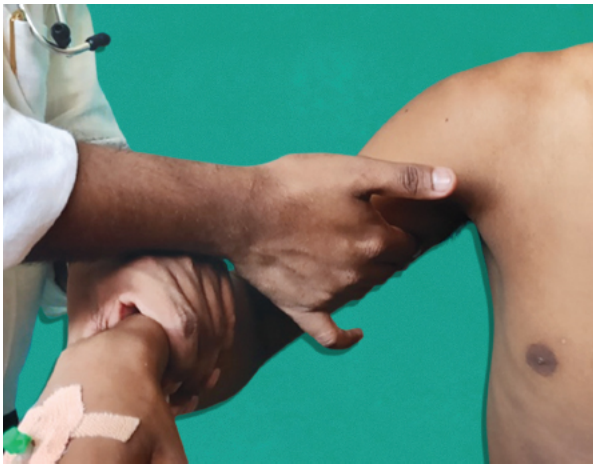
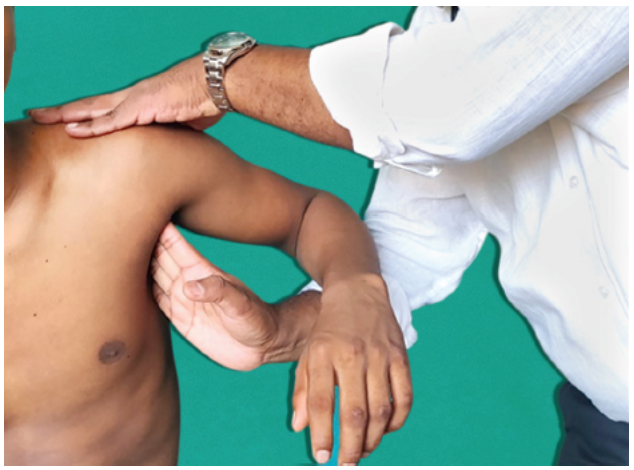


Fig. 2C.41: Method of examining right lateral group (axillary) lyn nodes.



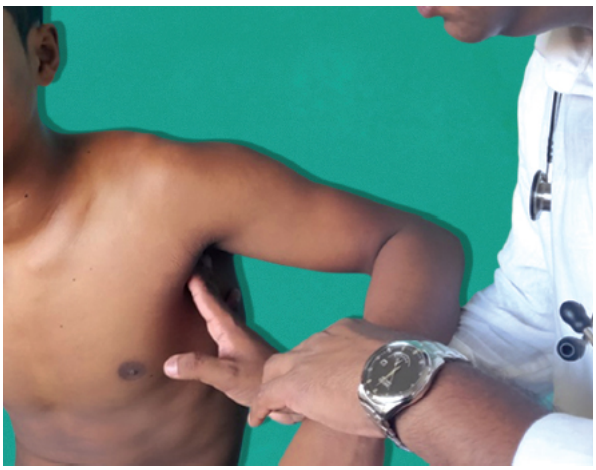


Fig. 2C.42: Method of examining left apical group (axillary) lymph nodes.

Fig. 2C.43: Method of examining left central group (axillary) lym nodes.

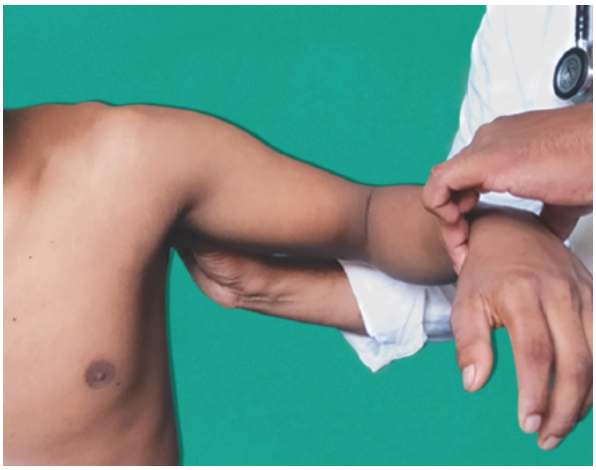
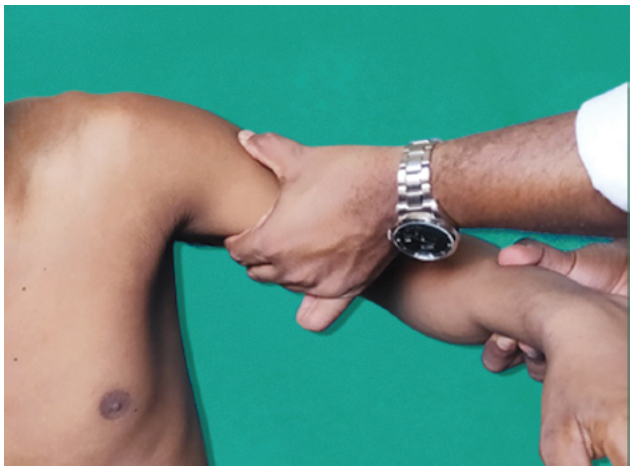


Fig. 2C.44: Method of examining left anterior group (axillary) lymph nodes.

Fig. 2C.45: Method of examining left posterior group (axillary lymph nodes.



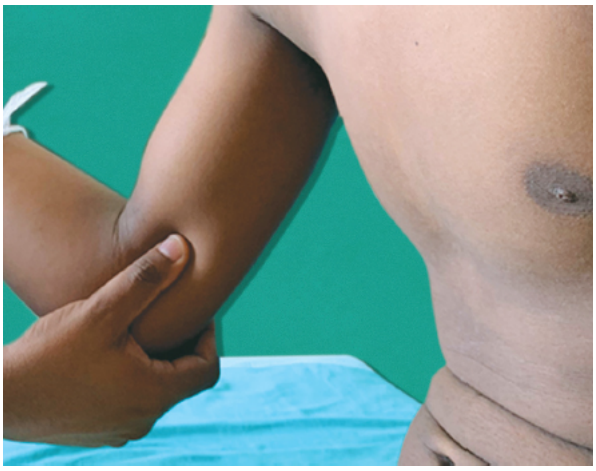


Fig. 2C.46: Method of examining left lateral group (axillary) lymph nodes.

Fig. 2C.47: Method of palpation of epitrochlear lymph nodes (t thumb).

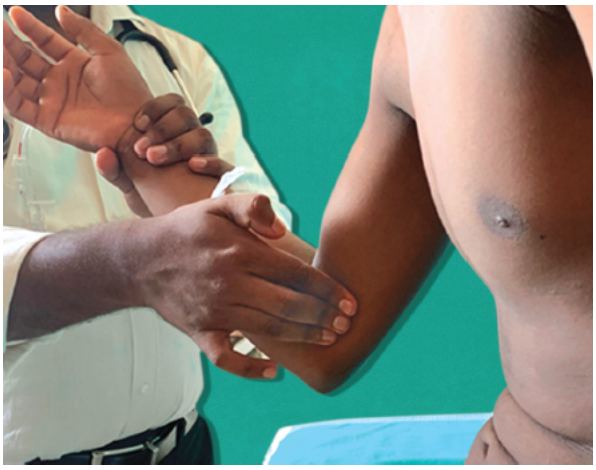


Fig. 2C.48: method of palpation of epitrochlear lymphnodes (b three fingers).

Epitrochlear Group of Lymph Nodes

- Situated on medial aspect of the elbow, about 4–5 cm above the humeral trochlea.
- Epitrochlear station drains the lymph from the last two or three fingers and from the medial aspect of the hand itself.
- For examining the right elbow—rest the right elbow of the patient on the right hand palm of the examiner and feel the lymph node with thumb as shown in the **f igure 2C.47** or by placing three fingers as shown in the **f igure 2C.48**.
- Systemic causes of epitrochlear lymphadenopathy:
  - Secondary syphilis
  - Non-Hodgkin's lymphoma (NHL)
  - Human immunodeficiency virus
  - Disseminated tuberculosis
  - Sporotrichosis
  - Cat scratch disease.

Inguinal Lymph Nodes

Horizontal group	Vertical group
Palpated along the inguinal ligament	Palpated vertically downwards from the midpoint of inguinal ligament
Drains: • External genitalia • Scrotum • Perineum • Anal canal below dentate line	**Drains:** • Lower limb

Popliteal Lymphadenopathy

- Palpate the popliteal fossa with the knee in semiflexed position
- Systemic diseases associated with enlargement include:
  - NHL
  - Disseminated TB
  - HIV.

Para-Aortic Lymphadenopathy

- Relax abdomen.
- With 2 hands placed over the epigastrium—one should feel for the enlarged lymph nodes by deep palpation.
- Enlarged in:
  - Lymphomas
  - Testicular malignancies
  - Tuberculosis.

Mesenteric Lymph Nodes

- Examined along the line of attachment of the mesentery, from the right iliac fossa medially toward the umbilicus.
- Enlarged in:
  - HIV
  - Lymphomas
  - Ulcerative colitis.

Mediastinal Lymph Nodes

- **D'Espine sign** is a bronchophony/whispering pectoriloquy heard over the vertebral spines (on the back) below the level of tracheal bifurcation; below the fourth thoracic spine (T_4) in adults.
- It indicates tracheobronchial (mediastinal) lymphadenopathy.

Nutritional deficiencies	
Vitamin deficiency	***Manifestation***
Fat-soluble vitamins	
Vitamin A, Retinol	Night blindness, keratomalacia, and Bitot's spots
Vitamin D, ergo/cholecalciferol	• Rickets/osteomalacia • Bone pain, costochondral beading • Proximal myopathy
Vitamin E, tocopherol	Hemolysis, posterior column signs, ataxia, muscle wasting, retinitis pigmentosa-like changes, and night blindness
Vitamin K, phylloquinone, and other menaquinones	Bruising, purpura, nose, and GI bleeds
Water-soluble vitamin (B-complex and vitamin C)	
B_1 (Thiamine)	• Wernicke/Korsakoff • Beriberi • Nystagmus • Sixth cranial nerve palsy • Ataxia • Acidosis • Dementia • Paraesthesiae • Neuropathy • Cardiac failure • Anemia
B_2 (Riboflavin)	• Ariboflavinosis • Angular stomatitis, glossitis, and magenta tongue
B_3 (Niacin)	• Pellagra • Dermatitis on sun-exposed areas • Dementia • Poor appetite, difficulty sleeping

	• Confusion, sore mouth
B_4 (Adenine)*	• Immune dysfunction • Aging
B_5 (Pantothenic acid)	• Nausea • Abdominal pain • Paraesthesiae, burning feet
B_6 (Pyridoxine)	• Poor appetite • Lassitude • Oxaluria
B_7 (Biotin)	Dermatitis, Depression, lassitude, Muscle pains, Electrocardiogram abnormalities, blepharitis
B_8 (Inositol)*	Depression and other psychiatric manifestations
B_9 (Folic acid)	Macrocytic anemia, Thrombocytopenia and Megaloblastic bone marrow
B_{10} (PABA)*	• Free radical damage • Sun burns and skin rashes
B_{11} (Salicylic acid) *	Works in tandem with vitamin B_{12}
B_{12} (Cobalamin)	• Subacute combined degeneration of spinal cord • Macrocytic anemia, icterus, knuckle pigmentation
Vitamin C (Ascorbic acid)	• Scurvy • Poor wound healing, fatigue, limb pain, scorbutic rosary • Difficulty sleeping, gingivitis, perifollicular purpura • Hyperkeratosis
* Vitamin B4, 8, 10, and 11 are no longer labeled as vitamins, as they do not fit the official definition of vitamin.	
Minerals	
Iron	• Koilonychia • Smooth tongue • Anemia • Esophageal web
Copper	• Microcytic hypochromic anemia • Neutropenia • Scurvy-like bone lesions, osteoporosis
Zinc	• Acrodermatitis enteropathica • Peristomal/perinasal/perineal • Erythema, thin hair • Diarrhea, apathy, anorexia • Growth failure • Hypoglycemia • Distorted or diminished taste (hypogeusia)
Chromium	Peripheral neuropathy, hyperglycemia
Selenium	Cardiomyopathy
Iodine	Goiter
Others	
Protein deficiency	• Pitting edema

	• Hair: thinning, easily pluckable with dyspigmentation or flag sign, and change in texture to silken, sparse hair. • Dermatosis with desquamation of the so-called flaky-paint type, with or without hyperpigmentation

D. ANTHROPOMETRY

HEIGHT

Method of measurement of length/height

- Recumbent length **(Fig. 2D.1)** is measured using an infantometer with a fixed head piece and horizontal backboard, and an adjustable foot piece. The **recorder supports the child's head** while the **examiner positions the feet** and ensures that the head lies in the Frankfort horizontal plane.
- Standing height **(Fig. 2D.2)** is an assessment of maximum vertical size. This stature measurement is collected on all sample persons (SPs) aged 2 years and older who are able to stand unassisted. Standing height is measured using a stadiometer with a fixed vertical backboard and an adjustable head piece. Instruct the SP to stand with the **heels together and toes apart**. The toes should point slightly outward at approximately a 60°angle. Check that the back of the **head, shoulder blades, buttocks, and heels make contact with the backboard**.

Short Stature

Short stature is defined as a height that is below the 2.5th percentile or two or more standard deviations below the mean for age and gender for a given population. A growth velocity that is below the 5th percentile for age and gender is called growth deceleration (e.g. <5 cm/year after the age of 5 years). Dwarfism is defined as short stature for the age of the patient. Most common causes of dwarfism are familial short stature and constitutional delay of growth and puberty.

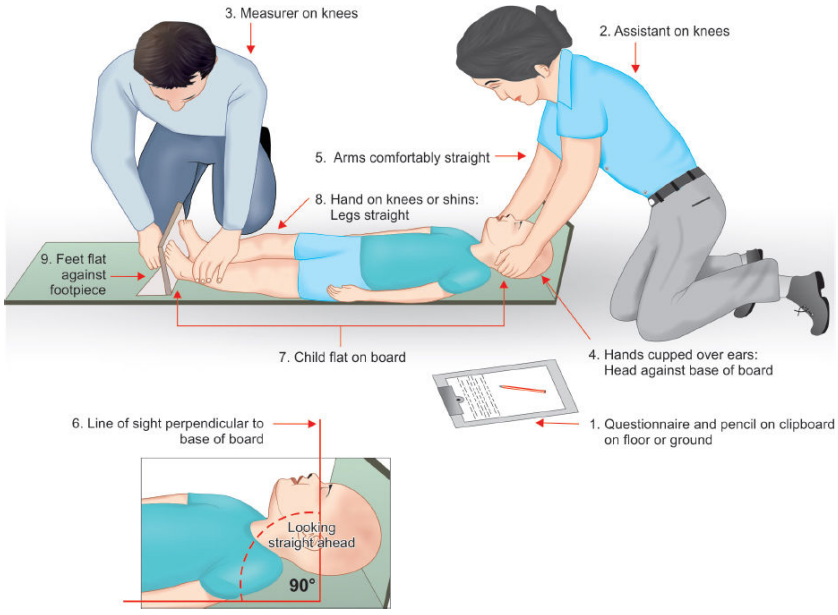


Fig. 2D.1: Measurement of recumbent length.

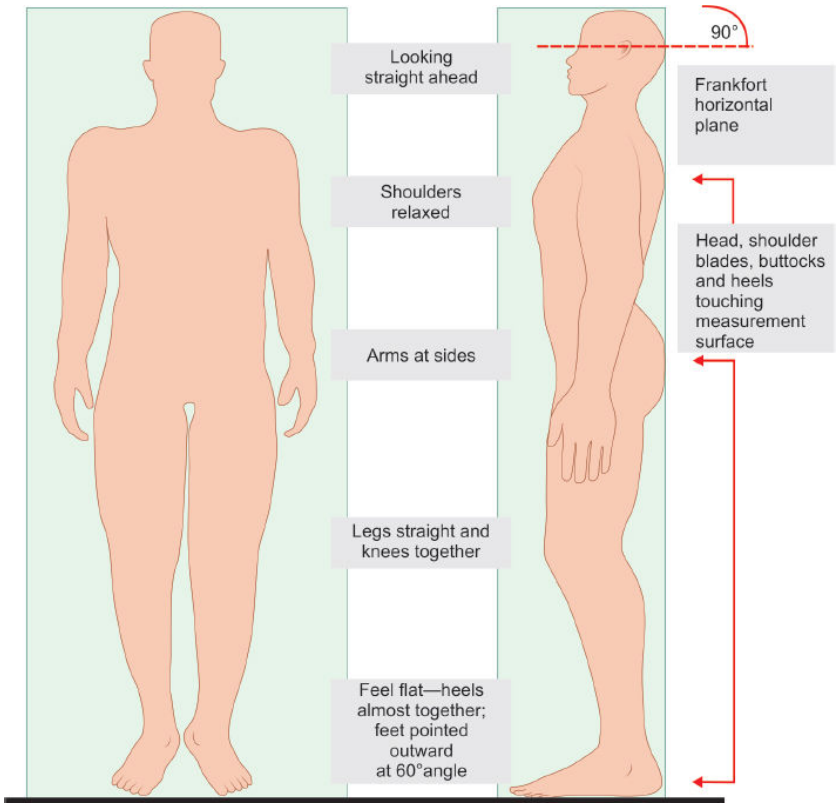


Fig. 2D.2: Measurement of vertical height.

Cause of short stature	
Constitutional (hereditary)	• Gurkhas, African pygmies
Endocrine	• Cretin (ratio between upper and lower segments is ≤1 with mental retardation) • Pituitary dwarf (short limbed, normal intelligence but may be associated with infantilism) • Froehlich's syndrome (obese, diabetes insipidus, hypogonadism) • Cushing syndrome
Genetic	• Turner syndrome • Noonan syndrome • Hurler's syndrome • Morquio's syndrome • Multiple lentigines syndrome
Skeletal	• Ellis–Van Creveld syndrome (chondrodystrophic dysplasia, short arms, and legs) • Achondroplasia (short and bowed legs and arms, waddling gait) • Osteogenesis imperfecta
Acquired (in children)	• Rickets • Pott's spine

Note: For detailed list and differential diagnosis of short stature refer to page No. 74–75 in Exam Preparatory Manual for Undergraduates by Archith Boloor.

Tall Stature

When the height of an individual is far in excess of the average normal for the age and race (≥2 standard deviation of the mean height), the individual is considered to be tall in stature.

Causes of tall stature

Tall stature with equal upper and lower segments or equal arm span to height ratio	Tall stature with unequal upper to lower segment (ratio of ≤0.8) or arm span to height (ratio of ≥1.05)
• Constitutional tall stature • Pituitary giants • Sexual precocity • Thyrotoxicosis	• Marfan syndrome (MFS) • Homocystinuria • Klinefelter's syndrome

ARM SPAN

Method of Measurement of Arm Span

It is the distance between the tips of the middle fingers of one hand to the other when held abducted in horizontal plane. The arm span to height ratio is normally equal or ≤1.05.

Clinical implication of arm span versus height ratio:

Age	Ratio
At birth	The arm span is typically less than length (by at least 2.5 cm)
10 years of age in boys and 12 years of age in girls	The arm span exceeds height

Cause of increased arm span-height ratio:

- Klinefelter syndrome
- Homocystinuria
- Marfan's syndrome
- Sotos syndrome
- Hypogonadism

UPPER SEGMENT AND LOWER SEGMENT

Method of Measurement

The upper segment of the body is measured from the top of the head to pubic symphysis/pubic ramus and the lower segment is measured from the pubic ramus to the floor.

Clinical implication of upper segment:lower segment (US:LS) ratio:

Age	Ratio
Birth	1.7
3 years	1.33
5 years	1.17
10 years	1.0
>10 years	<1.0

Causes of increased and decreased US:LS ratio:

Increased US:LS ratio	Decreased US:LS ratio
Children with rickets, achondroplasia, and Turner syndrome (because of decreased limb length)	Marfan syndrome (because of increased limb length)

SKINFOLD THICKNESS

Method of Measurement

- Approximately half of the total amount of fat tissue in the human body is located below the surface of the skin.
- This makes it possible to predict total body fat from skin-fold thicknesses with a relative high degree of accuracy using a simple two-compartmental method.
- This accuracy is confirmed by CT scan as well as ultrasonic and radiographic techniques used to measure subcutaneous fat.
- In general, when measuring skinfold thickness. The assessor, using the forefinger and the thumb, grasps and lifts the subcutaneous tissue and skin from the underlying muscle.
- Places the pincers of the skinfold caliper **(Fig. 2D.3)** applying a constant pressure, 2 cm below the fingers at a depth of 1 cm.
- Holds this position for 3–4 seconds.
- Takes three measurements for accuracy.
- Provides the actual skinfold thickness in mm.

Triceps Skinfold (TSF) (Fig. 2D.4)

- A measure of subcutaneous fat stores taken at the midpoint of the posterior aspect of the humerus.
- Correlates closely with percentage of body fat and with total body fat.
- Triceps skinfold thickness varies between 6 mm and 12 mm in lean individuals and between 40 mm and 50 mm in obese individuals.
- Subject should be **standing** with **arms hanging loosely at the sides.**
- Assessor to be positioned behind the subject.
- To locate the triceps skinfold site, **locate the site previously marked for the mid-arm circumference (MAC) measurement.**
- The triceps skinfold site is on the posterior surface of the arm, midway between the shoulder and the elbow.
- **Using the forefinger and the thumb** the assessor **grasps and lifts the subcutaneous tissue and skin 2 cm above TSF site.**
- Place the **pincers of the skinfold caliper** at the **TSF point at a depth of 1 cm.**

Fig. 2D.3: Different types of skinfold calipers.

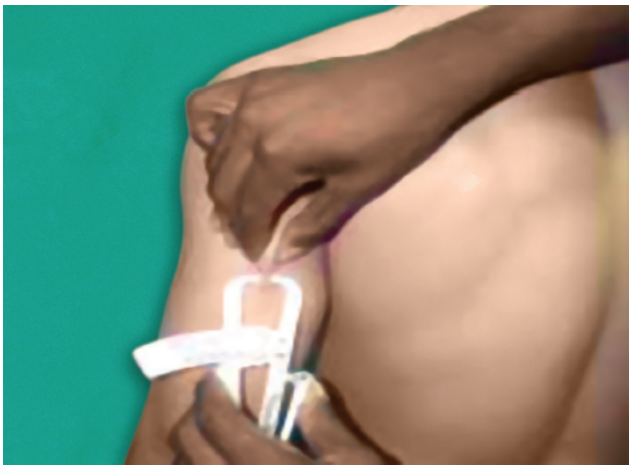


Fig. 2D.4: Triceps skinfold (TSF).

- Hold this position for 3–4 seconds.
- Take three measurements for accuracy.
- Provide the actual skinfold thickness in mm.

BODY MASS INDEX

Calculation

Formula is weight (kg)/Height (m^2)

Body Mass Index

	World Health Organization (WHO)	Southeast Asian Countries (SEAC)
Underweight	<18.5	<18.5
Normal	**18.5–24.9**	**18.5–22.9**
Overweight	25–29.9	23–24.9
Preobese	—	25–29.9
Obese	**≥30**	**≥30**
Obese 1	30–40	30–40
Obese 2 (morbid)	40.1–50	40.1–50
Obese 3	>50	>50

Metabolic syndrome	
National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III) 2005*	WHO 1999
Essential criteria	
—	Insulin resistance
Additional criteria	
(≥3 of following)	**(≥2 of following)**
Waist circumference (WC) • >90 cm (males)	**Waist-hip ratio (WHR)** • 0.9 (males)

• >80 cm (females)	• >0.85 (females) • BMI ≥30
Glucose ≥100 mg/dL or on Rx	
Triglyceride (TG) ≥150 mg/dL or on Rx	TG ≥150 mg/dL
High-density lipoprotein (HDL) <40 (males) <50 (females) or on Rx	HDL <35 (males) <40 (females)
Hypertension (HTN) ≥130/85 or on Rx	HTN ≥140/90

*Most commonly followed.

WAIST-HIP RATIO (FIG. 2D.5)

Method of Measurement

Waist Circumference

- Locate the narrowest point between ribs and iliac crests.
- Ensure that the tape measure is at the same height around the waist.
- Measure and state the measurement correctly to the nearest centimeter.
- **≥90 cm (adult male) and ≥80 cm (adult female) considered having abdominal obesity for south Asians.**
- **Differences in cut points abdominal obesity for south Asians and Europids.**

Abdominal obesity	South Asians	Europids
Men	WC ≥90 cm	WC ≥102 cm
Women	WC ≥80 cm	WC ≥88 cm

Hip Circumference

- Hip measurement is taken at the widest lateral extension of the hips.
- Ensure that the tape measure is horizontal.
- Measure and state the measurement correctly to the nearest centimeter.
- Calculate waist-hip ratio to two decimal places.

Clinical Implication

- 0.9 (males) or >0.85 (females) are criteria for metabolic syndrome.

MID-ARM CIRCUMFERENCE (FIGS. 2D.6 AND 2D.7)

- Locate the midpoint of the arm.
- Nondominant arm elbow flexed at 90° with palm facing upwards.
- Measurer stands behind the subject and locates the lateral tip of the acromion and the most distal point on the olecranon process.
- Place a tape measure so that it passes between these two landmarks and mark the midpoint.
- The subject stands erect with arms hanging freely at the sides and the palms facing the thighs.
- Place the tape measure perpendicular to the long axis of the arm at the marked midpoint and measure the circumference to the nearest mm (e.g. 18.1 cm).
- Provide the actual MAC in cm.

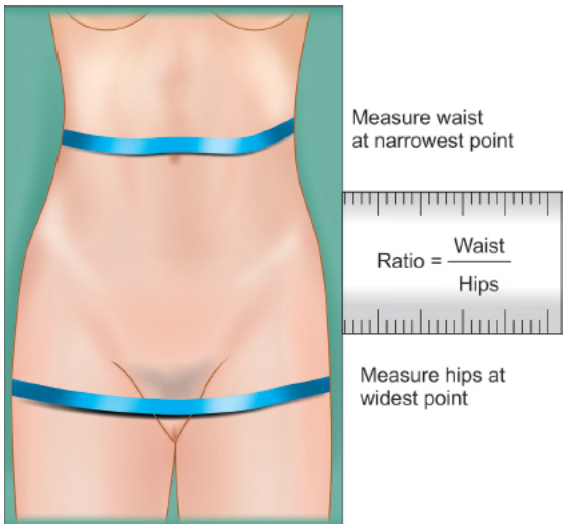


Fig. 2D.5: Examination of waist-hip ratio.

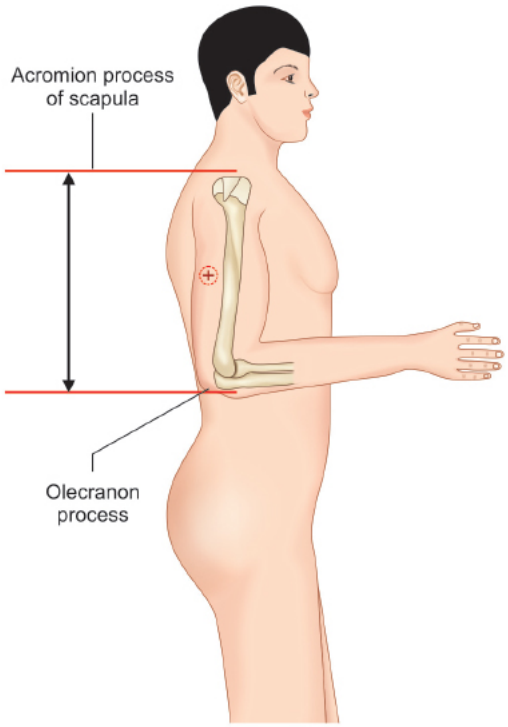


Fig. 2D.6: Method of marking midpoint for measuring mid-arm circumference.

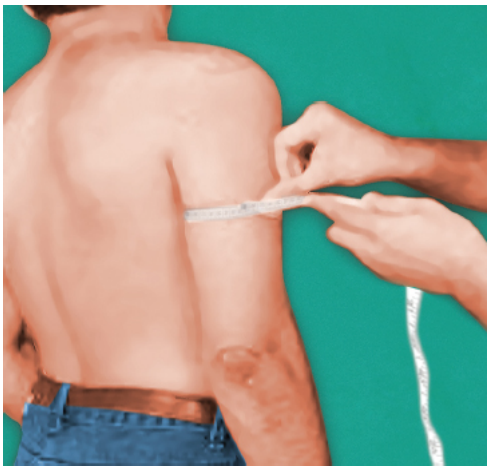


Fig. 2D.7: Method of measuring mid-arm circumference.

NECK CIRCUMFERENCE

- Neck circumference (NC) measurement, as a simple and time-saving screening measure, could be used to identify overweight and obese population.
- Measured on a plane as horizontal as possible, at a point just below the larynx (thyroid cartilage), and perpendicular to the long axis of the neck (the tape line in front of the neck should be placed at the same height as the tape line in the back of the neck).

- Varies based on population. Among south Asians, an NC of >34.9 cm for men and >31.25 cm for women were the best predictors for identifying metabolic syndrome.

NECK HEIGHT RATIO

- Neck length was measured as the linear distance between two easily recognizable and fixed bony points—the external occipital protuberance and the spinous process of C7 vertebra; with the patient standing upright and neck held in neutral position.
- Normal ratio of neck: height is 1:13 (Bird index).
- Short neck is an important feature of conditions like Turner, Noonan, Klippel–Feil, and mucopolysaccharidoses.
- Neck height ratio (NHtR) has also been suggested to be a measure of upper body adiposity like NC.

MISCELLANEOUS TOPICS

Significant Weight Loss

- >10% of body weight × 6 months
- 5 kg or more × 1 month

Cachexia

Complex metabolic syndrome associated with underlying illness and is characterized by the loss of muscle with or without loss of fat mass.

Emaciation

Extreme weight loss and unnatural thinness due to a loss of the fatty, adipose tissue beneath the skin and muscle throughout body.

Weight for Age (W/A)

- General appreciation of nutritional status
- For growth monitoring.

Height for Age (H/A)

- Measure of linear growth deficit or **stunting**
- Slow progress
- Used for community diagnosis.

Weight for Height/Length (W/H)

- Measure of weight deficit according to length
- Measure of wasting
- Used for individual and community diagnosis.

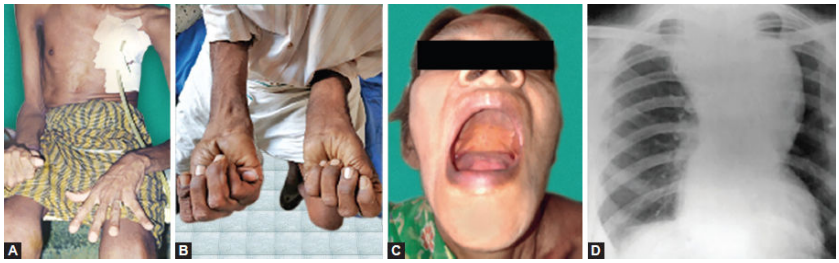
MARFAN'S SYNDROME: DIAGNOSTIC CRITERIA AND FEATURES (FIGS. 2D.8A TO D)

Diagnostic criteria (Modified Ghent criteria)	
In the **absence of family history** of MFS, the presence of one of any of the following criteria is diagnostic for MFS	In the **presence of family history** of MFS, the presence of one of any of the following criteria is diagnostic for MFS
1. Aortic criterion and ectopia lentis	1. Ectopia lentis

2. Aortic criterion and a causal FBN1 mutation	2. Systemic score ≥7 points
3. Aortic criterion and a systemic score ≥7	3. Aortic criterion
4. Ectopia lentis and a causal FBN1 mutation	

Aortic Criteria

Aortic diameter Z score ≥2 (above 20 years old), Z score ≥3 (below 20 years), or aortic root dissection.



Figs. 2D.8A to D: Features of Marfan's syndrome. (A) Wrist sign; (B) Thumb sign; (C) High-arched palate; (D) Chest X-ray showing aortic root dilatation.

Systemic Scoring

- A systemic score ≥7 indicates major systemic involvement.
- Calculate based on the following table:

Features	Points
Wrist **AND** thumb sign	3
Wrist **OR** thumb sign	1
Pectus carinatum deformity	2
Pectus excavatum or chest asymmetry	1
Hindfoot deformity	2
Plain pes planus	1
Pneumothorax	2
Dural ectasia	2
Protrusio acetabuli	2
Reduced upper segment/lower segment ratio **AND** increased arm span/height **AND** no severe scoliosis	1
Scoliosis or thoracolumbar kyphosis	1
Reduced elbow extension (≤170° with full extension)	1
Facial features [at least three of the following five features: dolichocephaly (reduced cephalic index or head width/length ratio), enophthalmos, downslanting palpebral fissures, malar hypoplasia, retrognathia)	1
Skin striae	1
Myopia >3 diopters	1
Mitral valve prolapse (all types)	1

CHAPTER 3

Respiratory System Examination

A. CASE SHEET FORMAT

HISTORY TAKING

Name:

Age:

Sex:

Residence:

Occupation:

Chief complaints:

1. _____ × days
2. _____ × days
3. _____ × days

History of presenting illness:

Cough:

- Duration
- Onset
- Progression
- Variation
  - Diurnal variation
  - Seasonal variation
  - Postural variation
- Aggravating factors
- Relieving factors

Expectoration:

- Duration
- Onset
- Progression
- Variation
  - Diurnal variation
  - Seasonal variation
  - Positional variation
- Aggravating and relieving factors
- Quantity of sputum
- Color
- Smell
- Blood tinged
  - How often
  - Quantity
  - Fresh or altered

Dyspnea:

- Duration
- Onset
- Grade
- Progression
- Aggravating factors
- Relieving factors
- Orthopnea
- Trepopnea
- Platypnea
- Paroxysmal nocturnal dyspnea (PND)
- Any respiratory system complaints
  - Wheeze
  - Cough with expectoration

Chest pain:
- Duration
- Onset
- Site
- Type of pain
- Radiation
- Diurnal variation (nocturnal angina)
- Variation with respiration
- Aggravating factors
- Relieving factors
- Associated symptoms
  - Nausea, vomiting, sweating
- Dyspepsia
- Local tenderness

Wheeze:
- Duration
- Onset
- Progression
- Episodic or continuous
- Variation
- Allergy
- Skin rashes
- Aggravating and relieving factors

Fever:
- Episodic or continuous
- Grade
- Chill and rigors
- Aggravating factors
- Relieving factors
- Variation
  - Diurnal variation

History of:

- Nasal discharge
- Recurrent cold/epistaxis
- Recurrent headaches
- Weight loss
- Anorexia
- Evening rise of temperature
- Smoking
- Belching
- Regurgitation of food
- Hoarseness of voice

Past history:
- Asthma
- Chronic obstructive airway disease
- Tuberculosis
- History of contact with tuberculosis
- Diabetes mellitus (DM)
- Hypertension (HTN)
- Ischemic heart disease (IHD)
- Seizure disorder

Family history:
(draw pedigree chart representing three generations as discussed in the chapter 15)

Personal history:
- Bowel habits
- Bladder habits
- Appetite
- Loss of weight
- Occupational exposure
- Sleep
- Dietary habits and taboo
- Food allergies
- Smoking (in smoking Index or Pack years)
- Alcohol history (__ grams of alcohol/day or ___ units of alcohol/week)

Menstrual and obstetric history
- G__P__L__A__
- Age of menarche __
- Menopause at __
- Flow—amenorrhea/oligomenorrhea/menorrhagia

Summarize:

Differential diagnosis:
1.
2.
3.

GENERAL EXAMINATION

Patient
- Conscious
- Cooperative
- Obeying commands

BMI
- W (kg)/H^2 (m)
- Grading according to WHO for Southeast Asian countries

Vitals
- Pulse
  - Rate
  - Rhythm
  - Volume
  - Character
  - Vessel wall thickening
  - Radio-radial delay and radio femoral delay
  - Peripheral pulses
- Blood pressure
  - Right arm
  - Left arm
  - Right leg
  - Left leg
- Respiratory rate
  - Regular
  - Abdominothoracic (male) or thoracoabdominal (female)
  - Usage of accessory muscles
- Jugular venous pulse
  - Waveform
- Jugular venous pressure
  - __ cm of blood above sternal angle (+ 5 cm water)
- Pulse oximetry
- Pain

On physical examination
- Pallor:
- Icterus:
- Cyanosis:
- Clubbing:
- Lymphadenopathy:
- Edema:

Others head to toe:
- Oral cavity examination
- Use of accessory muscles of respiration
- External markers of tuberculosis
- External markers of malignancy
- Features suggesting type of respiratory failure

SYSTEMIC EXAMINATION

Upper Respiratory Tract Examination

- Nostrils:
- Nasal septum:
- Nasal polyps:
- Sinus tenderness:
- Tonsils:
- Post-pharyngeal wall:

Lower Respiratory Tract Examination

Inspection

- Shape and symmetry:
- Spine:
- Sub costal angle:
- Trachea:
- Apex beat:
- Respiratory movements:

Area	Right	Left
Upper anterior chest		
Lower anterior chest		
Upper posterior chest		
Lower posterior chest		

- Visible pulsations/sinus/scars:

Palpation

(warm the palms by rubbing against each other before palpation)

- Spine: Position and tenderness
- Trachea:
- Apex:

Respiratory movements:

Area	Right	Left
Supraclavicular		
Infraclavicular		
Mammary		
Suprascapular		
Infrascapular		

Dimensions/Measurements

T diameter	
AP diameter	
T: AP diameter	

Chest circumference	Expiration	
	Inspiration	
Right hemithorax	Expiration	
	Inspiration	
Left hemithorax	Expiration	
	Inspiration	
Chest expansion	Right hemithorax	
	Left hemithorax	
	Total	
Spinoscapular distance	(Right side) and (left side)	
Spinoacromial distance	(Right side) and (left side)	

Vocal fremitus:

Areas	Right	Left
Supraclavicular		
Clavicular		
Infraclavicular		
Mammary		
Axillary		
Infra-axillary		
Suprascapular		
Interscapular		
Infrascapular		

- Tactile fremitus:
- Friction fremitus:
- Tenderness:
- Subcutaneous emphysema:
- Rib crowding:
- Bony tenderness:

Percussion

Areas	Right	Left
Supraclavicular		
Clavicular		
Infraclavicular		
Mammary		
Axillary		
Infra-axillary		
Suprascapular		

Interscapular		
Infrascapular		

- Shifting dullness:
- Tidal percussion:
- Traube's space:
- Kronig's isthmus:
- Liver dullness:
- Liver span:

Heart border on:

- Right side:
- Left side:

Auscultation

Breath sounds:

Vesicular/bronchovesicular/bronchial (tubular/cavernous/amphoric)

Comment on intensity of breath sound—normal/increased/decreased

Areas	Right	Left
Supraclavicular		
Clavicular		
Infraclavicular		
Mammary		
Axillary		
Infra-axillary		
Suprascapular		
Interscapular		
Infrascapular		

Vocal resonance:

Areas	Right	Left
Supraclavicular		
Clavicular		
Infraclavicular		
Mammary		
Axillary		
Infra-axillary		
Suprascapular		
Interscapular		
Infrascapular		

Adventitious sounds:
- Crepitations
- Rhonchi (inspiratory or expiratory/polymorphic or monomorphic)
- Rubs

Additional tests:
- Coin test:
- Bronchophony:
- Egophony:
- Whispered pectoriloquy:
- Succussion splash:
- Post-tussive crepts:

Other Systems

Cardiovascular system:
- Inspection:
- Palpation:
- Percussion:
- Auscultation:

Gastrointestinal system:
- Inspection:
- Palpation:
- Percussion:
- Auscultation :

Nervous system:
- Higher mental functions:
- Cranial nerves:
- Sensory system:
- Motor system:
- Reflexes:
- Cerebellar system:
- Meningeal signs:

NOTES

B. DIAGNOSIS FORMAT

ANATOMICAL DIAGNOSIS

- Lung (right/left/bilateral) disease with (upper/middle/lower) lobe
- Pleural disease

PATHOLOGICAL DIAGNOSIS

Consolidation/fibrosis/collapse/obstructive lung disease/restrictive lung disease/effusion/pneumothorax.

ETIOLOGICAL DIAGNOSIS

Tuberculosis/bronchogenic carcinoma/smoking/occupation/trauma.

COMPLICATIONS

Respiratory failure (type I or type II)/corpulmonale.

EXAMPLES

Example 1

Right upper lobe fibrosis post-tubercular etiology, no evidence of respiratory failure or corpulmonale.

Example 2

Bilateral obstructive lung disease—emphysema secondary to smoking with evidence of type 2 respiratory failure and corpulmonale.

Example 3

Left-sided pleural effusion secondary to malignancy with no evidence of respiratory failure or corpulmonale.

NOTES

C. DISCUSSION ON CARDINAL SYMPTOMS

Symptoms discussed include:

1. Cough
2. Expectoration
3. Hemoptysis
4. Dyspnea
5. Chest pain (with respect to respiratory system)
6. Others

COUGH

Definition: A sudden and variable expiratory thrust of air from the lungs through the air passages associated with phonation, which momentarily interrupts the physiological pattern of breathing.

Mechanism of cough production: Cough reflex initiated by chemical/mechanical stimuli **(Flowchart 3C.1)**. This is carried by the afferents which are type C and type 1 fibers and innervate pharynx, larynx, large airways, terminal bronchiole and lung parenchyma. Afferents travel via vagus and superior laryngeal nerve. Nucleus tractus solitarius (NTS) in brainstem is the cough center. Efferents travel via vagus, phrenic, spinal motor nerves to the larynx, trachea, bronchi, diaphragm producing cough.

Mechanical events during cough production: The mechanical events involved in a typical cough are rapid successions of:

1. A fairly deep initial inspiration
2. The tight closure of the glottis, reinforced by the supraglottic structures
3. The quick and forceful contraction of the expiratory muscles
4. The sudden opening of the glottis while the contraction of the expiratory muscles continues.

Classification

- **Based on etiology:** The etiology can be classified into respiratory causes and non-respiratory causes.
- **Based on duration of cough:** Cough has been classified into acute (less than 3 weeks), subacute (3–8 weeks), and chronic (more than 8 weeks; **Box 3C.1**).

Box 3C.1: Chronic cough with normal chest X-ray.

- Cough variant asthma
- Tropical eosinophilia
- Upper airway cough syndrome
- Aspiration
- Habitual cough
- Foreign body
- Drugs, angiotensin converting enzyme inhibitors
- Chronic bronchitis
- Chronic idiopathic cough

- **Based on expectoration:** It is also classified into productive or dry cough depending on the presence or absence of expectoration, respectively **(Table 3C.1)**.

Flowchart 3C.1: Algorithm showing cough reflex.

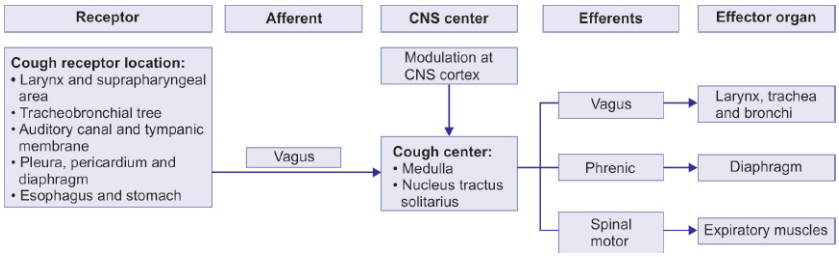


Table 3C.1: Classification of cough based on etiology.			
Cough	***Duration***	***Respiratory causes***	***Non-respiratory causes***
Acute cough	Less than 3 weeks	• Tracheobronchitis • Bronchopneumonia • Viral pneumonia • Acute-on-chronic bronchitis • Pulmonary embolism **Sudden onset:** • Bronchial asthma • Asthmatic bronchitis • Whooping cough • Foreign body	• LVF • GERD
Subacute cough	3–8 weeks	• Tuberculosis, pneumonia (bacterial, viral, fungal) • *B. pertussis* • Bronchiectasis • Postviral tussive syndrome	• GERD • Tourette's syndrome • Intentional cough
Chronic cough*	Lasting for more than 8 weeks	• COPD, asthma • ILD • Tuberculosis • Lung cancer • Pneumoconiosis (asbestosis, silicosis, anthracosis, etc.) • Mesothelioma of lung • Upper airway cough syndrome	• Drug induced (ACE inhibitors, beta blockers, NSAIDs) • Habit cough syndrome

**Chronic cigarette smoking is the most common cause of chronic cough.*

(LVF: left ventricular failure; GERD: gastroesophageal reflux disease; COPD: chronic obstructive pulmonary disease; ILD: interstitial lung disease; ACE: angiotensin converting enzyme; NSAIDs: nonsteroidal anti-inflammatory drugs)

Table 3C.2: Different types of cough.	
Types	***Features***
Dry cough	Pleural disorders, diseases of interstitium, mediastinal lesions
Productive cough	Suppurative lung disease, airway diseases
Brassy/Gander cough	Metallic sound due to compression of trachea by intrathoracic space occupying lesions or aortic aneurysms also known as leopards growl
Bovine cough	Loss of expulsive nature as in a tumor pressing on the recurrent laryngeal nerve

Paroxysmal cough	Whooping cough, chronic bronchitis, foreign body, bronchial asthma
Barking cough	Involvement of epiglottis, croup (laryngotracheobronchitis), hysteria
Spluttering cough	Tracheoesophageal fistula, cough while swallowing
Hacking cough	Heavy smokers, chronic pharyngitis or laryngitis
Otogenic cough	Due to stimulation of Arnold's nerve in the external auditory meatus (impacted wax/foreign body)

EXPECTORATION/SPUTUM

Sputum can be described under the following headings:

- Quantity
- Quality
- Odor

Quantity	
Normal	10–15 mL/24 hour
Bronchorrhea	Production of more than 100 mL/day • Bronchiectasis • Lung abscess • Bronchoalveolar carcinoma • Organophosphorus poisoning
Quality	
Mucoid	Chronic bronchitis, bronchial asthma
Mucopurulent	Infections
Purulent	Lung abscess, bronchiectasis
Rust-colored purulent sputum	Pneumococcal pneumonia
Currant-jelly and sticky sputum	*Klebsiella pneumoniae*
Blood-tinged foamy sputum	Pulmonary edema (pink frothy)
Greenish	*Pseudomonas*
Granules—yellow/black	Actinomycosis
Anchovy sauce (brown)	Amebic abscess rupturing into lung
Black (melanoptysis)	Carbon particles discolor the sputum gray (as in cigarette smokers) or black (as in coal miners or with smoke inhalation)
Odor	
Foul smelling sputum	Anaerobic infection seen in lung abscess, bronchiectasis

Special Points

- Chronic expectoration of large amounts of purulent and foul-smelling sputum is strongly suggestive of bronchiectasis.
- Sudden production of such sputum in a febrile patient indicates a lung abscess.

Table 3C.3: Causes of hemoptysis.

Structure involved	*Common causes*	*Uncommon causes*
Bronchial disease	Bronchial carcinoma, bronchiectasis, acute and chronic bronchitis	Bronchial adenoma, foreign body
Parenchymal disease of lung	Pulmonary tuberculosis (Rasmussen's aneurysm—dilation of a pulmonary artery in a tuberculous cavity), lung abscess, pneumonia (particularly *Klebsiella*), fungal infections (aspergilloma and invasive aspergillosis), pulmonary contusion/laceration (traumatic)	Parasites (e.g. hydatid disease, flukes), trauma, actinomycosis, mycetoma
Vascular diseases of the lung	Pulmonary infarction	Goodpasture's syndrome, polyarteritis nodosa, idiopathic pulmonary hemosiderosis, primary pulmonary hypertension
Cardiovascular disease	Acute left ventricular failure	Mitral stenosis, aortic aneurysm, pulmonary thromboembolism
Hematological disorders		Leukemia, hemophilia, anticoagulants, hemorrhagic diathesis

- **Three-layer sputum** consisting of a foamy upper layer, mucous middle layer, and viscous purulent bottom layer is pathognomonic of bronchiectasis.
- **Postural variation** in sputum: Bronchiectasis, lung abscess.

HEMOPTYSIS

Definition: Hemoptysis is defined as coughing of blood originating from below the vocal cords. Hemoptysis can range from blood-streaking of sputum to the presence of gross blood in the absence of any accompanying sputum. The different causes of hemoptysis are given in **Table 3C.3**.

The clinical clues of hemoptysis, differences between true and false hemoptysis and differences between hemoptysis and hematemesis are described in **Table 3C.4** to **Table 3C.6**, respectively.

Table 3C.4: Clinical clues of hemoptysis.

Clinical clues	*Suggested diagnosis*
Anticoagulant use	**Medication effect, coagulation disorder**
Tobacco use	**Acute bronchitis, chronic bronchitis, pneumonia, lung cancer**
Dyspnea on exertion, fatigue, orthopnea, paroxysmal nocturnal dyspnea, frothy pink sputum	**Congestive heart failure, left ventricular failure and mitral stenosis**
Fever, productive cough	**Upper respiratory tract infection, acute bronchitis, pneumonia, lung abscess**
History of cancer (e.g. breast, colon, or kidney)	**Endobronchial metastasis from carcinoma**
History of chronic lung disease, recurrent lower respiratory tract infection, cough with copious purulent sputum	**Bronchiectasis, lung abscess**
Pleuritic chest pain, calf tenderness	**Pulmonary embolism or infarction**
Toxic symptoms	**Tuberculosis**
Weight loss	**Emphysema, lung cancer, tuberculosis, bronchiectasis, lung abscess**
Melena, alcoholism, chronic use of nonsteroidal anti-inflammatory drugs (NSAIDs)	**Gastritis, gastric or peptic ulcer, esophageal varices**
Association with menses	**Catamenial hemoptysis**
Cachexia, clubbing, hoarseness	**Lung cancer, small cell carcinoma**

Clubbing	**Lung cancer, bronchiectasis, lung abscess**
Dullness to percussion, fever, crepitations	**Pneumonia**

Table 3C.5: Differences between true and false hemoptysis.

True hemoptysis	*False hemoptysis*
Below vocal cords	Above vocal cords
Persists as blood tinged sputum	Does not persist
May be mixed with sputum	Not mixed with sputum
History of cardiopulmonary disease	Obvious by ENT examination
Chest X-ray may be abnormal	Normal chest X-ray

Table 3C.6: Differences between hemoptysis and hematemesis.

Hemoptysis	*Hematemesis*
Coughing of blood. Cough precedes hemoptysis	Vomiting of blood. Nausea and vomiting precedes hematemesis
History of cardiopulmonary disease	History of gastrointestinal disease
Bright red in color	Dark brown in color
Sputum remains blood stained after the attack for few days	Usually followed by melena
Mixed with sputum	Mixed with gastric contents
Blood is frothy due to admixture of air	Airless and not frothy
Alkaline	Acidic
Sputum contains hemosiderin laden macrophages	No
Melena absent	Melena present

Massive hemoptysis: Life-threatening (or) massive hemoptysis is defined as coughing of blood >150 mL/episode (or) > 600 mL/24 hour. Only 5% of hemoptysis is massive but mortality is 80%. Clinical definition of massive hemoptysis is any bleeding that result in a threat to life because of airway or hemodynamic compromise due to bleeding. The different causes of massive hemoptysis are given in **Box 3C.2**.

DYSPNEA

Definition

“Dyspnea” is a term used to characterize a subjective experience of breathing discomfort that is comprised of qualitatively distinct sensations that vary in intensity (undue awareness of unpleasant breathing).

Box 3C.2: Causes of massive hemoptysis.

- Pulmonary tuberculosis
- Pulmonary infarction
- Bronchiectasis
- Bronchogenic carcinoma
- Cystic fibrosis

- Lung abscess
- Necrotizing pneumonia
- Mitral stenosis
- Pulmonary arteriovenous malformation

Mechanism of Dyspnea

Chemoreceptors	
Peripheral	Carotid and aortic bodies (sensitive to changes pO_2, pCO_2 and H^+)
Central	Medulla (sensitive only to changes in pCO_2, not pO_2, change in pH of cerebrospinal fluid)
Increased work of breathing	
Airflow obstruction	Bronchial asthma, chronic obstructive pulmonary disease (COPD), tracheal obstruction
Decreased pulmonary compliance	Pulmonary edema, fibrosis, allergic alveolitis
Restricted chest expansion	Ankylosing spondylitis, respiratory paralysis, kyphoscoliosis
Increased ventilatory drive	
Increased physiological dead space (V/Q mismatch)	Consolidation, collapse, pleural effusion (PE), pulmonary edema
Hyperventilation due to receptor stimulation	
Chemoreceptors	Acidosis, hypoxia (shock, pneumonia), hypercapnia
J receptors at alveolocapillary junction	Pulmonary edema, pulmonary embolism, pulmonary congestion (activates Hering-Breuer reflex which terminates inspiratory effort before full inspiration is achieved—rapid and shallow)
Muscle spindles in intercostal muscles	Tension-length disparity
Central	Exertion, anxiety, thyrotoxicosis, pheochromocytoma
Impaired respiratory muscle function	
Diseases with impaired muscle function	Poliomyelitis, Guillain-Barre syndrome (GBS), myasthenia gravis

Table 3C.7: Differences between paroxysmal nocturnal dyspnea (PND) orthopnea.

	Paroxysmal nocturnal dyspnea	***Orthopnea***
Definition	Episode of sudden onset of dyspnea 2–2.5 hours after sleep	Dyspnea in recumbent posture
Timing	Patient wakes up from rapid eye movement (REM) sleep	Occurs soon after lying down
Method of relief	Sits up with legs hanging down, stands up, air hunger, self ventilates of comfort	Gets up, uses more pillows, sleeps in erect posture
Mechanism	Depressed respiratory center. Sympathetic overactivity during REM → catecholamine surge resulting in tachycardia → interstitial pulmonary congestion → respiratory center lags behind → perceived as acute dyspnea. There is sudden transient increase in PCWP	Shifting of venous blood (>400 mL) into pulmonary circulation, V/Q mismatch, compression of diaphragm, postural diastolic dysfunction. There is a slow sustained rise in pulmonary capillary wedge pressure (PCWP)
Associated symptoms	Angina, perspiration, palpitation, rarely hemoptysis	All the symptoms of congestive cardiac failure (CCF)
Oxygen	Transient hypoxia	Normal

saturation		
Differential diagnosis	Night mares/panic attacks/nocturnal hypoglycemia/obstructive sleep apnea (OSA)	COPD/gross obesity/acute asthma/gross ascites

Orthopnea

Dyspnea develops in recumbent position and is relieved by sitting up or by elevation of the head with pillows.

The severity can be graded by the number of pillow used at night, e.g. three pillow orthopnea.

Pathophysiology of Orthopnea

- Pulmonary congestion during recumbency (cannot be pumped out of LV) seen in congestive heart failure (CHF), chronic obstructive pulmonary disease (COPD) and bronchial asthma.
- Increased venous return.
- Diaphragm elevation leading to decreased vital capacity.

Conditions Associated with Orthopnea

Orthopnea is classically seen in left heart failure but can also occur in constrictive pericarditis, COPD, bilateral diaphragmatic palsy, asthma triggered by gastric reflux, and gross ascites.

Paroxysmal Nocturnal Dyspnea

Attacks of dyspnea occur at night and awaken the patient from sleep. The important differences between orthopnea and PND are given in **Table 3C.7**.

Mechanism (Fig. 3C.1)

- It is due to decreased responsiveness of respiratory center in brain during sleep and pulmonary congestion (due to increased sympathetic activity during REM sleep), that occurs 2–3 hours after onset of sleep.

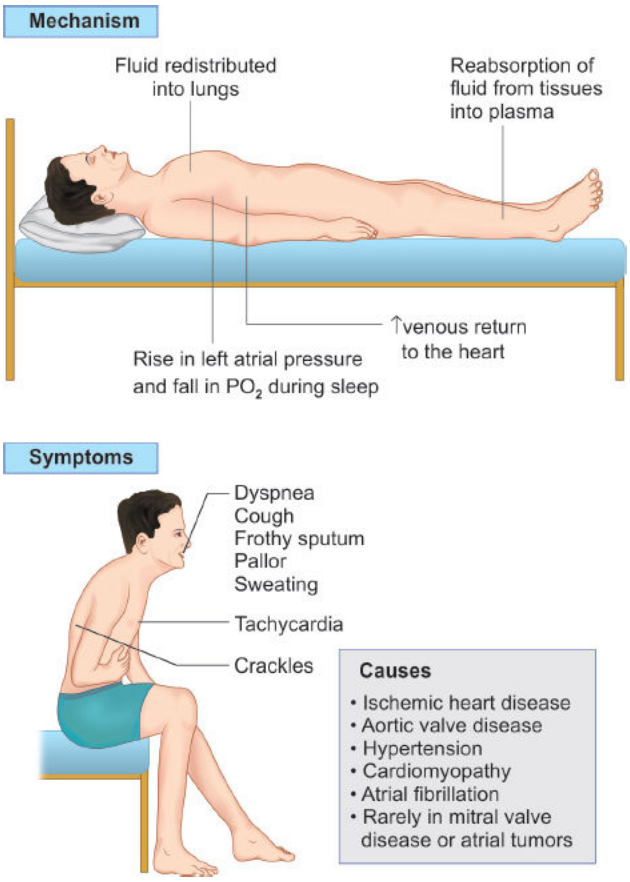


Fig. 3C.1: Mechanism of paroxysmal nocturnal dyspnea (PND).

- Absorption of edema fluid with increase in right ventricular output causing over filling of the lungs.
- Takes 10–30 minutes for recovery after upright posture.
- Specific sign of LV dysfunction and includes ischemic heart disease, aortic valve disease, hypertension, cardiomyopathy.
- It has low sensitivity (<30%) but 75% specificity to diagnose heart disease.

Differential Diagnosis for Paroxysmal Nocturnal Dyspnea

- Left heart failure
- Nocturnal episodes of asthma
- Postnasal discharge with attendant severe cough
- Sleep apnea with arousal
- Nightmares
- Nocturnal angina with dyspnea (angina equivalent)
- Nocturnal aspiration in gastroesophageal reflux disease
- Nocturnal episodes of recurrent minute pulmonary emboli
- Nocturnal hypoglycemia.

Trepopnea

Aggravation of dyspnea when lying on one side and relieved by lying on opposite side.

Causes

- Unilateral lung disease: Uninvolved normal lung receives more blood supply due to gravity.

- Congestive heart failure: Lying on right side enhances venous return and sympathetic activity.
- Lung tumor: Gravity induced compression of blood vessels or lung.

Platypnea

Dyspnea on sitting or standing and relieved by supine position.

Causes

- Venous to arterial shunting (lung bases)
- Intracardiac shunts (ASD, pneumonectomy)
- Intrapulmonary right to left shunt [hepatopulmonary syndrome, pulmonary embolism (PE), COPD]
- Acute respiratory distress syndrome (ARDS).

Bendopnea

A newly described symptom in patients with heart failure is mediated via a further increase in ventricular filling pressures during bending in subjects whose sitting ventricular filling pressures are already high, particularly in patients with low cardiac index **(Fig. 3C.2)**.

Fig. 3C.2: A patient sits in a chair, bends at the waist, and touches his or her feet. Bendopnea is considered present if dyspnea occurs within 30 seconds of bending.

Approach to dyspnea

Onset and duration	
Minutes to hours (rapid onset)	Pneumothorax, acute asthma, pulmonary embolism (PE), pulmonary edema, foreign body
Hours to days (gradual onset)	Pneumonia, pleural effusion, anemia, Guillain–Barre syndrome (GBS)
Months to years (slow onset)	Pulmonary tuberculosis (PTB), COPD, carcinoma, fibrosing alveolitis
Severity	
Medical Research Council (MRC) (Table 3C.8)	Discussed below
Modified Medical Research Council (mMRC) (Table 3C.9)	
New York Heart Association (NYHA) (Table 3C.10)	

Aggravating and relieving factors	
Improves on weekend/holidays	Occupational asthma, extrinsic alveolitis
Recumbency/sleep	Orthopnea/paroxysmal nocturnal dyspnea (PND)
Associated symptoms (Table 3C.11)	
Pleuritic chest pain	Pneumonia, pulmonary infarction, rib fracture, pneumothorax
Central non-pleuritic chest pain	Myocardial infarction, massive pulmonary embolism
Cough or wheeze	Asthma, pulmonary embolism, pneumothorax

Table 3C.8: Medical Research Council grading of breathlessness.

1. Note troubled by breathlessness except on strenuous exertion
2. Short of breath when hurrying on level ground or walking up slight hill
3. Walks slower than people of same age or stops after 15 minutes when walking at own pace on level
4. Stops after 100 yards (90 m) or after few minutes in level ground
5. Too breathless to leave house, dress or undress

Table 3C.9: Modified Medical Research Council grading of breathlessness.

Grade	*Description of breathlessness*
Grade 0	I only get breathless with strenuous exercise
Grade 1	I get short of breath when hurrying on level ground or walking up a slight hill
Grade 2	On level ground, I walk slower than people of the same age because of breathlessness, or I have to stop for breath when walking at my own pace on the level
Grade 3	I stop for breath after walking about 100 yards or after a few minutes on level ground
Grade 4	I am too breathless to leave the house or I am breathless when dressing

Pitfalls of mMRC Grading

- The mMRC dyspnea scale quantifies disability attributable to breathlessness, and is useful for characterizing baseline dyspnea in patients with respiratory diseases.
- It describes baseline dyspnea, but does not accurately quantify response to treatment of COPD.

Table 3C.10: New York Heart Association (NYHA) classification of breathlessness.

NYHA Class	*Patients with cardiac disease (Description of heart failure related symptoms)*
Class I (Mild)	Patients with cardiac disease but without resulting in limitation of physical activity. Ordinary physical activity does not cause undue fatigue, palpitation, dyspnea or anginal pain
Class II (Mild)	Patients with cardiac disease resulting in **slight limitation** of physical activity. They are comfortable at rest. Ordinary physical activity results in fatigue, palpitation, dyspnea, or anginal pain
Class III (Moderate)	Patients with cardiac disease resulting in **marked limitation** of physical activity. They are comfortable at rest. Less than ordinary activity causes fatigue, palpitation, dyspnea or anginal pain
Class IV (Severe)	Patients with cardiac disease resulting in the inability to carry on any physical activity without discomfort. Symptoms of heart failure or the anginal syndrome may be present even at rest. If any physical activity is undertaken, discomfort is increased

Table 3C.11: Causes of acute and chronic dyspnea.

Acute dyspnea	*Chronic dyspnea*

Cardiovascular system	
Cardiogenic acute pulmonary edema	Chronic heart failure, myocardial ischemia
Respiratory system	
• Acute severe bronchial asthma • Acute exacerbation of COPD • Spontaneous pneumothorax • Pneumonia • Acute pulmonary embolism • Acute respiratory distress syndrome • Inhaled foreign body (especially in children) • Lobar collapse • Laryngeal edema (e.g. anaphylaxis) or obstruction	• Chronic obstructive pulmonary disease (COPD) • Chronic bronchial asthma • Bronchial carcinoma • Interstitial lung disease (e.g. sarcoidosis, fibrosing alveolitis, extrinsic allergic alveolitis, pneumoconiosis) • Chronic pulmonary thromboembolism • Lymphatic carcinomatosis • Large pleural effusion(s) • Severe anemia • Obesity • Deconditioning
Non-respiratory, Non-cardiac causes	
Metabolic acidosis (e.g. diabetic ketoacidosis, lactic acidosis, uremia, overdose of salicylates, ethylene glycol poisoning)	Psychogenic hyperventilation (anxiety or panic-related)

Box 3C.3: Acute severe breathlessness

- Pulmonary edema
- Massive pulmonary embolism
- Acute severe asthma
- Acute exacerbation of COPD
- Severe pneumonia
- Tension pneumothorax
- Foreign body/mucous plug
- Epiglottitis (children)
- Metabolic acidosis
- Psychogenic

CHEST PAIN

Chest pain discussed in detail under Chapter 4.

Respiratory causes

- Upper sternal—tracheitis
- Pleuritic-associated with breathing
- Neurologic-invasion of nerves.

Pleuritic chest pain is characterized by sudden and intense sharp, stabbing, or burning pain in the chest when inhaling and exhaling. It is exacerbated by deep breathing, coughing, sneezing, or laughing. When pleuritic inflammation occurs near the diaphragm, pain can be referred to the neck or shoulder. Pleuritic chest pain is caused by inflammation of the parietal pleura (dry pleurisy) and can be triggered by a variety of causes.

Pulmonary embolism, myocardial infarction, pericarditis, aortic dissection, pneumonia, and pneumothorax are the six serious conditions that cause pleuritic pain.

OTHER SYMPTOMS

Noisy breathing (partial obstruction of airway):

Laryngeal level	Stridor (inspiratory sound)
Oropharyngeal level	Stertor
Tracheal level	Rattling
Bronchial level	Wheezing (inspiratory/expiratory)

Hoarseness of voice:

- Inflammatory: Acute and chronic laryngitis
- Smoke inhalation
- Neoplastic—carcinoma/laryngeal papillomatosis
- Recurrent Laryngeal nerve damage: Post-thyroidectomy carcinoma of lung/breast
- Neurological: Myasthenia gravis, hypothyroidism
- Rheumatoid arthritis—involvement of cricoarytenoid joint
- Habitual dysphonias
- Reinke's dysphonia
- Singer's nodules/vocal cord polyps
- Gastroesophageal reflux disease (GERD).

Hiccoughs

Respiratory causes include basal pneumonia and pleurisy.

Snoring

Feature of obstructive sleep apnea.

NOTES

D. DISCUSSION ON EXAMINATION

GENERAL EXAMINATION

Built and Nourishment

Body mass index (BMI), anthropometry has been discussed in detail in Chapter 2D of General Examination.

Respiratory diseases associated with emaciation:

1. Respiratory diseases associated with HIV
2. Pulmonary tuberculosis
3. Malignancy.

Pickwikian syndrome (obesity hypoventilation syndrome):

1. Obesity
2. Hypoxia
3. Pulmonary HTN.

Vital Examination (with Respect to Respiratory system)

Pulse:

- Rate—tachycardia (any pneumonia, febrile illness, hypoxia)
- Irregular pulse seen in multifocal atrial tachycardia, atrial fibrillation
- Bounding pulse—CO_2 retention
- Pulsus paradoxus—acute exacerbation of COPD/asthma.

Respiratory rate:

(for details on respiratory rate refer chapter on vitals examination).

Blood pressure:

- Wide pulse pressure—in hypercapnia
- Low blood pressure—seen with hypoxia, acute respiratory distress
- Postural hypotension—Addison's disease, paraneoplastic.

Jugular venous pressure:

- Elevated: In cor pulmonale, tricuspid regurgitation
- Nonpulsatile jugular venous pressure (JVP): Superior vena cava (SVC) obstruction.

Temperature:

- Evening rise of temperature: Tuberculosis
- High spiking fevers: Lung abscess, empyema, pneumonias.
- Temperature fall by crisis: Pneumonias.

Pallor:

- Tuberculosis
- Malignancy
- Any cause of massive hemoptysis.

Polycythemia:

Chronic respiratory diseases are usually associated with polycythemia

So if patient with COPD has anemia look for other causes like GI bleed, CKD or coexistent malignancy.

Icterus:

- Hepatitis secondary to antitubercular (ATT) drugs
- Atypical pneumonias (hemolytic jaundice)
- As a part of multiple organ dysfunction syndrome (MODS)
- Rarely metastasis to liver.

Edema:

- Cor pulmonale
- Bronchiectasis leading to hypoproteinemia (due to loss of protein in the sputum and nephrotic syndrome secondary to amyloidosis)—100 mL of sputum can cause 3–4 g of protein loss.
- Hypercapnia-induced dilation of the precapillary sphincters.
- Reduced renal blood flow with relatively preserved glomerular filtration rate and elevated levels of renin, aldosterone, arginine vasopressin and atrial natriuretic peptide.

Cyanosis, clubbing, and lymphadenopathy described in detail in the chapter 2D of General Examination.

Lymphatic drainage of lung	
Most of the lung (right upper lobe, Right middle lobe, Right lower lobe, Left lower lobe)	Right tracheobronchial → right bronchomediastinal → right supraclavicular lymph node
Left upper lobe	Left tracheobronchial → left bronchomediastinal → left supraclavicular lymph node
Lymphatic drainage of pleura (Fig. 3D.1)	
Cervical pleura	Axillary lymph nodes
Parietal pleura	• Anterior: Internal mammary nodes • Posterior: Extrapleural nodes
Diaphragmatic pleura	• Internal mammary nodes, cardiophrenic nodes • Para-aortic, intercostal and posterior mediastinal nodes
Mediastinal pleura	Internal mammary nodes

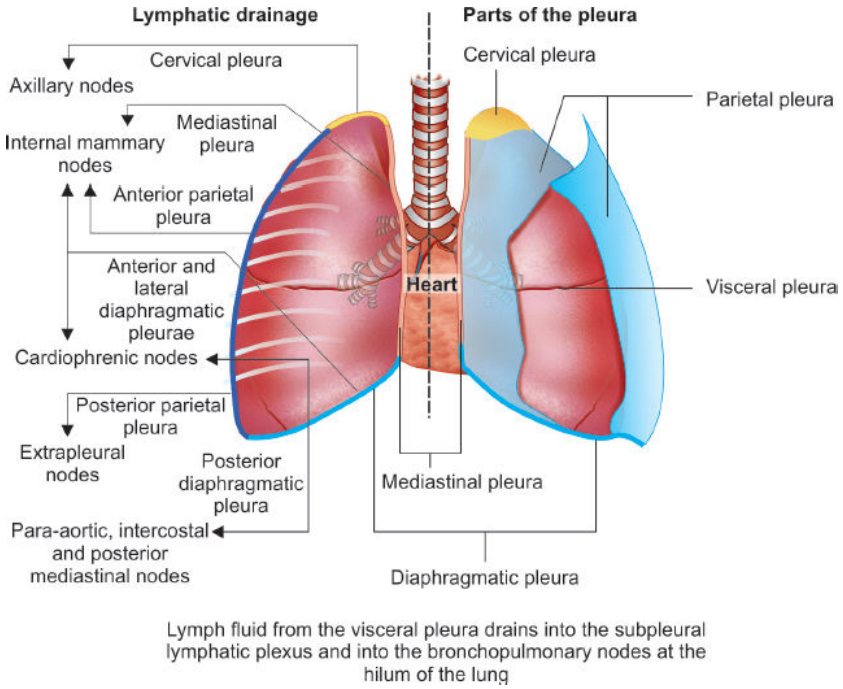


Fig. 3D.1: Parts of pleura with corresponding lymphatic drainage.

Oral cavity examination:

- Halitosis seen in suppurative lung diseases
- Tobacco staining of the teeth
- Poor oral hygiene
- Oral markers of malignancy—leukoplakia, erythroplakia, submucous fibrosis.
- Cyanosis or polycythemia.
- Oral candidiasis—due to inhaled steroids.
- Posterior pharyngeal wall/tonsils—infection.

External markers of tuberculosis:

- Matted lymph nodes
- Erythema nodosum
- Phlyctenular conjunctivitis
- Choroid tubercle
- Discharging sinuses
- Scrofuloderma
- Lupus vulgaris
- Beaded vas deferens
- Positive Mantoux test
- Generalized tinea versicolor
- Uveitis.

External markers of malignancy:

- Cachexia
- Grade IV clubbing (HPOA)
- Hard lymph nodes
- Acanthosis nigricans

- Horner's syndrome
- SVC obstruction features—non-pulsatile, dilated JVP, facial flushing and edema, conjunctival suffusion, papilledema, dilated veins on the chest wall.

Features of respiratory failure:

	Type 1	Type 2
Definition	Hypoxemic respiratory failure (type 1) is characterized by an arterial oxygen tension (PaO_2) lower than 60 mm Hg with a normal or low arterial carbon dioxide tension ($PaCO_2$)	Hypercapnic respiratory failure (type 2) is characterized by a $PaCO_2$ higher than 50 mm Hg
Sensorium	Anxious agitated	Drowsy to comatose
Peripheries	Cold	Warm
Pulse	Feeble	Bounding
Blood pressure	Low	Wide pulse pressure
Cyanosis	+	–
Asterixis	–	+
Respiratory rate	Tachypneic	Normal to low
Papilledema	–	+
Cause	• ARDS • Pneumonia • Acute severe asthma • Tension pneumothorax	• COPD • Obesity • Respiratory paralysis
Type 3 (perioperative): Functional residual capacity falls below closing volume as a result of atelectasis in postoperative patients. This is generally a subset of type 1 failure but is sometimes considered separately because it is common		
Type 4 (shock): Secondary to cardiovascular instability		

Features of Cor Pulmonale

Right ventricular dilatation:
- Parasternal heave
- Epigastric pulsation.

Right ventricular failure:
- Raised JVP
- Pedal edema
- Tender hepatomegaly
- Ascites
- Sustained abdominojugular reflux is first sign of RVF.

EXAMINATION OF RESPIRATORY SYSTEM

Examination of Upper Respiratory Tract

Demarcation of upper and lower respiratory tract:
- Externally: Demarcated by cricoid cartilage
- Internally: Demarcated by glottis.

Significant findings in the upper respiratory tract:

- Nasal turbinate hypertrophy or polyps causing airway obstruction
- Sinus tenderness suggestive of sinusitis
- Kartageners syndrome:
  - Recurrent sinusitis with ciliary dyskinesia
  - Bronchiectasis
  - Situs inversus
  - Male infertility
- Wegeners granulomatosis
  - Necrotizing granuloma
- Samter's triad
  - Aspirin sensitivity
  - Bronchial asthma
  - Ethmoidal polyps
- Young's syndrome
  - Sinopulmonary disease
  - Azoospermia
- Churg-Strauss syndrome
  - Asthma/allergic rhinitis
  - Eosinophilia
  - Vasculitis
  - Granuloma

Inspection (Lower Respiratory Tract)

Surface marking of lung

Right side 3 lobes	Left side 2 lobes
• Right upper lobe (RUL) • Right middle lobe (RML) • Right lower lobe (RLL)	• Left upper lobe (LUL) • Left lower lobe (LLL)

Demarcating lower lobe of either side (Figs. 3D.2 to 3D.5):

Lower lobe of either lungs can be demarcated from other lobes by drawing a curvilinear line (major interlobar fissure/oblique fissure) joining 3 bony points:

1. Starting from T2/T3 spinous process, curvilinear line along the medial border of scapula
2. Crossing the 5th rib in the Mid axillary line
3. Reaching the 6th rib in mid clavicular line

Part of lung below this line is lower lobe.

Marking right middle lobe:

Draw a straight line (minor interlobar fissure/horizontal fissure) from the 4th rib at right sternal border towards the midaxillary line cutting the major interlobar fissure at 5th rib. The triangular area represents RML.

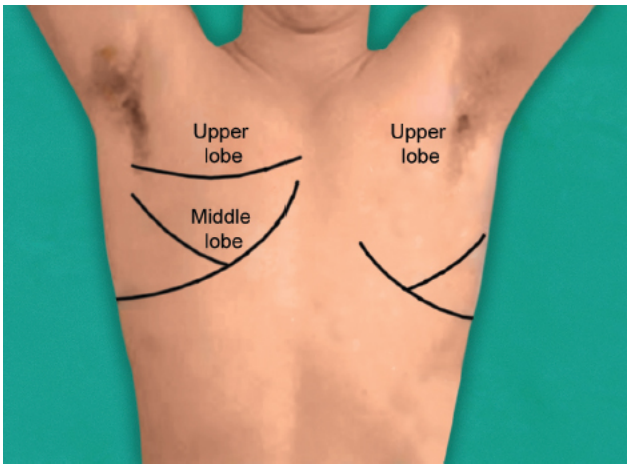


Fig. 3D.2: Anterior view of chest showing surface marking of lung fissures and lobes.

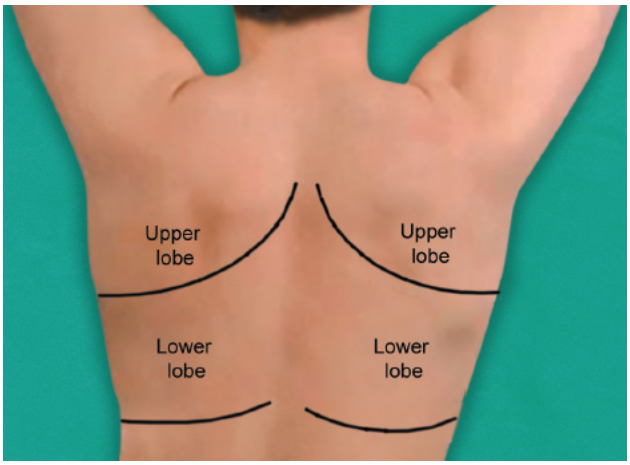


Fig. 3D.3: Posterior view of chest showing surface marking of lung fissures and lobes.

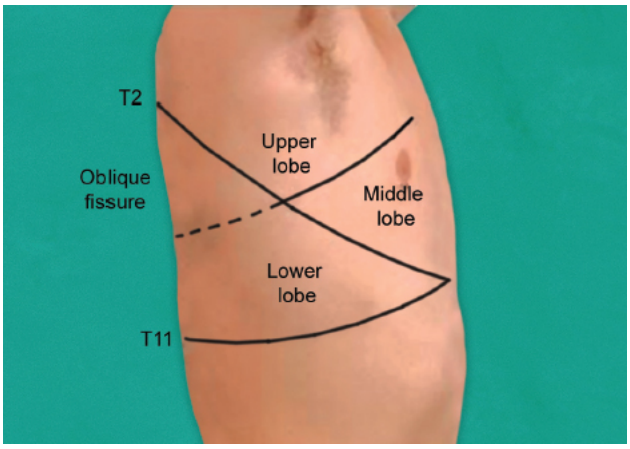


Fig. 3D.4: Right lateral view of chest showing right major interlobar (IL) fissure and right minor IL fissure.

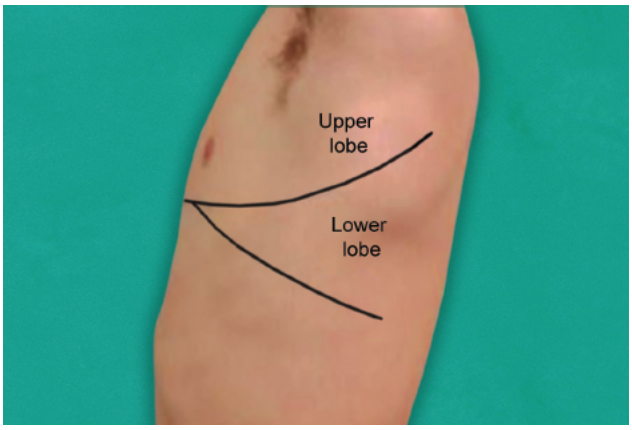


Fig. 3D.5: Left lateral view of chest showing left major interlobar fissure.

Level of lower border	Midclavicular line	Midaxillary line	Scapular
Lung (Figs. 3D.6 and 3D.7)	6th rib	8th rib	10th rib
Pleura	8th rib	10th rib	12th rib

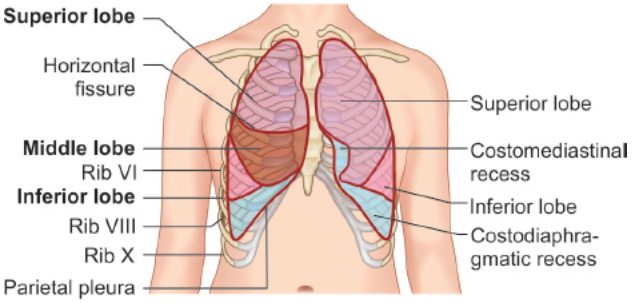


Fig. 3D.6: Lower margin of lung in midclavicular line and midaxillary line.

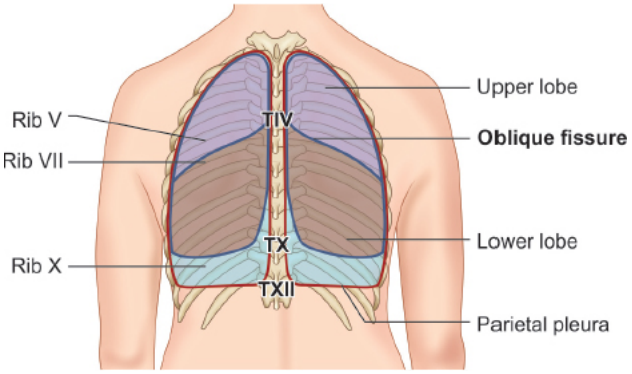


Fig. 3D.7: Lower margin of lung in scapular line.

Examination of chest:

Front examination	Back examination	Axillary examination
Predominantly to look for upper and middle lobe	Predominantly to look for lower lobe pathology	All three lobes can be assessed

Examined with patient in upright sitting position with hand by the side	Examined with patient in sitting upright with hands placed on the opposite shoulder and neck flexed	Examined with patient in the sitting position with hands raised above the shoulder and placed on the occiput

Position of patient during examination can be:

- Sitting—most of the examination is done in this position
- Standing—spine and shoulder droop
- Supine—shifting dullness.

Normal chest (Fig. 3D.8)

- Spine—central
- Shape
  - Circular—infants and early childhood
  - Elliptical—adults
  - Circular—old age
- Vertical length > transverse diameter > AP diameter
- Transverse: AP = 7:5 (called as **Hutchinson's index**)
- Subcostal angle ≤ 90 (more acute in males).

Deformities of chest	
1. Flat chest (alar chest)	Anterioposterior ratio is 2:1
2. Pectus carinatum (Fig. 3D.9) (Pigeon chest/keel chest)	Forward protrusion of sternum seen in rickets and childhood respiratory disease like asthma. Can also be seen in Marfan syndrome
3. Pectus excavatum (Fig. 3D.9) (Funnel chest, cobbler's chest)	Funnel like depression at the lower end of the chest, seen in Marfan syndrome. Displaces the heart to the left. Ventilation capacity of the lung is restricted
4. Rachitic chest	• Funnel shaped • Keel breast • Harrison sulci (horizontal groove where the diaphragm attaches to the ribs—seen in rickets, chronic asthma and COPD) • Vertical grooves on either side of sternum • Rachitic rosary (bead like enlargement of costochondral junction especially 4/5/6 ribs) —painless and seen in vitamin D deficiency
5. Scorbutic rosary	• Sharp angulation of the ribs arising due to backward displacement of sternum • Painful and seen in vitamin C deficiency
6. Barrel-shaped chest (Fig. 3D.8)	COPD—emphysema • Anteroposterior: Transverse diameter is 1:1 • Exaggerated thoracic kyphosis • Wide subcostal angle
7. Phthinoid chest	Combination of alar and flat chest
8. Flail chest	Paradoxical movement of the chest in fracture of 3 or more consecutive ribs
9. Shield-like chest	Turner's and Noonan syndrome

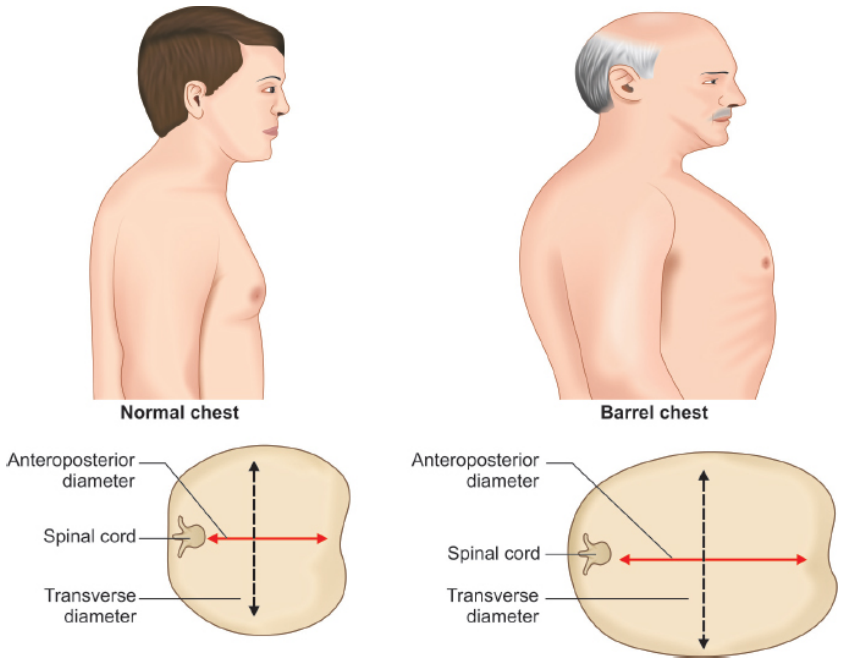


Fig. 3D.8: Normal- and barrel-shaped chest.

Asymmetry of chest	
Deformity of spine	• Scoliosis • Kyphoscoliosis • Gibbus
Unilateral bulge	• Pleural effusion • Pneumothorax • Compensatory hypertrophy • Malignancy of lung or pleura
Unilateral flattening	• Fibrosis • Collapse • Fibrothorax • Pneumonectomy • Agenesis of one lung (McLeod's syndrome/Swyer-James syndrome) • Mastectomy • Absent pectoralis (Poland's syndrome)
Local bulging (fullness)	• Supraclavicular fullness (Pancoast tumor/lymphadenopathy/massive pleural effusion/tension pneumothorax) • Empyema necessitans (cough impulse present) • Aortic aneurysm • Malignant infiltration • Pericardial effusion • Surgical emphysema
Local retraction	• Apical tuberculosis (**Morenheim's** fossa/infraclavicular fossa) • Lung fibrosis

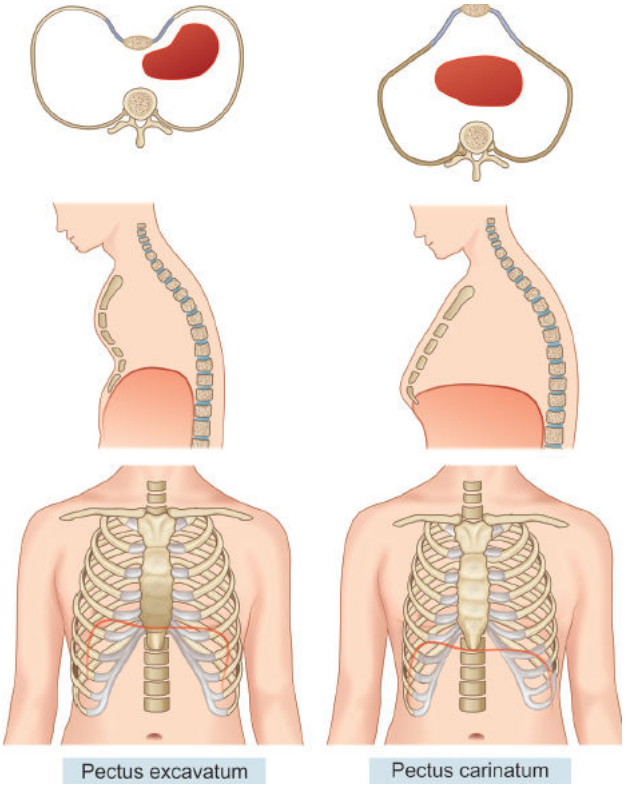


Fig. 3D.9: Pectus excavatum and pectus carinatum.

Trachea:

Normally central or slightly deviated to right.

Trail sign (Fig. 3D.10):

In the presence of tracheal deviation, there is prominence of the clavicular head of sternocleidomastoid of same side. The investing layer of cervical fascia splits to enclose the sternocleidomastoid and then falls back and continues as the pretracheal fascia. When there is tracheal shift to one side, the fascia covering the ipsilateral sternocleidomastoid relaxes. The sternocleidomastoid goes into a state of contraction making the clavicular head prominent.

- Clinical implication of tracheal shift: It suggests upper mediastinal shift.
- Indicates upper lobe fibrosis or collapse.

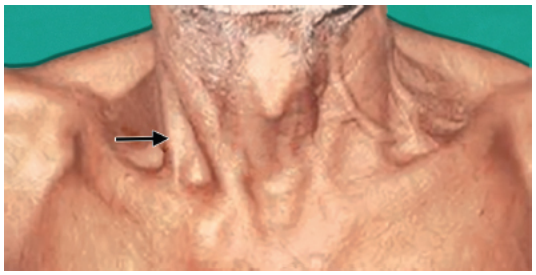


Fig. 3D.10: Trail sign showing undue prominence of sternocleidomastoid on the right side due to tracheal shift to right.

Apical impulse:

- Normally 10 cm from sternal margin.
- Clinical implication: Suggests lower mediastinal shift.

Examination of drooping of shoulder (Fig. 3D.11):

Examine the standing patient from behind to look for position of shoulder. Drooping of shoulder indicates volume loss on that side (collapse/fibrosis/fibrothorax/pneumonectomy). Rarely, it can be seen with paralysis of trapezius.

Associated features include:

- Prominent medial border of scapula on the affected side
- Space between medial border of scapula and spine is decreased
- Inferior angle of scapula is at the lower level (normally it is at level of T7 vertebra).

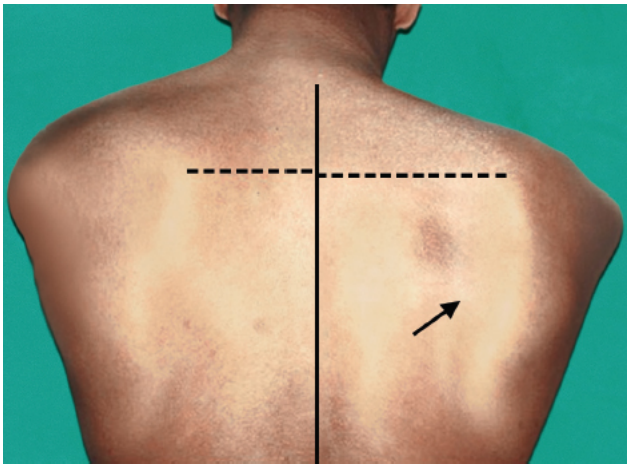


Fig. 3D.11: Shoulder drooping on right side.

Examination of spine:

- Look for position of spine
- Look for scoliosis/kyphosis/lordosis/Gibbus (Fig. 3D.12)
- In emphysema there is exaggerated thoracic kyphosis.

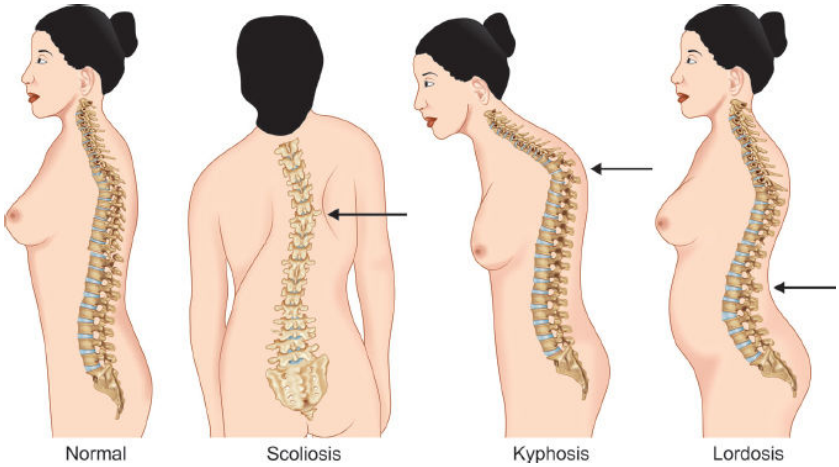


Fig. 3D.12: Spine deformities.

Causes of scoliosis

Neuromuscular causes	• Spina bifida • Marfan syndrome • Cerebral palsy • Friedreich's ataxia • Spinocerebellar degeneration • Charcot-Marie-Tooth disease • Syringomyelia • Poliomyelitis • Muscular dystrophy (Duchenne's, facioscapulohumeral, myotonic dystrophy)
Degenerative	• Osteoporosis • Post-spine surgery
Osteopathic	Klippel Feil syndrome
Congenital scoliosis	• Down's syndrome • Prader-Willi syndrome
Respiratory diseases	Fibrosis Fibrothorax
Idiopathic	–

Differentiation of congenital versus acquired scoliosis:
On bending forwards acquired scoliosis disappears but congenital scoliosis persists.

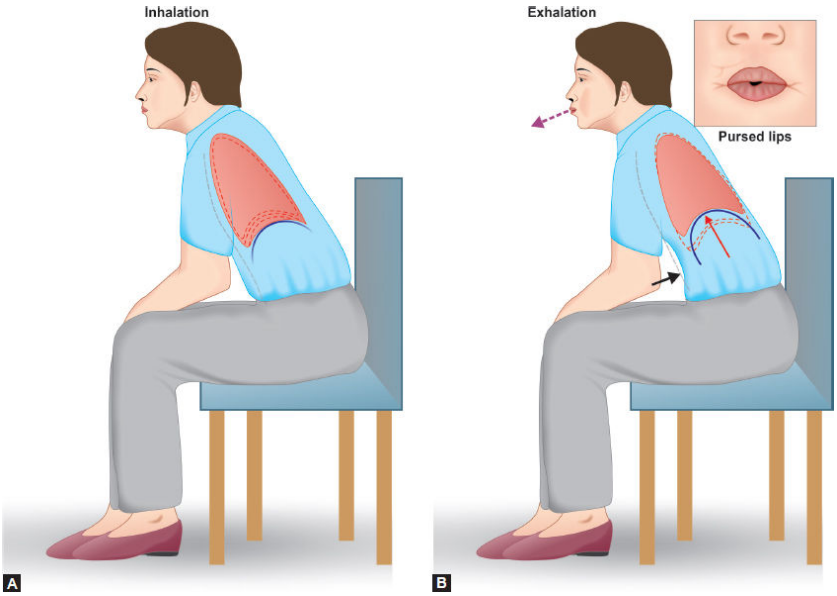
Respiratory movements:
(describe as equal/diminished in a particular area).

Area	Right	Left
Supraclavicular		
Clavicular		
Infraclavicular (arbitrarily up to 3rd rib)		
Mammary (arbitrarily 3rd to 6th rib)		
Axillary (up to 6th rib)		
Infra-axillary (beyond 6th rib)		
Suprascapular		
Infrascapular		
Interscapular		
Scapular (mentioned in some books)		

Note: There is no inframammary area.

Abnormal signs in respiratory system	
1. Sitting up and catching the edge	Described in COPD where the patient sits up and fixes shoulders to use latissimus dorsi for expiration
2. Tripod position (Fig. 3D.13A)	Patient is sitting in leading forward posture with their outstretched hands on their knees. This position fixes and lifts the shoulder girdle and improves the function of pectoralis major and minor
3. Hoover sign	Paradoxical inspiratory indrawing of lateral rib cage (costal margin). It is a sign of chronic airflow obstruction. Pulmonary hyperinflation leads to loss of apposition of the diaphragmatic fibers resulting in horizontal

	orientation of fibers. When these horizontally oriented fibers contract, the costal margins get pulled inwards
4. Pursed lip breathing (Fig. 3D.13B)	Seen in COPD to increase the intra-alveolar pressure to maintain a positive intraluminal pressure which reduces the airway collapse, airway resistance and residual volume and hence improves ventilation
5. Dahl's sign	Patches of hyperpigmentation/bruising above the knees due to constant tenting position of the hands and elbows
6. Litten's sign	To look for the diaphragmatic movement Sit to one side of the patient lying in supine position and look at the diaphragmatic movements
7. Excessive usage of SCM and scalene	COPD or asthma
8. Paradoxical respiration	Indrawing of abdominal wall when the rib cage moves outwards. Best felt by bimanual palpation with one hand over the patient's chest and other on the abdomen. Indicates respiratory muscle weakness



Figs. 3D.13A and B: Tripod position with pursed lip breathing.

Inspiratory intercostal retraction (Fig. 3D.13C):

Mild degree of intercostal retraction in the lower chest is normal. Bilateral lower intercostal retractions is seen in COPD.

Unilateral intercostal retraction	Bilateral intercostal retraction
• Collapse • Fibrosis • Adherent pericarditis (**Broadbent's sign**—indrawing of lower anterior chest wall with each ventricular systole)	• Indicates upper airway obstruction (adenoids/foreign body) • Hyperinflation of chest (COPD)

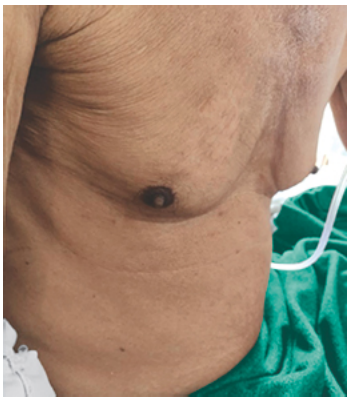


Fig. 3D.13C: Intercostal retractions.

Visible pulsations/scars/sinuses:

Visible pulsation or vessels	
Collaterals around scapula	Coarctation of aorta (**Suzman's sign**)
Engorged veins over the anterior part of chest	**SVC obstruction seen in** • Bronchogenic carcinoma • Mediastinal growth • Mediastinal lymph nodes • Aortic aneurysm • Chronic mediastinal fibrosis
Pulsatile swelling in anterior chest wall	Aortic aneurysm
Visible scars	
• Previous surgery (lobectomy) • Pleural fluid aspiration site • Lymph node biopsy site	
Sinuses	
• Abscess draining points • Empyema thoracis (usually in tuberculosis/actinomycosis)	

Palpation (Lower Respiratory Tract)

Trachea:

- Normal length: 4–5 cm above suprasternal notch
- Normal cricoid to suprasternal notch distance is 3–4 finger breadth (decreased in COPD due to hyperinflation).

Method of palpation:

Keep the index and ring finger of the right hand on medial ends of the clavicle
↓
With middle finger trace the trachea from above downwards **(Fig. 3D.14)**
↓
Then, insinuate the middle finger between the trachea and sternal head of sternocleidomastoid, and feel for resistance **(Fig. 3D.15)**

Note: **Implication of tracheal shift**—upper mediastinal shift

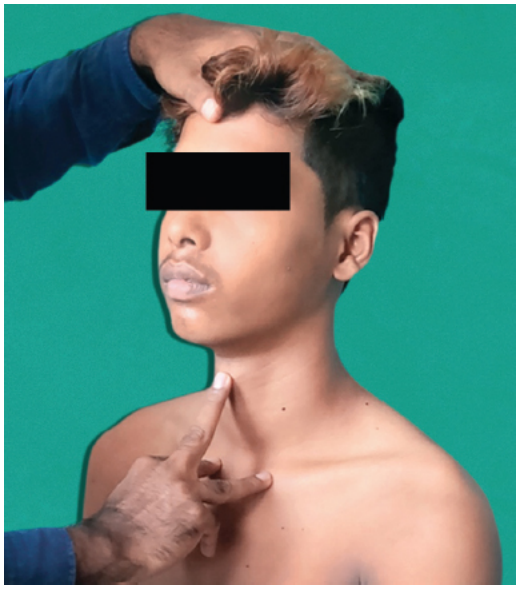


Fig. 3D.14: Tracing the trachea down with the middle finger.

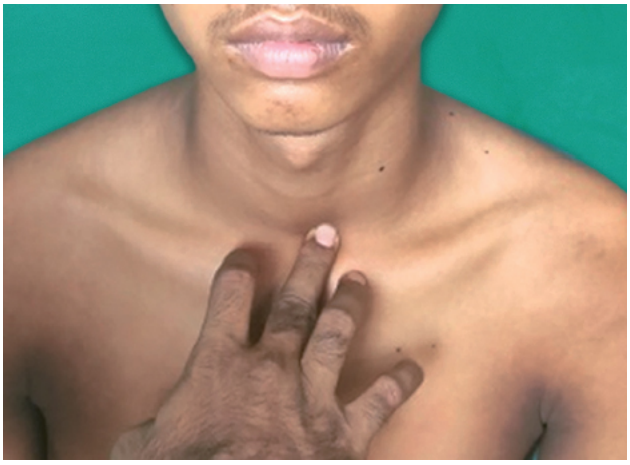


Fig. 3D.15: Insinuate the middle finger between the trachea and sternal head of sternocleidomastoid, and feel for resistance.

Oliver's sign (tracheal tug sign) (Fig. 3D.16):
Stand behind patient and hold cricoid cartilage give a slight upward thrust.

Positive test	Downward pull with each heart beat suggestive of aortic aneurysm
Negative test	Normal
False positive	Mediastinal tumor attached to abdominal aorta
False negative	Thrombosed aortic aneurysm

- **Tracheal descent on inspiration** (**Campbell sign**)**:** Due to downward pull of the depressed diaphragm in long standing hyperinflation of lung.

- **Laryngeal fixation:** Increased pressure on cricoid cartilage due to inflammatory or neoplastic lesion in mediastinum.

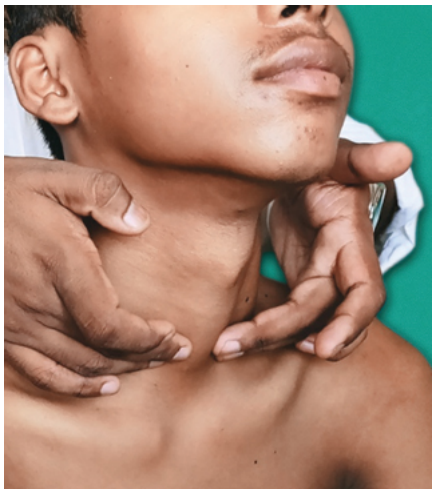


Fig. 3D.16: Demonstration of Oliver's sign.

Apical impulse:
- Confirm the position of apex
- Comment on character
- Watch for thrills and other palpable heart sounds
- **Implication of apical impulse shift:** It suggests lower mediastinal shift.

Apex not felt/seen in respiratory diseases
1. Emphysema
2. Left sided pleural effusion
3. Left sided pneumothorax.

Mediastinal shift with respect to respiratory diseases	
Shift to same side	• Fibrosis • Collapse
Shift to opposite side	• Pleural effusion • Pneumothorax • Tumor or mass
No shift of mediastinum	Unilateral disease • Pneumonia Bilateral disease • COPD • Asthma • Bronchiectasis • Interstitial lung disease

Examination of respiratory movements	
Upper anterior chest (Figs. 3D.17A and B)	• Examined by placing the palms in the infraclavicular areas • Look for superoanterior movement of the palms • This examines the **pump handle** movement of the upper lobes
Lower anterior chest (Figs. 3D.18A and B)	• Grasp the sides of the chest and approximate the tips of the thumbs in the mammary area with loose fold of skin in between

	• Watch for separation of the thumbs and compare the movements with each respiration • It demonstrates the **bucket handle** movements of the lower chest
Upper posterior chest (Fig. 3D.19)	• Examine from the back by placing hand in the supraclavicular fossa and watch for movements superiorly • This demonstrates the movement of the apical segment
Lower posterior chest (Fig. 3D.20)	• Grasp the sides of the chest and approximate the tips of the thumbs in the infrascapular area with loose fold of skin in between • Watch for separation of the thumbs and compare the movements with each respiration • This demonstrate the lower lobe movements

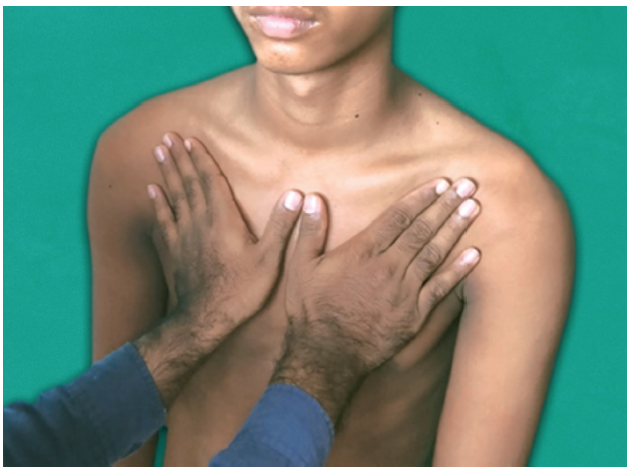


Fig. 3D.17A: Examination of respiratory movements of upper anterior chest.

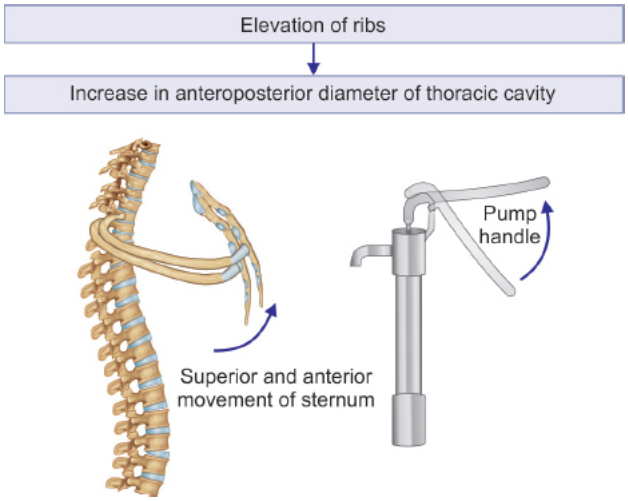


Fig. 3D.17B: Pump handle movement.

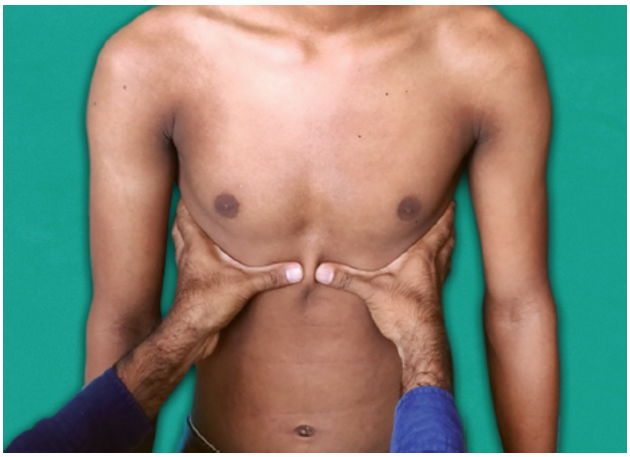


Fig. 3D.18A: Examination of respiratory movements of lower anterior chest.

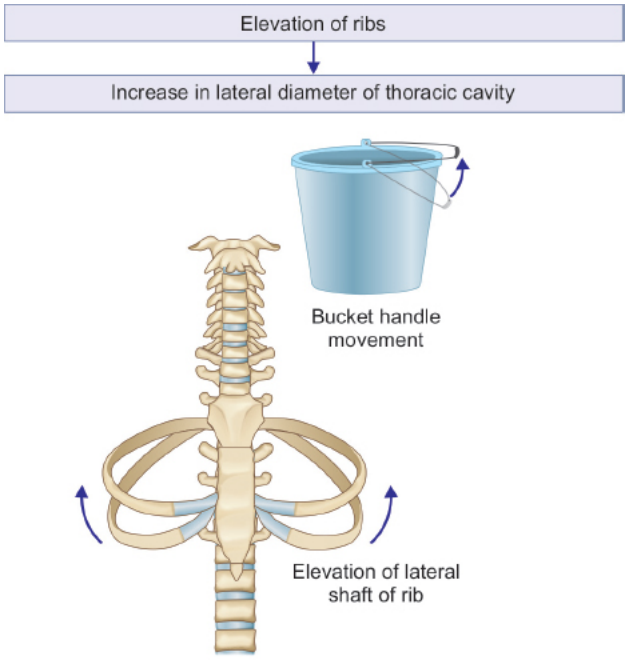


Fig. 3D.18B: Bucket handle movement.

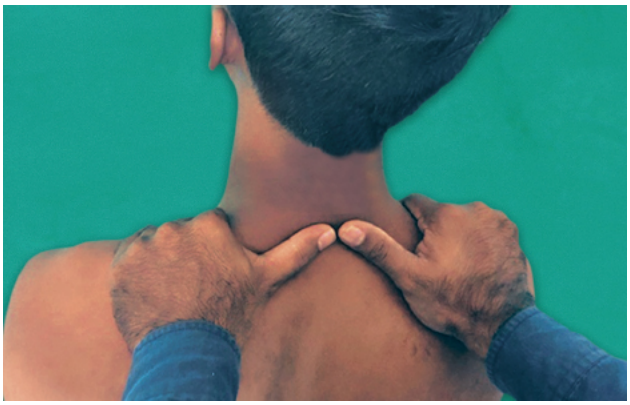


Fig. 3D.19: Examination of respiratory movements of upper posterior chest.

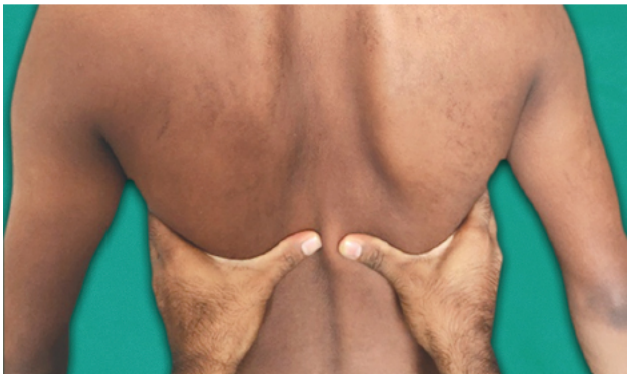


Fig. 3D.20: Examination of respiratory movements of lower posterior chest.

Diaphragmatic movements:

- Place one hand on chest and other hand on the abdomen **(Fig. 3D.21)**
- Normally—both hands are lifted during inspiration
- If chest rises but abdomen remains static—suggests an abdominal pathology which is fixing the abdomen
- If chest rises but abdomen retracts—suggests diaphragmatic palsy.

Causes of decreased chest movements	
Unilateral	***Bilateral***
• Pleural effusion • Empyema • Pneumothorax • Fibrosis • Collapse	• COPD • Asthma • Interstitial lung disease • Ankylosing spondylitis • Systemic sclerosis

Measurements of chest diameters	
AP diameter (Fig. 3D.22)	Use two cardboards and place as shown in **Figure 3D.22**. Normal ratio of AP:T = 5:7
Transverse diameter (Fig. 3D.23)	
Chest expansion (Fig. 3D.24)	Normal = 5–8 cm (adult), decreases with age (e.g. 60 years ≥3 cm is considered normal) COPD/ILD expansion is <1.5 cm
Hemithorax expansion (Fig. 3D.25)	Stand on side and place the tape from spine to midsternal as shown in **Figure 3D.25**.

Note: Chest expansion should be assessed as the difference of measurement between deep inspiration to deep expiration.

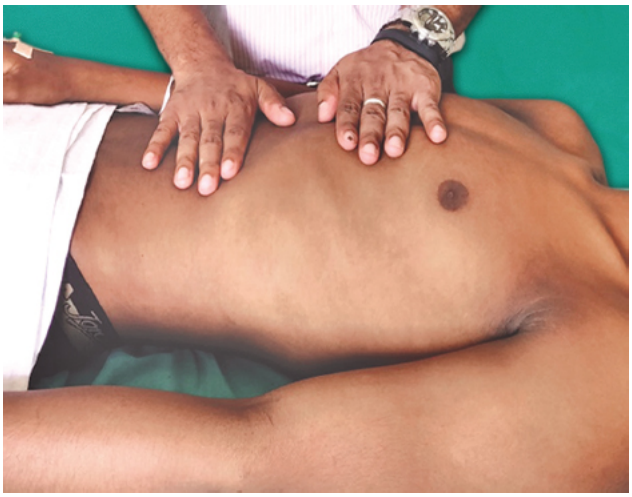


Fig. 3D.21: Examination of diaphragmatic movements.

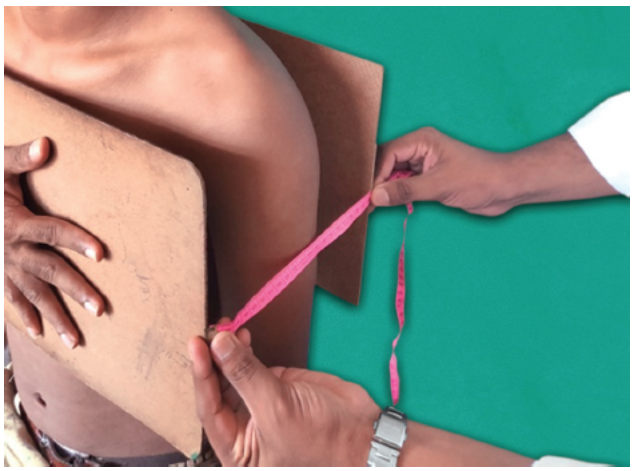
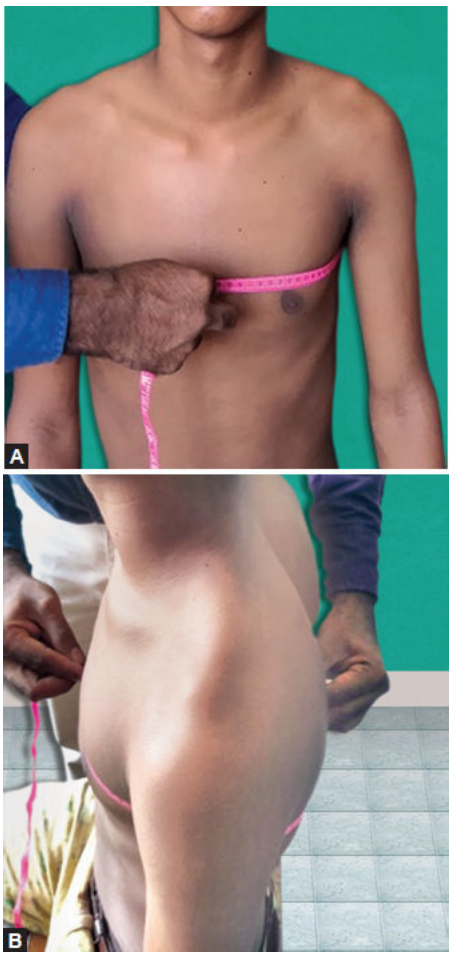


Fig. 3D.22: Examination of anteroposterior diameter.



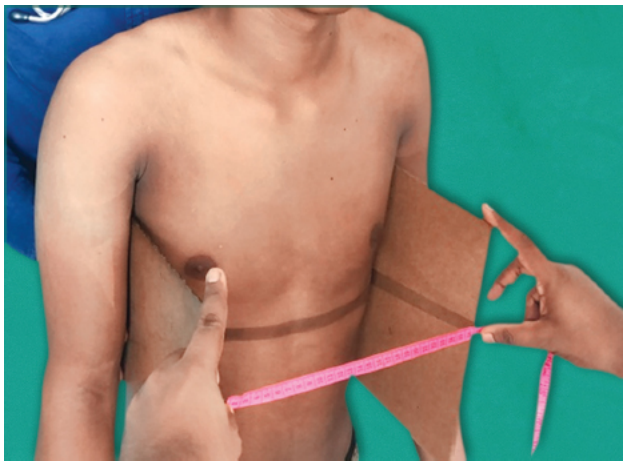


Fig. 3D.23: Examination of transverse diameter.

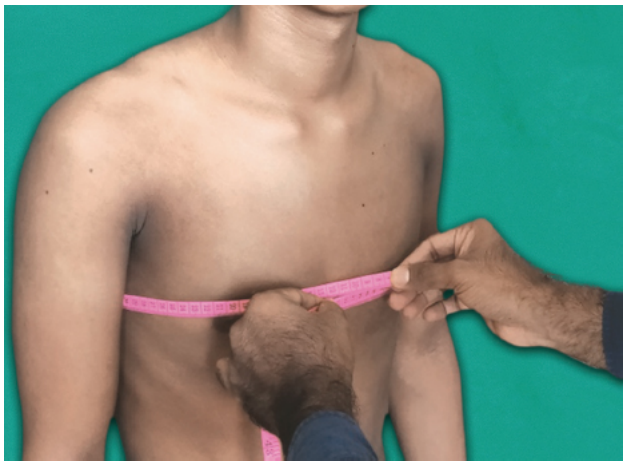


Fig. 3D.24: Examination of chest expansion (crossed tape).

Figs. 3D.25A and B: Examination of hemithorax circumference.

"THE MOST IMPORTANT EXAMINATION FINDING IS TO CHECK FOR HEMITHORAX EXPANSION AND HEMITHORAX MEASUREMENT."

Remember: "The side that moves less is the site of disease."

Increased hemithorax size with decreased hemithorax movement	*Decreased hemithorax size with decreased hemithorax movement*	*Normal hemithorax size with decreased hemithorax movement*
• Pleural effusion • Pneumothorax	• Fibrosis • Collapse	• Consolidation

Examination of spinoscapular distance (Fig. 3D.26): It is the distance between the spine and the scapular line (scapular line is the vertical line passing through the inferior angle of scapula).

Examination of spino-acromion distance (Fig. 3D.27): It is the distance measured between the spine and the tip of acromion process.

Fig. 3D.26: Examination of spinoscapular distance.

Fig. 3D.28: Demonstration of vocal fremitus.

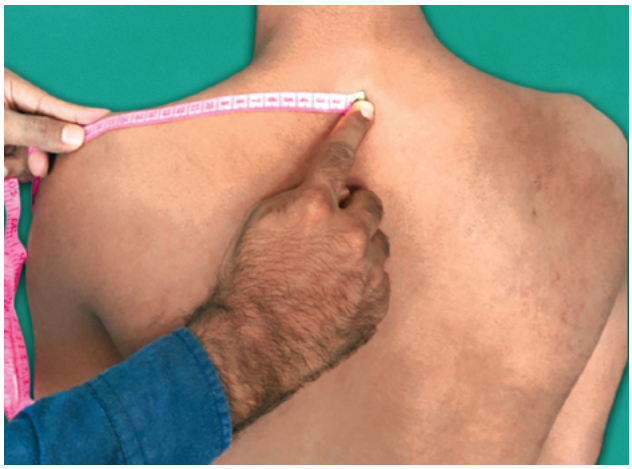


Fig. 3D.27: Examination of spino-acromion distance.

Vocal fremitus

- The sounds produced by vocal cords are transmitted along the tracheobronchial tree and heard/felt over the chest wall.
- Place the ulnar border of the hands on identical areas on both sides of the chest **(Fig. 3D.28)**.
- Ask the patient to repeat "one-one-one-"

Vocal fremitus	
Increased	***Decreased***
• Consolidation • Large cavity • Bronchopleural fistula	• Pleural effusion • Pneumothorax • Fibrosis • Collapse • Asthma • Emphysema • Thick pleura

Tactile fremitus

- These are palpable adventitious sounds

- It could be coarse crepitations or rhonchi.

Friction fremitus
These include palpable pericardial rub or pleural rub (e.g. dry pleurisy).

Tenderness:
Seen in
1. Empyema (intercostal tenderness)
2. Local inflammation of soft tissue
3. Osteomyelitis/rib fractures/costochondritis (Tietze syndrome)
4. Tumor infiltration
5. Amoebic liver abscess
6. Subphernic abscess

Detection of subcutaneous emphysema:
Spongy crepitant feeling on palpation
1. Injury to chest wall
2. Pneumothorax
3. Rupture of esophagus

Rib crowding/intercostal widening:
- Stand behind the patient and place the fingers in the intercostal spaces simultaneously on both sides as shown in **Figure 3D.29**.
- Observe for the separation of the fingers

Rib crowding		Intercostal widening	
Unilateral	***Bilateral***	***Unilateral***	***Bilateral***
• Atelectasis • Collapse • Fibrosis • Pneumonectomy	• Interstitial lung disease • Fibrosis (bilateral)	• Pneumothorax • Pleural effusion	Emphysema

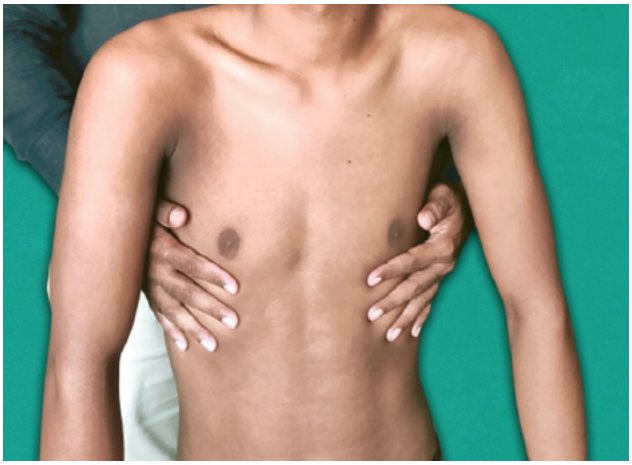


Fig. 3D.29: Examination of rib crowding.

Percussion (Lower Respiratory Tract)

Preferably done in sitting position, supine position is needed for demonstrating shifting dullness.

Position of patient for percussion:

- **Anterior chest (Fig. 3D.30):** Sits up straight with hands by his side
- **Axilla (Fig. 3D.31):** Raise the arm over the head and place over the back of head
- **Posterior of chest (Fig. 3D.32):** Sits up with hands crossed and placed over the opposite shoulders.

Rules of Percussion

1. **Direction of percussion**: Always percuss from resonant to non-resonant area.
2. **Pleximeter** is usually the middle phalanx of middle finger of left/nondominant hand and is firmly placed on the surface while rest of fingers are slightly lifted off.
3. **Plexor/plessor** (percussing finger) is middle finger of the right/dominant hand.
4. The movement of the plexor hand should be sudden and originating from the wrist.
5. The pleximeter must be kept parallel to the border to be percussed.
6. Percuss around 2–3 times over each area.
7. Percussion has to be heard as well as felt.
8. Always percuss the identical areas of chest for comparison.
9. The distance between the pleximeter finger and the ear should preferably be maintained.

Types of percussion

Heavy percussion	Light percussion
Posterior part of chest	Anterior part of chest and abdomen

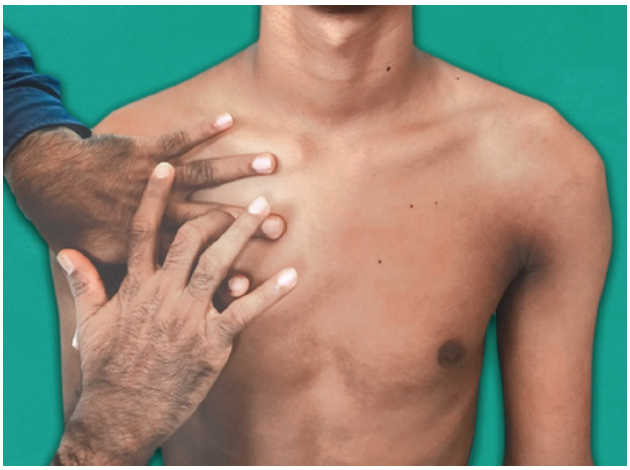


Fig. 3D.30: Demonstration of percussion of anterior chest.

Fig. 3D.31: Demonstration of percussion of axillary area.

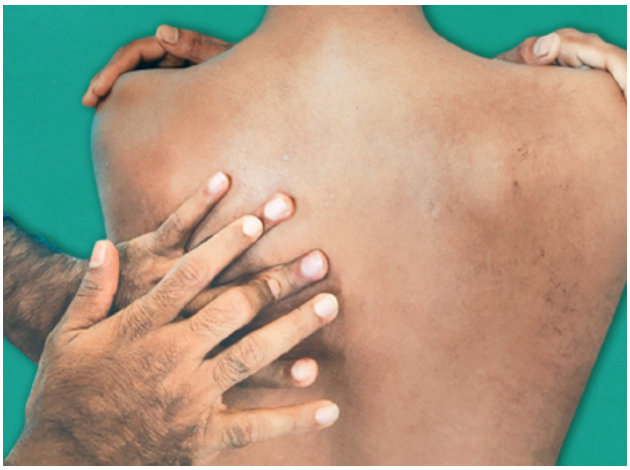


Fig. 3D.32: Demonstration of percussion over the posterior chest.

Direct percussion	Indirect percussion	Auscultatory percussion
Directly over the bony structures like clavicle	By percussing over the plexímeter finger with the plexor/plessor	Was first described by Laennec and used to delineate the size of organs by placing the stethoscope directly above the structure to be outlined, followed by percussion from the periphery towards the organ of interest

Direct percussion (Fig. 3D.33):

- Percuss the middle third of the clavicle with plexor finger.
- Stretch the skin over the clavicle using the left hand as shown in **Figure 3D.33**.
- Normally middle third of the clavicle is resonant whereas the medial and lateral thirds are dull (because of muscles attached).

Impaired note	Heard in apical fibrosis
Dull note	Mass lesion like pancoast tumor
Widening of zone of resonance	Heard in pneumothorax or emphysema

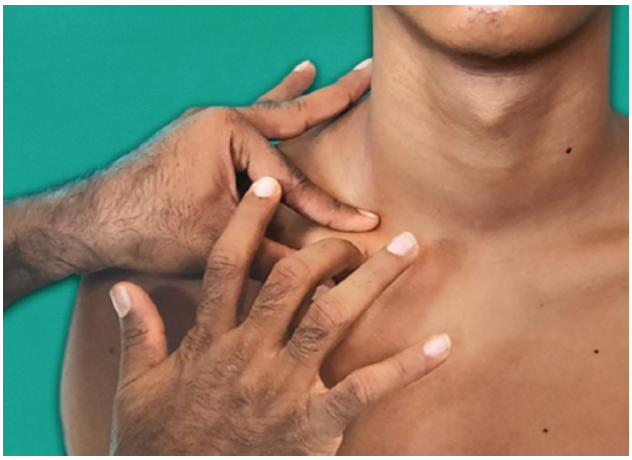


Fig. 3D.33: Demonstration of direct percussion over the clavicle.

Flicking percussion: Flicking using thumb and finger—done for percussion of the abdomen, cardiac border and to check for metallic note of pneumothorax.

Guarino's method of auscultatory percussion:

- Examined with patient sitting up and examiner facing the back of the patient
- Place the stethoscope around 3 cm below the last rib in the scapular line as shown in **Figure 3D.34**.
- Now percuss with the free hand (by finger flicking or with pulp of the finger) along 3 or more parallel lines from the apex of each hemithorax perpendicularly downward towards the base to note the dullness.

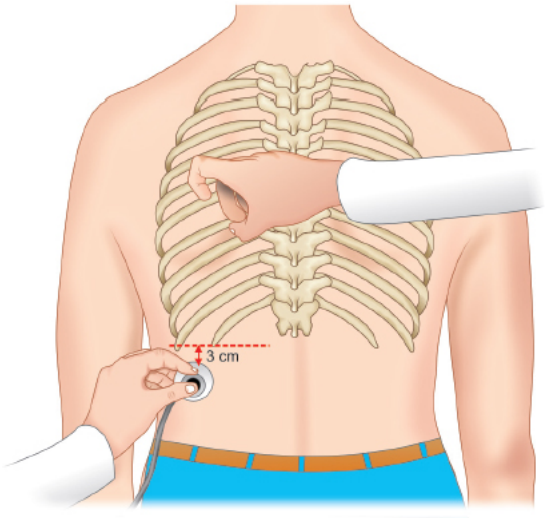


Fig. 3D.34: Guarino's method of auscultatory percussion in pleural effusion.

Lung Resonance

Normal:

- Vesicular resonance
- Front of chest more resonant
- Lesion >5 cm from chest wall or <2–3 cm in size will not alter the percussion note.

Abnormal types of percussion notes	
Quantitative	***Qualitative***
• Tympanic note • Subtympanic note • Hyper-resonant note • Impaired note • Dull/woody dull note • Stony dull note	• Crackpot • Amphoric • Bell tympany

Quantitative types	
Tympanic note	• It is a drum-like note • Normally seen over the stomach, intestine—Traube's space • In chest—superficial cavity, subcutaneous emphysema (metallic tympanic note)
Subtympanic (skodaic) note	• It is Boxy quality • Seen just above pleural effusion
Hyper-resonant note	• Intermediate between normal and tympanic note • Bilateral—emphysema • Unilateral—pneumothorax, compensatory emphysema • Large bullae
Impaired note	• Airless areas (fibrosis, collapse)
Dull note	• Consolidation • Thick pleura
Flat dull	• Can be elicited by percussing over the thigh • Seen in pleural effusion
Stony dullness	• Pain over the pleximeter finger with resistance felt by plexor • Large pleural effusion • Large solid tumor
Qualitative types	
Cracked pot resonance	• Normally seen in chest of infants or child during the act of crying • Pathological lung cavity with communication with bronchus due to sudden expulsion of air form the cavity to bronchus • Artificially imitated by beating clasped hands over the knee
Amphoric	• Low pitched hollow note • Normally seen in trachea and cheek distended with air • Pathologically seen in pneumothorax and large cavity
Bell tympany	• High pitched metallic or tympanic note • Seen in massive pneumothorax • Place coin on affected side of chest and percuss with another coin while simultaneously auscultating the back

Dullness in presence of fluid in lung	
Straight line dullness	Hydropneumothorax
S-shaped curve of Ellis	Pleural effusion

5-7-9 rule:

The upper border of liver dullness is at 5th intercostal space (ICS) in mid clavicular line, 7th ICS in the midaxillary line and 9th ICS in the scapular line.

Topographical percussion of lung

Apical percussion:

- **Kronigs isthmus:** It is a band of resonance in the supraclavicular area bounded anteriorly by the posterior border of the clavicle, medially by the neck muscles, posteriorly by the anterior border of trapezius, extended laterally till the acromioclavicular joint.
- Stand behind the patient, place the pleximeter finger over the neck and percuss from lateral to medial as shown in **Figure 3D.35**.
- On percussion there is dull zone medially and laterally, and only middle part is resonant.
- Dullness in this area suggests apical tuberculosis, Pancoast tumor or apical fibrosis.
- The zone of resonance may be widened in emphysema or apical pneumothorax.

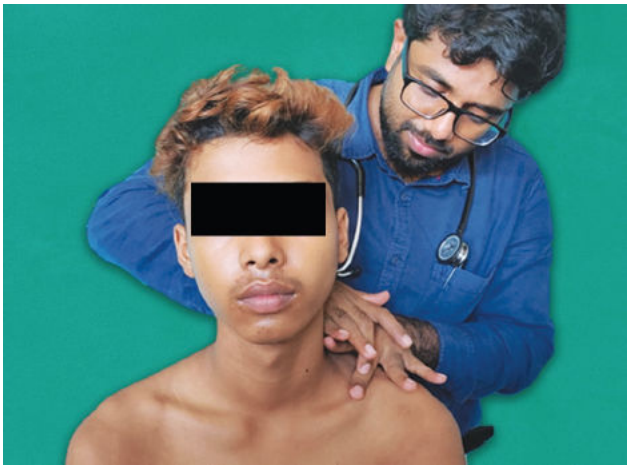


Fig. 3D.35: Percussion of apical area (Kronig's isthmus).

Tidal percussion:

- Tidal percussion is a measure of diaphragmatic excursion
- It is used to differentiate whether the causes of dullness are above the diaphragm (subpulmonic effusion) or below (subphrenic collections)
- With patient in, percuss the right side of the chest from above downwards till you get the liver dullness. Normally, it is in 5th intercostal space.
- Ask the patient to take a deep inspiration and hold his breath.
- Now percuss the same area
- Normally, dullness moves down by 1–2 intercostal spaces as shown in **Figure 3D.34**.
- Tidal percussion is negative in right-sided subpulmonic effusion, diaphragmatic paralysis.
- In emphysema, since the lung is already fully expanded tidal percussion will be negative (**Figs. 3D.36A and B**).

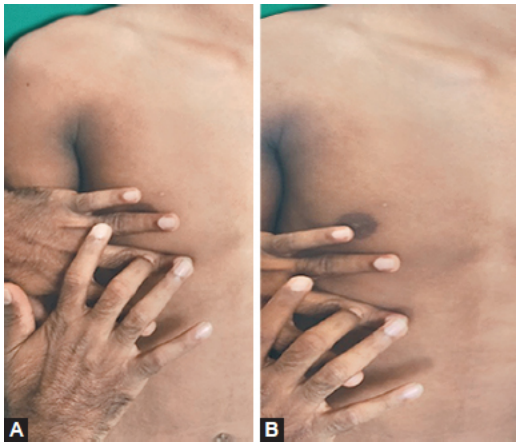
Percussion of Traube's space (Fig. 3D.37)

It is a semilunar space in the left anterior chest bounded by:

- Above by 6th rib
- Below by left costal margin
- Laterally by midaxillary line.

Normal Traube's space percussion	Tympanic note
Obliteration of Traube's space	• Left sided pleural effusion • Pericardial effusion • Massive splenomegaly • Enlarged left lobe of the liver • Full stomach or fundic mass

Upward shift of Traube's space	• Left diaphragmatic paralysis • Left lower lobe collapse or fibrosis



Figs. 3D.36A and B: Demonstration of tidal percussion: (A) Expiration; (B) Inspiration (Note the change in liver dullness from expiration to inspiration).

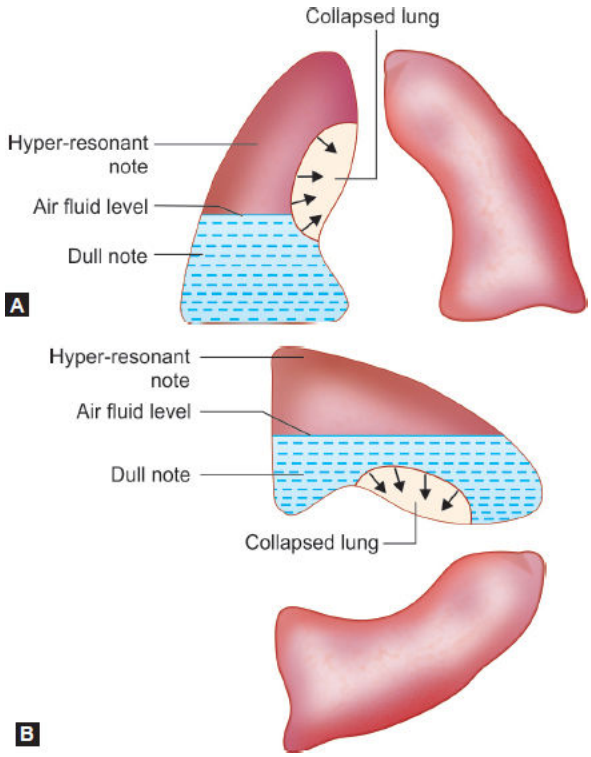
Fig. 3D.37: Percussion of Traube's space.

Shifting dullness:

It is classically described for hydropneumothorax. It can also be demonstrated in pleural effusion.

Steps:

- Percuss the anterior chest in sitting position, from above downward to get upper border of dullness. You will get a level of straight line dullness perpendicular to long axis of body as shown in **Figure 3D.38A**. Mark this level.
- Now, make the patient lie down in opposite lateral position/normal side (for around 5 minutes in case of hydropneumothorax and around 30 minutes in case of pleural effusion). Percuss over the affected side and note the change in the straight line dullness which will now be parallel to long axis of body as shown in **Figure 3D.38B**. Shifting dullness may be absent in case of empyema or loculated pleural effusion.



Figs. 3D.38A and B: Right hydropneumothorax: (A) Sitting position; (B) Left lateral position.

Special findings in percussion:

Special finding	Clinical condition
Shifting dullness	Hydropneumothorax
S-shaped curve of Ellis (Damoiseau's curve)	Pleural effusion (moderate)
Obliteration of Traube's space	Pleural effusion (left sided)
Grocco's triangle (Fig. 3D.39) (Paravertebral triangle of dullness)	**Boundaries of Grocco's triangle:** • **Medially:** The mid-spinal line from the level of the effusion to the level of the tenth dorsal vertebra • **Below:** A horizontal line extending outwards from the tenth dorsal vertebra along the lower limit of lung resonance • **Laterally:** A curved line connecting these two lines **Clinical condition:** Seen over the back of the chest, on the opposite side of effusion in moderate to massive pleural effusions
Garland's triangle (Fig. 3D.39)	• Small area of resonance next to the spine found in patients with large unilateral pleural effusions • Lower relaxed part of the lung in moderate or large pleural effusion is tympanic or subtympanic
William's tracheal resonance	**Description:** • Area of tympany over the first or second intercostal space, close to sternum **Seen in:** • Patch of consolidation or fibrosis interposed between the trachea or a major bronchus and the chest wall • Referred to as "**pulled trachea syndrome**" in fibrotic apical tuberculosis
Wintrich's sign	**Description:**

	• Percussion note over an area during inspiration appears clearer and higher-pitched with the mouth open than with it closed **Seen in:** • Lung cavity communicating with a bronchus, pneumothorax or mediastinal tumor
Gerhardt's sign	**Description:** • Percussion note over an area appears lower pitched with the patient recumbent than with him standing or sitting **Seen in:** • Lung cavity containing both fluid and air.
Friedreich's sign	**Description:** • Percussion note over an area becomes higher in pitch during forced inspiration than during expiration **Seen in:** • Lung cavity

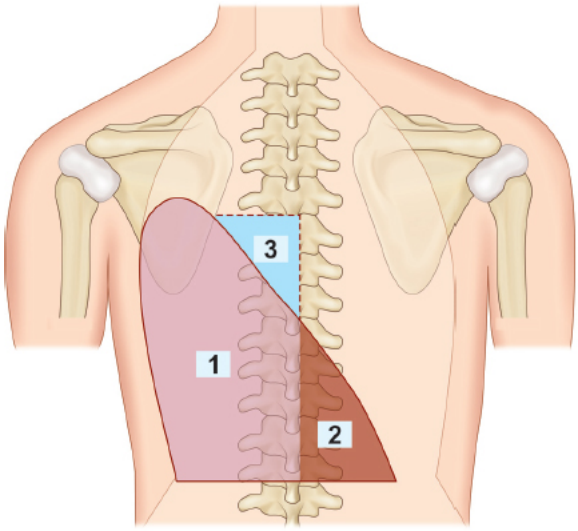


Fig. 3D.39: Special findings in percussion: (1) Effusion, (2) Rauchfuss-Grocco triangle, (3) Garland triangle.

Auscultation (Lower Respiratory Tract)

Position of patient:

In upright position	Front	Sitting or standing
	Back	Preferably sitting and leaning forward with neck flexed and arms crossed in front
In recumbent position	Back	Turn the patient sideways or slip the steth underneath the patient

Breathing advice:

Ask the patient to breathe through the mouth. If not cooperating ask the patient to count numbers or cough successively and then observe during deep inspiration.

Normal physiology of breath sounds :

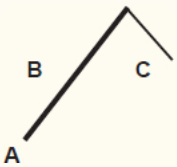
Mechanism of sound production	
In larger airways (pharynx, large airways of trachea and lung)	***In smaller airways***

Sounds are generated due to turbulence	Higher frequencies are lost due to dampening when they travel from higher to smaller airways
They are the source of sound	They are just filter sounds and not the source of sound
Sound frequencies are of range 200–2,000 Hz	Sound frequencies are of range 200–400 Hz
Heard over the upper sternum	Heard over most other areas of lung

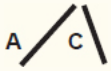
Grading of breath sound intensity	
0	Absent breath sounds
1	Barely audible breath sound
2	Faint but definitely audible breath sound
3	Normal breath sound
4	Louder than normal breath sound

Graphical representation of breath sounds	
Upstroke	Inspiratory element
Downstroke	Expiratory element
Length	Duration or timing
Thickness	Loudness or intensity
Angle between upstroke and downstroke made with a vertical line	• Pitch of respiratory sound • Lower the angle higher is the pitch

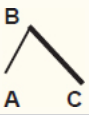
Types of normal breathing	
Vesicular breathing	Most areas of chest
Tracheal/bronchial breathing	• Larynx • Trachea • Between C7 to T3
Bronchovesicular	• Anteriorly 1st and 2nd intercostal space • Posteriorly between the scapula

Vesicular breath sounds	
Characteristics	• Rustling or breezy quality • Longer duration of inspiratory phase (which includes both tubular and alveolar phase) • Higher pitch of inspiratory sound • I:E = 4:1 • Absence of pause between I and E
Distribution	Most of chest
Intensity	• Louder: infraclavicular, axillary and infrascapular areas • Diminished: Lower margins of lung and over the scapular areas
Mode of production	Distension and separation of alveolar walls by the in rushing current of air
Graphical representation	 A. Tubular phase of inspiration B. Alveolar phase of inspiration

	C. Expiration

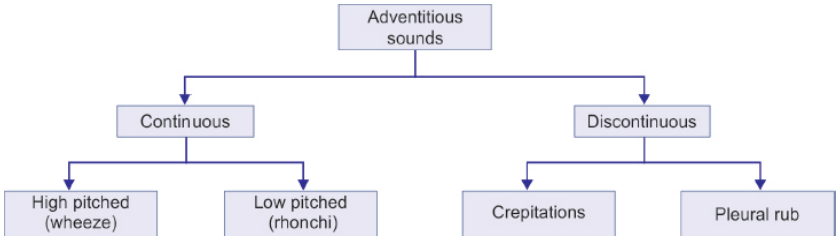
Tracheal (bronchial) breath sounds	
Characteristics	• Character is aspirate or guttural • Expiration in longer • Expiration is louder • Expiration has high pitch • I:E = 1:1 • There is a pause between inspiration and expiration (due to **absence of alveolar phase**)
Distribution	• Larynx • Trachea
Mode of production	Due to in and out movement of air through narrow aperture of glottis
Graphical representation	 A. Tubular phase of inspiration B. ABSENT C. Expiration

Type of bronchial breathing		
Tubular	***Amphoric***	***Cavernous***
High pitched sounds at the bronchioles are conducted to the chest wall without modification, e.g. • Consolidation • Above the level of pleural effusion • Massive pericardial effusion (Ewart's sign)	Low pitched bronchial breathing with high pitched overtones producing a metallic quality, e.g. • Open pneumothorax due to bronchopleural fistula • Large communicating cavity	Low pitched sound with a peculiar hollow quality, e.g. cavity

Bronchovesicular breath sounds (also known as vesicular breath sounds with prolonged expiration)		
Characteristics	• Intermediate in character between vesicular and bronchial breath sounds • Expiratory phase is louder, longer, higher pitched than inspiratory, or hollow character	
Distribution	• Upper part of sternum • Up to 3rd/4th dorsal spines between scapula • At times over the lung apices particularly on right side	
Mode of production	Usually seen when air containing lung tissue is interposed between a large bronchus and the chest wall—thus combining the characteristics of both vesicular and bronchial breath sounds	
Graphical representation		A. Tubular phase of inspiration B. Alveolar phase of inspiration C. Expiration
It is the hallmark auscultatory finding of obstructive lung disease like chronic obstructive pulmonary disease and asthma		

Diminished intensity of breath sounds	
Defect in production	***Defect in transmission***
• Bronchial obstruction • Emphysema • Respiratory muscle paralysis	• Pleural effusion • Pneumothorax • Thickened pleura • Thick chest wall • Fibrosis

Adventitious Sounds



Continuous adventitious sounds:

- Lasts for more than 250 ms
- Musical in quality
- Mechanism of production of sound: Important prerequisite for the production of wheeze is airflow limitation. Narrowing of airways along with increased intrathoracic pressure results in airflow limitation producing sinusoidal oscillations.
- For example: Wheeze and rhonchi.

Wheeze	Rhonchi
High pitched sounds	Low pitched sounds
400 Hz	200 Hz
Hissing quality (sibilant)	Snoring quality (sonorous)
Predominantly arise from small airways obstruction	Usually produced when air moves through tracheo-bronchial passages in the presence of mucus or respiratory secretions

Classification of wheezes/rhonchi:

1. Monophonic or polyphonic
2. Inspiratory or expiratory

Monophonic	Polyphonic
Single tones	Diffuse, multiple tones, both phases
Due to local pathology producing bronchial obstruction 1. Tumor 2. Foreign body aspiration 3. Bronchostenosis 4. Mucous plug 5. Lymph node compression	Due to dynamic compression 1. COPD 2. Bronchial asthma 3. Tropical pulmonary eosinophilia 4. Hypersensitivity pneumonitis 5. Eosinophilic pneumonia 6. Churg-Strauss syndrome

Discontinuous Adventitious Sounds (Rales/Crepitations/Crackles)

- These are discontinuous, explosive, nonmusical and harsh in quality
- Mainly inspiratory (can be in expiratory or both).

Mechanism of crepitation:

1. Bubbling sounds produced by passage of air through accumulated secretions.
2. Sudden snapping opening of successive small airways when airflow is through it.

Fine crepitations	Coarse crepitations
Due to snapping opening of successive small airways	Due to bubbling sounds produced by passage of air through accumulated secretions
High pitched (soft)	Low pitched (loud)
Smaller airways	Larger airways

Heard during inspiration	Heard during inspiration and expiration
Not modified by coughing	Modified by coughing
Not palpable	Palpable
For example 1. Indux crepitations (initial stages of pneumonia) 2. Pulmonary edema (early phase) 3. Interstitial lung disease 4. Asbestosis 5. Hypersensitivity pneumonitis 6. Sarcoidosis	**For example** 1. Redux crepitations (resolution phase of pneumonia) 2. Pulmonary edema (late phase) 3. Bronchiectasis 4. Lung abscess 5. Bronchitis

Inspiratory crepitations		Expiratory crepitations
Early	• Acute bronchitis • Chronic bronchitis	• **R**edux crepitations (**R**esolution phase of pneumonia) • Pulmonary edema (late phase) • Bronchiectasis • Lung abscess • Bronchitis
Mid	• Bronchiectasis Resolving phase of pneumonia	
Late	• Interstitial lung disease • Asbestosis • Early pneumonia • Pulmonary edema	

Few named crepitations	
Coarse leathery	Bronchiectasis
Velcro crepts	Interstitial lung disease
Posture induced crackles	Appearance of fine crackles while changing of posture (sitting to supine or supine with passive leg elevation). Ausculate in the posterior axillary line in the 8th, 9th and 10th intercostal spaces after 3 minute of supine position. It indicates ischemic heart disease with heart failure
Post-tussive crepitations	Crepitations which are not present normally but appear after a bout of cough. Seen in early pneumonia, early tuberculosis and lung abscess

Stridor

- High pitched whistling or grating sound which is produced by upper airway obstruction.
- It is louder over the neck than the chest wall.
- Indicates extrathoracic upper airway obstruction (like vocal cord paralysis, supraglottic growths, etc.)
- It usually seen during inspiration, however, can be seen in expiration in intrathoracic tracheobronchial obstruction.

Pleural rub

- It is harsh discontinuous, localized, nonmusical, superficial grating sound due to rubbing of the inflamed pleural surfaces against each other.
- It is heard in both phases of respiration and disappears on holding the breath.

Causes

- Dry pleurisy
- Consolidation
- Infarction

Differences between pleural rub and crepitations:

Pleural rub	Crepitations
Both inspiratory and expiratory phases	Inspiratory/expiratory or both
Localized to small area	Wide spread
No change after coughing	May clear after coughing
Pressure on stethoscope increases the sound	No effect
Associated with pleuritic chest pain and local tenderness	No pain or tenderness

Vocal resonance:

- Make the patient sit
- Place the stethoscope firmly on the chest wall
- Ask the patient to speak “one-one-one” or “ninety nine” repeatedly
- Compare corresponding areas anteriorly, in axilla and posteriorly.
- Increased vocal resonance

Vocal resonance	
Increased	***Decreased***
• Consolidation • Large cavity • Bronchopleural fistula	• Pleural effusion • Pneumothorax • Fibrosis • Collapse • Asthma • Emphysema • Thick pleura

Note: in upper lobe fibrosis, VR is increased due to the pulled trachea.

Variations of vocal resonance	
Bronchophony	Increase in loudness as well as clarity of the sound **Seen in:** • Consolidation • Just above level of pleural effusion • On spine up to T4
Aegophony	Selected amplification of high frequency sounds. “E” is heard as “A” **Seen in:** • Consolidation (it is the auscultatory sign of consolidation)
Whispering pectoriloquy	When the whispered sound in the chest wall is heard clearly and distinguishably as if uttered directly into the external ear **Seen in:** • Consolidation • Cavity with communication with bronchus

Other Auscultatory Features

Post-tussive suction:

It is a sign of superficial collapsible cavity seen in active tuberculosis. When you auscultate a cavernous bronchial breathing (which indicates a cavity), ask the patient to cough. A suction sound will be heard if the cavity collapses.

Prerequisites for post-tussive suction:

- Superficial cavity
- Thin-walled cavity

- Has to be communicating with bronchus
- Surrounding lung should be normal.

Succussion splash (Hippocrates succussion):
- It is seen in hydropneumothorax
- First percuss and get the air fluid level in hydropneumothorax
- Keep the diaphragm at the air-fluid level
- Hold the opposite shoulder of the patient and shake vigorously as shown in **Figure 3D.40**.
- Tinkling or splashing sound will be heard.
- Other conditions like large cavity with fluid, diaphragmatic hernia can also produce succussion splash.

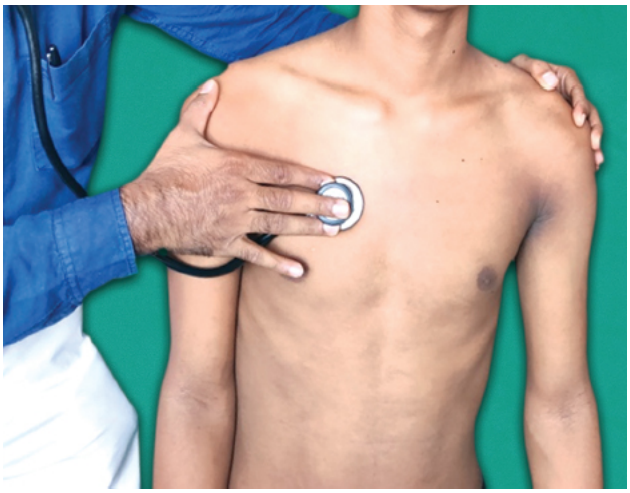


Fig. 3D.40: Demonstration of succussion splash.

Coin test:
- High pitched metallic or tympanic note
- Place one coin flat on affected side of chest (posteriorly/anteriorly) and percuss with another coin perpendicularly on it, while simultaneously auscultating from the opposite direction of the same affected side as shown in **Figure 3D.41**.
- Seen in massive pneumothorax/hydropneumothorax.

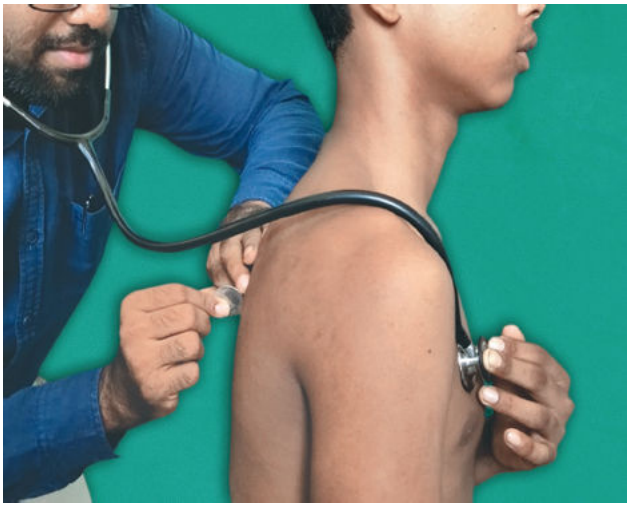


Fig. 3D.41: Demonstration of coin test.

Scratch sign:

- Used for diagnosis of pneumothorax
- Patient sitting, place the diaphragm of the stethoscope in the midpoint of sternum or spine
- Scratch the chest wall from mid axillary line towards the sternum on either side.
- Sound will be louder on the side of pneumothorax.

Hamman's mediastinal crunch:

- Loud cracking or clicking sound heard in the 3rd to 5th intercostal spaces near the left sternal border synchronous with the heartbeat.
- It is the sign of mediastinal emphysema (pneumomediastinum) or can also be seen in left-sided pneumothorax.

Forced expiratory time (FET):

- It is a simple inexpensive and sensitive bedside test to detect airflow obstruction.
- Instruct the patient to inhale up to the total lung capacity and then blow it as fast and complete as possible.
- Place the bell of stethoscope in suprasternal notch and time the audible expiration.
- A value less than 5 seconds indicates FEV1/FVC more than 60%, whereas FET more than 6 sec indicates FEV1/FVC less than 50%.

Summary of findings in pleural effusion based on the severity			
Finding	***Mild effusion (<300 mL)***	***Moderate effusion (300–1,500 mL)***	***Massive effusion (>1,500 mL)***
Tachypnea	No	Present	Significant
Chest expansion	Normal	Decreased on the effected side	Significantly decreased on the effected side
Tactile fremitus	Normal	Decreased	Absent
Breath sounds	Vesicular	Decreased	Absent or bronchial
C/L tracheal or mediastinal shift	Absent	Absent	Present
Bulging intercostal spaces	No	Sometimes	Present
Egophony	No	Yes	Yes

E. RESPIRATORY SYSTEM: SUMMARY OF FINDINGS IN COMMON RESPIRATORY DISEASES

	Findings	Fibrosis	Collapse	Pleural effusion	Pneumothorax	Hydropneumothorax	Conso
Inspection	**Trachea/Mediastinum**	Pulled to same side	Pulled to same side	Pushed to opposite side	Pushed to opposite side	Pushed to opposite side	Central
	Retraction/bulge	Retraction on the affected side	Retraction on the affected side	Bulging/fullness on the affected side	Bulging/fullness on the affected side	Bulging/fullness on the affected side	—
Palpation	**Chest expansion**	Reduced on the effected side	Reduced on the effected side	Reduced on the effected side	Reduced on the effected side	Reduced on the effected side	Reduce the effe side
	Hemithorax dimension	Reduced on the effected side	Reduced on the effected side	Increased on the effected side	Increased on the effected side	Increased on the effected side	Normal dimens
	Vocal fremitus	Reduced	Reduced	Reduced	Reduced	Reduced	Increas
Percussion	**Percussion note**	Impaired note over fibrosed lung	Dull note over the collapsed lung	Stony dull note over the pleural effusion and skodiac resonance at the level of pleural effusion	Hyper-resonant note over the pneumothorax	Hyper-resonant note above the air fluid level and dull note below the air fluid level.	Woody note ov consoli
	Special findings	William's tracheal resonance		• Ellis curve pattern of upper level of effusion Grocco's • triangle • Obliteration of Traube's space • Garland's triangle	Bell tympany can be appreciated (Coin test positive)	Shifting dullness, Straight line dullness, Succussion splash, Bell tympany can be appreciated (Coin test positive)	
Auscultation	**Breath sounds**	Diminished breath sounds	Absent breath sounds	Absent breath sounds	Absent breath sounds	Absent breath sounds	Tubular sounds
	Adventitious sounds/special findings	Fine crepitations	—	—	Bell tympany can be appreciated (Coin test positive)	Bell tympany can be appreciated (Coin test positive)	Crepita heard
	Vocal resonance	Reduced	Reduced	Reduced	Reduced	Reduced	Increas (Bronch egopho whispe pectori

NOTES

Cardiovascular System Examination

CHAPTER 4

A. CASE SHEET FORMAT

HISTORY TAKING

Name:

Age:

Sex:

Residence:

Occupation:

Chief complaints (describe in chronological order):

1. _____ × days
2. _____ × days
3. _____ × days

Dyspnea:

- Duration
- Onset
- Grade
- Progression
- Aggravating factors
- Relieving factors
- Orthopnea
- Trepopnea
- Platypnea
- Bendopnea
- Paroxysmal nocturnal dyspnea
- Associated symptoms
  - Wheeze
  - Cough with expectoration

Chest pain:

- Duration
- Onset
- Site
- Type of pain
- Radiation
- Diurnal variation (nocturnal angina)
- Aggravating factors
- Relieving factors

- Associated symptoms
  - Nausea, vomiting, sweating
- Dyspepsia
- Local tenderness
- Angina equivalents.
  - Dyspnea
  - Diaphoresis
  - Discomfort in lower jaw
  - Dyspeptic symptoms
  - Fatigue

Palpitations:

- Duration
- Onset
- Fast or slow
- Regular or irregular
- Precipitating factors
- Associated symptoms
  - Stoke Adams
- Post-palpitation diuresis

Syncope:

- Duration
- Onset
- No of attacks
- Awareness
- Precipitating factors
- Associated symptoms

Pedal edema:

- Duration
- Onset
- Progression
- Aggravating factors
- Relieving factors
- Is it preceded by facial puffiness or followed by facial puffiness?

Hemoptysis

Cyanosis

Decreased urine output

Gastrointestinal symptoms

Right hypochondrial pain

Fatigability

Fever

Rheumatic fever history

Infective endocarditis

Cyanotic spells

Squatting after exertion

Past history:
- Asthma
- Chronic obstructive airway disease
- Tuberculosis
- History of contact with tuberculosis
- Diabetes mellitus
- Hypertension
- Ischemic heart disease (IHD)
- Seizure disorder
- History of sudden cardiac death.

Family history:
Third generation pedigree chart to be drawn

Personal history:
- Bowel habits
- Bladder habits
- Appetite
- Loss of weight
- Occupational exposure
- Sleep
- Dietary habits and taboo
- Food allergies
- Smoking Index or Pack years
- Alcohol history (if yes mention in grams of alcohol)

Treatment history:
- Drugs using
- Frequency of drug (e.g. drug taken 5 times a week most likely to be digoxin)
- Duration of usage
- Any blood test to be monitored (e.g. INR for warfarin)
- Any intramuscular injections (once in 3 weeks IM injection most likely to be benzathine penicillin for rheumatic heart disease prophylaxis)

Menstrual and obstetric history
- Gravida, parity, live births, abortions (GPLA)
- Age of menarche __
- Menopause at __
- Duration

Summarize:

Differential diagnosis:
1.
2.

3.

GENERAL EXAMINATION

Patient

- Conscious
- Coherent
- Cooperative
- Obeying commands

Body Mass Index (BMI)

- Weight (kg)/H^2 (meters)
- Grading according to WHO for Southeast Asian countries
- Arm span
- Upper segment: Lower segment ratio

Vitals Examination

- Pulse
  - Rate
  - Rhythm
  - Volume
  - Character
  - Vessel wall thickening
  - Radio-radial delay and radio-femoral delay
  - Peripheral pulses
- Blood pressure
  - Right arm
  - Left arm
  - Leg—right and left
  - Postural drop in BP
- Respiratory rate
  - Regular/irregular
  - Abdominothoracic (male) or thoracoabdominal (female)
  - Usage of accessory muscles
- Jugular venous pressure
  - __ cm of water (blood) above sternal angle (+ 5 cm from the right atria)
- Jugular venous pulse
  - Waveform
- Pulse oximetry

Physical Examination

- Pallor:
- Icterus:
- Cyanosis:
- Clubbing:
- Lymphadenopathy:
- Edema:

Others

Signs of infective endocarditis

Signs of rheumatic fever

SYSTEMIC EXAMINATION

Inspection

- Chest shape and symmetry
- Breast abnormalities
- Spine deformity
- Precordial prominence
- Cardiovascular pulsations
  - Apical pulse
  - Pulsation in aortic and pulmonary area
  - Sternoclavicular pulsations
  - Left parasternal pulsations
  - Epigastric pulsations
  - Ectopic pulsations
- Distended veins

Palpation

- Confirmation of shape and symmetry
- Palpation of precordium
- Palpation of cardiovascular pulsation for sounds, thrills and rubs
- Tracheal tug

Percussion

- Right heart border
- Left heart border
- 2nd IC space
- Sternal percussion

Auscultation

- **Apex (mitral area)**
  - S1
  - S2
  - S3, S4
  - OS/clicks
  - Murmur
    1. Timing
    2. Grade
    3. Quality
    4. Pitch
    5. Configuration
    6. Radiation
    7. Best heard with diaphragm or bell
    8. Patient positon

9. With breath held in inspiration or expiration
10. Dynamic auscultation

- **Tricuspid area**
  - S1
  - S2
  - S3, S4
  - OS/clicks
  - Murmur
    1. Timing
    2. Grade
    3. Quality
    4. Pitch
    5. Configuration
    6. Radiation
    7. Best heard with diaphragm or bell
    8. Patient positon
    9. With breath held in inspiration or expiration
    10. Dynamic auscultation
- **Erb's neo aortic area**
  - S1
  - S2
  - S3, S4
  - OS/clicks
  - Murmur
    1. Timing
    2. Grade
    3. Quality
    4. Pitch
    5. Configuration
    6. Radiation
    7. Best heard with diaphragm or bell
    8. Patient positon
    9. With breath held in inspiration or expiration
    10. Dynamic auscultation.
- **(R) 2nd intercostal space (aortic area)**
  - S1
  - S2
  - S3, S4
  - OS/clicks
  - Murmur
    1. Timing
    2. Grade
    3. Quality
    4. Pitch
    5. Configuration
    6. Radiation
    7. Best heard with diaphragm or bell
    8. Patient positon

9. With breath held in inspiration or expiration
10. Dynamic auscultation.

- **(L) 2nd intercostal space (pulmonary area)**
  - S1
  - S2
  - S3, S4
  - OS/clicks
  - Murmur
    1. Timing
    2. Grade
    3. Quality
    4. Pitch
    5. Configuration
    6. Radiation
    7. Best heard with diaphragm or bell
    8. Patient positon
    9. With breath held in inspiration or expiration
    10. Dynamic auscultation.
- **Other areas**
  - Axilla
  - Epigastrium
  - Clavicle
  - Carotid
  - Back (interscapular area)

OTHER SYSTEM EXAMINATION

Respiratory:
- Inspection:
- Palpation:
- Percussion:
- Auscultation :

Gastrointestinal system:
- Inspection:
- Palpation:
- Percussion:
- Auscultation :

Nervous system:
- Higher mental functions:
- Cranial nerves:
- Sensory system:
- Motor system:
- Reflexes:
- Cerebellar system:
- Meningeal signs:

B. DIAGNOSIS FORMAT

ACQUIRED/CONGENITAL HEART DISEASE

For Acquired Heart Disease

- Acquired heart disease possible etiology (rheumatic/ischemic/cardiomyopathy/degenerative)
- Valvular involvement (MS/MR/AS/AR/others) with severity grading
- With/without evidence of pulmonary artery hypertension (grading)
- Patient in or not in atrial fibrillation (if AF present look for signs of thromboembolism)
- With or without evidence of heart failure (right/left/congestive)
- With or without signs of infective endocarditis
- With or without signs of active rheumatic carditis
- Patient is in NYHA (New York Heart Association) class (I/II/III/IV)

Example: Acquired valvular heart disease, possibly rheumatic etiology, with severe mitral stenosis and moderate mitral regurgitation, with severe pulmonary artery hypertension, patient in atrial fibrillation and congestive cardiac failure, with no signs of infective endocarditis, thromboembolism or active rheumatic carditis. Patient is in NYHA class III.

For Congenital Heart Disease

- Congenital cyanotic/acyanotic heart disease
- Type of defect (shunt/obstructive)
- With/without evidence of pulmonary artery hypertension (grading)
- Patient in or not in atrial fibrillation (if AF present look for signs of thromboembolism)
- With or without evidence of heart failure (right/left/congestive)
- With or without signs of infective endocarditis
- Patient is in NYHA class (I/II/III/IV).

Note: Mention if any features of dysmorphies or syndromes.

Example: Congenital acyanotic heart disease, atrial septal defect with pulmonary artery hypertension, with no evidence of eisenmengerisation, patient not in atrial fibrillation, no evidence of heart failure or infective endocarditis. Patient in NYHA class II. Patient has features of Holt–Oram syndrome.

NOTES

C. DISCUSSION ON CARDIAC CYCLE

SYSTOLE AND DIASTOLE

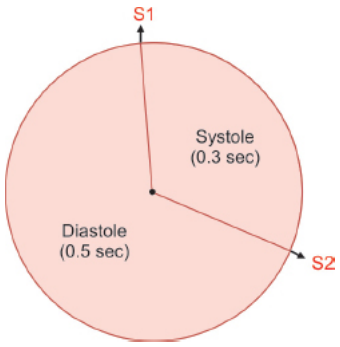


Fig. 4C.1: Systole and diastole.

In **Figure 4C.1**, cardiac cycle is represented as cyclical events beginning from S1 and ending back at S1 in clockwise fashion. Assuming the heart rate of 72 beats/min, each cardiac cycle is of 0.8 seconds duration. 0.3 seconds is ventricular systole and 0.5 seconds is ventricular diastole. Systole is represented by S1 to S2 in clockwise direction and diastole is represented by S2 to S1 in clockwise direction. And these events continuously repeat.

EVENTS OF CARDIAC CYCLE

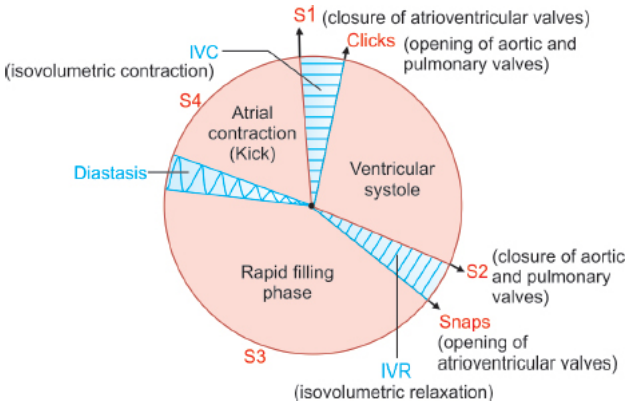
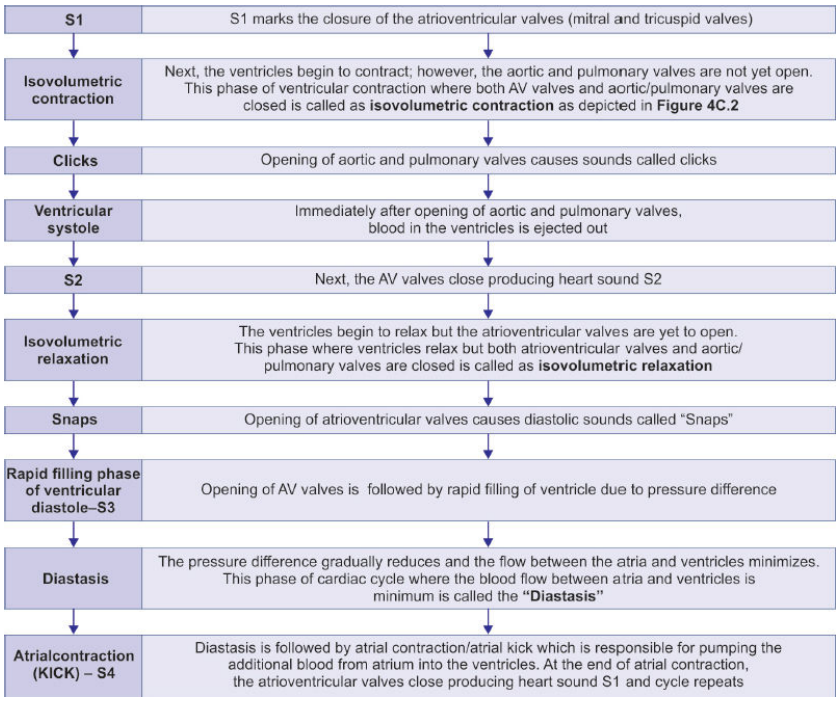


Fig. 4C.2: Major events during cardiac cycle.

Let us describe the cardiac events in clockwise fashion beginning from S1



Jugular Venous Pressure Waveform—timing with Other Cardiac Events

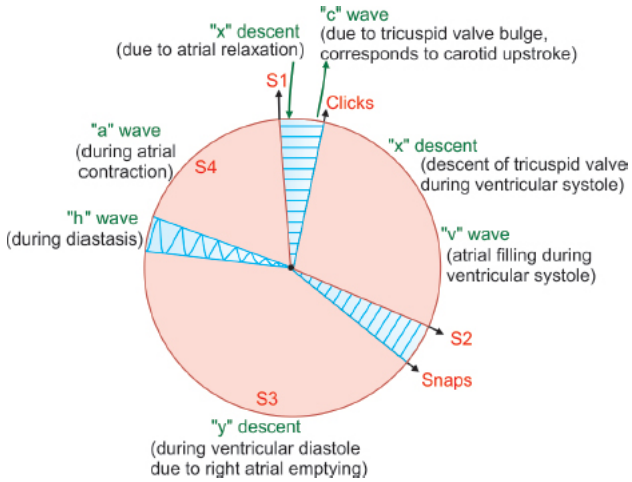


Fig. 4C.3: Timing of JVP with cardiac events.

Now, let us superimpose waves of jugular venous pressure (JVP) onto the cardiac cycle. JVP has the following waves, starting from a, x, c, x', v, y, and h which repeat in a cyclical fashion. Clinically appreciable waves are four, two in systole (i.e. "x" descent and "v" wave) and two in diastole (i.e. "y" descent and "a" wave). The timing of JVP with respect to cardiac cycle has been depicted in **Figure 4C.3**. The waves in JVP include:

"a" wave	• It coincides with atrial contraction • It is seen in diastole and • It precedes S1
X wave (initial x descent)	• It is due to atrial relaxation • It is seen in systole • It follows S1

C wave	• It is due to bulge of tricuspid valve • It is seen in systole • Coincides with carotid upstroke • Absent in humans
X' wave (x descent following 'c' wave)	• It is due to descent in floor of RA with downward pull of TV with continued ventricular contraction • It is seen in systole • It follows clicks (if audible)
V wave	• It is due to atrial filling during ventricular systole • Seen in systole • It precedes S2
Y wave	• It is due to RA emptying during ventricular diastole • Seen in diastole • It follows opening snaps (if audible)
H wave (Hirschfelder wave)	• It is positive wave during the diastasis • Seen in diastole • Not clinically appreciable

CARDIAC MURMURS—TIMING WITH OTHER CARDIAC EVENTS (FIG. 4C.4)

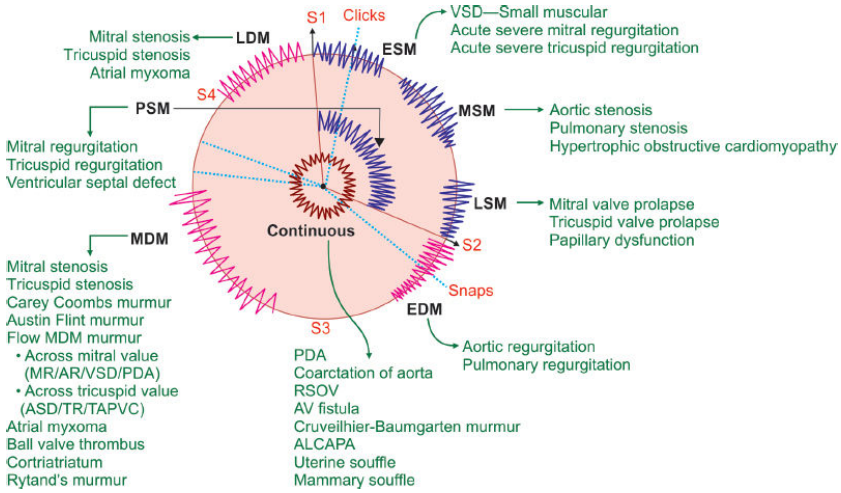


Fig. 4C.4: Timing of cardiac murmurs and pictorial representation on the diagram of cardiac cycle.

To remember murmurs:

Note 1: **ESM/PSM—**due to valve abnormalities of mitral and tricuspid valve (regurgitant lesions); **MSM—** due to valve abnormalities of aortic and pulmonary valve (stenotic lesions); **LSM—**due to prolapse of mitral and tricuspid valve; **EDM—**due to valve abnormalities of aortic and pulmonary valve (regurgitant lesions); **MDM—**due to valve abnormalities of mitral and tricuspid valve; **LSM—**atrial myxomas.

Note 2: **Early murmurs** are regurgitant lesions; **Mid murmurs** are stenotic lesions; **Late murmurs** are prolapse/papillary dysfunction/myxomas

ECG WAVEFORM—TIMING WITH OTHER CARDIAC EVENTS (FIG. 4C.5)

- Atrial contraction follows the **P wave** of the ECG.
- Isovolumetric contraction and systole follows the **QRS wave** of the ECG.
- Diastole follows the **T wave** of ECG.

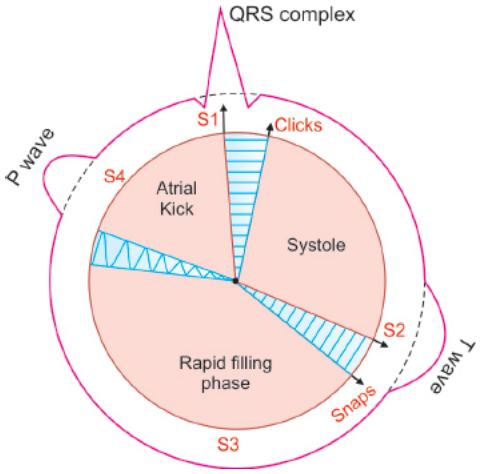


Fig. 4C.5: Timing of waves of ECG and pictorial representation on the diagram of cardiac cycle.

STANDARD REPRESENTATION OF ALL CARDIAC EVENTS IN CARDIAC CYCLE (FIG. 4C.6 AND TABLE 4C.1)

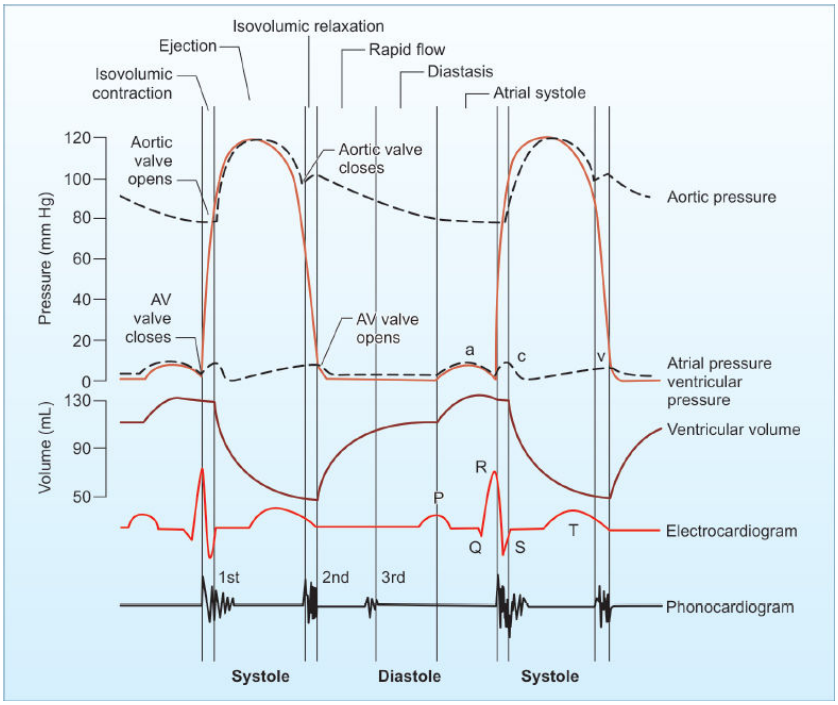


Fig. 4C.6: Events of cardiac cycle during systole and diastole (phonogram, electrocardiogram, volumes and pressure changes).

Table 4C.1: Pressure changes during cardiac cycle.			
Pressures (mm Hg)			
Right atrium		***Left atrium***	
Mean	2	**Mean**	8

		a wave	13
		c wave	12
		v wave	15
Right ventricle		***Left ventricle***	
Peak systolic	30	**Peak systolic**	130
End-diastolic	6	**End-diastolic**	10
Pulmonary artery		***Aorta***	
Mean	15	**Mean**	95
Peak systolic	25	**Peak systolic**	130
End-diastolic	8	**End-diastolic**	80
Pulmonary capillaries		***Systemic capillaries***	
Mean	10	**Mean**	25

NOTES

D. DISCUSSION ON CARDINAL SYMPTOMS

CHEST PAIN

Chest pain is a common symptom of cardiac disease. It can be due to noncardiac causes such as anxiety or diseases involving the respiratory, musculoskeletal or gastrointestinal systems.

Causes of Chest Pain (Fig. 4D.1)

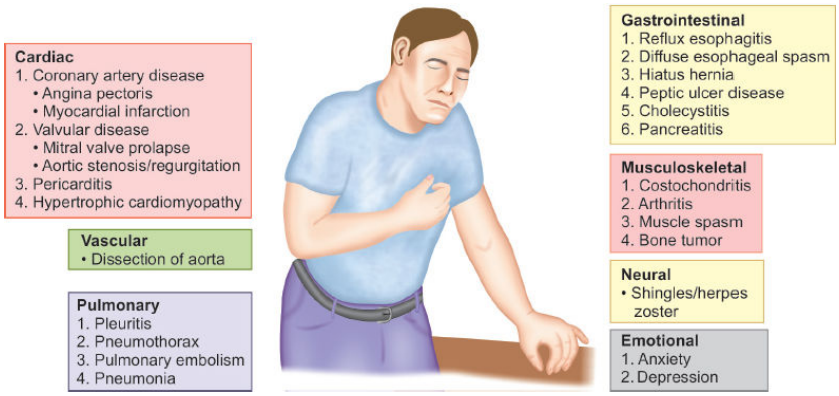


Fig. 4D.1: Causes of chest pain.

Differential Diagnosis of Chest Pain (Table 4D.1)

Table 4D.1: Differential diagnosis of chest pain.

Potentially life-threatening causes	*Common nonlife-threatening causes*
• Acute coronary syndromes: Acute myocardial infarction (MI), ST-segment elevation MI, non-ST-segment elevation MI • Unstable angina • Pulmonary embolism • Aortic dissection • Myocarditis (most common cause of sudden death in the young) • Tension pneumothorax • Acute chest syndrome/crisis in sickle cell anemia • Pericarditis • Boerhaave's syndrome (perforated esophagus) • Gastrointestinal: Perforated peptic ulcer, acute pancreatitis, acute cholecystitis	• Gastrointestinal – Biliary colic – Gastroesophageal reflux disease – Peptic ulcer disease • Pulmonary – Pneumonia – Pleuritis • Musculoskeletal pain: Costochondritis (Tietze's syndrome), intercostal myalgia/neuralgia, fracture of the ribs (cough, trauma), secondaries in the ribs, Bornholm disease • Thoracic radiculopathy: Texidor's twinge (precordial catch syndrome) • Emotional: Anxiety • Neural: Shingles/herpes zoster

Differential Features of Ischemic Cardiac and Noncardiac Pain (Table 4D.2)

Table 4D.2: Differential features of ischemic cardiac and noncardiac pain.

Features	*Ischemic cardiac pain*	*Noncardiac pain*
Site	Central, diffuse	Peripheral, localized
Character of pain	Tight, squeezing, dull, constricting, choking or 'heavy'	Sharp, stabbing, catching
Precipitation/provocation	Exertion, emotion	Spontaneous, not related to exertion

Radiation	Jaw/neck/shoulder	Usually no radiation
Relieving factors	Rest (in less than 5 minutes), nitrates	Not relieved by rest or by nitrates
Associated features	Breathlessness, diaphoresis	Depends on the cause

Differentiating Features of the Common Causes of Chest Pain (Table 4D.3)

Table 4D.3: Differentiating features of the common causes of chest pain.

Disease	*Description*	*Location*	*Radiation*	*Associations*
Acute coronary syndromes	Crushing, tightening, squeezing, or pressure like	Retrosternal, left anterior chest or epigastric	Right (R) or left (L) shoulder, R or L arm/hand/jaw	Dyspnea, diaphoresis, nausea
Pulmonary embolism	Heaviness, tightness	Whole chest (massive) or focal chest (segmental)	None	Dyspnea, unstable vital signs, feeling of impending doom if massive or just tachycardia, tachypnea if segmental
Aortic dissection	Ripping, tearing	Midline, substernal	Interscapular area of back	Secondary arterial branch occlusion (paraplegia)
Pericarditis/cardiac tamponade	Sharp, constant or pleuritic	Substernal	None	Fever, dyspnea, pericardial friction rub
Pneumothorax	Sudden, sharp, lancinating, pleuritic	One side of chest	Shoulder, back	Dyspnea
Perforated esophagus	Sudden, sharp, after forceful vomiting	Substernal	Back	Dyspnea, diaphoresis, signs of sepsis

Types of angina	
Angina	Angina is a symptom of myocardial ischemia that is recognized clinically by its character, its location and its relation to provocative stimuli
Stable angina	Angina is stable when it is not a new symptom and when there is no deterioration in frequency, severity or duration of episodes
Unstable angina	This is a form of acute coronary syndrome. It has at least one of these three features: 1. It occurs at rest (or with minimal exertion), usually lasting more than 10 minutes 2. It is severe and of new onset (i.e. within the prior 4–6 weeks) 3. It occurs with a crescendo pattern (i.e. distinctly more severe, prolonged, or frequent than before)
Variant angina/prinzmetal angina	Caused due to coronary vasospasm
Microvascular angina/cardiac syndrome X	Angina-like chest pain in the context of normal epicardial coronary arteries on angiography
Episodic angina	This syndrome is one in which pains having the characters of angina of effort occur at longer or shorter intervals
Nocturnal angina	Seen in severe aortic regurgitation (AR) Proposed mechanisms are: 1. Prolonged diastole at night: Regurgitation time is prolonged 2. Dilated left ventricular (LV), increased LV mass, increased demand 3. Diastolic coronary stealing, Venturi effect of AR jet
Angina decubitus	It is angina that occurs when a person is lying down (not necessarily only at night) without any apparent cause. Occurs because gravity redistributes fluids in the body
Second wind, or warm up, angina	Describes patients with ischemic heart disease and exertional angina that forces them to stop; after the first bout of angina, they are able to continue with minor, or even without any, further symptoms ischemic

	preconditioning and collateral recruitment are proposed mechanisms
Linked angina	It is associated with: 1. Gastroesophageal and duodenal disorders and diseases 2. Gallbladder disease 3. Cervical spondylitis
Refractory angina	Angina that cannot be controlled with optimal medical therapy and where revascularization is not feasible
Status anginosus	It is a clinical term denoting periods of frequently recurring anginal pain at rest, indistinguishable from the pain of cardiac infarction or from its prodromal manifestation, but without the electrocardiographic and laboratory evidences of classical cardiac infarction
Vincent's angina	Fusospirochetal infection of the pharynx and palatine tonsils, causing "ulceromembranous pharyngitis and tonsillitis"
Ludwig's angina	Severe diffuse cellulitis that presents as an acute onset and spreads rapidly, bilaterally affecting the submandibular, sublingual, and submental spaces
Abdominal angina	Postprandial pain that occurs in the mesenteric vascular occlusive disease
Angina sine dolore	A painless episode of coronary insufficiency. It is associated with diabetes mellitus and also called silent ischemia

Canadian cardiovascular society (CSS) functional classification of angina	
Class I	Ordinary activity (e.g. walking, climbing stairs at own pace) does not bring on angina. Angina occurs only with strenuous, rapid, or prolonged exertion at work or during recreation
Class II	Slight limitation of ordinary activity. Symptoms occur when walking or climbing stairs rapidly, walking up a hill, walking up stairs after a meal, in cold weather, in wind, or when under emotional stress, or only a few hours after waking, and climbing more than one flight of ordinary stairs at a normal pace and in normal conditions
Class III	Marked limitation of ordinary activity. Symptoms occur after walking 50–100 yards on the level, or climbing more than one flight of ordinary stairs in normal conditions
Class IV	Inability to carry on any physical activity without discomfort. Angina may be present at rest

Angina Equivalents

These are commonly seen in elderly and diabetics (with autonomic neuropathy) where ischemic angina is absent and they present with:

- Shortness of breath
- Perspiration/diaphoresis
- Syncope
- Gastrointestinal (GI) symptoms—upper abdominal pain, nausea, and vomiting
- Fatigue
- Confusion.

PALPITATIONS

Definition

Palpitation is the term used to describe an uncomfortable increased awareness of one's own heartbeat or the sensation of slow, rapid or irregular heart rhythms.

- Palpitations do not always indicate the presence of arrhythmia and conversely, an arrhythmia can occur without palpitations.
- Palpitations are usually noted when the patient is quietly resting.
- Palpitation can be either intermittent or sustained and either regular or irregular.
- A change in the rate, rhythm or force of contraction can produce palpitations.

Causes of Palpitations (Table 4D.4)

Table 4D.4: Causes of palpitations.	
Cardiac causes • **Cardiac arrhythmias** – Premature atrial and ventricular contractions – Supraventricular and ventricular arrhythmias • **Structural heart diseases** – Atrial myxoma, valvular heart disease – Congenital heart disease, cardiomyopathy – Mitral valve prolapse, pacemaker	**Drug induced** • Alcohol (use or withdrawal) • Atropine • Amphetamines • Caffeine, nicotine • Cocaine • Beta agonists, theophylline
Psychosomatic disorders • Generalized anxiety, major depression, panic disorder	**Endocrine** • Hyperthyroidism, hypoglycemia, pheochromocytoma
High output states • Anemia, beriberi, fever, pregnancy, thyrotoxicosis	**Miscellaneous and idiopathic** • Emotional stress, hyperventilation, premenstrual syndrome, strenuous physical activity

Duration and Frequency of Palpitations

- Duration may be either short-lasting or persistent.
- Note the onset and offset of palpitations.
- Frequency: It may occur daily, weekly, monthly, or yearly.

Types of palpitations	
Extrasystolic palpitations	Ectopic beats, usually produce feelings of “missing/skipping a beat” and/or a “sinking of the heart” interspersed with periods during which the heart beats normally. Patients report that the heart seems to stop and then start again. It can often even be seen in young individuals, usually without any disease of the heart, and generally benign
Tachycardiac palpitations	These are the rapid fluctuation like “beating wings” in the chest. It may be regular (e.g. in atrioventricular tachycardia, atrial flutter, or ventricular tachycardia) or irregular or arrhythmic (e.g. in atrial fibrillation)
Anxiety-related palpitations	They are usually associated with anxiety epidsodes. They begin and end gradually

Associated Symptoms and Circumstances

- Palpitations developing after sudden changes in posture are usually due to orthostatic intolerance or to episodes of atrioventricular nodal re-entrant tachycardia.
- Occurrence of syncope or other symptoms, such as severe fatigue, dyspnea, or angina, in addition to palpitations, is more common with structural heart disease.
- Hypersecretion of natriuretic hormone results in polyuria/postpalpitation diuresis in atrial fibrillation.
- Palpitations associated with anxiety or during panic attacks are usually due to sinus tachycardia secondary to the mental disturbance.
- Palpitations may be produced by an increase in the sympathetic drive during physical exercise.

Typical descriptions of palpitations	
Flip-flopping in the chest	Palpitations are sensed as the heart seeming to stop and then start again, producing a pounding or flip-flopping sensation. This type of palpitation is generally caused by supraventricular or ventricular premature contractions.
Rapid fluttering	It is due to a sustained ventricular or supraventricular arrhythmia, including sinus tachycardia

in the chest	
Pounding in the neck	An irregular pounding feeling in the neck is caused by atrioventricular dissociation, with independent contraction of the atria and ventricles, resulting in occasional atrial contraction against a closed tricuspid and mitral valve. This produces cannon A waves, which are intermittent increases in the "A" wave of the jugular venous pulse. **Cannon A** waves may be seen with ventricular premature contractions, third degree or complete heart block, or ventricular tachycardia (VT)

DYSPNEA

Discussed in detail in section of symptomatology, Chapter 2C.

SYNCOPE

Definition

Syncope is defined as a transient loss of consciousness due to inadequate cerebral blood flow with loss of postural tone. It is associated with spontaneous return to baseline neurologic function without any resuscitative efforts.

- **Presyncope** is the term used for lightheadedness in which the individual thinks he/she may black out.
- **Classical vasovagal syncope:** Syncope triggered by emotional or orthostatic stress such as venipuncture (experienced or witnessed), painful or noxious stimuli, fear of bodily injury, prolonged standing, heat exposure, or exertion.

Mechanism

- Global hypoperfusion of cerebral cortices or focal hypoperfusion of the reticular activating system.
- About one-third of individuals may develop a syncopal episode during their lifetime.
- Its incidence increases with age (sharp rise at age 70 years).
- Cardiac syncope has a high incidence (about 24%) of subsequent cardiac arrest.

Causes of True Syncope (Table 4D.5)

Table 4D.5: Causes of true syncope.

Cardiac causes	*Noncardiac causes*
• **Cardiac arrhythmias:** Ventricular tachycardia, paroxysmal supraventricular tachycardia, long QT syndrome, Brugada syndrome, bradycardia (Mobitz type II or 3rd degree heart block) • **Structural cardiac or cardiopulmonary disease:** Cardiac valvular disease (AS, MS, PS), obstructive cardiomyopathy, atrial myxoma, acute aortic dissection, pericardial disease/tamponade, pulmonary embolus/pulmonary hypertension, acute myocardial infarction/ischemia	• **Neurocardiogenic syncope 'vasovagal or vasodepressor syncope':** Classical vasovagal syncope, situational syncope, carotid sinus syncope, glossopharyngeal neuralgia, micturation syncope • **Orthostatic hypotension:** Autonomic failure which may be primary (e.g. pure autonomic failure, multiple system atrophy, Parkinson's disease with autonomic failure) or secondary (e.g. diabetic neuropathy) • **Neurovascular syncope:** Vascular steal syndromes

Causes of Pseudosyncope (Box 4D.1)

Box 4D.1: Causes of pseudosyncope.

- **Seizures**.
- **Metabolic or toxic abnormalities:** Hypoglycemia and encephalitis.

- **Neurologic syncope:** Subarachnoid hemorrhage, transient ischemic attack, complex migraine headache.
- **Psychogenic syncope**
- **Drug induced loss of consciousness:** Drugs of abuse and alcohol.

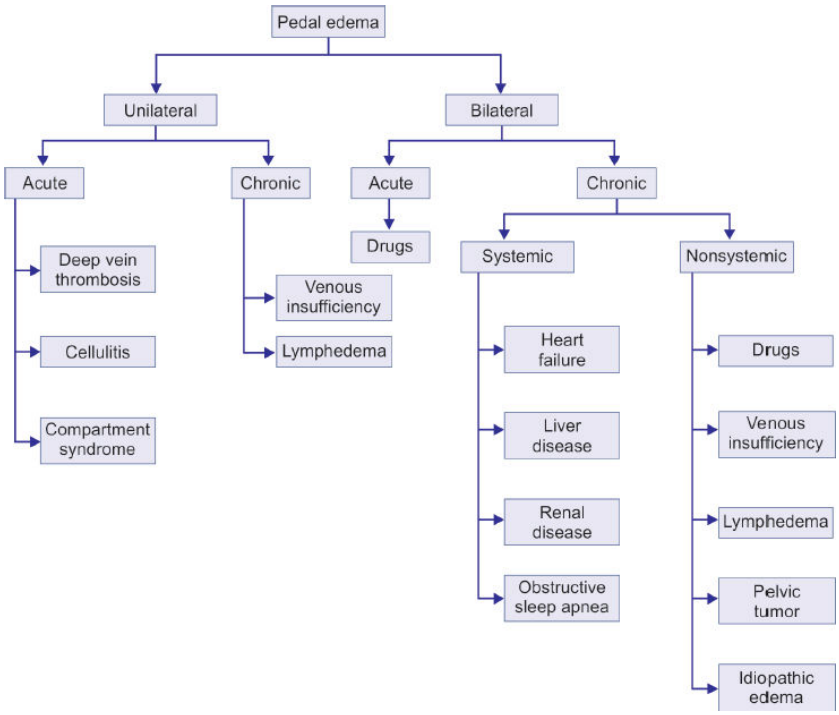
PEDAL EDEMA

Definition

Edema is defined as the abnormal fluid accumulation in the interstitial space that exceeds the capacity of physiological lymphatic drainage. Pedal edema is a common presentation of various systemic and nonsystemic diseases.

Approach to pedal edema (Flowchart 4D.1)	
Site and distribution	Whether the pedal edema is unilateral or bilateral • Unilateral edema results mainly due to local causes like deep vein thrombosis (DVT), cellulitis, compartment syndrome, and filarial lymphatic obstruction • Bilateral pedal edema is mainly due to systemic causes like congestive cardiac failure, anemia, chronic kidney disease, and chronic liver disease
Duration of illness	• Short duration of the illness indicates an acute cause like cellulitis, DVT, compartment syndrome, etc. which usually occurs in 72 hours
Association with pain	• **Painless:** Edema due to feart failure, hypoproteinemia, and lymphedema • **Painful:** Deep vein thrombosis and cellulitis. *A dull aching type of pain is seen in chronic venous insufficiency*
Variability of edema	• Venous edema due to congestive cardiac failure and venous insufficiency is aggravated by standing and improves with overnight limb elevation during sleep • ***Idiopathic edema*** which is seen in females and increases throughout the day due to upright posture
History of systemic illness	• Symptoms of systemic diseases like exertional dyspnea, orthopnea, paroxysmal nocturnal dyspnea, and chest pain point to cardiac failure • History of oliguria and puffiness of face suggest renal etiology • Long-term alcohol consumption, yellowish discoloration of eyes and urine, and abdominal distension points to cirrhosis of liver • Symptoms of endocrine disorders like hypothyroidism are often missed • Similar history about all other systemic causes of pedal edema should be elicited in detail • Patients who are bed ridden for a prolonged period of time have dependent edema over the sacral area
History of drug intake	Drugs like calcium channel blockers, nonsteroidal anti-inflammatory drugs (NSAIDs) and steroids
History of trauma and radiation	Trauma and radiation can cause cellulitis and compartment syndrome leading to pedal edema
Miscellaneous causes	Obstructive sleep apnea can also cause pedal edema due to right ventricular failure

Flowchart 4D.1: Algorithm for approach to pedal edema.



Other Symptoms

- **Symptoms of low cardiac output**: Fatigue, dizziness, and syncope
- **Symptoms of pulmonary hypertension:** Exertional fatigue, exertional chest pain, and exertional dyspnea
- **Fever:** Rheumatic fever and infective endocarditis
- **Symptoms of heart failure**: Fatigue, anorexia, weight gain, leg swelling, exertional fatigue, decreased urine output, perspiration, confusion, cough, hemoptysis, and wheezing.

NOTE

E. DISCUSSION ON EXAMINATION

GENERAL EXAMINATION

Vitals

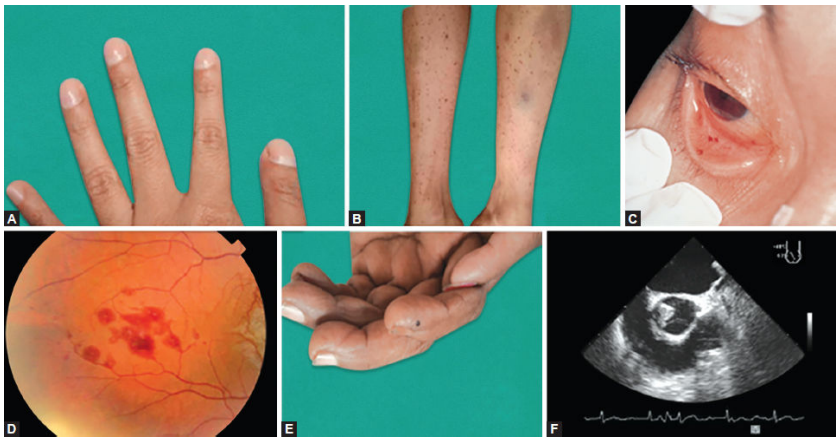
Pulse, blood pressure and jugular venous pressure:
(Discussed in detail in chapter 2B).

Anthropometry:
(Discussed in the chapter 2D).

Physical Examination

Signs of infective endocarditis (Figs. 4E.1A to F):
- Fever
- Pallor
- Clubbing
- Splinter hemorrhages
- Mucosal petechiae
- Janeway lesions
- Osler's nodes
- Roth spots on fundus.

Signs of rheumatic fever:
- Fever
- Arthritis
- Erythema marginatum
- Subcutaneous nodules
- Tachycardia.



Figs. 4E.1A to F: Signs of infective endocarditis: (A) Clubbing; (B) Petechiae; (C) Subconjunctival hemorrhage; (D) Roth spots; (E) Osler's nodes; (F) Echocardiography showing vegetation.

Stigmata of congenital heart disease

Syndrome	Cardiac defects	Other features
Down syndrome (trisomy 21) (CHILD HAS MANY PROBLEM)	ECD, VSD	• **C**ataract • **H**ypotonia • **H**ypothyroidism • **I**ncreased gap between 1st and 2nd toe (sandal gap) • **L**eukemia • **D**uodenal atresia • **H**irschsprung's disease • **A**lzheimer's disease • **S**imian crease • **M**ental retardation • **M**icrognathia • **A**tlantoaxial instability • **Ny**stagmus • **P**rotruding tongue • **P**oor hearing • **R**ound face • **R**espiratory infections • **O**cciput is flat • **O**blique palpebral fissure • **B**rushfield spots • **B**rachycephaly • **L**ow nasal bridge • **L**anguage problem • **E**picanthic fold • **E**ar folded • **M**ongolian slant • **M**yoclonus
Marfan syndrome	Aortic aneurysm, aortic and/or mitral regurgitation	Arachnodactyly with hyperextensibility, subluxation of lens and other joint deformities
William's syndrome	• Supravalvular AS • PA stenosis	Varying degrees of mental retardation, so-called elfin facies (consisting of some of the following: upturned nose, flat nasal bridge, long philtrum, flat malar area, wide mouth, full lips, widely spaced teeth, periorbital fullness), hypercalcemia of infancy
Rubella syndrome	• PDA and pulmonary stenosis	**Triad of the syndrome:** Deafness, cataract, and CHDs Others include: Intrauterine growth retardation, microcephaly, microphthalmia, hepatitis, neonatal thrombocytopenic purpura
Noonan's syndrome (Turner-like syndrome)	PS (dystrophic pulmonary valve), LVH (or anterior septal hypertrophy)	Similar to Turner's syndrome but may occur in phenotypic male and without chromosomal abnormality
LEOPARD syndrome (multiple lentigines syndrome)	PS, HOCM, long PR interval	Lentiginous skin lesion, ocular hypertelorism, pulmonary stenosis, abnormal genitalia, retarded growth, deafness
Holt-Oram syndrome (cardiac-limb syndrome)	ASD, VSD	Defects or absence of thumb or radius
Ellis–van Creveld syndrome (chondroectodermal dysplasia)	ASD, single atrium	Short stature of prenatal onset, short distal extremities, narrow thorax with short ribs, polydactyly, nail hypoplasia, neonatal teeth
DiGeorge syndrome	Interrupted aortic arch, truncus arteriosus, VSD, PDA, TOF	Hypertelorism, short philtrum, down slanting eyes, hypoplasia or absence of thymus and parathyroid, hypocalcemia, deficient cell-mediated immunity
Cornelia de Lange's (de Lange's)	VSD	Hirsutism, prenatal growth retardation, microcephaly, anteverted nares, downturned mouth, mental retardation

syndrome		
CHARGE syndrome	TOF, truncus arteriosus, aortic arch anomalies (e.g. vascular ring, interrupted aortic arch)	Coloboma, choanal atresia, growth or mental retardation, genitourinary anomalies, ear anomalies, genital hypoplasia

(AS: aortic stenosis; ASD: atrial septal defect; ECD: endocardial cushion defect; HOCM: hypertrophic obstructive cardiomyopathy; LVH: left ventricular hypertrophy; PA: pulmonary artery; PS: pulmonary stenosis; TOF: tetralogy of Fallot; VSD: ventricular septal defect); CHDs: congenital heart diseases; PDA: patent ductus arteriosus)

SYSTEMIC EXAMINATION

All cardiovascular examination has to be simultaneously timed with carotid pulse. Findings synchronous with carotid upstroke is systolic and if it is asynchronous, it is diastolic.

Inspection and Palpation of Heart

Palpation of CVS (Fig. 4E.2)

Tips of fingers	For localizing the pulsations
Metacarpal heads	For appreciating the thrills
Heel of hand	For appreciating the heave

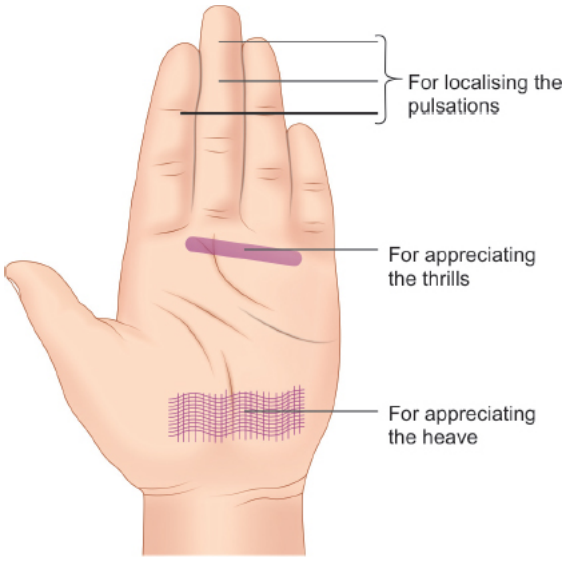


Fig. 4E.2: showing sites of hand for palpation of pulses, thrills and heave.

Chest deformity and associated clinical diseases:

Chest deformity	Associated diseases
Barrel shaped	Chronic obstructive pulmonary disease and cor pulmonale
Broad shield like chest	• Turner syndrome • Noonan syndrome
Pectus carinatum	• Marfan's syndrome • Noonan syndrome

Pectus excavatum	• Marfan's syndrome • Homocystinuria
Straight back syndrome	• Loss of normal kyphosis • Expiratory splitting of S2 • Midsystolic murmur • Prominent pulmonary artery
Male gynecomastia	Digitalis or spironolactone
Female hypomastia	Mitral valve prolapse (MVP)

Topographical Areas of the heart (Fig. 4E.3):

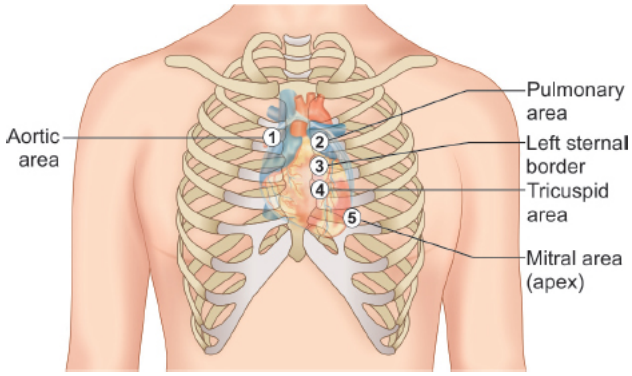


Fig. 4E.3: Illustration showing areas of heart.

Precordial Bulge

- Patient in supine position, stand at the foot end of the bed and look for precordial bulge
- If present, indicates right ventricular dilatation in childhood
- Classically seen only with congenital heart diseases like atrial septal defect (ASD)
- Costal cartilage fuses by 16 years of age, so cardiac diseases which are acquired beyond 16 years may not have a precordial bulge
- Acquired heart disease that can produce precordial bulge is juvenile mitral stenosis.

Causes of precordial bulge:

Cardiovascular causes	
Ribs involved, e.g. cardiac enlargement of long duration	Ribs not involved, e.g. pericardial effusion
Noncardiovascular causes	
• Skeletal deformity • Bronchogenic carcinoma • Mediastinal growth	

Apical Impulse

Definition

It is the outermost and lowermost point of maximum cardiac impulse (PMI) in early systole which imparts a perpendicular gentle thrust to a palpating finger followed by a slight medial retraction in the late systole.

Method of Examination of Apical Impulse

First observe the **position** of apical impulse, then comment on the **character**.

- Patient should be in supine position
- First palpate the apex with the palm (Fig. 4E.4), then localize it with fingertip (Fig. 4E.5)
- Observe the amplitude and duration of the lift of the palpating finger
- If apical impulse is not palpable in supine position, the patient can be put in left lateral position and examination done.

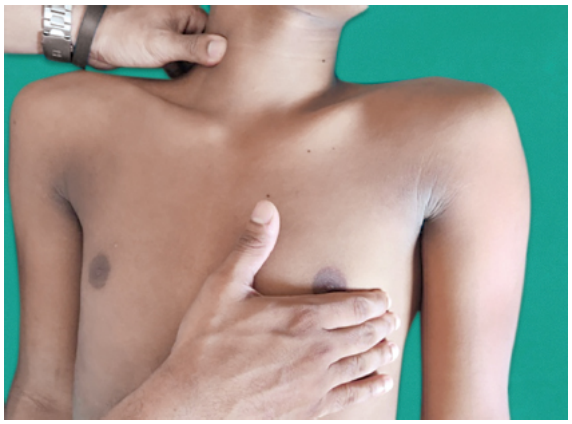


Fig. 4E.4: Palpating the apex with palm flat on the chest.

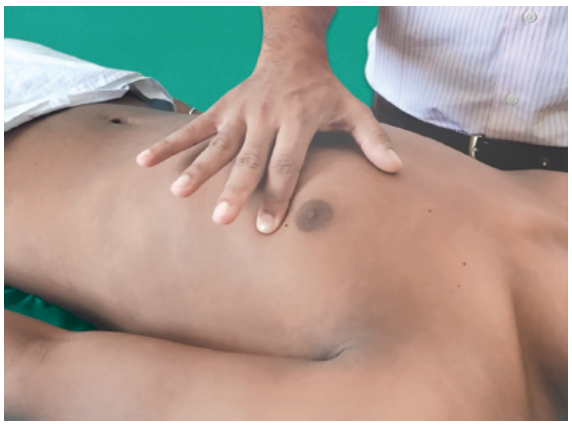


Fig. 4E.5: Localizing the apex with the fingertip.

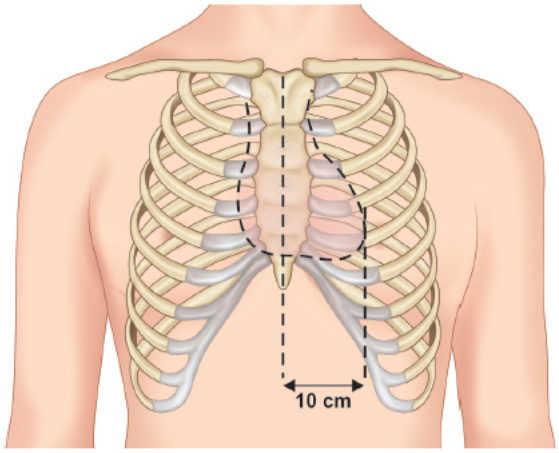


Fig. 4E.6: Location of cardiac impulse.

Features of normal cardiac impulse:

Location	Left 5th ICS, 1–2 cm medial to MCL (or) ≤10 cm from the midsternal line **(Fig. 4E.6)**
Extent	<3 cm diameter or one ICS
Duration	<50% of systole

(ICS: intercostal space; MCL: midclavicular line)

Mechanism of normal apical impulse:

Anterior and counter clockwise rotation of left ventricle (LV) due to isovolumic contraction during early systole and medial retraction due to clockwise rotation of the LV during late systole.

Abnormalities of apex (Fig. 4E.7)	
Absent (Not seen nor felt)	**Cardiovascular causes** • Pericardial effusion • Dextrocardia **Noncardiac causes** • Behind rib • Obesity or thick chest wall • COPD/emphysema • Left sided pleural effusion • Left sided pneumothorax
Tapping	Mitral stenosis (palpable S1—**closing snap**)
Hyperdynamic	• Increased in amplitude • Duration is >1/3–<2/3 of systole • Occupies more than one intercostal space (hence called **diffuse apex**) Occurs in LV **volume overload** conditions **Physiological** • Thin chest • Pectus excavatum • High output states **Pathological** • AR • MR • VSD • PDA • AV fistula

Heaving	• Increase in amplitude • Duration is >2/3 of systole • Confined to one intercostal space Occurs in LV **pressure overload** • AS • Systemic hypertension • HCM • Coarctation of aorta
Double apical impulse	• HOCM • LV aneurysm • LV dyssynergy
Triple or quadruple or wavy impulse	HOCM
Retractile	Severe TR
See-saw apex	LV aneurysm

(AR: aortic regurgitation; AS: aortic stenosis; AV fistula: arteriovenous fistula; COPD: chronic obstructive pulmonary disease; HOCM: hypertrophic obstructive cardiomyopathy; LVH: left ventricular hypertrophy; MR: mitral regurgitation; PDA: patent ductus arteriosus; VSD: ventricular septal defect); LV: left ventiricular; TR: tricuspid regrurgitation)

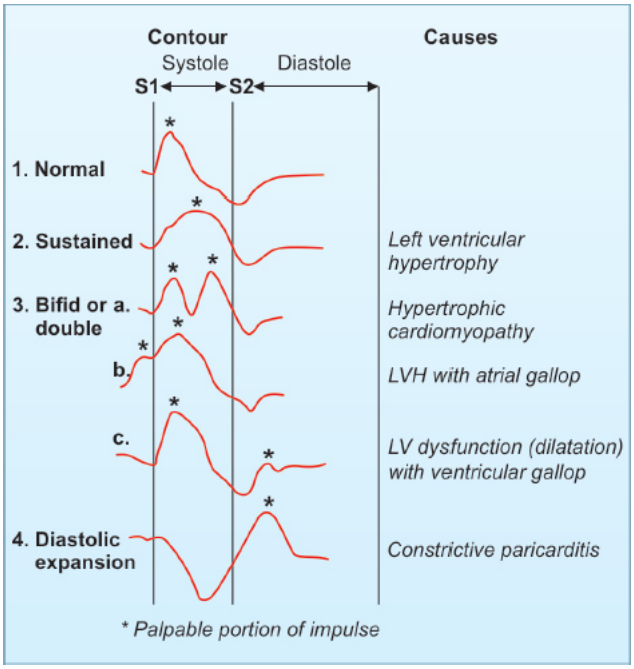


Fig. 4E.7: Apicogram showing different types of cardiac apex.

Which Ventricle is Causing the Apical Impulse?

- The heart during systole, becoming smaller, generally withdraws from the chest wall except for the apex. The effect of this withdrawal on the chest wall can be observed as an inward movement of the chest wall during systole called "**Retraction**".
- The presence of lateral retraction identifies the apical impulse to be formed by the right ventricle, which is an abnormal state.
- A wide area apex beat with medial retraction implies left ventricular enlargement.

Right ventricular (RV) apex vs. left ventricular (LV) apex:

RV apex	*LV apex*
Apex rotated and shifted laterally	Apex may be shifted down and out
Lateral retraction	Medial retraction

Note: In adhesive pericarditis/constrictive pericarditis—systolic retraction of the apex followed by diastolic expansion is—**Skoda's sign.**

Displacement of apex	
Upward displacement	• Children • Ascites • Abdominal tumor • Pericardial effusion
Downward displacement	• Mediastinal growth • Aortic aneurysm
Lateral displacement	If trachea is also shifted along with the displacement of apex beat, then it is due to mediastinal shift as a result of conditions such as lung fibrosis, collapse, pneumothorax or skeletal abnormalities If the trachea is central but the apex is displaced, the causes may be: • **Left ventricular enlargement**: The apex will be displaced *downwards and laterally.* • **Right ventricular enlargement**: The apex will displaced *laterally*

Left Parasternal (LPS) Pulsation/heave

- Produced either by right ventricle (RV) or left atrium (LA).
- Normally RV activity is neither visible nor palpable.

Examination of LPS Area

- Heel of hand with wrist cocked up **(Fig. 4E.8)** or ulnar border of hand is applied over 3/4/5 ICS in left sternal margin **(Fig. 4E.9)** and felt for the pulsations.
- In children or thin patients, parasternal heave can be demonstrated by placing a pen over the parasternal area parallel to the sternal margin and watched for the movement of the tip of the pen.
- In case of difficulty in appreciating the parasternal heave from breathing, ask the patient to momentarily hold the breath.

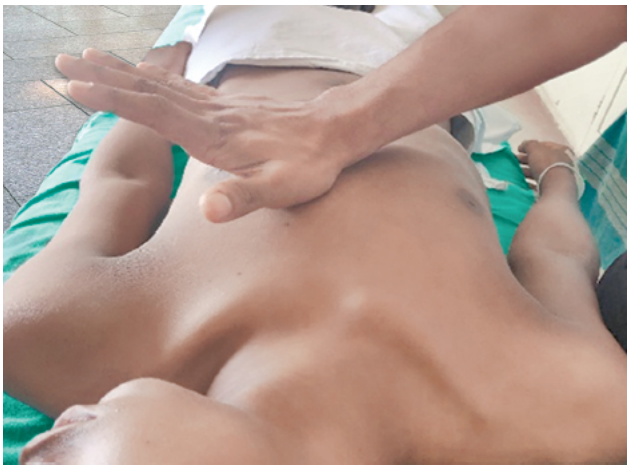


Fig. 4E.8: Examination of parasternal heave (with heel of the hand in cocked up position).

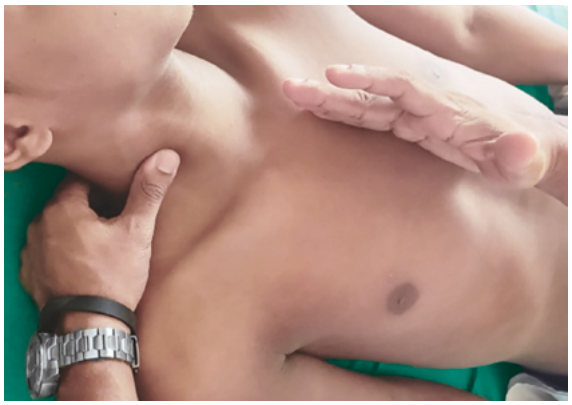


Fig. 4E.9: Examination of parasternal heave (by placing ulnar border).

All India Institute of Medical Science (AIIMS) Grading of Parasternal Heave

Grade I	*Grade II*	*Grade III*
• Visible • Not palpable	• Visible • Palpable • Obliterable	• Visible • Palpable • Not obliterable
Ill-sustained	>50% of systole	Full systole

How to Differentiate RV and LA Parasternal Heave?

RV parasternal heave	*LA parasternal heave*
• Synchronous with apex • Systolic	• Not synchronous with apex • Diastolic

Conditions where LPS pulsations are seen	
Physiological	• Children • Reduced AP diameter
Right ventricular hypertrophy associated	**Pressure overload** • Pulmonary HTN • Pulmonary stenosis **Volume overload** • TR • ASD • VSD
Normal RV	• **Moderate to severe MR** (jet or squid effect)–regurgitant jet of blood into LA pushes the RV anteriorly • **Regional wall motion abnormality (RWMA) of LV**–dyskinetic motion of LV septum pushes RV forwards during the systole

Note:

1. There is no parasternal heave in TOF
2. In MS with MR there is both LAE and RVH, hence very prominent parasternal heave seen

(AP: anteroposterior; ASD: atrial septal defect; HTN: hypertension; LAE: left atrial enlargement; LV: left ventricular; MR: mitral regurgitation; RVH: right ventricular hypertrophy; TR: tricuspid regurgitation; VSD: ventricular septal defect); LA: left atrium; RV: right ventiricular)

Aortic and Pulmonary Pulsations (Base of the Heart)

Examined in sitting and leaning forward position with breath held in expiration (**Erb's maneuver**—described in auscultation section).

Aortic area	*Pulmonary area*
Right 2nd ICS area	Left 2nd ICS area
Visible pulsations	
• Aneurysm of aorta • Chronic AR	• Pulmonary HTN • Pulmonary artery dilatation • Pulmonary artery aneurysm • Hyperdynamic pulmonary artery circulation
Palpable heart sounds	
• A2 (sHTN) • Ejection click (bicuspid aortic valve)	• P2 (pHTN)—**diastolic shock** • Ejection click (pulmonary stenosis)
Palpable murmurs	
• AS • AR (dilated root—AR)	• PS • PDA (Gibsons area—left 1st ICS) • Graham steel murmur

(AR: aortic regurgitation; AS: aortic stenosis; HTN: hypertension; pHTN: pulmonary hypertension; sHTN: systemic hypertension; ICS: intercostal space; PDA: patent ductus arteriosus; PS: pulmonary stenosis)

Sternoclavicular Pulsations

Suprasternal pulsations	• Aneurysm of arch of aorta • Thyroidea ima artery
Right sternoclavicular joint	• Aortic dissection • Aneurysm of aorta • Aortic regurgitation • Right aortic arch • Blalock-Taussig shunt

Epigastric Pulsations

- The subxiphoid region should be palpated by placing the thumb/index finger/palm of the hand over the epigastrium with the fingertip pointing towards the patient's head **(Fig. 4E.10)**.
- Gentle pressure is applied downward (posteriorly) and upward towards the head.
- The patient should be asked to take a deep inspiration in order to move the diaphragm down. This facilitates the palpation of the right ventricle.
- If the impulse were palpable pushing the tip of the thumb/fingertips downward (toward the feet), it would indicate a palpable right ventricular impulse.
- Transmitted abdominal aortic pulsations will cause the impulse to strike the pulp/palmar aspect of the thumb/hand.
- Transmitted hepatic pulsations are felt from the right side onto lateral surface of the examining finger.

Causes of epigastric pulsations	
Cardiac causes	RVH (due to any cause)
Aortic causes	• Thin build • Aneurysm of descending aorta • Aortic regurgitation
Hepatic causes	• Presystolic/diastolic: TS • Systolic: TR

(RVH: right ventricular hypertrophy; TR: tricuspid regurgitation; TS: tricuspid stenosis)

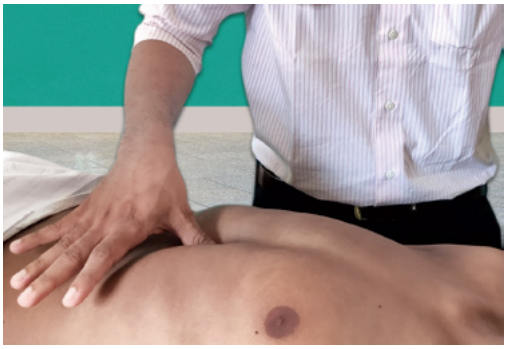


Fig. 4E.10: Demonstration of epigastric pulsations.

Other Pulsations

At back	• **Suzman's sign** in coarctation of aorta • Pulmonary arteriovenous fistula
At neck	• Aortic regurgitation • Carotid aneurysm • Subclavian artery aneurysm

Thrills

- Thrills are palpable murmurs (grade IV or more intensity).
- It is described as purring of the cat.
- Best felt with head of the metacarpal bones.
- Can be systolic, diastolic or continuous.

Area	Timing	Cause
Mitral (apex)	• Systolic	• Severe MR
	• Diastolic	• MS
Left sternal border	• Systolic	• VSD
Pulmonary area	• Systolic	• PS
Aortic area	• Systolic	• AS
	• Diastolic	• Acute severe AR
Left 1st ICS	• Continuous	• PDA or rupture of sinus of Valsalva
Note: As a rule, thrills in the apex of heart are diastolic and thrills in the base of the heart are systolic (exceptions are systolic thrill of acute severe MR and diastolic thrill of acute severe AR).		

(AR: aortic regurgitation; AS: aortic stenosis; ICS: intercostal space; MR: mitral regurgitation; MS: mitral stenosis; PDA: patent ductus arteriosus; PS: pulmonary stenosis; VSD: ventricular septal defect)

Other Sounds Palpable at Apex

Low frequency sounds	
LV S3	• LVF, MR
LV S4 (LVEDP >15–18 mm Hg)	• AS • HCM

	• MR/AR • CAD
Pericardial knock	Constrictive pericarditis
High frequency sounds	
S1	Tapping apex of MS
OS	Early diastolic sound in MS
Ejection systolic click	AS (congenital—bicuspid aortic valve)
Tumor PLOP	LA/RA myxoma
Murmurs (thrills)	
Systolic	• MR • AS • VSD
Diastolic	MS

(AR: aortic regurgitation; AS: aortic stenosis; CAD: coronary artery disease; HCM: hypertrophic cardiomyopathy; LA: left atrial; LV: left ventricular; LVF: left ventricular failure; MR: mitral regurgitation; MS: mitral stenosis; PDA: patent ductus arteriosus; RA: right atrial; VSD: ventricular septal defect).

Other Palpable Sounds in Parasternal Area

Low frequency sounds	
RV S3 (increased flow to ventricles)	• RV failure • Chronic TR • ASD
RV S4 (against increased pressures of ventricle)	• PS • Decreased RV compliance
High frequency sounds	
OS	TS
Murmurs (thrills)	
Systolic	TR
Diastolic	TS

(ASD: atrial septal defect; OS: opening snap; PS: pulmonary stenosis; RV: right ventricular; TR: tricuspid regurgitation; TS: tricuspid stenosis)

Note:

Palpable S1	Tapping apex
Palpable S2	Diastolic shock (palpable P2)
Constrictive pericarditis	Diastolic knock or pericardial knock

Dilated vessels:

1. Dilated veins: caudal flow [superior vena cava (SVC) obstruction]; cranial flow [inferior vena cava (IVC) obstruction]
2. Collaterals are seen with coarctation of the aorta (COA)

For example, **Suzman's sign**—seen in COA where **collaterals** are seen in interscapular and infrascapular region.

Scars (Fig. 4E.11)

Median sternotomy (Generally done when there is need for connecting a heart lung machine)	Coronary artery bypass grafting (CABG)
Lateral thoracotomy	All valve replacement surgeries Patent ductus arteriosus (PDA) surgery scar

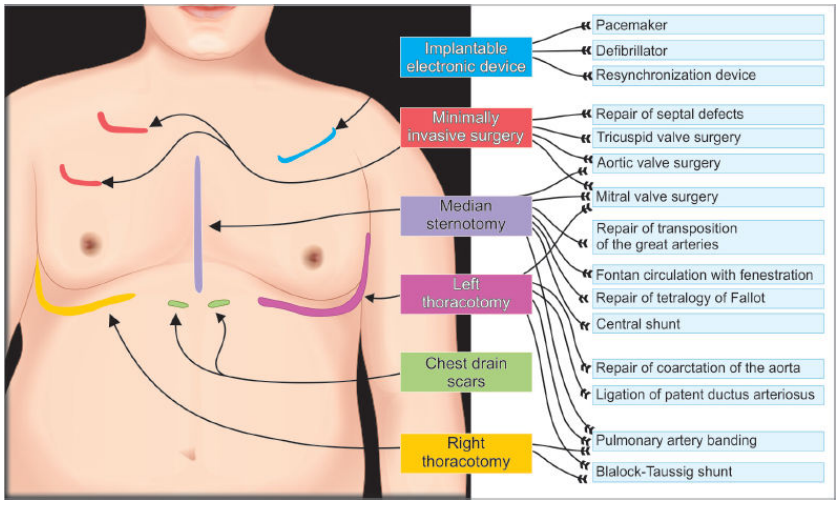


Fig. 4E.11: Image showing different surgical scars for cardiac disease.

Tracheal Tug (Oliver's Sign)

Raise the chin of patient and apply the upward pressure on two sides of cricoid cartilage **(Fig. 4E.12)**.

Positive	Downward pull with each heartbeat	Aortic aneurysm
False positive		Due to mediastinal mass
False negative	Do not move with heartbeat	Thrombosed aortic aneurysm

Percussion

Determination of Heart Border

Right heart border:

- Percuss from above downward in midclavicular line up to the liver dullness **(Fig. 4E.13)**.
- Start percussing one space above the liver dullness **(Fig. 4E.14)**, from the right midclavicular line to the sternum keeping the pleximeter finger parallel to the sternal edge **(Figs. 4E.15A and B)**.
- Repeat this in two more consecutive spaces above.

Fig. 4E.12: Demonstration of Oliver's sign.

Dullness corresponding to right sternal margin	Normal
Dullness outside the right sternal edge	• Pericardial effusion • Dextrocardia • Cardiac enlargement • Right atrial enlargement • Mediastinal mass • Lung pathology

Left heart border:

- Palpate the apex.
- In same ICS go to the midaxillary line and start percussing medially.
- Direction of percussion should be parallel to the apparent left heart border **(Figs. 4E.16A and B)**.

Normally	Corresponds to the apex
Dullness outside apex seen in	• Large pericardial effusion • Left ventricular aneurysm

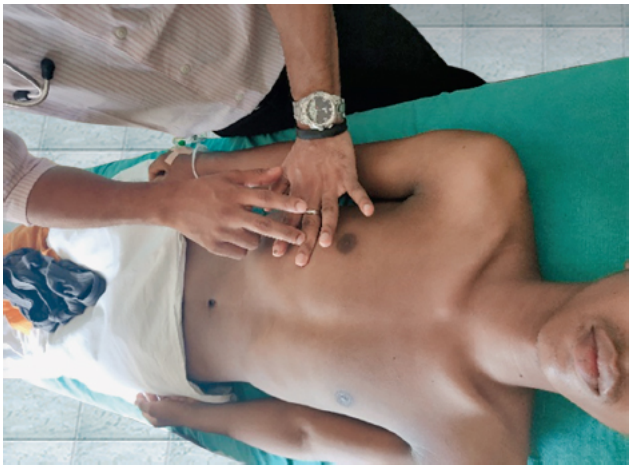


Fig. 4E.13: Percuss from above downward in midclavicular line up to the liver dullness.

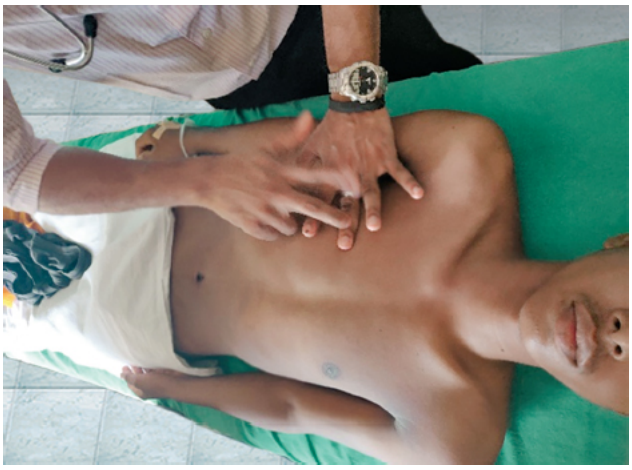


Fig. 4E.14: Now, go one space above the liver dullness.

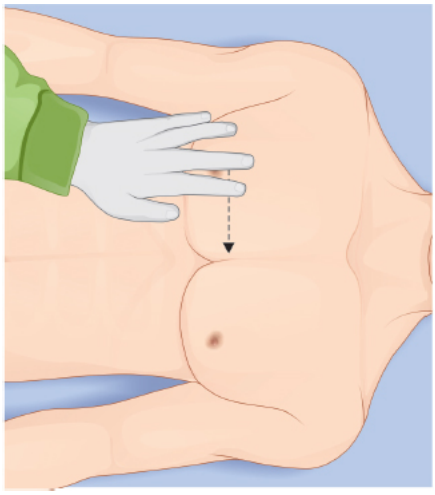


Fig. 4E.15A: Illustration showing direction of percussion of right heart border.

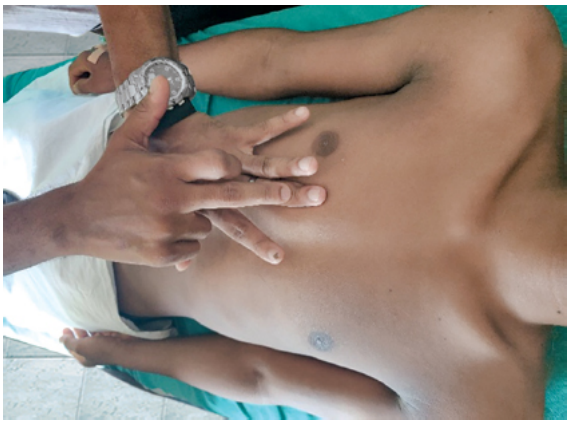


Fig. 4E.15B: Change the direction of percussing finger parallel to heart border and move medially till you get dullness (due to right heart border).

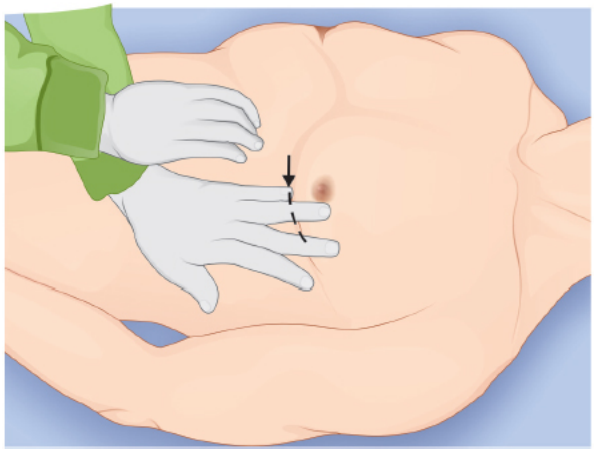


Fig. 4E.16A: Illustration showing direction of percussion of left heart border.

Fig. 4E.16B: Percussion for left heart border from mid axillary line and start percussing medially with percussing finger parallel to the apparent heart border.

Note: Position of pleximeter while percussing the heart border showing should be always parallel to the presumed borders of heart as showed in **Figure 4E.17**.

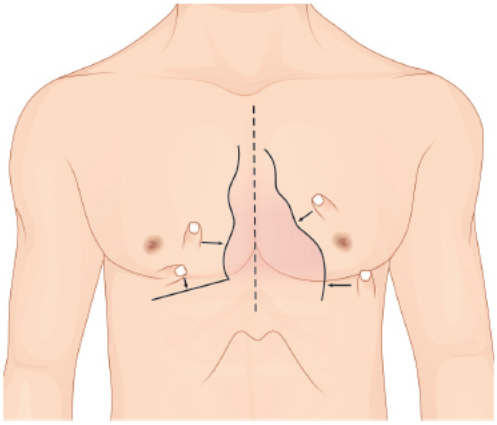


Fig. 4E.17: Illustration showing placement of pleximeter finger during percussion of heart borders.

Percussion of Aortic and Pulmonary Areas

- For aortic area: Start percussing parallel to the right sternal edge and percuss laterally.

- For pulmonary area: Start percussing parallel to the left sternal edge and percuss laterally.
- Normally it is resonant.

Aortic area	*Pulmonary area (Fig. 4E.18)*
Resonant (normal)	Resonant (normal)
Dullness • Dilated aorta • Aortic aneurysm • Superior mediastinal mass	Dullness • Dilated PA • PAH • PDA

(PA: pulmonary artery; PAH: pulmonary arterial hypertension; PDA: patent ductus arteriosus)

Note:
*__Rotch sign__—seen with moderate to large pericardial effusion causing obliteration of cardiohepatic angle.

Fig. 4E.18: Percussion of left 2nd intercostal space.

Auscultation

Hearing of human beings:
- Capability is 20–20,000 Hz
- Sensitivity is 1,000–5,000 Hz

Minimum time gap to differentiate two sounds by human ear is 20 ms.

Characters of cardiac sounds:
- **Loudness:** Implies amplitude or intensity.
- **Pitch:** Implies frequency.

Difference between low and high frequency heart sounds	
Low frequency	***High frequency***
<125 Hz	>300 Hz
Low pitch	***High pitch***
Rough Rumbling	Soft Blowing
For example: S3, S4, pericardial knock MDM (TS/MS)	**For example:** S1, S2, ESC, OS Systolic murmur of (MR, AR)
Better appreciated with **Bell** of stethoscope by applying low	Better appreciated with **Diaphragm** of stethoscope by applying

pressure over the chest piece.	firm pressure over the chest piece

(AR: aortic regurgitation; ESC: early systolic click; OS: opening snap; MDM: mid-diastolic murmur; MR: mitral regurgitation; MS: mitral stenosis; TS: tricuspid stenosis)

Topographical areas of heart (Fig. 4E.19)	
Mitral area	Corresponds to apex (normally in left 5th ICS 1–2 cm medial to mid clavicular line
Tricuspid area	Lower left sternal edge corresponding to 5th ICS
Aortic area	Right 2nd ICS
Neoarotic area (Erb's neo aortic area)	Left 3rd ICS
Pulmonary area	Left 2nd ICS
Other areas	
Axilla	PSM of MR
Epigastrium	PSM of TR
Carotid artery	• Conduction of AS murmur • Carotid bruit
Gibson's area	• Left 1st ICS (PDA)
Roger's area	• Left 4th ICS (VSD)
Interscapular area	• Coarctation of aorta • Aneurysm of descending aorta
Subclavian artery (supraclavicular area)	Bruit over this area heard in aortoarteritis
Femoral artery	Durozier's murmur of AR

(AR: aortic regurgitation; AS: aortic stenosis; ICS: intercostal space; MR: mitral regurgitation; PDA: patent ductus arteriosus; PSM: Pansystolic murmur; TR: tricuspid regurgitation; VSD: ventricular septal defect)

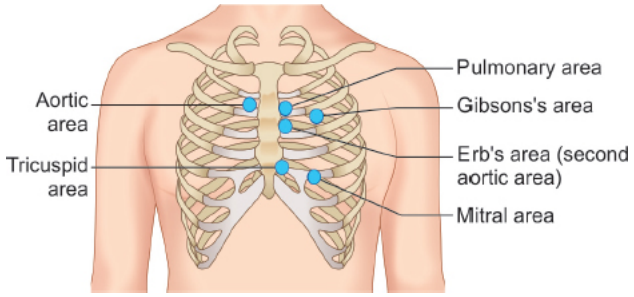
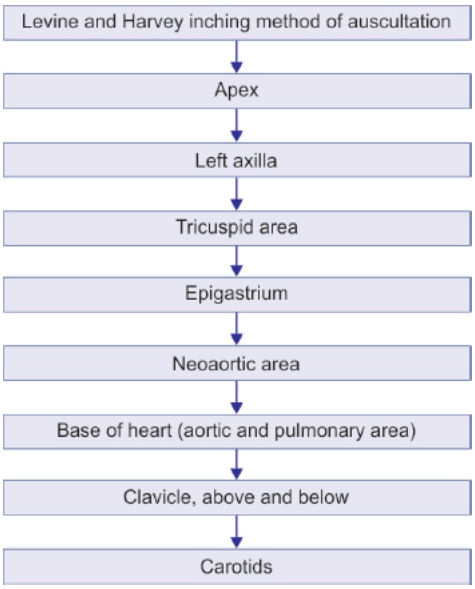


Fig. 4E.19: Illustration of areas of auscultation.

Sequence of Auscultation



Position of patient during auscultation	
Left lateral decubitus	Mitral area
Supine	Tricuspid area
Sitting and leaning forward (Erb's maneuver)	Aortic or pulmonary area

CARDIAC CYCLE AND HEART SOUNDS

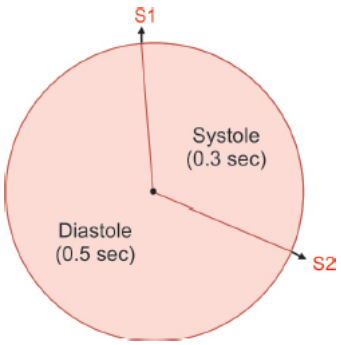


Fig. 4E.20: Cardiac cycle.

Cardiac Cycle Duration (Fig. 4E.20)

Assuming heart rate of 72, each heartbeat is approximately 0.8 seconds in which 0.5 seconds is diastole and 0.3 seconds is systole.

Heart sounds (Figs. 4E.21A and B)	
S1	• Closing of mitral and tricuspid valves • Marks the onset of ventricular systole
S2	Closing of aortic and pulmonary valves
S3	Rapid filling phase of ventricle
S4	Filling of ventricle due to atrial contraction

Others	
Clicks	Systolic sounds are called clicks which can be either ejection click or nonejection clicks
Snaps	Diastolic sounds indicating opening of mitral and tricuspid valves.
Pericardial knock	• Diastolic sounds (early) • Seen in constrictive pericarditis

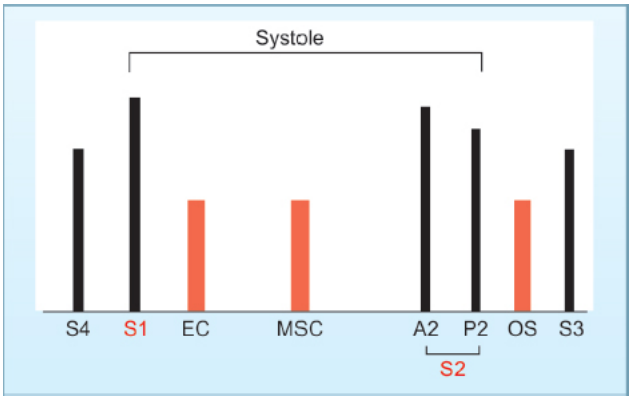


Fig. 4E.21A: Image showing different heart sounds. (EC: ejection click; MSC: mid systolic click; OS: opening snap)

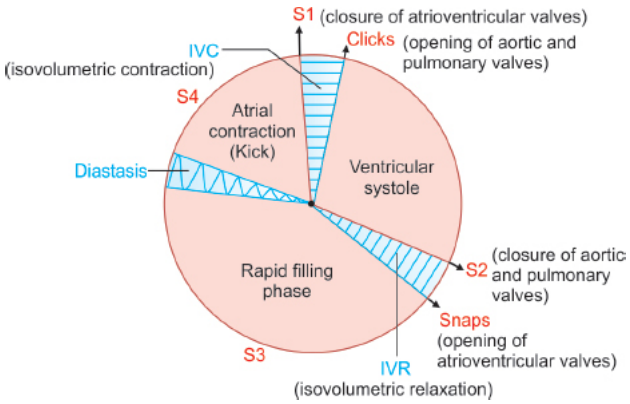


Fig. 4E.21B: Different cardiac events and heart sounds.

Heart Sounds

First Heart Sound (S1)

- Two audible components (M1 and T1)
- Two inaudible components (muscular in origin coinciding with beginning of LV contraction and opening with semilunar valves respectively)
- Order of appearance (1st inaudible component → M1 → T1 → 2nd inaudible component)
- M1–T1 interval = 20 ms
- It is loudest at apex
- Coincides with carotid upstroke
- Determinants of S1
  - Structural integrity of valve
  - Position of the valve at the onset of ventricular systole

- PR interval (inversely proportional)
- Increased ionotropic activity of heart (directly proportional)
- Loss of isovolumetric contraction leads to soft S1 (MR, AR, VSD)
- Thoracic cavity and chest wall (high frequency murmurs are more attenuated with soft tissues).

Variations of S1		
Loud	***Soft***	***Variable***
• MS (mild to moderate), TS • ASD (loud T1) • Tachycardia • Short PR interval • Hyperdynamic circulation • Thin people	• Muffled in pan-systolic murmurs—MR, TR (here valves are wide and do not coaptate) • MS (severe calcific) • AR (increased LV filling and premature closure of mitral valve) • Bradycardia • Long PR, heart blocks, • Obesity, emphysema, effusion	• Atrial fibrillation • Ventricular tachycardia (AV dissociation) • Complete heart blocks (cannon sound)

When do you say loud S1? When S1 is heard with the same intensity as of mitral area in the base of heart (aortic and pulmonary areas)	
Splitting of S1	
Wide splitting	***Reverse splitting (T1→ M1)***
• Ebstein's anomaly • ASD • Complete RBBB • LV pacing	• Ectopics • Severe MS • Complete LBBB • RV pacing
Note: In ebstein's anomaly one can hear S1 split, S2 split, OS, S4 and pulmonary ejection click.	

(AR: aortic regurgitation; ASD: atrial septal defect; AV: atrioventricular; LV: left ventricular; MR: mitral regurgitation; TR: tricuspid regurgitation; MR: mitral regurgitation; MS: mitral regurgitation; MS: mitral stenosis; TS: tricuspid stenosis; RBBB: right bundle branch block; LBBB: left bundle branch block)

Second Heart Sound (S2)

- Two components (A2 and P2)
- A2 → P2
- A2-P2 time interval is <30 ms (expiration) and 40–50 ms (inspiration).
- Heard best in base of the heart (pulmonary and aortic areas).
- The loudest component of S2 in pulmonary area is A2.
- The loudest component of S2 in aortic area is A2.
- **Hang out interval**: The time interval from the crossover of pressures between ventricles and the arteries to the actual closure of valves is called hang out interval.
- Mechanism of normal split of S2:
  - During inspiration there is an increase in the capacitance of pulmonary vascular bed à this results in the delay of rise of pulmonary arterial pressure resulting in prolonged pulmonary hang out interval.
  - Early A2 (contributes around 27%).
  - Delayed P2 (contributes for 73%).
- Physiological split is inspiratory and disappears on standing, due to decreased venous return (while pathological split persists on standing).

Variations of S2 (Fig. 4E.22)	
A2	
Loud	***Soft***

• Hyperdynamic state, sHTN • Aneurysm of aorta • Aortic root dilatation (e.g. syphilis, ankylosing spondylosis) • TGA • Pulmonary atresia	• AS • AR • Aortic sclerosis (elderly) • Thick chest wall, obesity, emphysema
When do you say loud A2? Normally A2 is loudest at the base (aortic and pulmonary area). A2 is considered to be loud if the intensity in the mitral area is same as the base of the heart	
P2	
Loud	***Soft***
• Hyperkinetic states • pHTN • Dilation of pulmonary trunk • Aneurysm of pulmonary artery • Thin chest wall • Condition with L → R shunt	• PS • Dysplastic pulmonary valve • Thick chest wall, obesity, emphysema
When do you say loud P2? Normally A2 is louder than P2 even in pulmonary area but if P2 is as loud as A2 in pulmonary area, it is considered as loud P2	
Single S2	
• Severe AS, aortic atresia • Severe PS, pulmonary atresia • Fallot's tetralogy (A2 becomes loud and P2 disappears)	

(AR: aortic regurgitation; AS: aortic stenosis; pHTN: pulmonary hypertension; PS: pulmonary stenosis; sHTN: systemic hypertension; TGA: transposition of the great arteries)

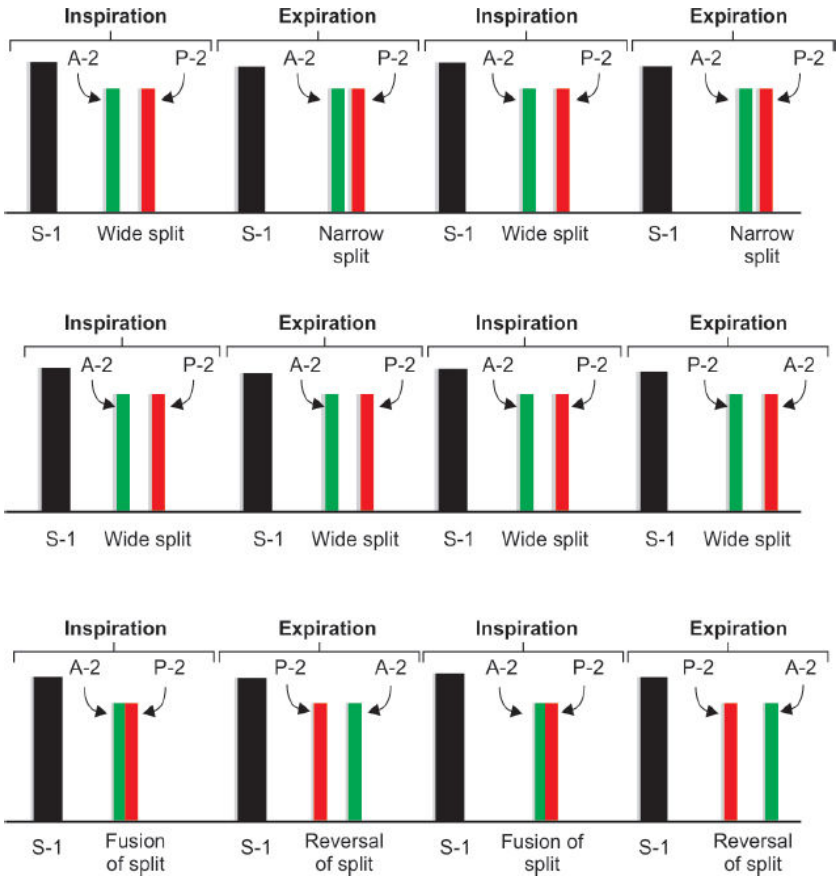


Fig. 4E.22: Variations of 2nd heart sound.

Splitting of 2nd heart sound		
Narrow split	***Wide and variable split***	***Wide and fixed split***
Severe pHTN	• **Chest deformity**: Funnel chest and straight back syndrome • **Due to early A2**: MR, VSD • **Due to late P2**: RBBB, LV pacing, ectopics from LV	• ASD • Severe RV failure • Acute pulmonary embolism

Note: **Why do you get wide fixed split in ASD?**	
Wide split *is due to*	**Fixed split *is due to***
1. Increased RV ejection time 2. Prolonged pulmonary hangout interval 3. RBBB	1. Free communication between two atria equalizes the pressure during inspiration and expiration 2. Already prolonged pulmonary hangout interval cannot be further prolonged
Paradoxical split (reverse split)	
• P2 comes before A2 • Split is prominent and wider during expiration, while it narrows during inspiration • Causes due to either early P2 or late A2	
Early P2	***Late A2***
• Complete LBBB • RV pacing • PVCs of RV	• Severe AS • Severe sHTN • HCM

(AS: aortic stenosis; ASD: atrial septal defect; HCM: hypertrophic cardiomyopathy; LBBB: left bundle branch block; LV: left ventricular; MR: mitral regurgitation; pHTN: pulmonary hypertension; PVCs: premature ventricular contractions; RBBB: right bundle branch block; RV: right ventricular; sHTN: systemic hypertension; VSD: ventricular septal defect)

Valvular diseases and S2	
MS	• Mild to moderate → normal • Severe MS with pHTN → loud P2
MR	• Mild to moderate → normal • Severe → wide and variable • MR + CAD/HOCM → reverese split
AS	• Severe AS → reverse split (severe AS)
AR	• Root pathology → A2 loud—tambour • Valvular pathology → A2 soft

(AR: aortic regurgitation; AS: aortic stenosis; CAD: coronary artery disease; HOCM: hypertrophic obstructive cardiomyopathy; MR: mitral regurgitation; MS: mitral stenosis; pHTN: pulmonary hypertension)

THIRD HEART SOUND (S3)

- Third heart sound (S3) is a low-pitched early diastolic sound best heard with the bell. Also called as ventricular sound or protodiastolic sound/gallop.
- It coincides with rapid ventricular filling immediately after opening of the atrioventricular valves and is therefore heard after the second sound as ‘lub-dub-dum’.
- It is almost never heard at the base of heart (aortic and pulmonary area).
- Less palpable than S4.
- It is sign of ventricular systolic dysfunction.
- Prerequisite
  - Nonobstructed AV valve.
- Best head with bell
  - LVS3—left lateral position at apex during expiration.
  - RVS3—left sternal edge in supine position during inspiration.

Causes of S3		
Physiological and hyperdynamic states	***Pathological LV S3***	***Pathological RV S3***
• Children • Under 40 years • Athletes • Pregnancy • Other hyperdynamic states	• Left ventricular failure • Aortic regurgitation • Mitral regurgitation • Ischemic heart disease • Cardiomyopathy	• Right ventricular failure • Endomyocardial fibrosis

PERICARDIAL KNOCK

- Cause–sudden cessation of ventricular filling
- Seen in–constrictive pericarditis
- Timing–comes earlier than S3
- Frequency–higher than S3.
- **Diastolic knock** is a palpable pericardial knock in constrictive pericarditis.
- Correlate with other clinical findings like:

- Rapid ‘y’ descent
- Kussmaul sign
- Systolic retraction of apex (broadbent’s sign)
- Congestive hepatomegaly with ascites.

FOURTH HEART SOUND (S4)

- It is a low frequency late diastolic or presystolic sound heard during atrial contraction.
- It is also called as a presystolic or an atrial diastolic gallop (even though it is ventricular in origin).

Prerequisites:

- Healthy contracting atrium.
- Nonobstructive AV valve.
- Noncompliant (stiff) ventricle.
- Theories of production of S4:
  - Ventricular theory (rapid deceleration of incoming blood).
  - Impact theory (dynamic impact of the heart with chest wall).
- Best head with bell.
- LVS4—left lateral position at apex during expiration.
- RVS4—left sternal edge in supine position during inspiration.
- S4 may be confused with spilt S1. Firm pressure by the diaphragm of stethoscope eliminates S4 but not split S1.

Causes of S4:

- Physiological: >60 years
- Pathological:

Pathological S4	
RV S4	***LV S4***
Right ventricular hypertrophy due to: • Pulmonary hypertension • Pulmonary stenosis	• Systemic hypertension • Hypertrophic cardiomyopathy • Ischemic heart disease (especially acute myocardial infarction) • Acute mitral regurgitation • Anemia, thyrotoxicosis and AV fistula

Note:

1. Triple gallop rhythm: S1, S2, S3 (or S4) with HR >100
2. Summation rhythm: S1, S2, S3, S4 with HR >100

CLICKS AND SNAPS

Clicks	***Snaps***
High pitched systolic sounds	High pitched diastolic sounds
Produced by aortic and pulmonary valve opening	Produced by mitral and tricuspid valve opening

Clicks

Clicks	Ejection clicks	Non-ejection clicks
Timing	**Early systolic**	**Mid to late systolic**

Pathology	Vascular (dilated vessel)	Valvular (diseased valve)	Valve prolapse
Left sided causes	Systemic hypertension Aneurysm of aortic root	Bicuspid aortic valve	Mitral valve prolapse
Right sided causes	Dilated pulmonary artery (idiopathic or secondary to pulmonary arterial hypertension)	Congenital pulmonary stenosis	Tricuspid valve prolapse

Note: Pulmonary valvular ejection click seen in congenital pulmonary stenosis is the only event occurring in the right side of the heart which is better heard on expiration.

Opening Snaps

- High pitched diastolic sound occurring 0.04–0.12 seconds after A2 (S3 occurs 0.12 seconds after A2) due to opening of mitral or tricuspid valves.
- Occurs after S2 and before S3.
- **Mechanism of opening snap (OS):**
    1. Stenotic anterior mitral/tricuspid valve leaflet suddenly bulging downward into the ventricular cavity like a dome, with a snapping sound when the valve is rapidly opened during diastole. So, OS is heard only if leaflets are mobile.
    2. OS occurs when movement of valve suddenly stops, at point when ventricular pressure drops below that of atrial pressure.

In mitral stenosis (MS):

- It is the most important auscultatory sign of valvular involvement in MS (pathognomonic sign).
- Absent OS indicates the calcification of body of the mitral leaflets.
- The time interval between A2 and OS is inversely proportional to the severity of the MS.
- **Best heard:** During expiration, just medial to the cardiac apex with the diaphragm of the stethoscope.

Other conditions with OS:

- Mitral regurgitation (10%)
- Tricuspid stenosis,
- Atrial septal defect.

Differences between OS, split S2 and S3:

	Opening snap (OS)	S2 split	S3
Area	Medial to apex	Base of heart	At the apex
On standing	A2-OS increases	A2-P2 decreases	Disappears
Pitch	High	High	Low
Best heard	Diaphragm	Diaphragm	Bell

Other sounds:

Tumor plop	Seen in myxomas
Prosthetic valve sounds	• Metallic S1 heard with mechanical mitral valve • Metallic S2 heard with mechanical aortic valve

Note: Bioprosthetic valves heart sounds are normal.

PERICARDIAL RUB

It is the sound produced due to sliding (apposition) of the two inflamed layers (visceral and parietal pericardium) of the pericardium.

- **Phases:** It is triphasic
  1. midsystolic,
  2. mid-diastolic and
  3. presystolic.
- **Character:** It is scratchy, grating, leathery or creaking in character. Its intensity varies over time, and with the position of the patient.
- **Best heard:** With diaphragm of stethoscope on the left sternal border (3rd and 4th intercostal space) leaning forward at the end of expiration. It may be audible over any part of the precordium but is often localized. It can be better appreciated with patient in knee elbow position.
- A pleuropericardial rub is a similar sound that occurs in time with the cardiac cycle but is also influenced by respiration and is pleural in origin.

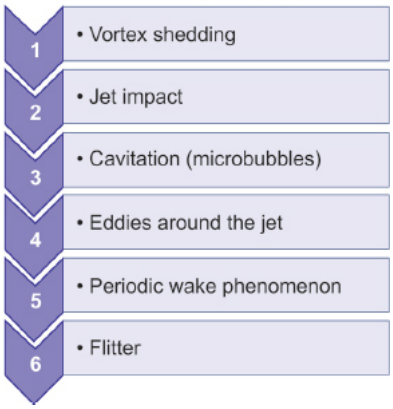
MURMURS

Sudden deceleration of blood produces heart sounds while heart murmurs are produced by turbulent flow (Reynold's number >2,000) across an abnormal valve, septal defect or outflow obstruction, or by increased volume or velocity of flow through a normal valve.

Mechanism

- Increased blood velocity
- Decreased blood viscosity
- Valve—narrowed or incompetent; organic or relative
- Abnormal connection
- Vibration of loose structure
- Diameter of vessel increased or decreased.

Rushmer RF postulated 6 mechanism of production of murmurs:



Murmurs are described under the following headings:

1. Timing
2. Grade
3. Quality
4. Pitch
5. Configuration
6. Radiation/conduction

7. Best heard with diaphragm or bell
8. Patient position
9. With breath held in inspiration or expiration
10. Variation with other maneuvers
11. Location of maximum intensity

1. Timing (Fig. 4E.23)

Timing refers to the portion of the cardiac cycle that the murmur occupies. Murmurs may be systolic, diastolic, or continuous.

Systolic murmurs may be:

- Early systolic murmurs
- Midsystolic murmurs
- Late systolic murmurs
- Pansystolic murmurs.

Systolic Murmurs

Murmur and description	Example
Early systolic murmurs (begin with the first heart sound and extend to middle or late systole)	• VSD (small muscular VSD/large VSD with pulmonary hypertension • Acute severe MR • Acute severe TR
Midsystolic/ejection systolic murmurs (begin following a murmur-free interval in early systole and end with a murmur-free interval (of variable duration) in late systole	• Aortic stenosis • Pulmonary stenosis • HOCM
Late systolic murmurs (begin during the last half of systole and may or may not extend to the second heart sound)	• Mitral valve prolapse • Tricuspid valve prolapse • Papillary muscle dysfunction
Pansystolic murmurs (begin with the first heart sound and extend to or through entire systole, muffling S1. They are sometimes called **Holosystolic murmur** but in holosystolic murmur S1 is distinct (e.g. VSD)	• Mitral regurgitation • Tricuspid regurgitation • Ventricular septal defect • Rare—early PDA/PDA with Eisenmenger

(HOCM: hypertrophic obstructive cardiomyopathy; MR: mitral regurgitation; PDA: patent ductus arteriosus; TR: tricuspid regurgitation; VSD: ventricular septal defect)

Diastolic murmurs may be:

- Early diastolic
- Mid-diastolic
- Late diastolic/presystolic

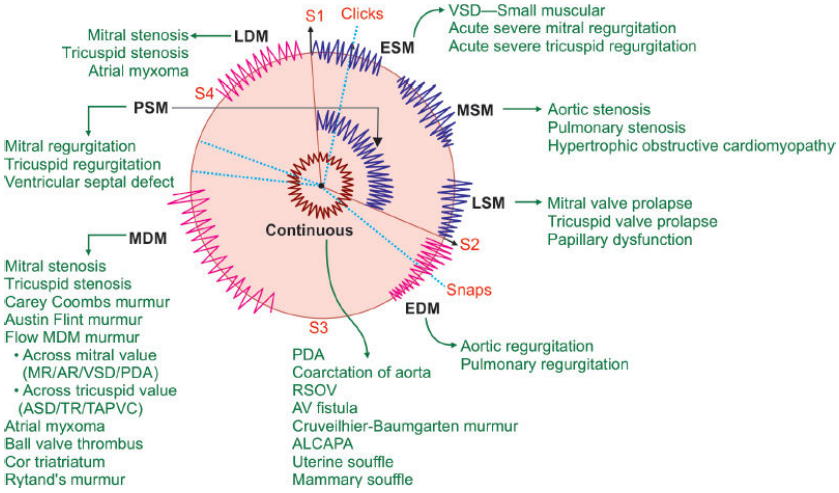


Fig. 4E.23: Timing of murmurs and examples.

Diastolic Murmur

Murmur	Example
Early diastolic murmur	1. Aortic regurgitation 2. Pulmonary regurgitation
Mid-diastolic murmur	1. Mitral stenosis 2. Tricuspid stenosis 3. Carey Coombs murmur of acute rheumatic fever 4. Austin Flint murmur of chronic aortic regurgitation 5. Flow MDM murmur: a. Across mitral valve: MR, AR, VSD, PDA b. Across tricuspid valve: ASD, TR, TAPVC 6. Atrial myxoma 7. Ball valve thrombus 8. Cortriatriatum 9. Rytand's murmur of complete heart block
Late diastolic murmurs/Presystolic murmur	1. Mitral stenosis 2. Tricuspid stenosis 3. Myxoma

(AR: aortic regurgitation; MDM: mid-diastolic murmur; MR: mitral regurgitation; PDA: patent ductus arteriosus; TAPVC: total anomalous pulmonary venous connection; TR: tricuspid regurgitation; VSD: ventricular septal defect)

Continuous Murmurs

The continuous murmur is the murmur that begins in systole and continues without interruption, **encompassing the second sound,** throughout diastole or part of diastole.

Continuous murmurs
A. Systemic to pulmonary communication 1. Patent ductus arteriosus 2. Aortopulmonary window 3. Anomalous origin of left coronary artery from pulmonary artery (ALCAPA) 4. Tricuspid atresia 5. Truncus arteriosus 6. Shunts for tetralogy of Fallot (TOF) surgery—Waterson, Potts, or Blalock-Taussig shunt
B. Systemic to right heart connection

1. Coronary AV fistula
2. Rupture sinus of Valsalva

C. Left atrium to right atrium connection
1. Lutembacher syndrome

D. Arteriovenous fistula
1. Systemic
2. Pulmonary

E. Normal flow through constricted arteries
1. Coarctation of aorta
2. Peripheral pulmonary stenosis
3. Renal artery stenosis

F. Increased flow through normal vessels
1. Venous
   i. Cervical venous hum
   ii. Cruveilhier–Baumgarten murmur
2. Arterial
   i. Mammary soufflé
   ii. Uterine soufflé
   iii. Thyrotoxicosis
   iv. Tumors—hepatoma, hypernephroma

Differential Diagnosis of Continuous Murmur

Systolic-diastolic murmurs	*To and fro murmurs*
Murmur in systolic and murmur in diastolic but S2 is heard distinctly. The two murmurs are separated by small silence differentiating them from continuous murmurs.	
Occurs through different orifices	Occurs through same orifice
• VSD with AR • MR with MS	• AS with AR • Pulmonary hypertension with pulmonary regurgitation

(AR: aortic regurgitation; AS: aortic stenosis; MR: mitral regurgitation; MS: mitral stenosis; VSD: ventricular septal defect)

2. Grading of Murmurs

Systolic Murmurs

Levine and Freeman grading of systolic murmurs		
Grade	*Description*	*Thrill*
Grade 1	Murmur so faint that it can be heard only with special effort	**Absent**
Grade 2	Murmur is faint but is immediately audible	
Grade 3	Murmur that is moderately loud	
Grade 4	Murmur that is very loud	**Present**
Grade 5	A murmur that is extremely loud and is audible with one edge of the stethoscope touching the chest wall	
Grade 6	A murmur that is so loud that it is audible with the stethoscope just removed from contact with the chest wall	

Diastolic Murmurs (by AIIMS)

Grade	*Description*	*Thrill*
Grade 1	Very soft	**Absent**

Grade 2	Soft	
Grade 3	Loud	
Grade 4	Very loud	**Present**

3. Character/Quality

Quality refers to the tonal effect of the murmurs. Frequently used descriptors are *blowing, musical, squeaking, whooping, honking, harsh, rasping, grunting, and rumbling.*

4. Frequency or Pitch

- Relates to the velocity of the blood at the site of origin of the murmur and is designated as high, medium, or low. In general, the higher the velocity, the higher the pitch of the murmur.
- Murmurs that emanate from areas of stenosis where velocity is lower are typically low to medium pitched.

5. Configuration (Figs. 4E.24 to 4E.26):

Configuration of a murmur refers to its shape.

- To a large degree it is a function of intensity and duration.
- Crescendo murmurs progressively increase in intensity.
- Decrescendo murmurs progressively decrease in intensity.
- With crescendo-decrescendo murmurs (diamond or kite-shaped murmurs), a progressive increase in intensity is followed by a progressive decrease in intensity.
- Plateau murmurs maintain a relatively constant intensity.

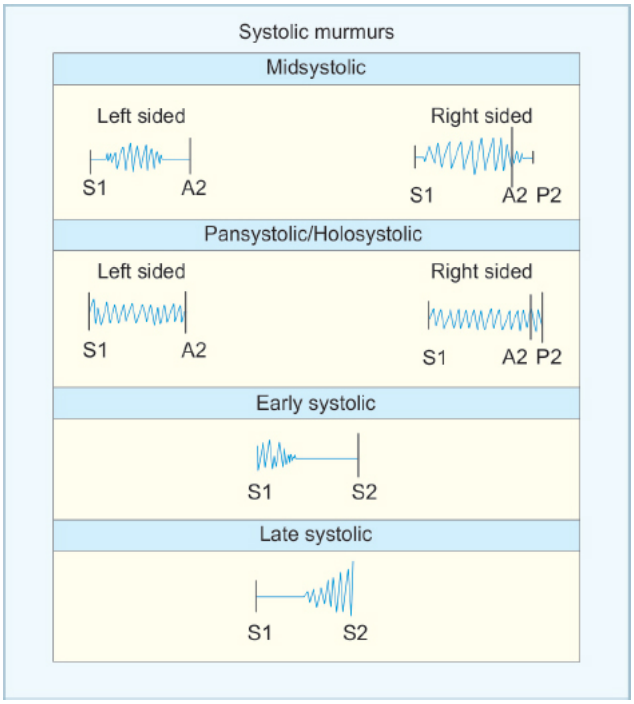


Fig. 4E.24: Configuration of systolic murmurs.

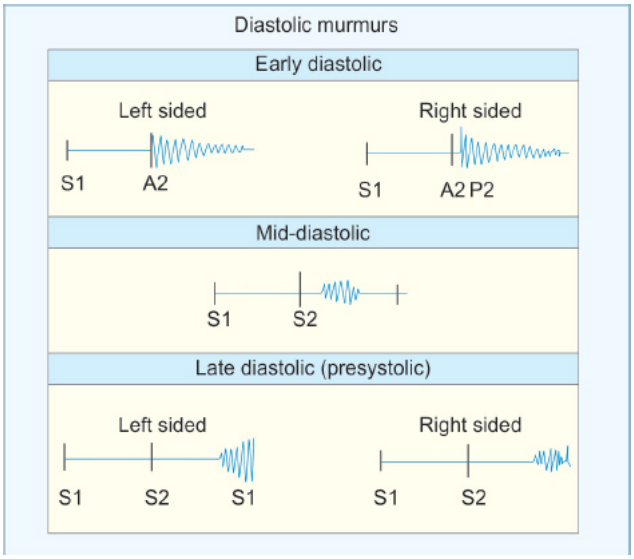


Fig. 4E.25: Configuration of diastolic murmurs.

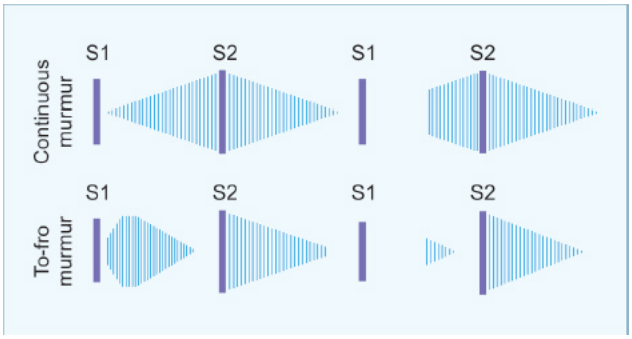


Fig. 4E.26: Configuration of continuous and to-fro murmurs.

6. Radiation/Conduction (Fig. 4E.27)

Reflects the intensity of the murmur and the direction of blood flow.

Radiation	Conduction
It is through noncardiac structures	It is through anatomical continuity
Intensity decreases with distance	Intensity remains same or decreases with distance
Mitral regurgitation murmur (PSM) radiates to axilla. Tricuspid regurgitation radiates to epigastrium	Aortic stenosis murmur (ESM) conducts to the carotid

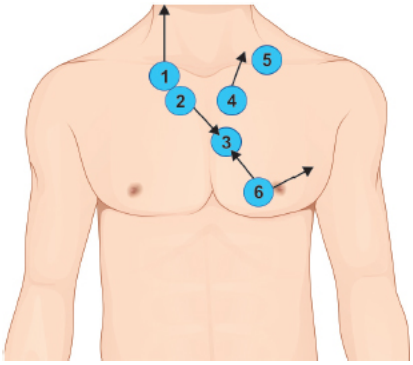


Fig. 4E.27: Radiation of murmurs: (1) ESM of AS conducting to carotids; (2) EDM of AR in right 2nd ICS radiating to left 3rd ICS; (3) PSM of TR radiating to upper left sternal border; (4) ESM of PS conducting towards clavicle; (5) Murmur of PDA at infraclavicular area radiates to back; (6) PSM of MR radiating to axilla or base of heart.

7. Best Heard with Bell or Diaphragm

Best heard with bell	Best heard with diaphragm
MDM of MS and TS (Other sounds: S3, S4, pericardial knock)	Systolic murmur of MR, TR, AS and diastolic murmur of AR (Other sounds: S1, S2, ESC, OS)

(AR: aortic regurgitation; AS: aortic stenosis; MDM: mid-diastolic murmur; MR: mitral regurgitation; MS: mitral stenosis; TR: tricuspid regurgitation; TS: tricuspid stenosis)

8. Variation with position

Left lateral recumbent position	Sitting and leaning forward	Lying flat or passive leg raising in supine position
Accentuates *Sounds:* • S1 • LVS3 and LVS4 • OS of MS *Murmurs:* • MS • MR • Click and murmur of MVP • Austin Flint murmur	**Accentuates** *Murmurs:* • AR • PR	**Accentuates** *Sounds:* • S3 and S4 *Murmurs:* • Valvular AS/PS • TR **Attenuates** • EDM of AR • Murmur of HOCM • MVP murmur and click are delayed

(AR: aortic regurgitation; AS: aortic stenosis; EDM: early diastolic murmur; HOCM: hypertrophic obstructive cardiomyopathy; MR: mitral regurgitation; MS: mitral stenosis; MVP: mitral valve prolapse; OS: opening snap; TR: tricuspid regurgitation; TS: tricuspid stenosis)

9. Variation with Respiration

Breathing produces a greater effect on the right side of the heart than the left side.

RIght-sided murmurs increase on **I**nspiration	**LE**ft-sided murmurs increase on **E**xpiration
Inspiration increases venous return to the right side of the heart by increasing flow in the vena cava but decreases venous return to the left side of the heart due to pooling of blood in pulmonary venous capacitance vessels	Expiration decreases venous return to the right side of the heart by reducing vena cava flow, but increases venous return to the left side of the heart due to collapse of pulmonary venous capacitance vessels
• TS • TR (Carvallo's sign*) • PR • Mild or moderate PS	• MS • MR • AS • AR

• Severe PS	• VSD • Pericardial rub

(AR: aortic regurgitation; AS: aortic stenosis; MR: mitral regurgitation; MS: mitral stenosis; PS: pulmonary stenosis; TR: tricuspid regurgitation; TS: tricuspid stenosis; VSD: ventricular septal defect)

Note:

1. Carvallo's sign*—when the murmur of tricuspid valve regurgitation gets louder with deep inspiration.
2. The effects of inspiration on systolic murmurs can be accentuated by employing Muller's maneuver (forced inspiration on a closed glottis).

10. Variation with Other Maneuvers

- The physiologic maneuvers are breathing, standing, sudden squatting, isometric hand grip exercise, Valsalva maneuver (described at the end), passive leg raising, and attention to the beat following a post-extrasystolic pause.
- The pharmacological interventions used most commonly in clinical practice are amyl nitrite administration and intravenous infusion of alpha-adrenergic agonists (phenylephrine or methoxamine).

Valvular disease	Accentuated by	Attenuated by
MS	• Expiration • Exercise, squatting, amyl nitrate, isometric hand grip	Inspiration, sudden standing
MR	• Expiration • Squatting • Isometric exercise	• Sudden standing • Valsalva • Amyl nitrate
AS	• Expiration • Post-PVC beat • Squatting • Lying flat from standing	• Valsalva • Standing • Handgrip
AR	• Expiration • Sitting up and leaning forward • Squatting • Isometric exercise • Vasopressors	• Amyl nitrate • Valsalva
MVP	Murmur and click later • If LV volume increases • Squatting • Postectopic • Isometric exercise (intensity increases)	Murmur and click earlier if LV volume decreases • Standing • Valsalva
HOCM	• Expiration • Valsalva strain • Standing • Postectopic • Amyl nitrate	• Inspiration • Sustained handgrip • Squatting • Methoxamine

(AR: aortic regurgitation; AS: aortic stenosis; HOCM: hypertrophic obstructive cardiomyopathy; LV: left ventricular; MVP: mitral valve prolapse; PVC: premature ventricular contraction; MR: mitral regurgitation; MS: mitral stenosis)

11. Location of Maximum Intensity of Murmur

- Location refers to the point on the precordium where the murmur is heard with maximum intensity.
- Many systolic murmurs are audible over multiple areas of the precordium. Localizing their point of maximum intensity may aid greatly in determining their site of evolution.

Example:

In aortic stenosis/aortic sclerosis—gallavardin phenomenon seen. Two distinct systolic murmurs are heard; one high pitched murmur in the aortic area and the other musical systolic murmur in the mitral area. This is due to periodic wake phenomenon or the Hour-glass murmur.

Examples for How to Describe a Murmur

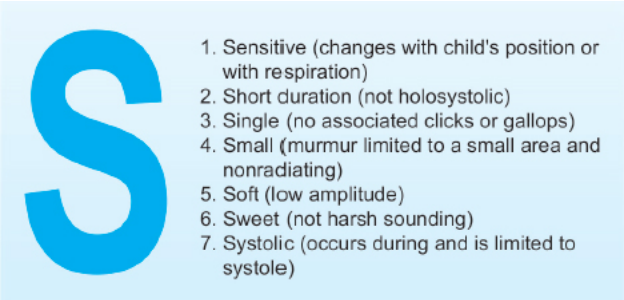
The murmur of mitral stenosis is a mid-diastolic low-pitched rough rumbling murmur with presystolic accentuation best audible at the apex (mitral area), in the left lateral position with the bell of the stethoscope, breath held in expiration. The murmur increases on isometric hand grip.

The murmur of aortic regurgitation is a soft, high-pitched, early diastolic, decrescendo murmur usually heard best at the third intercostal space on the left (Erb's point) with the diaphragm of the stethoscope at end expiration with the patient sitting up and leaning forward.

Innocent Murmurs

Innocent murmurs are those those murmurs which are not due to recognizable lesions of the heart or blood vessels. They are most common in children and adolescents.

The Seven S's of innocent murmurs



Examples of Innocent Murmurs

Systolic	1. Vibratory systolic murmur (Still's murmur) 2. Pulmonic systolic murmur (pulmonary trunk) 3. Mammary soufflé 4. Peripheral pulmonic systolic murmur (pulmonary branches) 5. Supraclavicular or brachiocephalic systolic murmur 6. Aortic systolic murmur
Diastolic	All diastolic murmurs are pathological (not innocent)
Continuous	1. Venous hum 2. Continuous mammary soufflé

Named murmurs	
Carey Coombs murmur	Mid-diastolic murmur, in rheumatic fever
Austin Flint murmur	Mid-late diastolic murmur, in aortic regurgitation (AR)
Graham-Steel murmur	High pitched, diastolic, in pulmonary regurgitation
Rytand's murmur	Mid-diastolic atypical murmur, in complete heart block
Docks murmur	Diastolic murmur, left anterior descending (LAD) artery stenosis
Mill wheel murmur	Due to air in right ventricle (RV) cavity following cardiac catheterization
Stills murmur	Inferior aspect of lower left sternal border, systolic ejection sound, vibratory/musical quality in subaortic stenosis, small ventricular septal defect
Gibson's murmur	Continuous machinery murmur of patent ductus arteriosus (PDA)

Key–Hodgkin murmur	Diastolic murmur of aortic regurgitation. Hodgkin correlated this diastolic murmur with retroversion of the aortic valve leaflets, seen in syphilitic aortic regurgitation
Cabot–Locke murmur	Diastolic murmur heard best at the left sternal border. heard in anemic patients. The murmur resolves with treatment of anemia
Roger's murmur	It is the loud pansystolic murmur which is heard maximally at the left sternal border in small ventricular septal defect (VSD).
Pontains murmur	Cervical venous hum in severe anemia
Cole-Cecil murmur	AR murmur in left axilla due to higher position of apex
Cruveilhier-Baumgarten venous hum	It is diagnostic of portal venous hypertension

Auscultation for Mitral Stenosis (Fig. 4E.28)

- Patient in left lateral position
- Breath held in expiration
- Using bell of stethoscope
- Time the murmur with carotid.

Auscultation of Tricuspid Area (Fig. 4E.29)

- Patient in supine position
- Breath held in inspiration
- Using diaphragm of stethoscope
- Murmur increases on hepatic compression or passive leg raise.

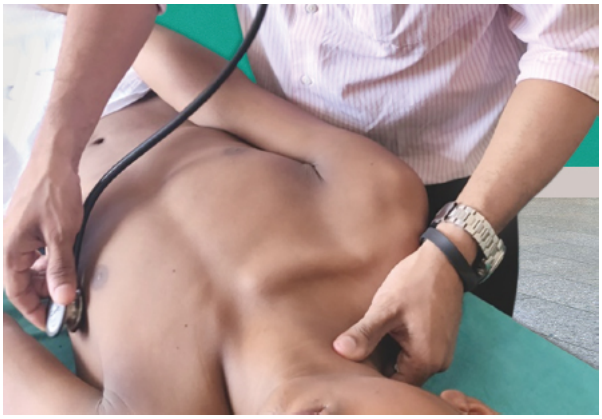


Fig. 4E.28: Auscultation of mitral area—mid-diastolic murmur of mitral stenosis.

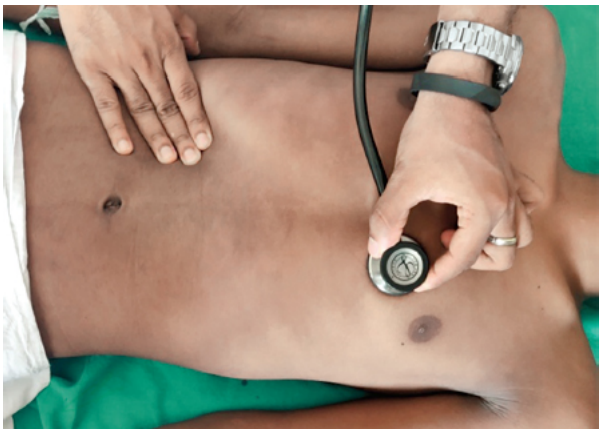


Fig. 4E.29: Auscultation of tricuspid regurgitation.

Auscultation of Aortic Area (Fig. 4E.30)

- Patient in sitting up and leaning forward position
- Breath held in expiration
- Using diaphragm of stethoscope
- Time the murmur with carotid.

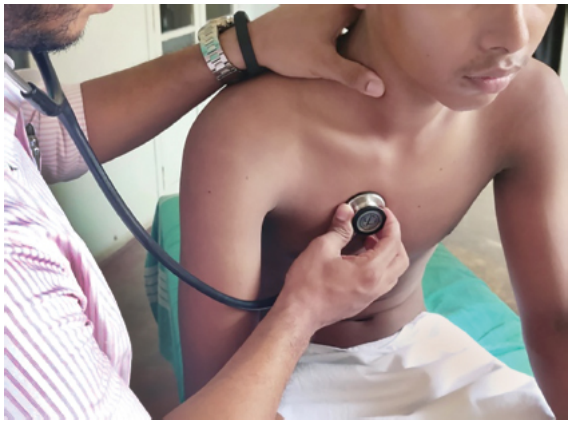


Fig. 4E.30: Auscultation of aortic area (Erb's maneuver).

Changing murmurs
Murmurs which change in character or intensity from moment to moment:
1. Carey Coombs murmur 2. Infective endocarditis 3. Atrial thrombus 4. Atrial myxomas

OTHER SYSTEM EXAMINATION

Respiratory system	• Hoarseness of voice (enlarged left atrium—Ortner's syndrome) • Hemoptysis • Left lower lobe collapse or consolidation (pericardial effusion) • Basal crepitations [left ventricular failure (LVF)]

	• Pleural effusion (LVF) • Rhonchi (pulmonary edema)
Gastrointestinal tract	• Tender hepatomegaly (right heart failure) • Splenomegaly (infective endocarditis) • Ascites (right heart failure) • Dysphagia (due to large left atrium)
Nervous system	• Stroke (hemiplegia/Horner's syndrome, cranial nerve palsies)

PULSATILE LIVER

Examination of Pulsatile Liver

- Patient in 45° recumbent position
- Two methods are described
  1. **Bimanual palpation (Fig. 4E.31):** Place one palm over the anterior surface of the right lower chest and other palm on the posterolateral surface of the right lower chest. Pulsations of the liver are felt between the two palms.
  2. **Make fist of the** right hand and placing the knuckles and fingers in the right lower intercostal spaces and feel for the pulsatile liver as shown in **Figure 4E.32**.

Systolic pulsation	Diastolic pulsations (presystolic)
• TR • AR	TS

(AR: aortic regurgitation; TR: tricuspid regurgitation; TS: tricuspid stenosis)

Valsalva Maneuver

The Valsalva maneuver is a forceful attempted exhalation against a closed glottis.

Instruction:
Take a deep breath, close your mouth and pinch your nose with the thumb and index finger and attempt to breathe out gently, keeping your cheek muscles tight, not allowing the air to escape by keeping the lips pursed.

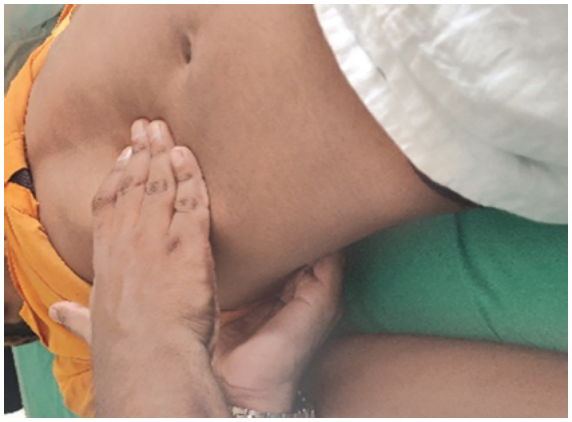


Fig. 4E.31: Bimanual method of palpation of pulsatile liver.

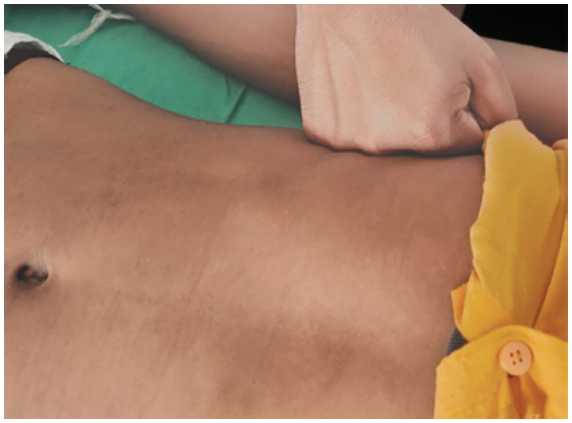


Fig. 4E.32: Examining the pulsatile liver by making fist and placing the knuckles and fingers in the intercostal spaces.

"Standard" or "quantitative":
Blowing out with an open glottis into a tube of a sphygmomanometer against the pressure of 40 mm Hg.

Phases of Valsalva Maneuver

Physiological effects on blood pressure, heart rate and phases of Valsalva maneuver are presented in **Figure 4E.33**.

Phases of Valsalva maneuver	
Phase 1	• The onset of blowing. • The pressure within the chest and abdomen increases and presses upon the arteries in the chest, which results in an increase in mean arterial blood pressure **(Fig. 4E.33)**. This activates the baroreceptor reflex, which results in an increase in parasympathetic (vagal) activity and hence in a drop in heart rate. • The increased intrathoracic pressure also reduces the amount of blood that comes into the right atrium (decreased venous return or preload)
Phase 2	A decrease of venous return results in a lower amount of blood that is ejected from the heart, which results in a decrease of central venous pressure and consequently in a decrease of mean arterial blood pressure. This activates the baroreflex, which results in a decrease of the parasympathetic (vagal) activity and consequent increase of the heart rate, and in an increase in sympathetic activity, which constrict the arteries (an increase of peripheral resistance) and results in a slight rise of the blood pressure at the end of phase 2 (2b).
Phase 3	Relaxation—the end of the maneuver. The intrathoracic pressure decreases, so the intrathoracic arteries widen, which results in a brief drop in blood pressure. At the same time, the venous blood fills the heart
Phase 4	The heart ejects the blood into the arterial system against increased peripheral resistance (which has developed in phase 2), so the blood pressure rises again (blood pressure overshoot). This activates the baroreflex, which results in a drop in heart rate (bradycardia). Eventually, both the blood pressure and heart rate normalize

Uses

- Eustachian tube dysfunction
- Heart murmurs: Valsalva increases murmurs in hypertrophic cardiomyopathy and mitral valve prolapse and decreases them in atrial septal defects and aortic stenosis.
- Congestive heart failure: Valsalva responses lost.
- Function of the autonomous nervous system:
  - An abnormal blood pressure response (for example, an absence of the blood pressure rise in phase 4) suggests an abnormality of the sympathetic system.
  - An abnormal heart rate response suggests an abnormality of the parasympathetic system. Valsalva maneuver that can be used as a provocative test to check for neurogenic orthostatic hypotension, Chiari malformation, the Valsalva maneuver (coughing) triggers a headache at the back of the head.

- Diagnosis of inguinal hernia, prolapse of the uterus, bladder or vagina, varicocele and intrinsic sphincter deficiency in stress urinary incontinence system.
- Valsalva maneuver can help: Equalize the pressure between the middle ear and the ambient pressure during scuba diving, driving from a steep hill, elevator descending, parachuting or plane landing or in individuals with Eustachian tube dysfunction.

Modified Valsalva Maneuver

Modified Valsalva maneuver is used to terminate an attack of supraventricular tachycardia (SVT); it includes blowing against a closed glottis followed by lying down face up and raising legs with the help of an assistant, may be effective in 19–54% of cases.

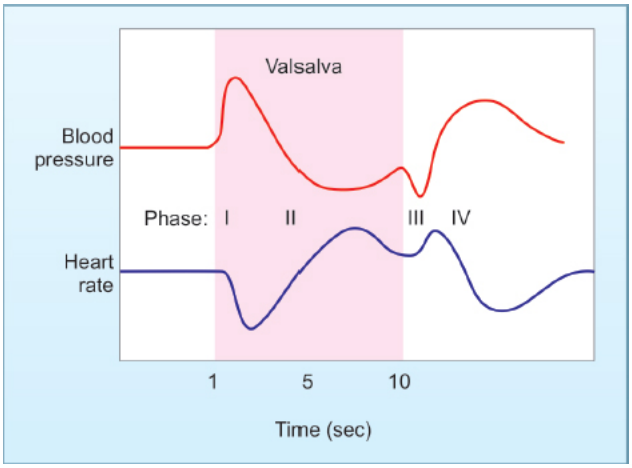


Fig. 4E.33: Mean arterial blood pressure and heart rate changes during the Valsalva maneuver.

Various phases of Valsalva maneuver and its associated changes:

Phase	1	2a	2b	3	4
Intrathoracic pressure	↑	↑	↑	N	N
Mean arterial blood pressure	↑	↓	↑	↓	↑
Heart rate	↓	↑	↓	↑	↓
Sympathetic activity	↓	↓	↑	↑	↑
Parasympathetic (vagal) activity	↑	↑	↓	↓	↑

Reversed Valsalva—Müller's maneuver

Muller's maneuver is the opposite of the Valsalva maneuver and includes forced exhalation followed by an attempted forceful inhalation with a closed mouth and nose or just with a closed glottis. The test can be used to evaluate weakness of the soft palate and throat walls in individuals with obstructive sleep apnea.

NOTES

F. SUMMARY OF FINDINGS IN COMMON CARDIOVASCULAR DISEASES

Findings		MS	MR	AS	AR	TR	ASD	
Pulse		• Low volume, • Irregularly irregular (if associated with AF)	• High volume, • Irregularly irregular (if associated with AF)	• Low volume, • **Pulsus parvus et tardus** • Anacrotic pulse • Apico-carotid delay—severe AS	• High volume, **Collapsing pulse** • **Water hammer pulse** • **Pulsus bisferiens**	Normal	• Normal • Irregularly irregular (if associated with AF)	• Hig
Blood Pressure		• Low BP • Mean of 3 readings to be taken if atrial fibrillation is present	• Wide pulse pressure • Mean of 3 readings to be taken if atrial fibrillation is present	• Low BP • Systolic decapitation • **Coanda effect**: Right upper limb BP >left upper limb BP (supravalvular AS)	• Wide pulse pressure • **Hills sign**—Lower limb BP >20 mm of upper limb BP	Normal	Normal	• Wid pres
JVP		• Raised in heart failure **Prominent a waves**—pulmonary hypertension without atrial fibrillation • **Absence of a wave**—atrial fibrillation • **Prominent v waves** (c-v waves) and rapid y descent → tricuspid regurgitation	• Raised in heart failure **Prominent a waves**—pulmonary hypertension without atrial fibrillation • **Absence of a wave**—atrial fibrillation • **Prominent v waves** (c-v waves) and rapid y descent → tricuspid regurgitation	• Usually normal • Raised in heart failure • Rarely prominent a wave—Bernheim effect	• Usually normal • Raised in heart failure	• Raised with most prominent 'giant' v wave in the jugular venous pulse (a **c-v wave** replaces the normal x descent). • **Earlobe pulsations** (Lancisi's sign)	**"M" pattern--** a and v waves have equal height, a wave becomes taller when pulmonary hypertension develops or associated mitral stenosis (MS).	Raise failure
Apex		**Tapping** apex	**Hyperdynamic** Down and out apex	Heaving	**Hyperdynamic** Down and out apex	Normal	Normal	Mild d down
Parasternal heave		Present (RVH or left atrial enlargement)	Present (RVH or left atrial enlargement)	No	No		Present	Prese
Thrills		Diastolic thrill at apex	Systolic thrill at apex in acute or severe MR	Systolic thrill over the aortic and carotid area	Diastolic thrill in aortic/neoaortic area	Systolic thrill in left lower sternal edge	nil	Left 4- parast
Heart sounds	S1	Loud	Soft	Normal	Soft	Soft	Loud	Soft
	S2	• Loud P2 (pulmonary hypertension) • Narrow split (pulmonary hypertension)	Loud P2 (pulmonary hypertension) Narrow split (pulmonary hypertension)	Soft A2 (valvular AS) Loud A2 (bicuspid aortic valve)	Normal Tambour A2 in syphilitic AR	Loud P2 with narrow split (pulmonary hypertension)	P2 loud Wide fixed split	P2 lou

				Paradoxical split (severe AS)				
	S3	RVS3 (present in failure)	RV/LVS3 (present in failure)	LVS3 in failure	LVS3 in severe AR	RVS3	RVS3	+/-
	S4	Never	Present in acute MR	Present. indicates severe AS	+/-	--	RVS4 (Eisenmenger's)	RVS4 (Eisen
	Others	Opening snap	OS in 10%	AEC in bicuspid aortic valve	---	--	PEC (Eisenmenger's)	PEC (Eisen
Murmurs		• **MDM** at mitral area • **PSM** at tricuspid area • **ESM** at pulmonary area • **EDM** (Graham Steel) at pulmonary area	• **PSM** in mitral area radiation to axilla/base • **Flow MDM** at mitral area • **PSM** at tricuspid area • **ESM** at pulmonary area • **EDM** (**Graham Steel**) at pulmonary area	• **ESM** in aortic area conducting to carotid • Systolic murmur at mitral area (**Gallavardain Phenomenon**)	• **EDM** in aortic/neoaortic area • **Flow ESM** in aortic area • **MDM** at mitral area (**Austin Flint**) • Diastolic murmur in left axilla (**Cole-Cecil murmur**)	**Blowing PSM:** At the lower-left sternal border that is increased during inspiration and reduced during expiration (**de-Carvallo's sign**).	**ESM** in pulmonary area and MDM in tricuspid area. Once Eisenmenger's —**EDM** in pulmonary area and PSM in tricuspid area	**PSM** h at the sterna (3rd, 4 5th int space
Other features		Palpable P2 (diastolic shock)	Palpable P2 (diastolic shock)	--	Peripheral signs	Pulsatile liver	Precordial bulge	Aortic insuffi approx 5%

(AR: aortic regurgitation; AS: aortic stenosis; ASD: atrial septal defect; ESM: ejection–systolic murmur; EDM: early diastolic murmur; MDM: mid-diastolic murmur; MR: mitral regurgitation; MS: mitral stenosis; PS: pulmonary stenosis; PDA: patent ductus arteriosus; PSM: pansystolic murmur; TR: tricuspid regurgitation; VSD: ventricular septal defect)

Gastrointestinal System

CHAPTER 5

A. CASE SHEET FORMAT

HISTORY TAKING

Name:

Age:

Sex:

Residence:

Occupation:

Chief Complaints

1. _____ × days
2. _____ × days
3. _____ × days

History of Presenting Illness

Abdominal distention:

- Duration
- Onset
- Progression
- Aggravating factors
- Relieving factors
- Associated symptoms
- Is it preceded by pedal edema or followed by it?

Pedal edema:

- Duration
- Onset
- Progression
- Aggravating factors
- Relieving factors
- Is it preceded by facial puffiness or followed by it?

Abdominal pain:

- Onset
- Site
- Type of pain
- Radiation
- Aggravating factors
- Relieving factors
- Associated symptoms

Nausea and vomiting:

- Episodes
- Contents
- Blood tinged or not

- How many hours after consumption of food associated with pain abdomen?
- Conditions with nausea and vomiting but not associated with pain abdomen:
  - Metabolic
  - Neurologic
  - Drug induced
  - Psychogenic

Other symptoms:

Heart burn, flatulence, and waterbrash

Hematemesis and melena

Dysphagia

Constipation and diarrhea

Altered bowel habit:

- Stool color
- Stool odor
- Stool frequency
- Blood tinged or melena

Jaundice—itching and high colored urine

Fever

Weight loss

Pain in oral cavity

Halitosis

Hiccups

Other relevant history

Past history:

- Asthma
- Chronic obstructive airway disease
- Tuberculosis
- History of contact with tuberculosis
- Diabetes mellitus (DM)
- Hypertension (HTN)
- Ischemic heart disease (IHD)
- Seizure disorder

Family history:

- Draw a three generations pedigree chart

Personal history:

- Bowel habits
- Bladder habits
- Appetite
- Loss of weight
- Occupational exposure
- Sleep
- Dietary habits and taboo
- Food allergies

- Smoking index or pack years
- Alcohol history

Menstrual and obstetric history
- G__P__L__A__
- Age of menarche __
- Menopause at __
- Flow—ameno/oligo/menorrhagia

Summarize:

Differential diagnosis:
1.
2.
3.

GENERAL EXAMINATION

Patient

- Conscious
- Coherent
- Cooperative
- Obeying commands

Body Mass Index (BMI)

- Weight (kg)/Height^2 (meters)
- Grading according to WHO for Southeast Asian countries

Vitals

- **Pulse**
  - Rate:
  - Rhythm:
  - Volume:
  - Character:
  - Vessel wall thickening:
  - Radio-radial delay and radio-femoral delay:
  - Peripheral pulses:
- **Blood pressure**
  - Right arm:
  - Left arm:
  - Right leg
  - Left leg
- **Respiratory rate**
  - Regular/irregular
  - Abdominothoracic/thoracoabdominal
  - Usage of accessory muscles:
- **Jugular venous pressure**
  - __ cm of blood above sternal angle (+ 5 cm water from right atrium)
- **Jugular venous pulse**

- Waveform (describe waves)

On Physical Examination

- Pallor:
- Icterus:
- Cyanosis:
- Clubbing:
- Lymphadenopathy:
- Edema:

Other Head to Toe Signs of Chronic Liver Cell Failure

1. Alopecia
2. Fetor hepaticus
3. Jaundice
4. Parotid swelling
5. Gynecomastia
6. Testicular atrophy
7. Loss of secondary sexual characters
8. Spider nevi
9. Palmar erythema
10. Dupuytren's contracture
11. Asterixis
12. Xanthelasma
13. Signs of chronic cholestasis (scratch marks due to pruritus).

SYSTEMIC EXAMINATION

The order of examination of abdomen is preferably done—Inspection→Auscultation→Palpation→Percussion (as the auscultatory findings might change post palpation and percussion).

Inspection:

- Spine
- Shape/distention (localized/generalized) and flanks (free/full)
- Skin over the abdomen
- Symmetry
- Umbilicus
- Movement of corresponding quadrants with respiration
- Dilated veins
- Visible mass
- Visible pulsations
- Visible peristalsis
- Scars or sinuses
- Divarication of recti

Palpation:

- Superficial palpation
  - Warmth
  - Tenderness

  - Guarding
  - Rigidity
- Deep palpation
  - Liver
    - Size
    - Shape
    - Border or edge
    - Surface
    - Tenderness
    - Consistency
    - Movement with respiration
    - Pulsation
  - Spleen
    - Location
    - Size
    - Shape
    - Consistency
    - Surface
    - Edge
    - Tenderness
    - Movement with respiration
  - Gallbladder
  - Other palpable mass
- Bimanual palpation
  - Kidneys
    - Location
    - Size
    - Shape
    - Consistency
    - Surface
    - Edge
    - Tenderness
    - Movement with respiration
- Dipping method (in case of large ascites)
- Hernia orifices
- Direction of flow in veins (if dilated veins present)
- Abdominal girth measurement
- Spino-umbilical distance
- Xiphisternum to umbilicus distance (x) in cms
- Umbilicus to pubic symphysis distance in cms (y)
- Ratio of x/y

Percussion:

- Liver
- Spleen
- Traube's space
- Fluid
  - Shifting dullness

  - Fluid thrill
  - Puddle sign

Auscultation:

- Bowel sounds
- Succussion splash
- Bruit
- Venous hum
- Friction rub

Examination of

- Scrotum
- Spine
- Supraclavicular fossa

Per Rectal Examination

Per Vaginal Examination

NOTES

B. DIAGNOSIS FORMAT

CIRRHOSIS/LIVER DISEASE

- Acute hepatitis <4 weeks
  or
  Subacute hepatitis
  or
  Chronic (cirrhosis/hepatitis >6 months)
  or
  Acute on chronic liver disease (ACLD)
- Compensated or decompensated
- Possible etiology—alcohol/postviral/toxin/nonalcoholic steatohepatitis (NASH)
- With complications—portal hypertension with or without gastrointestinal (GI) bleed/hepatic encephalopathy (preferable to mention stage)/spontaneous bacterial peritonitis/hepatocellular carcinoma/hepatorenal syndrome/others.

EXAMPLE

Decompensated chronic liver disease—cirrhosis secondary to alcohol, with portal hypertension, with upper gastrointestinal (UGI) bleed, patient in stage 2 hepatic encephalopathy with no evidence of spontaneous bacterial peritonitis or other complications.

NOTES

C. DISCUSSION ON CARDINAL SYMPTOMS

ABDOMINAL SWELLING

Abdominal swelling is a manifestation of numerous diseases. Patients may complain of bloating or abdominal fullness. Patients with abdominal distension from *ascites* may report the new onset of an inguinal or umbilical hernia. Dyspnea may result from pressure against the diaphragm.

Causes

The causes of abdominal swelling can be remembered conveniently as the *seven Fs*: flatus, fat, fluid, fetus, feces, full bladder, or a "fatal growth"/neoplasm.

Flatus	• The normal small intestine contains ~200 mL of gas made up of nitrogen, oxygen, carbon dioxide, hydrogen, and methane • *Aerophagia*, the swallowing of air, can result in increased amounts of oxygen and nitrogen in the small intestine and lead to abdominal swelling • Increased intestinal gas is the consequence of bacterial metabolism of excess fermentable substances such as lactose and other oligosaccharides, which can lead to production of hydrogen, carbon dioxide, or methane
Fat	• Weight gain with an increase in abdominal fat can result in an increase in abdominal girth • Visceral obesity is associated with metabolic syndrome, insulin resistance, and cardiovascular disease • It also can be a manifestation of certain diseases, such as Cushing's syndrome
Fluid	The accumulation of fluid within the abdominal cavity (ascites) often results in abdominal distension
Fetus	Pregnancy results in increased abdominal girth. Typically, an increase in abdominal size is first noted at 12–14 weeks of gestation, when the uterus moves from the pelvis into the abdomen
Feces	In the setting of severe constipation or intestinal obstruction, increased stool in the colon leads to increased abdominal girth. These conditions are often accompanied by abdominal discomfort or pain, nausea, and vomiting and can be diagnosed by imaging studies
Fatal growth/neoplasm	An abdominal mass can result in abdominal swelling. Neoplasms, abscesses, or cysts can grow to sizes that lead to increased abdominal girth. Enlargement of the intra-abdominal organs, specifically the liver (hepatomegaly) or spleen (splenomegaly), or an abdominal aortic aneurysm can result in abdominal distension
Full bladder	Bladder distension also may result in lower abdominal swelling. It will be associated with anuria

JAUNDICE

Discussed in detail in Chapter 2C: Physical Examination.

GASTROINTESTINAL BLEEDING

Gastrointestinal bleeding (GIB) presents as either overt or occult bleeding.

Overt GIB	Occult GIB
Overt GIB is manifested by *hematemesis*, vomitus of red blood, or "coffee-grounds" material; *melena*, black, tarry stool; and/or *hematochezia*, passage of red or maroon blood from the rectum	***Occult GIB*** may present with *symptoms of blood loss or anemia,* such as lightheadedness, syncope, angina, or dyspnea; or with iron deficiency anemia or a positive fecal occult blood test on routine testing

GIB is also categorized by the site of bleeding as:

1. UGIB (esophagus, stomach, and duodenum)

2. LGIB (colonic), small intestinal, or obscure GIB (if the source is unclear).

Hematemesis is the vomiting of blood, which may be obviously red or have an appearance similar to coffee grounds.

Melena is the passage of black, tarry stools due to altered blood (blood should remain in the gut for 14 hours approximately). It usually means bleeding episodes from sites above the ligament of Treitz. However, even up to middle of transverse colon can produce melena. It takes 60 mL or more of blood in the stomach to turn stools black. One episode of bleed can produce 5–7 episodes of melena.

Hematochezia is the passage of fresh blood per anus, usually in or with stools.

Upper Gastrointestinal Sources of Bleeding

Causes		
Esophageal causes	***Gastric causes***	***Duodenal causes***
• Esophageal varices • Esophagitis • Esophageal cancer • Esophageal ulcers • Malory–Weiss tear	• Gastric ulcer • Gastric cancer • Gastritis • Gastric varices • Dieulafoy's lesions • Gastric antral vascular ectasia • Portal hypertensive gastropathy	• Duodenal ulcer • Vascular malformations including aortoenteric fistulae • Hemobilia or bleeding from biliary tree • Hemosuccus pancreaticus or bleeding from the pancreatic duct • Severe superior mesenteric artery syndrome

Lower Gastrointestinal Bleeding (Fig. 5C.1)

Causes of LGI bleeding	
Colonic bleeding (95%)	***Small intestinal bleeding (5%)***
• Diverticular disease	• Angiodysplasia
• Anorectal disease (hemorrhoid, anal fissure, fistula in ano, solitary rectal ulcer, etc.)	• Crohn's disease and infectious disease
• Neoplasia (polyp, ulcerated lesions)	• Neoplasia (polyp, ulcerated lesions)
• Inflammatory bowel disease	• Radiation
• Infectious collitis	
• Angiodysplasia	• Meckel's diverticulum
• Radiation collitis/proctitis	• Aortoenteric fistula
• Other	• Mesenteric ischemia

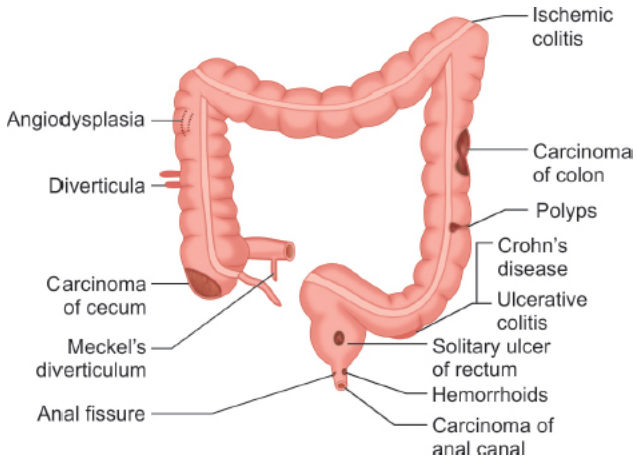


Fig. 5C.1: Lower gastrointestinal bleeding.

NAUSEA AND VOMITING (TABLE 5C.1)

Definitions

Nausea is the subjective feeling of a need to vomit. **Vomiting** (emesis) is the oral expulsion of gastrointestinal contents due to gut and thoracoabdominal wall contractions.

Mechanism of Initiation of Emesis

Brainstem nuclei—including the nucleus tractus solitarius; dorsal vagal and phrenic nuclei; medullary nuclei regulating respiration; and nuclei that control pharyngeal, facial, and tongue movements—coordinate initiation of emesis involving neurokinin NK1, serotonin 5-HT3, and vasopressin pathways.

Clinical Clues for Diagnosis

1. Gastroparesis and pyloric obstruction elicit vomiting within an hour of eating.
2. Emesis from intestinal blockage occurs later.
3. Vomiting occurring minutes after meal consumption prompts consideration of rumination syndrome.
4. With severe gastric emptying delays, the vomitus may contain food residue ingested days before.
5. Feculent emesis is noted with distal intestinal or colonic obstruction.
6. Bilious vomiting excludes gastric obstruction, whereas emesis of undigested food is consistent with a Zenker's diverticulum or achalasia.
7. Vomiting can relieve abdominal pain from a bowel obstruction, but has no effect in pancreatitis or cholecystitis.
8. Profound weight loss raises concern about malignancy or obstruction.
9. An intracranial source is considered if there are headaches or visual field changes.
10. Vertigo or tinnitus indicates labyrinthine disease.

Projectile vomiting is a type of severe **vomiting** in which stomach contents are forcefully propelled several feet away from the patient and is usually not associated with nausea. It is a classical feature of raised intracranial tension.

DIARRHEA

Definitions

Diarrhea is loosely defined as passage of abnormally liquid or unformed stools at an increased frequency. For adults on a typical Western diet, stool weight >200 g/d can generally be considered as

diarrhea.

Diarrhea may be further defined as *acute* if <2 weeks, *persistent* if 2–4 weeks, and *chronic* if >4 weeks in duration.

Table 5C.1: Causes of nausea and vomiting.		
Intraperitoneal	***Extraperitoneal***	***Medications/Metabolic disorders***
Obstructing disorders • Pyloric obstruction • Small bowel obstruction • Colonic obstruction • Superior mesenteric artery syndrome **Enteric infections** • Viral • Bacterial **Inflammatory diseases** • Cholecystitis • Pancreatitis • Appendicitis • Hepatitis **Altered sensorimotor functions** • Gastroparesis • Intestinal pseudo-obstruction • Gastroesophageal reflux • Chronic nausea vomiting syndrome • Cannabinoid hyperemesis syndrome • Rumination syndrome **Biliary colic** **Abdominal irradiation**	**Cardiopulmonary disease** • Cardiomyopathy • Myocardial infarction **Labyrinthine disease** • Motion sickness • Labyrinthitis **Intracerebral disorders** • Malignancy • Hemorrhage • Abscess • Hydrocephalus **Psychiatric illness** • Anorexia and bulimia nervosa • Depression **Postoperative vomiting**	**Drugs** • Cancer chemotherapy • Antibiotics • Antiarrhythmic drugs • Digoxin • Oral hypoglycemic agents • Oral contraceptives • Antidepressants • Anti-Parkinson's agents • Smoking cessation agents **Endocrine/metabolic disease** • Pregnancy • Uremia • Ketoacidosis • Thyroid and parathyroid disease • Adrenal insufficiency **Toxins** • Ethanol

Types of Diarrhea

1. **Inflammatory diarrhea** is characterized by frequent, small-volume, bloody stools and may be accompanied by tenesmus, fever, or severe abdominal pain. Inflammatory diarrhea is suspected with the demonstration of leukocytes or leukocyte proteins (e.g. calprotectin or lactoferrin) on stool examination.
2. **Fatty stools** are suggested by a history of weight loss, greasy or bulky stools that are difficult to flush, and oil in the toilet bowl that requires a brush to remove. **Floating stools** indicate gas production by colonic bacteria, not steatorrhea.
3. **Watery diarrhea** can be further classified as osmotic or secretory in origin. **Osmotic diarrhea** is due to the ingestion of poorly absorbed ions or sugars. **Secretory diarrhea** is due to disruption of epithelial electrolyte transport.

Large-volume versus small-volume diarrhea	
Large-volume diarrhea	***Small-volume diarrhea***
Right colonic or small bowel disorders	Left colonic disorders
The rectosigmoid reservoir is intact	Compromises the rectosigmoid reservoir capacity
Individual bowel movements are less frequent and larger	Frequent small-volume bowel movements

Normal rectosigmoid colon functions as a storage reservoir.

Acute diarrhea	***Chronic diarrhea***
More than 90% of cases of acute diarrhea are caused by infectious agents; these cases are often accompanied by vomiting, fever, and abdominal pain. The remaining 10% are caused by medications, toxic ingestions, ischemia, food indiscretions, and other conditions **(Table 5C.2)**	Diarrhea lasting >4 weeks warrants evaluation to exclude serious underlying pathology. In contrast to acute diarrhea, most of the causes of chronic diarrhea are noninfectious **(Table 5C.3)**

Table 5C.2: Causes of acute diarrhea.	
Viral infection	Viral gastroenteritis; Norovirus or rotavirus
Bacterial infection	*Campylobactor, Escherichia coli, Salmonella or shigella*
Parasitic infection	*Cryptosporidium, Entamoeba histolytica* or *giardia*
Traveler's diarrhea	Consuming food or drinks contaminated with bacteria, parasites or viruses
Medication	Antibiotics and long-term use of proton pump inhibitors, increased risk of *Clostridium difficile* infections
Food allergy or intolerance	Cow's milk, egg, seafood, soy or fructose or lactose intolerance
Digestive disorder	Celiac disease, Crohn's disease, irritable bowel syndrome or ulcerative colitis
Artificial sweetener	Mannitol, sorbitol or xylitol found in sugar-free candies or gums

Table 5C.3: Causes of chronic diarrhea.	
Fatty diarrhea	***Watery diarrhea***
• *Malabsorption syndromes*: – Mucosal diseases, (e.g., celiac disease, Whipple's disease) – Mesenteric ischemia – Short bowel syndrome – Small intestinal bacterial growth. • *Maldigestion*: – Inadequate luminal bile acid concentration – Pancreatic exocrine insufficiency ***Inflammatory diarrhea*** • Diverticulitis • Infectious diseases: – Invasive bacterial infections (e.g. tuberculosis and yersiniosis) – Invasive parasitic infections (e.g. amebiasis and strongyloidiasis) – Pseudomembranous colitis (*Clostridium difficile* infection) – Ulcerating viral infections (cytomegalovirus, herpes simplex virus). • Inflammatory bowel diseases: Crohn's disease, ulcerative colitis • Ischemic colitis • Neoplasia: carcinoma of colon, lymphoma • Radiation colitis	• Osmotic diarrhea: – Carbohydrate malabsorption – Osmotic laxatives • Secretory diarrhea • Bacterial toxins • Congenital syndromes (e.g. congenital chloridorrhea) • Disordered motility, regulation: – Diabetic autonomic neuropathy – Irritable bowel syndrome – Postsympathectomy diarrhea – Postvagotomy diarrhea • Diverticulitis • Endocrinopathies: Addison's disease, carcinoid syndrome, gastrinoma, hyperthyroidism, mastocytosis, medullary carcinoma of thyroid, pheochromocytoma, somatostatinoma, and vipoma • Laxative abuse (stimulant laxatives) • Medication and toxins

Mimics of Diarrhea

Pseudodiarrhea, or the frequent passage of small volumes of stool, is often associated with rectal urgency, tenesmus, or a feeling of incomplete evacuation, and accompanies IBS or proctitis.

Fecal incontinence is the involuntary discharge of rectal contents and is most often caused by neuromuscular disorders or structural anorectal problems.

Overflow diarrhea may occur in nursing home patients due to fecal impaction that is readily detectable by rectal examination.

CONSTIPATION

Definition

Constipation refers to bowel movements that are infrequent or hard to pass.

Obstipation is intractable constipation that has become refractory to cure or control. There is inability to pass any feces or flatus.

Tenesmus is stated by patients as the unpleasant symptom that there remains something to evacuate from the rectum despite passing a stool. It is often painful. It indicates rectal inflammation.

Etiology of constipation	
Functional (nonorganic) or retentive	Includes constipation due to fecal withholding behaviors and when all organic causes have been ruled out
Anatomic causes	Include anal stenosis or atresia, anteriorly displaced anus, imperforate anus, intestinal stricture, and anal stricture
Abnormal musculature	Related causes include prune belly syndrome, gastroschisis, Down syndrome, and muscular dystrophy
Intestinal nerve abnormality	Related causes include Hirschsprung disease, pseudo-obstruction, intestinal neuronal dysplasia, spinal cord defects, tethered cord, and spina bifida
Drugs	Like anticholinergics, narcotics, antidepressants, lead, and vitamin D intoxication
Metabolic and endocrine causes	Like hypokalemia, hypercalcemia, hypothyroidism, diabetes mellitus (DM), or diabetes insipidus
Other causes	Include celiac disease, cystic fibrosis, cow milk protein allergy, inflammatory bowel disease, scleroderma among others

DYSPEPSIA

Definition

Rome III criteria for dyspepsia
≥1 of the following:
1. Postprandial fullness 2. Early satiation (inability to finish a normal-sized meal) 3. Epigastric pain or burning

Table 5C.4: Causes of dyspepsia.
Luminal gastrointestinal tract
• Chronic gastric or intestinal ischemia • Food intolerance • Functional dyspepsia • Gastroesophageal reflux disease • Gastric or esophageal neoplasms • Gastric infections (e.g. cytomegalovirus, fungus, tuberculosis, and syphilis) • Gastroparesis (e.g. diabetes mellitus, postvagotomy, scleroderma, chronic intestinal pseudo-obstruction, postviral, and idiopathic)

• Irritable bowel syndrome • Peptic ulcer disease • Parasites (e.g. *Giardia lamblia*, *Strongyloides stercoralis*)
Medications
Acarbose, aspirin, other nonsteroidal anti-inflammatory drugs (including cyclooxygenase-2 selective agents), colchicine, digitalis preparations, estrogens, ethanol, glucocorticoids, iron, levodopa, niacin, narcotics, nitrates, orlistat, potassium chloride, quinidine, sildenafil, and theophylline
Pancreaticobiliary disorders
• Biliary pain: cholelithiasis, choledocholithiasis, and sphincter of Oddi dysfunction • Chronic pancreatitis • Pancreatic neoplasms
Systemic conditions
Adrenal insufficiency, congestive heart failure, diabetes mellitus, hyperparathyroidism, myocardial ischemia, pregnancy, renal insufficiency, and thyroid disease

DYSPHAGIA

Definition

Dysphagia, from the Greek dys (difficulty, disordered) and phagia (to eat), refers to the sensation that food is hindered in its passage from the mouth to the stomach.

Table 5C.5: Causes of oropharyngeal dysphagia.

Neuromuscular causes	*Structural causes*
• Amyotrophic lateral sclerosis (ALS) • Multiple sclerosis • Muscular dystrophy • Myasthenia gravis • Parkinson's disease • Polymyositis or dermatomyositis • Stroke • Thyroid dysfunction	• Carcinoma • Infections of pharynx or neck • Osteophytes or other spinal disorders • Prior surgery or radiation therapy • Proximal esophageal web • Plummer-Vinson syndrome • Thyromegaly • Zenker's diverticulum

Table 5C.6: Common causes of esophageal dysphagia.

Motility (neuromuscular) disorders	*Structural (mechanical) disorders*
Primary disorders: • Achalasia • Diffuse esophageal spasm • Hypertonic lower esophageal sphincter (LES) • Ineffective esophageal motility • Nutcracker (high pressure esophagus).	***Intrinsic factors***: • Carcinoma and benign tumors • Diverticula • Eosinophilic esophagitis • Esophageal rings and webs (except Schatzki ring) • Foreign body • Lower esophageal (Schatzki) ring • Medication-induced stricture • Peptic stricture
Secondary disorders: • Chagas disease • Reflux-related dysmotility • Scleroderma and other rheumatological disorders	***Extrinsic factors***: • Mediastinal mass • Spinal osteophytes • Vascular compression

ODYNOPHAGIA

Definition

Odynophagia, or painful swallowing, is a specific feature for esophageal involvement. It usually reflects an inflammatory process in the esophageal mucosa.

Table 5C.7: Causes of odynophagia.

Caustic ingestion: Acid alkali

Pill-induced injury:

- Alendronate and other bisphosphonates
- Aspirin and other NSAIDs
- Iron preparations
- Potassium chloride (especially slow release form)
- Tetracycline and its derivatives
- Quinidine
- Zidovudine

Infectious esophagitis:

- *Viral*: Cytomegalovirus, Epstein-Barr virus, herpes simples virus, and human immunodeficiency virus
- *Bacteria*: Mycobacteria (tuberculosis or *Mycobacterium avium* complex)
- *Fungal*: *Candida albicans*, histoplasmosis
- *Protozoan*: *Cryptosporidium*, *Pneumocystis*

Severe reflux esophagitis

Esophageal carcinoma

PAIN IN ABDOMEN

The history of a patient with abdominal pain includes determining whether the pain is acute or chronic and a detailed description of the pain and associated symptoms, which should be interpreted with other aspects of the medical history.

Acute versus Chronic Pain

There is no strict time period that will classify the differential diagnosis unfailingly. A clinical judgment must be made that considers whether this is an accelerating process, one that has reached a plateau, or one that is long-standing but intermittent. Patients with chronic abdominal pain may present with an acute exacerbation of a chronic problem or a new and unrelated problem. Pain of less than a few days' duration that has worsened progressively until the time of presentation is clearly "acute". Pain that has remained unchanged for months or years can be safely classified as chronic. Pain that does not clearly fit either category might be called subacute and requires consideration of a broader differential than acute and chronic pain.

Description of Pain

Pain is discussed under following headings:

1. **Location and radiation:** the location of abdominal pain helps narrow the differential diagnosis as different pain syndromes typically have characteristic locations (described in the tables below). For example, pain involving the liver or biliary tree is generally located in the right upper quadrant, but it may radiate to the back or epigastrium. Because hepatic pain only results when the capsule of the liver is "stretched", most pain in the right upper quadrant is related to the biliary tree. Pain radiation is also important: the pain of pancreatitis classically bores to the back, while renal colic radiates to the groin.
2. **Temporal elements**: the onset, frequency, and duration of the pain are helpful features. The pain of pancreatitis may be gradual and steady, while perforation and resultant peritonitis begins suddenly and is maximal from the onset.
3. **Quality**: the quality of the pain includes determining whether the pain is burning or gnawing, as is typical of gastroesophageal reflux and peptic ulcer disease, or colicky, as in the cramping pain of

gastroenteritis or intestinal obstruction.

4. **Severity**: the severity of the pain generally is related to the severity of the disorder, especially if acute in onset. For example, the pain of biliary or renal colic or acute mesenteric ischemia is of high intensity, while the pain of gastroenteritis is less marked. Age and general health may affect the patient's clinical presentation. A patient taking corticosteroids may have significant masking of pain, and older adult patients often present with less intense pain.
5. **Precipitants or palliation**: determining what precipitates or palliates the pain can help narrow the differential. The pain of chronic mesenteric ischemia usually starts within one hour of eating, while the pain of duodenal ulcers may be relieved by eating and recur several hours after a meal.
6. **Position/posture:** the pain of pancreatitis is classically relieved by sitting up and leaning forward. Peritonitis often causes patients to lie motionless on their backs because any motion causes pain. Obtaining a history of pain occurring in relationship to eating lactose- or gluten-containing foods may be helpful in identifying sensitivities to these food constituents. Patients with foodborne illness may become ill after eating certain foods.

Associated Symptoms

- **Other gastrointestinal symptoms:** we ask about associated nausea, vomiting, diarrhea, constipation, hematochezia, melena, and changes in stool (e.g. change in caliber). For patients with right upper quadrant pain or concern for liver disease, we also ask about jaundice and changes in the color of urine and stool. The bowel habit is an important part of the history for chronic abdominal pain. While many organic lesions can result in chronic diarrhea, irritable bowel syndrome (IBS) often presents with swings between diarrhea and constipation, a pattern that is much less likely with organic disease.
- **Genitourinary symptoms:** patients with symptoms such as dysuria, frequency, and hematuria are more likely to have a genitourinary cause for their abdominal pain.
- **Constitutional symptoms:** symptoms such as fever, chills, fatigue, weight loss, and anorexia would be concerning for infection, malignancy, or systemic illnesses [e.g. inflammatory bowel disease (IBD)].
- **Cardiopulmonary symptoms:** symptoms, such as cough, shortness of breath, orthopnea, and exertional dyspnea suggest a pulmonary or cardiac etiology. Orthostatic hypotension may indicate early shock or be associated with adrenal insufficiency.
- **Other:** patients with diabetic ketoacidosis will have symptoms of polyuria and thirst. Patients with suspected IBD should be asked about extraintestinal manifestations.

Other Medical History

- **Specific questions for women**: women should be screened for sexually transmitted diseases and risks for pelvic inflammatory disease (e.g. new or multiple partners). Premenopausal women should be asked about their menstrual history (last menstrual period, last normal menstrual period, and cycle length) and use of contraception. They should also be asked about vaginal discharge or bleeding, dyspareunia, or dysmenorrhea, as these symptoms suggest a pelvic pathology.
- **Past medical history**: a history of surgeries and procedures should be obtained to assess risk for differing etiologies (e.g. a history of abdominal surgery is a risk factor for obstruction). A history of cardiovascular disease (CVD) or multiple risk factors for CVD in a patient with epigastric pain raises concern for a myocardial ischemia.
- **Medications**: a comprehensive medication list should be elicited as this can inform the differential. For example, patients taking high doses of nonsteroidal anti-inflammatory drugs (NSAIDs) are at risk for gastropathy and peptic ulcer disease. Patients with recent antibiotic use or hospitalization are at risk for *Clostridioides* (formerly *Clostridium*) *difficile*. Patients on chronic steroids are at risk for adrenal insufficiency and may be immunosuppressed with atypical presentations of abdominal pain.
- **Other history:** Alcohol—it is important to ask about alcohol intake to assess for the possibility of liver disease and pancreatitis.
- **Family history**: family history should be asked as appropriate based on other history. For example, patients with history concerning for IBD or cancer should also be asked about family history.

- **Travel history:** a travel history is important to elicit in patients with symptoms consistent with gastroenteritis or colitis (e.g. nausea, vomiting, and diarrhea) to consider infectious etiologies.
- **Sick contacts:** often patients are in contact with someone with gastroenteritis before having similar symptoms. Patients with foodborne illness may also have close contact with similar illness.

Site of Pain and Possible Etiology

Causes of right upper quadrant (RUQ) abdominal pain.	
RUQ	***Clinical features***
Biliary	
Biliary colic	Intense dull discomfort located in the RUQ or epigastrium. Associated with nausea, vomiting, and diaphoresis. Generally lasts at least 30 minutes plateauing within 1 hour. Benign on abdominal examination
Acute cholecystitis	Prolonged (>4–6 hours), RUQ or epigastric pain, fever. Patients will have abdominal guarding and Murphy's sign
Acute cholangitis	Fever, jaundice, and RUQ pain
Sphincter of Oddi dysfunction	RUQ pain similar to other biliary pain
Hepatic	
Acute hepatitis	RUQ pain with fatigue, malaise, nausea, vomiting, and anorexia. Patients may also have jaundice, dark urine, and light-colored stools
Perihepatitis (Fitz-Hugh-Curtis syndrome)	RUQ pain with a pleuritic component. Pain is sometimes referred to the right shoulder
Liver abscess	Fever and abdominal pain are the most common symptoms
Budd–Chiari syndrome	Symptoms include fever, abdominal pain, abdominal distension (from ascites), lower extremity edema, jaundice, gastrointestinal bleeding, and/or hepatic encephalopathy
Portal vein thrombosis	Symptoms include abdominal pain, dyspepsia, or gastrointestinal bleeding

Causes of epigastric abdominal pain	
Epigastric	***Clinical features***
Acute myocardial infarction	May be associated with shortness of breath and exertional symptoms
Acute pancreatitis	Acute onset, persistent upper abdominal pain radiating to the back
Chronic pancreatitis	Epigastric pain radiating to the back
Peptic ulcer disease	Epigastric pain or discomfort is the most prominent symptom
Gastroesophageal reflux disease	Associated with heartburn, regurgitation, and dysphagia
Gastritis/gastropathy	Abdominal discomfort/pain, heartburn, nausea, vomiting, and hematemesis
Functional dyspepsia	The presence of one or more of the following: postprandial fullness, early satiation, epigastric pain, or burning
Gastroparesis	Nausea, vomiting, abdominal pain, early satiety, postprandial fullness, and bloating

Causes of left upper quadrant (LUQ) abdominal pain	
LUQ	***Clinical features***
Splenomegaly	Pain or discomfort in LUQ, left shoulder pain, and or early satiety

Splenic infarct	Severe LUQ pain
Splenic abscess	Associated with fever or LUQ tenderness
Splenic rupture	May complain of LUQ, left chest wall, or left shoulder pain that worsens with inspiration

Causes of lower abdominal pain		
Lower abdomen	***Localization***	***Clinical features***
Appendicitis	Generally right lower quadrant	Periumbilical pain initially that radiates to the right lower quadrant. Associated with anorexia, nausea, and vomiting
Diverticulitis	Generally left lower quadrant, right lower quadrant more common in Asian patients	Pain usually constant and present for several days prior to presentation. May have associated nausea and vomiting
Nephrolithiasis	Either	Pain most common symptom, varies from mild-to-severe. Generally flank pain but may have back or abdominal pain
Pyelonephritis	Either	Associated with dysuria, frequency, urgency, hematuria, fever, chills, flank pain, and costovertebral angle tenderness
Acute urinary retention	Suprapubic	Present with lower abdominal pain and discomfort, inability to urinate
Cystitis	Suprapubic	Associated with dysuria, frequency, urgency, and hematuria
Infectious colitis	Either	Diarrhea is the predominant symptom, but may also have associated abdominal pain which may be severe

Causes of diffuse abdominal pain	
Diffuse/poorly characterized	***Clinical features***
Bowel obstruction	• Most common symptoms are nausea, vomiting, crampy abdominal pain, and obstipation • Distended tympanic abdomen with high-pitched or absent bowel sounds.
Perforation of the gastrointestinal tract	Severe abdominal pain, particularly following procedures
Acute mesenteric ischemia	Acute and severe onset of diffuse and persistent abdominal pain often described as pain out of proportion to examination
Chronic mesenteric ischemia	Abdominal pain after eating (“intestinal angina”), weight loss, nausea, vomiting, and diarrhea
Inflammatory bowel disease (ulcerative colitis/Crohn’s disease)	Associated with bloody diarrhea, urgency, tenesmus, bowel incontinence, weight loss, and fever
Viral gastroenteritis	Diarrhea accompanied by nausea, vomiting, and abdominal pain
Spontaneous bacterial peritonitis	Fever, abdominal pain, and/or altered mental status
Dialysis-related peritonitis	Abdominal pain and cloudy peritoneal effluent. Other symptoms and signs include fever, nausea, diarrhea, abdominal tenderness, and rebound tenderness
Colorectal cancer	Variable presentation, including obstruction and perforation
Other malignancy	Vary depending on malignancy
Celiac disease	Abdominal pain in addition to including diarrhea with bulky, foul smelling, floating stools due to steatorrhea and flatulence
Ketoacidosis	Diffuse abdominal pain, nausea and vomiting
Adrenal insufficiency	Diffuse abdominal pain, nausea and vomiting
Foodborne illness	Mixture of nausea, vomiting, fever, abdominal pain, and diarrhea
Irritable bowel syndrome	Chronic abdominal pain with altered bowel habits

Constipation	Diffuse abdominal pain
Diverticulosis	May have symptoms of abdominal pain and constipation
Lactose intolerance	Associated with abdominal pain, bloating, flatulence, and diarrhea. Abdominal pain may be cramping in nature

NOTES

D. DISCUSSION ON EXAMINATION

GENERAL EXAMINATION

General Physical Examination in Gastroenterology and Hepatobiliary System

Pulse
- Tachycardia—anemia, hypovolemia
- Bradycardia—obstructive jaundice
- High volume pulse—cirrhosis of liver

Blood pressure
- Wide pulse pressure—cirrhosis
- Low blood pressure—sepsis, UGI bleed

Fever
- SBP
- Hepatoma
- Cirrhosis
- Hepatitis
- Abscess
- Pancreatitis
- Inflammatory bowel disease

Pallor
- GI bleed
- Anemia of chronic disease
- Macrocytic anemia—liver disease, B_{12} and folate deficiencies

Icterus
- Hepatic
- Posthepatic

Cyanosis
- Hepatopulmonary syndrome
- Pleural effusion

Clubbing
- Primary biliary cirrhosis
- Inflammatory bowel disease
- HCC

Lymphadenopathy
- Tuberculosis
- HIV
- Lymphoma

Pedal edema
- Cirrhosis

- Nephrotic syndrome
- CKD

Peripheral Signs of Chronic Liver Disease

Skin, nail and hands

1. Spider nevi (telangiectatic superficial blood vessels with central feeding vessel)
2. Clubbing of hands (especially biliary cirrhosis and hepatocellular carcinoma)
3. Leukonychia
4. Palmar erythema (blotchy appearance over the thenar and hypothenar eminence)
5. Brusing
6. Dupuytren's contracture (sign of alcoholism)
7. Scratch marks (cholestatic jaundice)

Endocrine—due to estrogen excess

1. Gynecomastia
2. Atrophy of testis
3. Loss of axillary and pubic hair

Others

1. Parotid and lacrimal gland swelling (alcoholic liver disease)
2. Fetor hepaticus (characteristic sweet smelling breath)
3. Asterixis

Signs of Cirrhosis of Liver

Jaundice

- Jaundice is not a common feature of cirrhosis, its more common with acute diseases.
- Mechanisms of jaundice in cirrhosis:
  - Failure to excrete bilirubin (mainly)
  - Intrahepatic cholestasis (superadded hepatitis/tumor)
  - Hemolysis due to hypersplenism (not a major contributor).

Hepatomegaly

- **Early stages**: Liver is enlarged, firm to hard, irregular, and non-tender. Hepatomegaly is not common in cirrhosis but common when the cirrhosis is due to alcoholic liver disease, nonalcoholic steatohepatitis (NASH) and hemochromatosis. Hepatomegaly may indicate transformation into hepatocellular carcinoma (HCC).
- **Late stages:** Liver decreases in size and non-palpable due to progressive destruction of liver cells and accompanying fibrosis.

Ascites

- Ascites due to liver failure and portal hypertension.
- It signifies advanced disease.
  (discussed in detail below)

Spider Naevi

Spider nevi (Fig. 5D.1) (Spider telangiectasia; vascular spiders; spider angiomas; arterial spiders, and nevus araneus)	
Description	Consists of a central arteriole from which numerous small vessels radiate peripherally-resembling spider's legs. Whole spider disappears when central arteriole is compressed with a pinhead. When compression is released filling occurs from center to periphery

Pathophysiology	Due to arteriolar changes induced by hyperestrogenism	
Location	Usually found only in the necklace area, i.e. above the nipples, territory drained by the superior vena cava, such as: head and neck, upper limbs, front and back of upper chest	
Size	Vary from pinhead to 0.5 mm in diameter	
Clinical demonstration	Applying pressure over the body of spiders with a glass slide (diascopy) **(Fig. 5D.2)**, or pin head **(Fig. 5D.3)** leading to pallor with refilling following the release of pressure	
Significance	They are a strong indicator of liver disease but can be found in other conditions	
Causes	***Liver disorders***	***Others***
	• Viral hepatitis • Alcoholic hepatitis • Hepatocellular carcinoma • Treatment with sorafenib	• Third trimester of pregnancy • Rheumatoid arthritis • Thyrotoxicosis • Also normally seen in 2% of healthy population
Differential diagnosis	Venous star, Campbell de Morgan spots, petechiae, and hereditary hemorrhagic telangiectasias	

Note:

- *Florid spider telangiectasia, gynecomastia, and parotid enlargement are most common in* ***alcoholic hepatitis.***
- *Florid spiders and new onset clubbing in a patient with cirrhosis indicates* ***hepatopulmonary syndrome****.*

Palmar Erythema (Liver Palm)

- Can be seen early but is of limited diagnostic value, as it occurs in many conditions associated with a hyperdynamic circulation (e.g. normal pregnancy).

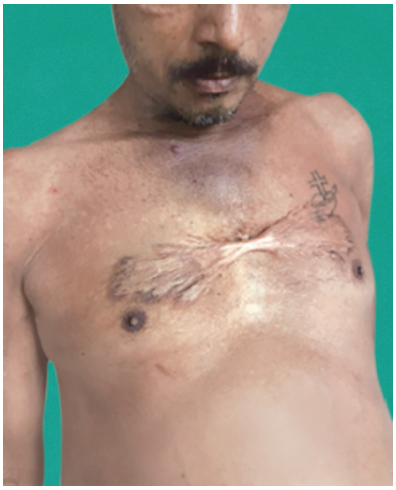


Fig. 5D.1: Cirrhosis of liver with ascites and spider nevi. Patient in addition has tattoo and keloid—which may suggest viral hepatitis as the cause of cirrhosis.

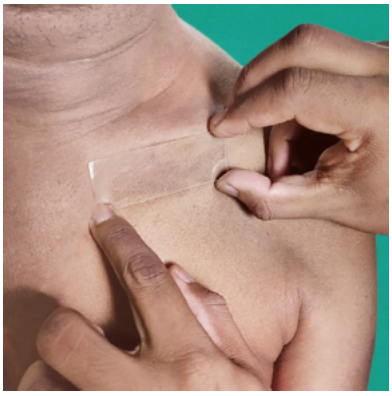


Fig. 5D.2: Demonstration of spider naevi (glass slide method).

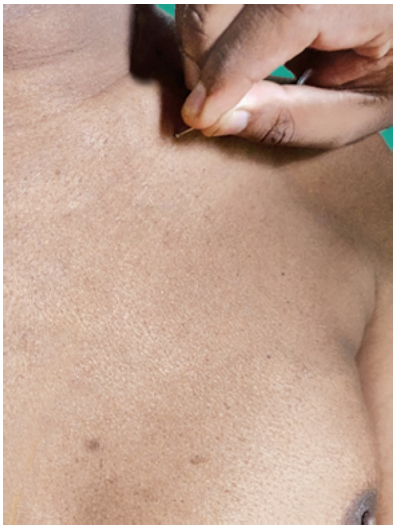


Fig. 5D.3: Demonstration of spider nevi (pin head method).

- **Cause:** Develops due to increased peripheral blood flow. In cirrhosis, circulatory changes results in increased peripheral blood flow and decreased visceral blood flow (especially to the kidneys).
- **Sites involved:** Prominent in the thenar and hypothenar eminences of palm. Spares the central portion of the palm. May be seen on the sole.

Endocrine Changes

- **Diminished body hair and loss of hair:** Seen mainly in males with loss of male hair distribution. Alopecia affects usually the face, axilla and chest and is due to hyperestrogenism. Causes of hyperestrogenism: Due to increased peripheral formation of estrogen resulting from diminished hepatic clearance of the precursor, androstenedione. Effects of hyperestrogenism: Alopecia, gynecomastia, and testicular atrophy.
- **Hyperglycemia**: 80% of cirrhotics have impaired glucose tolerance, 20% develop diabetes.
- **Gynecomastia (Fig. 5D.4):** Found in males (atrophy of breasts in females).
  - **Cause**: Due to increased estradiol/free testosterone ratio.
  - **Examination (Fig. 5D.5):** Appear as palpable nodule (4 cm, subareolar).
  - **Microscopy**: Proliferation of glandular tissue of breast.

Pseudogynecomastia is accumulation of subareolar fat tissue without palpable nodule. Seen in obesity and Cushing's syndrome:

Causes of gynecomastia

- Cirrhosis of liver
- Drugs:
  - Spironolactone
  - Cimetidine
  - Digoxin
  - Ketoconazole
  - Estrogens
  - Isoniazid/Antiandogens
- Physiological (puberty/ageing)
- Klinefelter's syndrome
- Hypogonadism
- Tumor:
  - Testes
  - Lung

Testicular Atrophy

Due to hyperestrogenic state, it is characterized by a small size compared with Prader's orchidometer **(Fig. 5D.6)**, soft testes with loss of testicular sensation (sickening sensation in epigastrium on squeezing the testes). The dimensions of the average adult testicle is 4.5 × 3.5 × 2.5 cm and the volume is 15–25 mL.

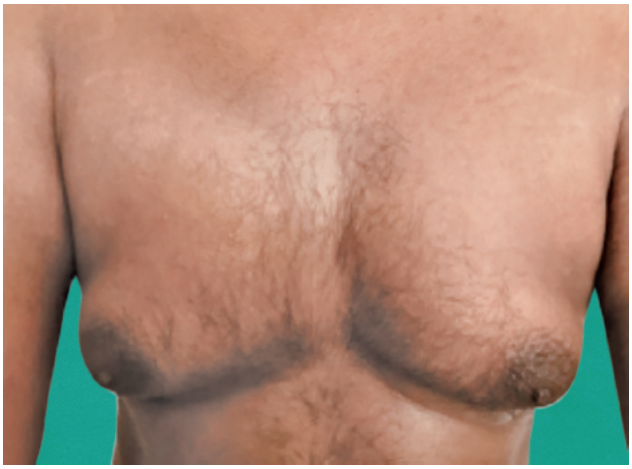


Fig. 5D.4: Gynecomastia.

Fig. 5D.5: Palpation breast bud in gynecomastia.

Fig. 5D.6: Prader's orchidometer.

Endocrine changes in females

Irregular menses, amenorrhea, and atrophy of breast.

Dupuytren's Contracture (It is a Sign of Alcoholism)

Pathophysiology	Fibrosis of palmar aponeurosis probably caused by local microvessel ischemia. Platelet and fibroblast-derived growth factors promote fibrosis
Sites involved	Flexion contracture of the fingers **(Fig. 5D.7)** (especially ring and little fingers)
Other causes of Dupuytren's contracture	Diabetes mellitus, rheumatoid arthritis, and manual labor (workers exposed to repetitive handling tasks or vibration).

Clubbing and Central Cyanosis

Due to development of pulmonary arteriovenous shunts that leading to hypoxemia **(Orthodeoxia—Platypnea syndrome)**.

Nail Changes

- **White (Terry's)** chalky and brittle nails **(Fig. 5D.8)**. And it can be easily demonstrated on comparison with normal person nails when placed side by side **(Fig. 5D.9)**.

- **Muehrcke's nails:** Characterized by transverse white lines that disappear on applying pressure and these lines do not move with growth of nail.
- **Clubbing** is present in primary biliary cirrhosis or hepatoma.

Parotid and Lacrimal Gland Enlargement (Fig. 5D.10)

Observed commonly in alcoholic cirrhosis due to associated autonomic dysfunction.

Anemia

It can be due to various causes:

- Acute and chronic blood loss from varices

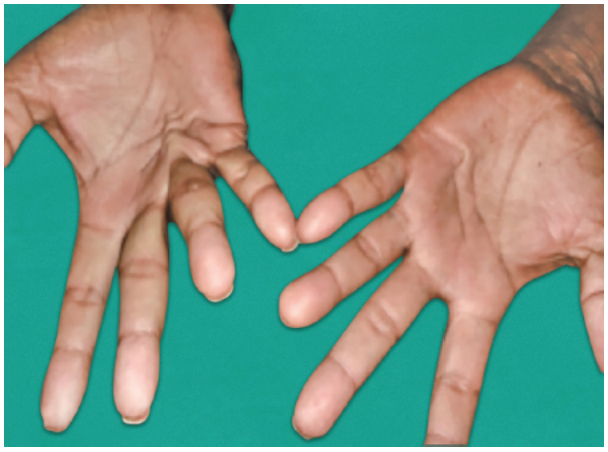


Fig. 5D.7: Dupuytren's contracture.

Fig. 5D.8: White nails.

Fig. 5D.9: Leukonychia—compare with nails of normal person (preferably hands to be placed side by side).

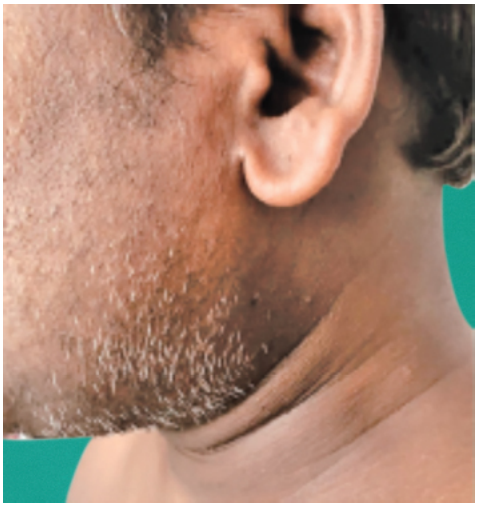


Fig. 5D.10: Diminished facial hair with parotid enlargement.

- Nutritional deficiency of vitamin B_{12} and folate
- Hypersplenism
- Bone marrow suppression by alcohol
- Hemolysis
- **Zeives syndrome:** Alcohol induced hemolytic anemia with hypercholestrolemia.

Fetor Hepaticus

- Sweet, pungent smell
- It is due to volatile **dimethylsulfide**, especially in portosystemic shunting and liver failure and hepatic encephalopathy.

Asterixis/Flapping Tremor

- Asterixis is a disorder of motor control characterized by an inability to actively maintain a position and consequent irregular myoclonic lapses of posture affecting various parts of the body independently.
- It is a type of negative myoclonus characterized by a brief loss of muscle tone in agonist muscles followed by a compensatory jerk of the antagonistic muscles.

- **Demonstration of asterixis of hand (Fig. 5D.11):** Asterixis is tested by extending the arms, dorsiflexing the wrists, and spreading the fingers to observe for the "flap" at the wrist. The flap is due to irregular myoclonic lapses of posture caused by involuntary 50–200 ms silent periods appearing in tonically active muscles.

Demonstration of asterixis of leg (Fig. 5D.12): Testing asterixis at the hip joint involves keeping the patient in a supine position with knees bent and feet flat on the table, leaving the legs to fall to the sides. Negative myoclonus of the lower limbs at the hip joints repetitively occurs and is appreciated by looking at the knees.

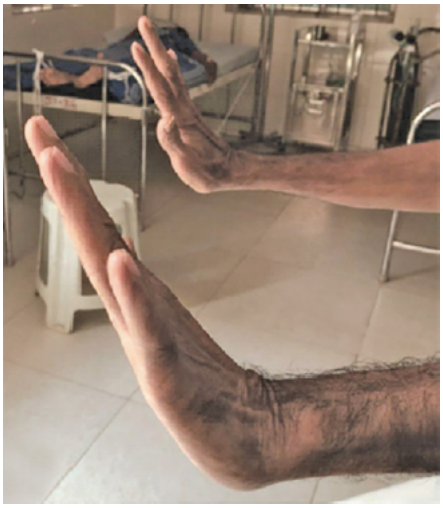


Fig. 5D.11: Demonstration of asterixis in hands.

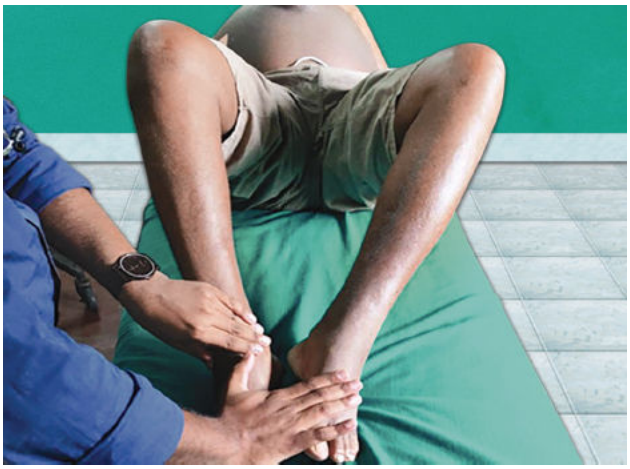


Fig. 5D.12: Demonstration of flapping tremors in legs—on leaving the legs to fall apart a negative myoclonus can be noticed by observing the knee.

Causes of asterixis (flapping tremor)	
Bilateral asterexis	***Unilateral asterexis***
Metabolic: Liver failure, azotemia, respiratory failure **Sedatives:** Benzodiazepines, barbiturates	**Focal brain lesions at:** • Thalamus • Corona radiata • Anterior cerebral artery territory

Anticonvulsants: Phenytoin (phenytoin flap), carbamazepine, valproic acid, gabapentin **Antipsychotics:** Lithium **Antibiotics:** Ceftazidime **Others:** Metoclopramide **Dyselectrolytemia:** Hypomagnesemia, hypokalemia **Bilateral structural brain lesions**	• Primary motor cortex • Parietal lobe • Cerebellum • Midbrain • Pons

Signs Pointing the Etiology of Cirrhosis

Signs	Etiology of cirrhosis
Parotid enlargement, dupuytren's contracture	**Alcohol**
Tattoo marks, jaundice	**Hepatitis B/C**
Metabolic syndrome	**NASH**
Xanthoma, xanthelasma, obstructive jaundice	**Primary biliary cirrhosis**
Skin hyperpigmentation, organomegaly, diabetes	**Hemochromatosis**
Emphysema and cirrhosis	**Alpha-1 antitrypsin deficiency**
Long standing heart failure	**Cardiac cirrhosis**
Tender liver with absent abdominojugular reflux	**Budd–Chiari syndrome**
Arthritis, skin changes, nephritis	**Autoimmune**
Deforming arthritis on treatment	**Methotrexate induced**
Kayser–Fleischer (KF) ring on cornea	**Wilson's disease**

Signs of Chronic Alcoholism

- Parotid swelling
- Dupuytren's contracture.

ORAL CAVITY EXAMINATION

A torch, tongue depressor, and gloves (for palpation) are needed.

Lips

- Angular stomatitis, cheilitis—iron deficiency, riboflavin deficiency
- Herpes labialis
- Circumoral pigmentation
  - Addison's disease.

Teeth

- Caries
- Color/staining—tobacco, tetracycline (yellow), fluorosis (chalk white), red/erythrodontia (porphyria)
- Shape of teeth—peg-shaped incisors and moon molars in congenital syphilis, widely spaced teeth in acromegaly.

Gums

- Gingivitis

- Gum bleeding—scurvy, vitamin K deficiency, acute leukemia, thrombocytopenias, coagulopathies, gingivitis
- **Gum hypertrophy**
  - Drugs—phenytoin, nifedipine, cyclosporine
  - Pregnancy
  - Acute myeloid leukemia (AML)—M4, M5
  - Chronic gingivitis
  - Tumors—epulis
- Ulcers and pyorrhea.

Tongue

- Macroglossia—acromegaly, myxedema, amyloidosis, down syndrome
- Coated tongue—typhoid, candidiasis
- Color of tongue
  - Pale—anemia
  - Red beefy—B_{12} deficiency
  - Magenta—B_2 deficiency
  - Bluish—cyanosis
  - Yellowish—jaundice
  - Strawberry—scarlet fever
- Dry tongue—dehydration, anticholinergics, diabetes
- Leukoplakia, hairy leukoplakia
- Fissuring
- Geographic tongue—desquamated epithelium
- Median rhomboid glossitis.

Buccal Mocosa

- Ulcers
- Pigmentation
- Candidiasis
- Koplik spots.

Palate/Pharynx

- Ulcers
- Postnasal drip
- White patch of tonsil:
  - Candidiasis
  - Diphtheria
  - Agranulocytosis
  - Infectious mononucleosis
  - Follicular tonsillitis
  - Vincents angina
  - Malignancy
  - Tonsilolith.

Causes of oral ulcers	
Aphthous ulcer	
Infections	**Gastrointestinal disease**

• Herpetic stomatitis • Chickenpox • Hand, foot, and mouth disease • Herpangina • Infectious mononucleosis • Human immunodeficiency virus (HIV) • Acute necrotizing gingivitis • Tuberculosis • Syphilis • Candida	• Celiac disease • Crohn's disease • Ulcerative colitis **Connective tissue disorders** • Lupus erythematosus • Behçet's syndrome • Reiter's disease
Dermatological disorders • Lichen planus • Pemphigus • Pemphigoid • Erythema multiforme • Dermatitis herpetiformis • Linear immunoglobulin A (IgA) disease • Epidermolysis bullosa	**Malignancy** **Drugs—**cytotoxic agents, antibiotics **Radiation** **Trauma**

Pigmentation of oral mucosa
• Addison's disease • Peutz–Jeghers syndrome • Hemochromatosis • Heavy metal—lead (Burtonian line) • Acanthosis • Drugs like hormones, oral contraceptives, cyclophosphamide, busulfan, bleomycin, clofazimine, chloroquine • Pregnancy • Laugier–Hunziker syndrome • Nevi • Malignant melanoma

SYSTEMIC EXAMINATION

The order of examination of abdomen is preferably done—Inspection→Auscultation→Palpation and Percussion.

(As the auscultatory findings might change post-palpation and percussion)

Inspection

Position of patient:

- Most of the gastrointestinal tract (GIT) examination (inspection) is done in supine position (standing position is adapted for examination of dilated veins).
- Expose from chest to mid-thigh preferably.
- Relax abdominal wall muscles by flexing the thigh with arms by the side of the patient.

Shape of abdomen:

Shape	Condition seen
Scaphoid	Normal
Generalized abdominal distention **[The 7 F's]**	1. Fluid 2. Fat 3. Flatus 4. Feces 5. Fetus 6. Full bladder 7. Fatal neoplasm

Localized abdominal distention	Indicates a organomegaly or mass
Fullness of flanks indicates	Free fluid

Skin over the abdomen:

Findings	Seen in
Discoloration	**Pancreatitis** • **Cullen's sign**—discoloration around umbilicus • **Grey turner's sign**—discoloration over the flanks
Ecchymosis or purpura	Coagulopathy
Striae atrophica or gravidarum (white or pink wrinkled linear marks)	• Recent change in size of the abdomen • Pregnancy • Ascites • Wasting diseases • Severe dieting
Purple striae	Cushing's syndrome (pigmented)
Linea nigra	Pigmentation of the abdominal wall in the midline below the umbilicus, seen in pregnancy
Erythema ab igne	• Brown mottled pigmentation produced by constant application of heat, usually a hot water bottle or heat pad, on the skin of the abdominal wall. • It is a **sign of chronic pain** as in chronic pancreatitis.
Paracentesis marks	Indicate diagnostic/therapeutic ascitic tapping
Sinuses	• Tuberculosis • Crohn's disease
Stretched shiny skin	Indicates tense ascites

Scars (Fig. 5D.13):

Few commonly employed incisions over the abdomen as showed in **Figure 5D.13**.

Quadrants of abdomen (Fig. 5D.14):

Abdomen can be grossly divided into four quadrants as shown in **Figure 5D.14** with help of transumbilical plane and median plane.

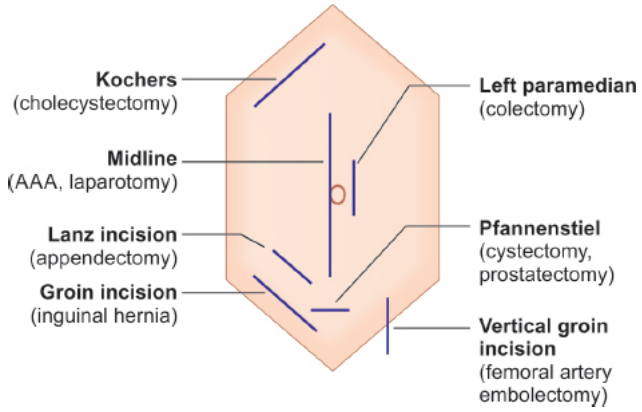


Fig. 5D.13: Surgical incisions commonly employed.

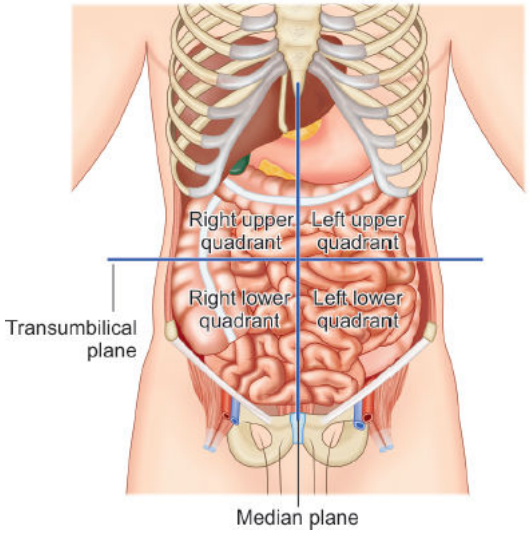


Fig. 5D.14: Four quadrants of the abdomen.

Regions of abdomen (Fig. 5D.15):

Abdomen can also be divided into nine regions with the help of right and left midclavicular line, transtubercular plane, and subcostal plane as shown in **Figure 5D.15**.

Umbilicus:

Finding	Seen in
Slightly retracted and inverted	Normal
Everted	Suggestive of tense ascites
Umbilical hernia	Indicate lax abdominal wall with gross ascites
Umbilical node	Sister Mary Joseph node seen in metastasis from GIT cancers
Normally, $\frac{\textit{Distance between xiphisternum and umbilicus}}{\textit{Distance between umbilicus and pubis symphysis}} = 1.6$	
Ratio decreased—umbilicus is displaced up (smiling umbilicus)	• Pelvic mass • Ovarian tumors
Ratio increased—umbilicus displaced down (weeping umbilicus)	• Upper abdominal mass • Ascites
Spinoumbilical distance (distance between ASIS to umbilicus)	• Normally equidistant • Shift of umbilicus to one side indicates tumors/mass originating from other side

Movement with Respiration

Method of examination: Shine a light, across the patient's abdomen, and watch for the abdominal wall movements.

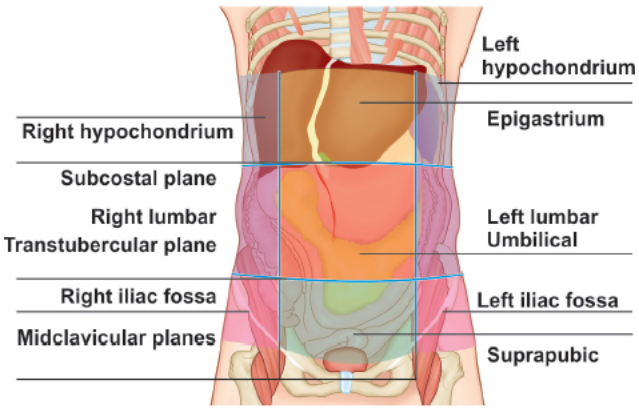


Fig. 5D.15: Planes and nine areas of the abdomen.

Finding	Seen in
Normal	• Gentle rise in the abdominal wall during inspiration and a fall during expiration • Corresponding areas move equally on both sides
Diminished or absent movements	• Generalized peritonitis **(the still, silent abdomen)**

Visible peristalsis:

Site of obstruction	Direction of peristalsis
Obstruction at the pylorus	• Peristalsis from left costal margin to right
Obstruction in the distal small bowel	• Right to left (or) • Irregular pattern

Note: Visible peristalsis may be a normal finding in very thin elderly patients with lax abdominal muscles.

Visible mass:

- **Figure 5D.16** demonstrates the underlying intra-abdominal structures with respect to the regions.

Divarication of recti (diastasis of recti):

It is a gap between the rectus abdominis muscle which becomes prominent on straining **(Fig. 5D.17)**. Make the patient lie supine and tense the abdominal muscles by lifting the head **(Fig. 5D.18)**, a midline defect can be seen and felt. It is common after postpartum, and also can be seen with tense ascites.

AUSCULTATION

Note that the abdomen should be auscultated prior to palpation. Auscultate in all four quadrants of the abdomen.

1. Bowel sounds
2. Bruits
3. Venous hum

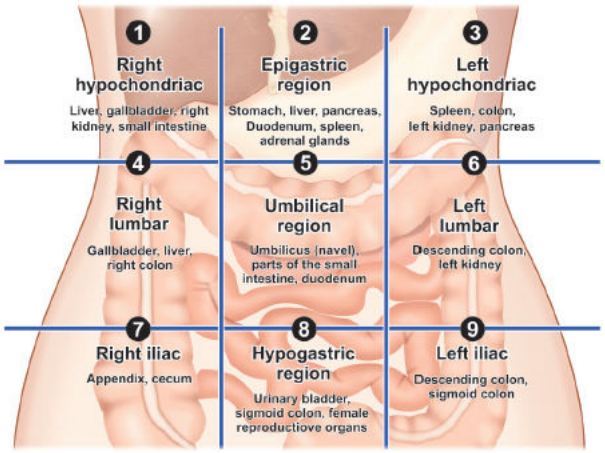


Fig. 5D.16: Pictorial representation of corresponding areas and underlying structures.

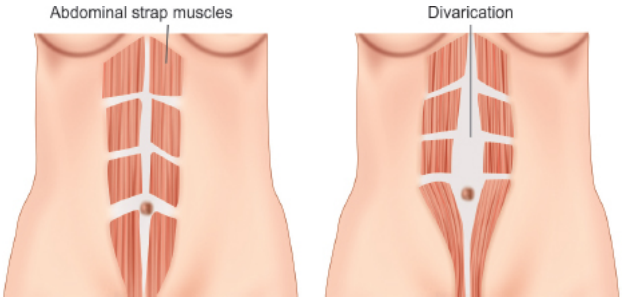


Fig. 5D.17: Divarication of recti.

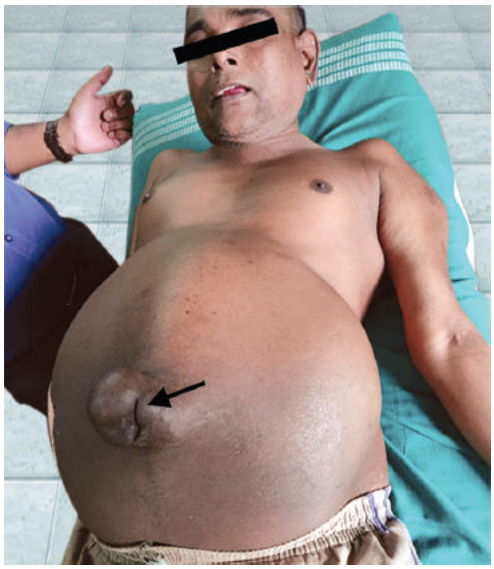


Fig. 5D.18: Midline defect suggestive of divarication of recti, on asking the patient to raise the head off the bed. Also patient has umblical hernia.

4. Rubs
5. Succussion splash.

1. **Bowel sounds (Fig. 5D.19):**

Normal	7–35 per minute
Increased (borborygmus)	• Intestinal obstruction • Diarrhea • Laxative use • Carcinoid syndrome • Massive GI bleed
Decreased	Paralytic ileus and peritonitis

Note: When bowel sounds are not present, one must auscultate for a full 3 minutes before saying that bowel sounds are absent.

2. **Bruits:**

Renal artery bruit (Fig. 5D.20)	• 2.5 cm above and lateral to the umbilicus in transpyloric plane • Indicates partial renal artery stenosis
Abdominal aorta (Fig. 5D.21)	Epigastrium in aortic aneurysm or aortoarteritis
Hepatic bruit (Fig. 5D.22)	• Hepatocellular carcinoma (HCC) • Acute alcoholic hepatitis • Hemangioma
Iliac bruit (Fig. 5D.23)	2.5 cm below and lateral to the umbilicus

3. **Venous hum:**
   Cruveilhier–Baumgarten murmur (Fig. 5D.24):
   - It is a continuous murmur, produced due to the opening of the paraumbilical vein in the falciform ligament.
   - It is heard midway between the xiphisternum and umbilicus on the right side of the epigastrium.

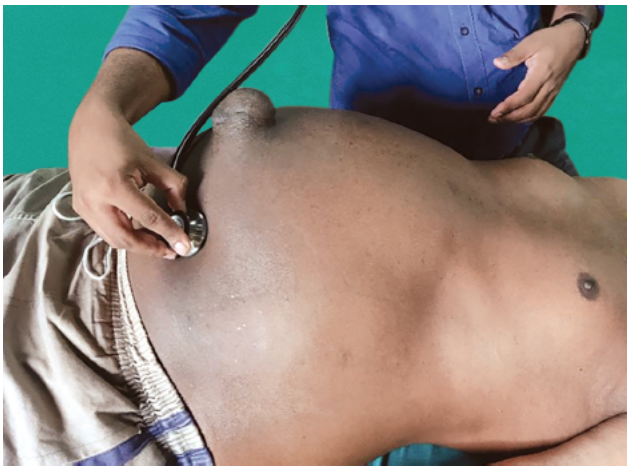


Fig. 5D.19: Auscultation of bowel sounds.

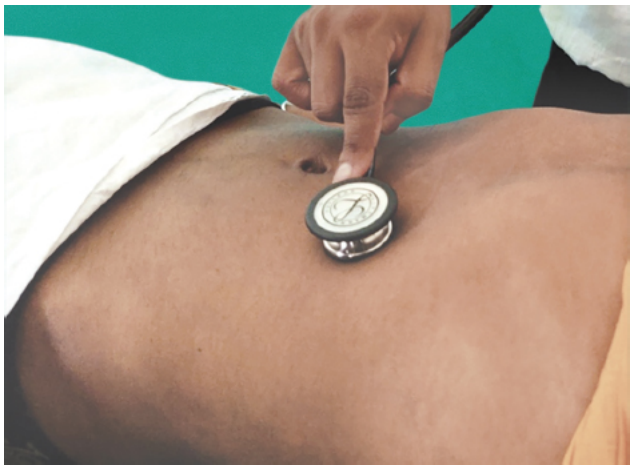


Fig. 5D.20: Renal artery bruit—2.5 cm above and later to umbilicus in transpyloric plane.

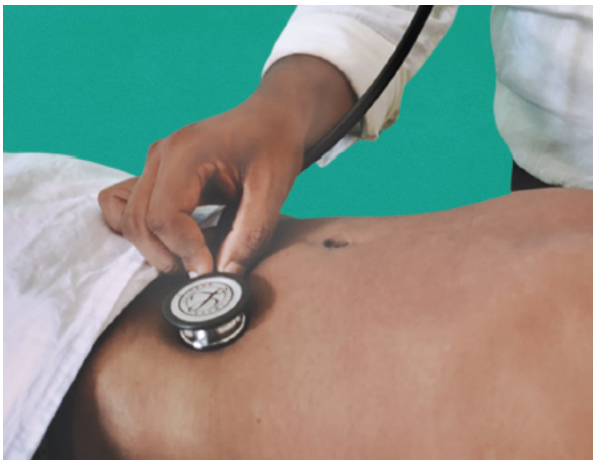


Fig. 5D.23: Iliac bruit—2.5 cm below and lateral to umbilicus

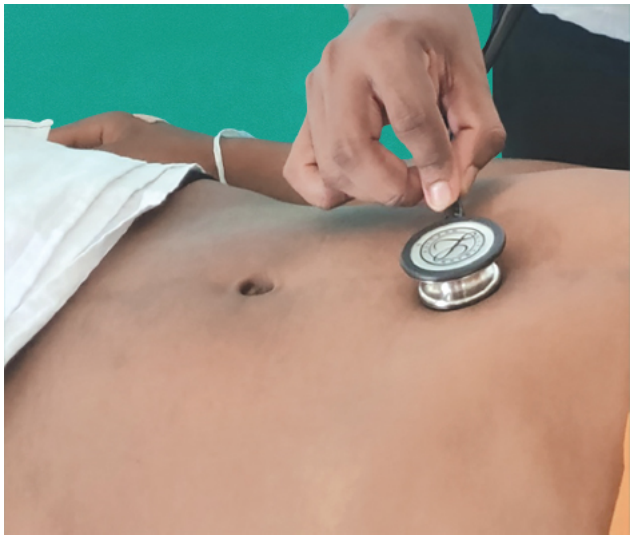


Fig. 5D.21: Abdominal aorta bruit in the epigastrium in the midline.

Fig. 5D.24: Cruveilhier–Baumgarten murmur heard midway between the xiphisternum and umbilicus on the right side of th epigastrium.

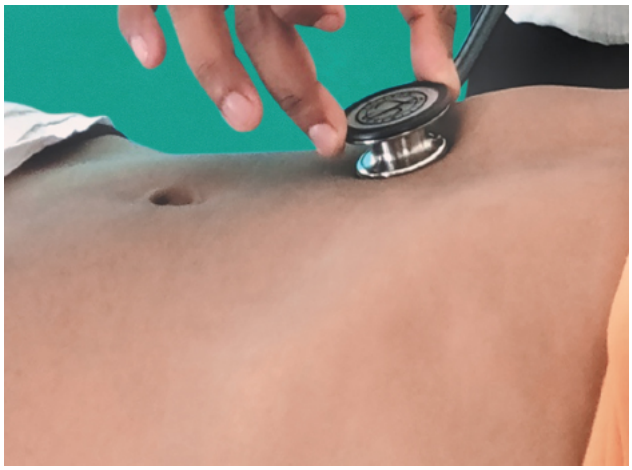


Fig. 5D.22: Hepatic bruit.

– A patent umbilical vein excludes an extrahepatic cause of portal hypertension because the umbilical vein arises from the intrahepatic portion of the left portal vein.

4. Rubs:

- **Hepatic friction rub** is a superficial, scratchy sound heard on the liver.

  Commonly seen with:

  » HCC
  » Postliver biopsy
  » Hepatic infarcts and
  » Gonococcal peritonitis (Fitz–Hugh–Curtis syndrome).

  – **Splenic rub** is a coarse, scratching sound coinciding with inspiration over the left upper quadrant due to splenic infarct.

  Commonly seen with:

  » Subacute bacterial endocarditis
  » Chronic myeloid leukemia.
  » Sickle cell anemia.
  » After splenic puncture (e.g. in diagnosis of chronic kala-azar).

5. **Succussion splash:**
   – When you auscultate the patient's epigastrium/left upper quadrant and then shake the patient a "splash-like" noise is heard
   – If heard after several hours after eating, it suggests delayed gastric emptying which may be due to gastric outlet obstruction.
   – Thoracic succussion splash has been described in achalasia cardia, hydropneumothorax, and large hiatal hernia.

PALPATION AND PERCUSSION OF THE ABDOMEN

The following scheme is suggested for palpating the abdomen:

- Start in left lower quadrant of abdomen and repeat in all quadrants as described below.
- Palpate lightly initially, followed by deep palpation.
- Feel for left kidney→spleen→right kidney→liver→aorta and para-aortic glands→common femoral vessels→urinary bladder→both groins→external genitalia.

EXAMINATION OF INDIVIDUAL ORGANS

Examination of Liver

Location

- Right hypochondriac region
- Epigastric region
- Left hypochondriac region.

Extent

- Upper border—6th rib anteriorly
- Inferior border—crosses midline at the level of transpyloric plane (at the level of L1 vertebrae).

INSPECTION

- Watch for the fullness in the right hypochondrium and epigastrium (epigastrium usually represents left lobe).
- Direction of enlargement is towards the right iliac fossa.

Palpation

Following methods of palpation have been discussed:

1. Traditional method/conventional method
2. Preferred method
3. Alternate method
4. Hooking method
5. Dipping method

1. Traditional method/conventional method (Fig. 5D.25):

- Place right hand on the right iliac fossa, parallel to the costal margin.
- Keep the hand steady during inspiration and feel for the liver edge as it descends with each inspiration.
- If edge is not felt, move the hand upwards towards costal margin by 1 cm during expiration.
- Repeat the procedure till the liver border is felt.

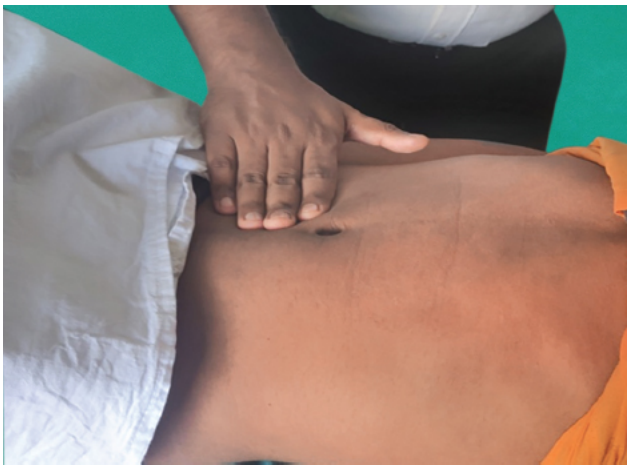


Fig. 5D.25: Traditional method of palpation of

2. Preferred method (Fig. 5D.26):

- Sit on the right side the patient facing the head end of the patient.
- Now place both hands side-by-side flat on the abdomen in the right subcostal region lateral to the rectus with the fingers pointing towards the ribs.

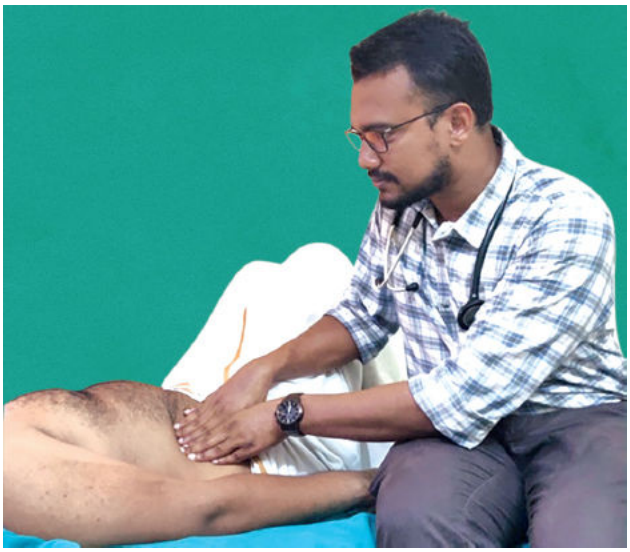


Fig. 5D.26: Preferred method of palpation of liver.

- If resistance is felt, move the hands further down until resistance disappears.
- Exert gentle pressure and ask the patient to inspire deeply.
- The border of the liver can be felt on the tips of the fingers.
- This procedure can be repeated from lateral to medial to trace the entire edge of the liver.

3. Alternate method (Fig. 5D.27):

- Place the right hand below and parallel to the right subcostal margin.
- The liver edge will then be felt against the radial border of the index finger.

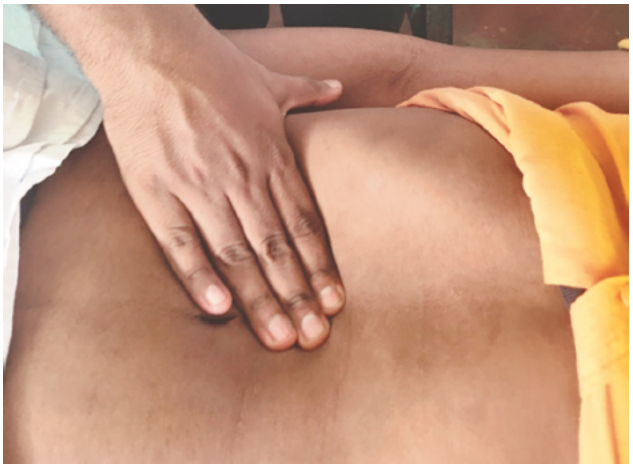


Fig. 5D.27: Alternate method of palpation of liver.

4. Hooking method of liver examination (Fig. 5D.28):

- Examiner stands at the patient's right shoulder, facing the foot end and examines the lower edge of the liver by curling the fingertips under the right costal margin.

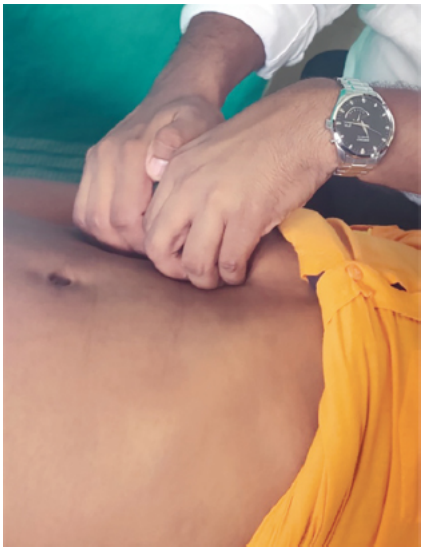


Fig. 5D.28: Hooking method of palpation of liver.

5. **Dipping method of liver palpation in ascites (Fig. 5D.29):**
   - Place both hands one over the other, over the area to be palpated.
   - Rapidly flex your metacarpophalangeal joints, so that your fingers suddenly dip into the patient's abdomen.
   - This displaces the fluid, enhancing the palpation of underlying organ.

Liver Span

- The liver span is the distance in centimeters between the upper border of the liver in the right midclavicular line, as determined by percussion (i.e. where lung resonance changes to liver dullness), and the lower border, as determined by either percussion or palpation **(Figs. 5D.30 to 5D.32)**.
- The upper border of the liver is assessed using a heavy percussion technique. Light percussion is used to locate the lower edge of the liver. Light percussion is required because heavy percussion may underestimate the lower extent of the liver border.
- The normal liver span is less than 13 cm.
- In midclavicular line: Normally 6–12 cm.
- In midsternal line (left lobe): Normally 4–8 cm.
- The clinical estimate of the liver span is usually an underestimation of the actual liver size by about 2–5 cm.

Liver span	Condition seen
Increased	Hepatomegaly
Decreased	Shrunken liver as in cirrhosis
False positive for enlarged liver	• Right sided pleural effusion • Right lower lobe consolidation
Note: In conditions like emphysema of the lung, the liver may be pushed down. The edge may be palpable, leading the examiner to believe that the patient has hepatomegaly when the real problem is a hyperinflated lung. Percussion will reveal that the upper border is lower than expected.	

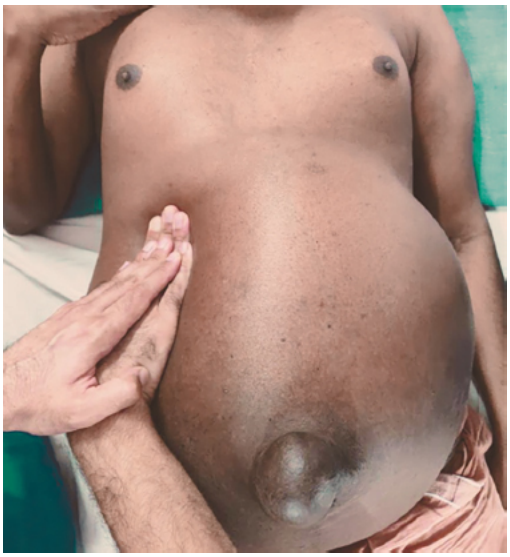


Fig. 5D.29: Dipping method of palpation of liver.

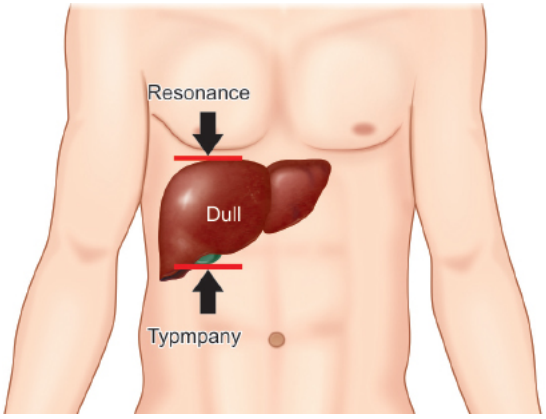


Fig. 5D.30: Liver span.

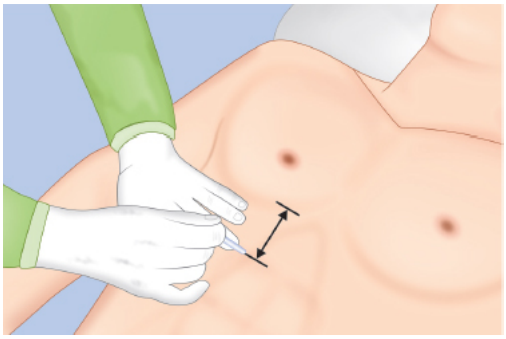


Fig. 5D.31: Percuss along the midclavicular line.

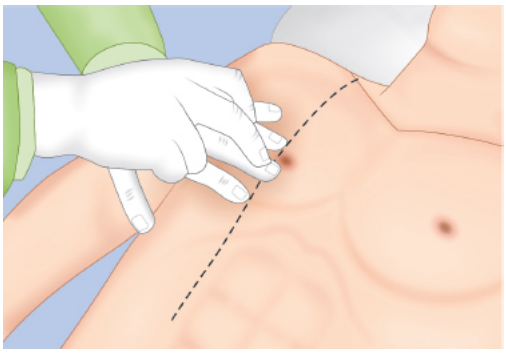


Fig. 5D.32: Mark the upper and lower border of dullness.

If the liver is enlarged and palpable, assess the following:

- **Location** of the edge in cm below the costal margin in the midclavicular or anterior axillary line.
- **Span (** in cm)
- **Tenderness** (tender/nontender).

Tender hepatomegaly	Painless hepatomegaly
• Right heart failure • Acute hepatitis (viral/alcoholic/drug induced) • Liver abscess (amoebic/pyogenic) • Hepatoma • Infarcts • Actinomycosis • Acute Budd–Chiari syndrome	• Fatty liver • Infiltrative and storage disorders • Malaria • Leukemia • Lymphoma

- **Margins** (regular, irregular, rounded or sharp). In cancers the liver edge may be irregular.

Rounded	Infiltrative disorders
Sharp	• Secondary metastases, acute hepatitis • Biliary obstruction • Chronic hepatitis

- **Surface** (smooth, nodular).

Smooth	• Malaria • Acute hepatitis • Infiltrative disorders, etc.
Nodular	• Metastatic cancers • Hepatoma • Alcoholic cirrhosis (micronodular) • Posthepatic cirrhosis (macronodular)

- **Consistency** (soft/firm/hard): In metastatic cancers and in obstructive jaundice, the liver is typically firm to hard.
- **Pulsatility** (pulsatile/not pulsatile): A pulsatile liver may be present in tricuspid regurgitation (systolic), tricuspid stenosis (diastolic), hepatocellular carcinoma, and hemangiomas.

Ausculto-Percussion Method (The Scratch Test)

- The diaphragm of the stethoscope is placed either over the xiphoid process or just superior to the costal margin along the midclavicular line.
- The examiner then gently scratches the skin along the right midclavicular line, starting in the lower abdomen and advancing towards the head **(Fig. 5D.33)**.

- The sound produced by the scratching changes in quality and intensity when over the liver, as sounds are much more easily transmitted through the solid organ.

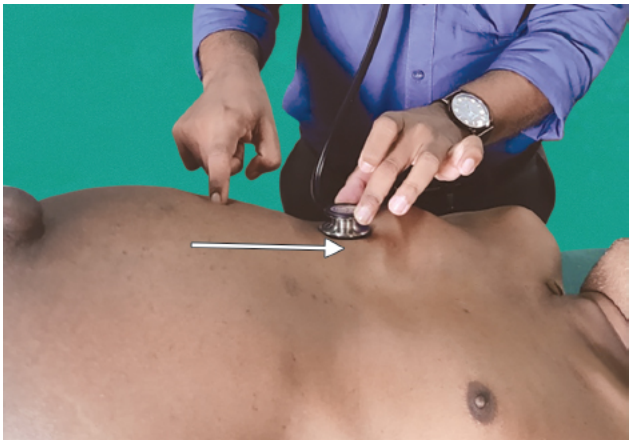


Fig. 5D.33: Demonstration of ausculto-percussion method.

Causes of Hepatomegaly (Fig. 5D.34)

Causes of hepatomegaly can be grossly grouped under the headings of infections, malignancies, infiltrative disorders, hematological disorders, and vascular disorders as shown in **Figure 5D.34**. Massive hepatomegaly (> 10 cm) seen with Hepatoma.

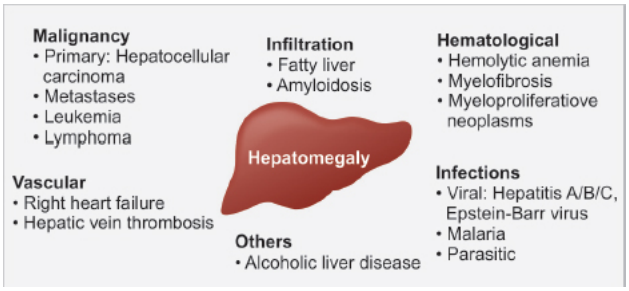


Fig. 5D.34: Causes of hepatomegaly.

Caudate Lobe (Fig. 5D.35)

- Arises from the right lobe of the liver, on the postero-superior surface

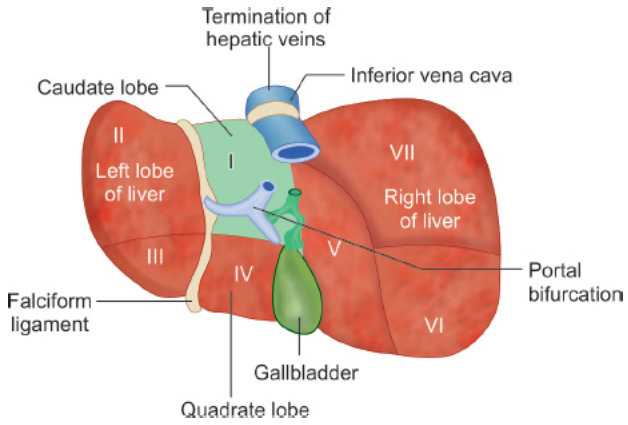


Fig. 5D.35: Caudate lobe location and boundaries.

- Hypertrophy of caudate lobe is characteristic of hepatic outflow obstruction (Budd–Chiari syndrome).

Riedel's Lobe (Fig. 5D.36)

- Congenital variant projecting from the right lobe of the liver
- May be mistaken for gallbladder or right kidney.

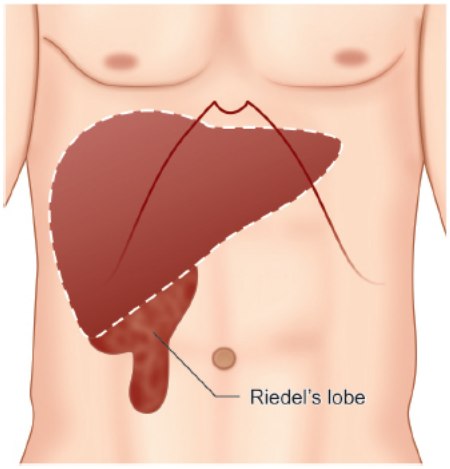


Fig. 5D.36: Anomalous lobe of the liver projecting from right lobe.

Examination of Spleen

Normal characteristics:

Dimensions	• 12 cm length, 7 cm width • 13 cm craniocaudal diameter
Weight	<250 g
Location (Fig. 5D.37)	• Along—9th, 10th, 11th ribs midaxillary line • Along the long axis of 10th rib
Extent	• **Anteriorly** (lower pole): Up to mid axillary line • **Posteriorly:** The superior angle of spleen is 4 cm lateral to T10 spine
Margin	There is a **notch** on the inferolateral border, and this may be palpated when the spleen is enlarged

Normal spleen is not palpable clinically except in following scenarios:

- Only occasionally palpable in 1–3% of New Guinea population.
- Tip may be palpable in newborn up to 3 months of age.

Splenic enlargement:
- Before becoming clinically palpable—spleen enlarges in superior and posterior direction.
- It has to enlarge two to three times of normal to become palpable.

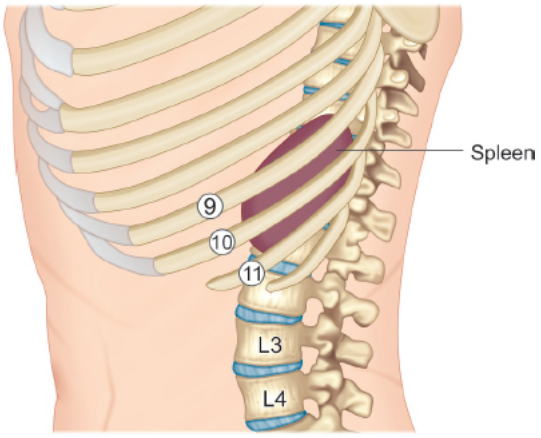


Fig. 5D.37: Surface marking of spleen.

- Once palpable, it appears (felt) below tip of 10th rib (beneath/under the left costal margin) and further enlarges downwards, medially (inwards), and forwards towards umbilicus (LHC to RIF).

Grading of enlargement/splenomegaly:

Based on largest dimension		
Moderate splenomegaly		***Severe splenomegaly***
11–20 cm		>20 cm
Based on distance from costal margin (Fig. 5D.38)		
Mild (tip) enlargement	***Moderate enlargement***	***Severe (marked) enlargement***
1–2 cm (<3 cm)	**3–7 cm** (3–8 cm) Between costal margin and umbilicus	**7+ cm** >8 cm below left costal margin >1000 g dry weight. Crossing midline

Note: Size of the spleen is measured from the left costal margin to the tip along the long axis of spleen.

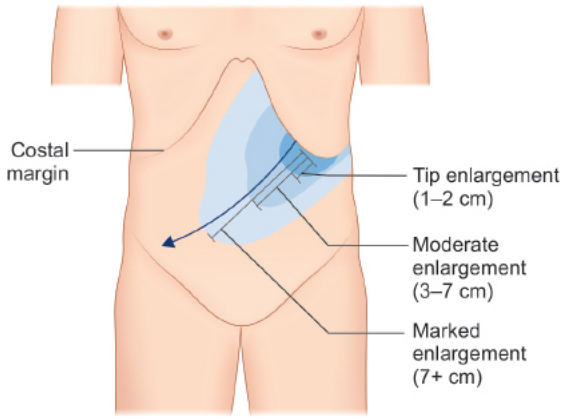


Fig. 5D.38: Grading of splenomegaly.

Hackett's grading system for palpable splenomegaly (Fig. 5D.39):

Grade	Description
Grade 0	Normal impalpable spleen
Grade 1	Spleen palpable only in deep inspiration
Grade 2	Spleen palpable on midclavicular line half way between umbilicus and costal margin
Grade 3	Spleen expands towards the umbilicus
Grade 4	Spleen goes past the umbilicus
Grade 5	Spleen expands towards pubic symphysis

Inspection:

Fullness may be seen emerging from left upper quadrant extending diagonally towards the right lower quadrant (RLQ).

Palpation:

Following methods of palpation have been discussed:

1. Classical method
2. Bimanual method
   a. In supine position
   b. In right lateral position
3. Hooking method
   a. In supine position
   b. In right lateral position
4. Middleton's maneuver
5. Dipping method.

Classical method (Fig. 5D.40):

- Patient in supine position, examine with single hand (right).
- Place the hand in the RLQ in RIF and move diagonally towards left upper quadrant.
- Hand should be firmly placed one the abdominal wall.
- Keep the hand steady during inspiration and feel for the splenic edge as it descends with each inspiration.

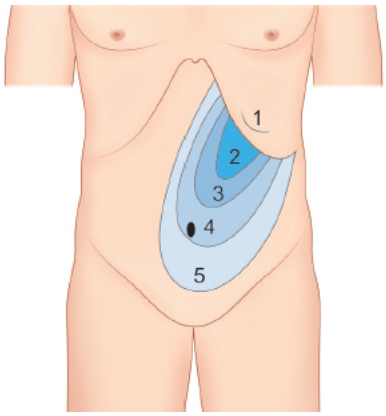


Fig. 5D.39: Hackett's grading system for palpable splenomegaly.

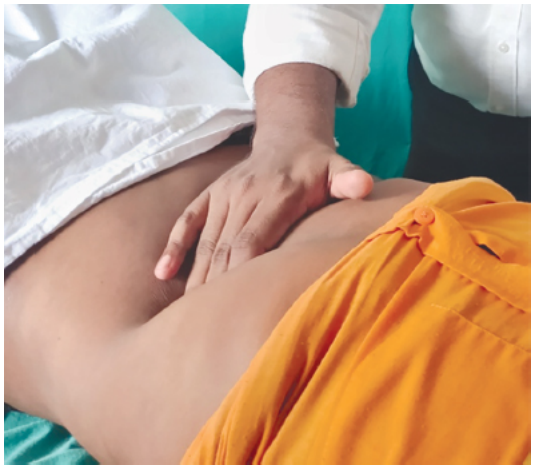


Fig. 5D.40: Demonstration of classical method of spleen palpation.

- If edge is not felt move the hand diagonally towards LUQ by 1 cm during expiration.
- Repeat the procedure.
- Tip of the fingers are used to feel the splenic tip.

Bimanual (supine position) (Fig. 5D.41):

- Place palm of left hand over the left lowermost rib cage posterolaterally, restricting the expansion of left lower ribs on inspiration.
- While applying firm pressure with the left hand, ask the patient to take deep inspiration.
- Insinuate the right hand beneath the left costal margin and feel for the splenic edge.

Bimanual (right lateral position):

- Done with patient lying in right lateral position with the left hip and knee flexed.
- Rest of maneuver is similar to above.

Fig. 5D.41: Demonstration of bimanual method (supine position) of spleen palpation.

Hooking method (supine position) (Fig. 5D.42):

- The physician hooks his fingers beneath the left costal margin as the patient inspires.

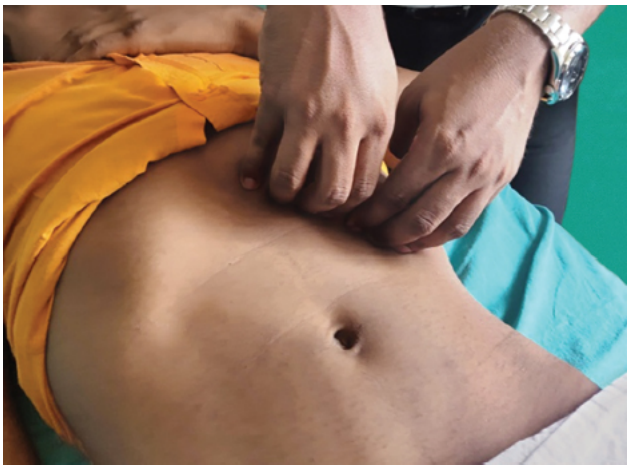


Fig. 5D.42: Demonstration of hooking method (supine position) of spleen palpation.

- For better appreciability, patient is asked to lie down on his left fist just inferior to his left scapula **(Middleton's maneuver) (Figs. 5D.43A and B)**
- From above, spleen may be continently palpable with two hands arching below the left costal margin while patient is asked to take deep breath in/out slowly.

Hooking maneuver (right lateral position):

- Examiner stands on left side facing towards the foot end
- With one hand hook the left lower costal margin and with other hand, give a counter-pressure from the posterolateral aspect.
- Now ask the patient to take take a deep inspiration and feel for the tip of the spleen, by hooking the fingers.

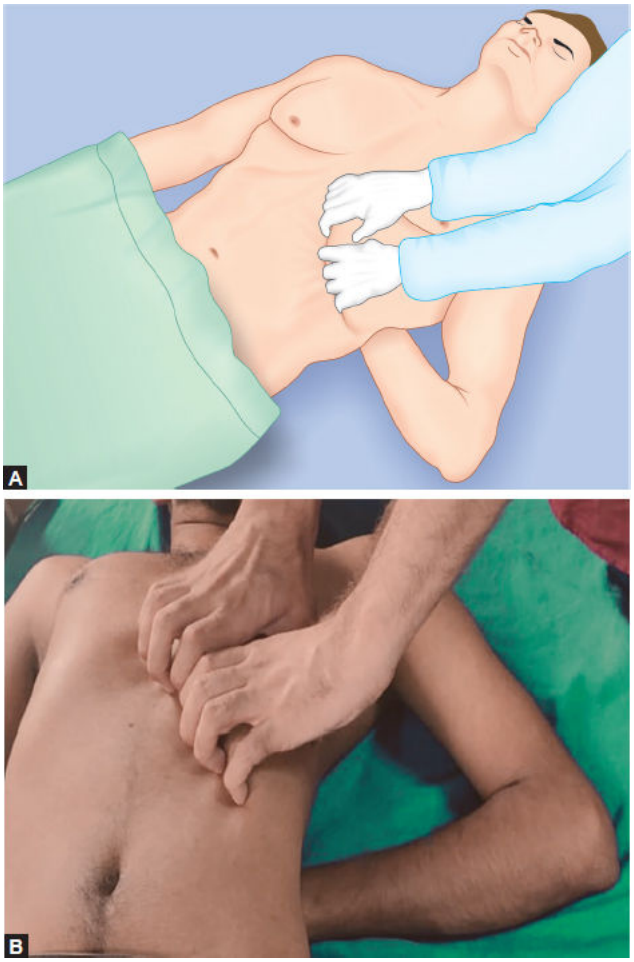
Dipping method:

- It is done in marked ascites
- Similar to dipping method of liver (as described below under the palpation of liver).

Following methods of percussion have been discussed:

1. Castell's method
2. Traube's space percussion
3. Nixon's method of percussion

1. **Percussion by Castell's method (spleen percussion sign)**
   - With patient in supine position, percuss in the lowest left intercostal (IC) space in the anterior axillary line **(Figs. 5D.44 and 5D.47)** (usually the 8th or 9th IC space—Castell's point)
   - This space should remain resonant during full inspiration.



Figs. 5D.43A and B: Demonstration of hooking method with Middleton's maneuver percussion.

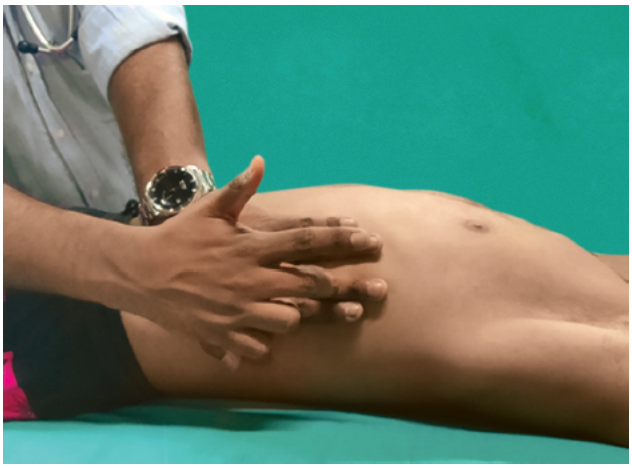


Fig. 5D.44: Percussing the lowest left intercostal space in anterior axillary line—Castell's method of splenic percussion.

- Dullness on full inspiration indicates possible splenic enlargement (a positive Castell's sign).
- Most sensitive of all clinical signs with sensitivity 82% and specificity 83%.

	Full inspiration	Full expiration
Normal	Resonant	Resonant
Mild splenomegaly*	Dull	Resonant
Moderate/severe splenomegaly	Dull	Dull
*Percussion sign is considered positive, when a change in percussion note is observed between full expiration and full inspiration.		

2. **Percussion of Traube's (semilunar) space**
   - It is a semilunar space in the left anterior chest bounded by:
     - » Above by 6th rib
     - » Below by left costal margin
     - » Laterally by midaxillary line.
   - With patient supine, percuss inferior to lung resonance from medial to lateral **(Figs. 5D.45 and 5D.47)** (as described by **Barkun**). Normally, a tympanic note heard due to gastric air bubble.

Obliteration of Traube's space	• Massive splenomegaly • Left-sided pleural effusion • Pericardial effusion • Enlarged left lobe of the liver • Full stomach or fundic mass
Upward shift of Traube's space	• Left diaphragmatic paralysis • Left lower lobe collapse or fibrosis

Fig. 5D.45: Percussion of Traube's space.

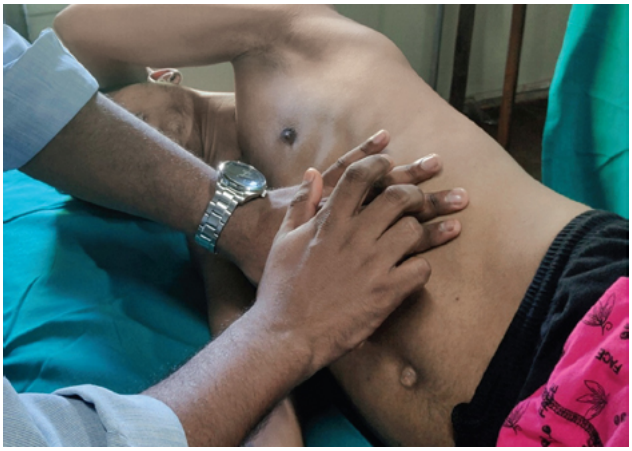


Fig. 5D.46: Percussing the posterior axillary line in right lateral position (Nixon's method).

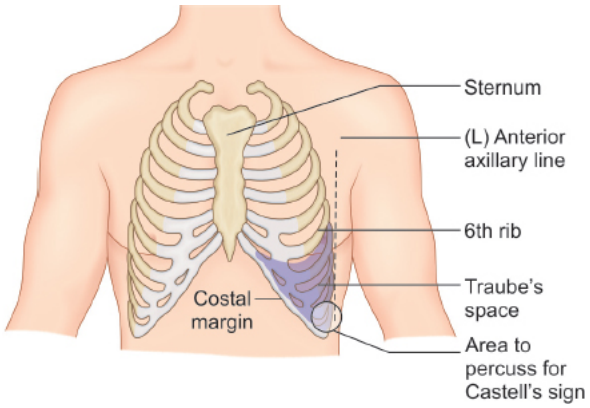


Fig. 5D.47: Landmarks of Traube's space and Castell's sign.

3. **Percussion by Nixon's method**
   - Patient is first placed in the right lateral decubitus position.

– Percussion starts at the midpoint of the left costal margin and is continued upward perpendicular to the left costal margin **(Fig. 5D.46)**.

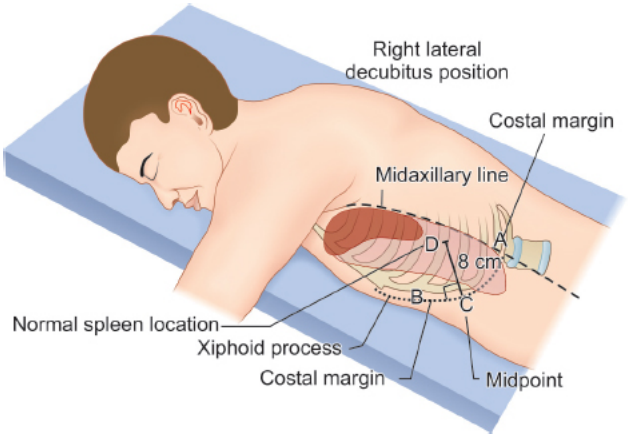


Fig. 5D.48: Landmarks for Nixon's method.

– Normally, the level of dullness does not extend further than 8 cm above the costal margin and splenomegaly is diagnosed if the dullness extends beyond 8 cm.

Causes of splenomegaly	
Mild splenomegaly	
Acute infections	Septic shock, infective endocarditis, enteric fever, infectious hepatitis, infectious mononucleosis, brucellosis, cytomegalovirus, toxoplasmosis
Chronic infections	Tuberculosis, syphilis, brucellosis, chronic bacteremia, HIV
Parasitic infestations	Malaria, kala-azar, and schistosomiasis
Inflammation	Rheumatoid arthritis, sarcoidosis, systemic lupus erythematosus (SLE)
Others	Congestive cardiac failure, thalassemia minor
Moderate splenomegaly	
Neoplastic	Lymphomas, acute leukemias, chronic lymphocytic leukemia, chronic myeloid leukemia
Non-neoplastic	Cirrhosis of liver (with portal hypertension), chronic hemolytic anemia, malaria, kala-azar, sarcoidosis, infectious mononucleosis, splenic abscess, amyloidosis, hemochromatosis, polycythemia vera
Severe (massive) splenomegaly	
Common causes	Chronic myeloid leukemia, myelofibrosis, kala-azar, primary splenic lymphomas (Hairy cell, mantle cell, marginal B cell), portal hypertension (extrahepatic portal vein thrombosis), hyper-reactive malarial splenomegaly (tropical splenomegaly)
Uncommon causes	Gaucher's disease, Niemann-Pick disease, thalassemia major, splenic cysts and tumors of spleen, *mycobacterium avium* complex (MAC) infection in HIV patients

Causes of Hepatosplenomegaly

Common causes of hepatosplenomegaly and associated features have been illustrated in **Figure 5D.49**.

Examination of Gallbladder

- Location: Lateral edge of rectus abdominis near the tip of right 9th costal margin
- Moves with respiration

- Upper border continues with liver

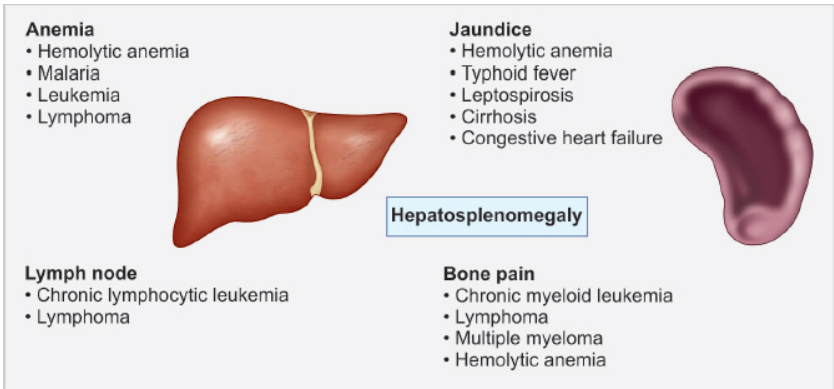


Fig. 5D.49: Causes of hepatosplenomegaly.

- Causes of enlarged gallbladder:
  - Carcinoma head of pancreas
  - Common bile duct (CBD) obstruction
  - Mucocele of gallbladder
  - Carcinoma of gallbladder
- **Murphy's sign**: In acute cholecystitis, at the height of inspiration, patient stops breathing with a gasp as a mass is felt.
- **Courvoisier's law**: In a jaundiced patient, if the gallbladder is palpable, it is unlikely to be due to a CBD gallstone obstruction.

Examination of Kidney

Examination of Left Kidney

- The right hand is placed anteriorly in the left lumbar region while the left hand is placed posteriorly in the left loin **(Fig. 5D.50)**.
- Ask the patient to take a deep breath in, press the left hand forward and the right hand backward, upward and inward.
- Left kidney is usually not palpable (except when low lying or enlarged).
- If palpable, it is described as bimanually palpable and ballotable.
- **Bimanually palpable:** As it can be felt as a swelling between both right and left hands.
- **Ballotable:** It can be pushed from one hand to the other. It is due to perinephric fat which allows the free movement of the kidney in the retroperitoneum.

Palpation of Right Kidney

- Place the right hand horizontally in the right lumbar region anteriorly with the left hand placed posteriorly in the right loin **(Fig. 5D.51)**.
- Push forwards with the left hand, press the right hand inward and upward and ask the patient to take a deep breath in.
- The lower pole of the right kidney, unlike the left, is commonly palpable in thin patients and is felt as a smooth, rounded swelling which descends on inspiration.
- It is also bimanually palpable and ballotable.

Fig. 5D.50: Palpation of left kidney.

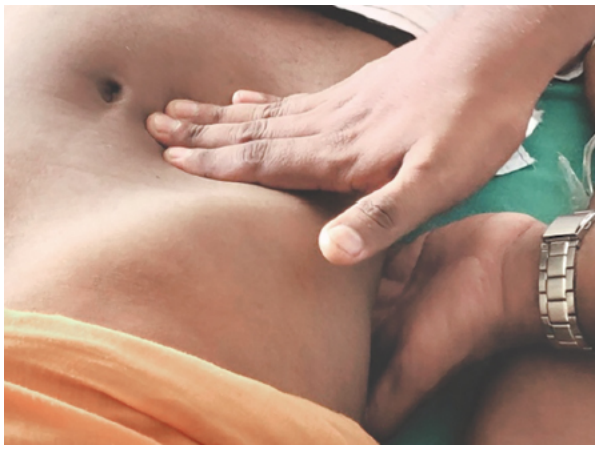


Fig. 5D.51: Palpation of right kidney.

Causes of unilateral and bilateral kidney enlargement:

Unilateral kidney enlargement	Bilateral kidney enlargement
1. Renal cell carcinoma 2. Hydronephrosis	1. Polycystic kidneys 2. Bilateral hydronephrosis

Differences between spleen and left kidney		
Characteristics	***Spleen***	***Left kidney***
Location	Left hypochondrium	Left lumbar
Direction of enlargement	Towards RIF	Towards left hypochondrium and LIF
Movement with respiration	+	–
Insinuation between left costal margin and organ	Not possible	Possible
Bimanual palpation	–	+
Ballotability	–	+
Crossing midline	Can cross midline	Never cross midline
Notch	+	–
Band of colonic resonance	–	+

Differences points between liver versus spleen versus kidney			
Features	***Liver***	***Spleen***	***Kidney***
Location	Right hypochondrium	Left hypochondrium	Lumbar
Direction of enlargement	Towards RIF	Towards RIF	Towards hypochondrium and iliac fossa
Movement with respiration	+	+	–
Insinuation of fingers between the costal margin and organ	Not possible	Not possible	Possible
Bimanually palpable	–	–	+
Ballotability	–	–	+

Anterior percussion	Dull	Dull	Tympanic

Examination of Free Fluid in Abdomen

Ascites

Definition:

Ascites is defined as the accumulation of free fluid in the peritoneal cavity. The peritoneal cavity can accumulate as much as 60 liters of fluid.

Massive ascites and tense ascites *are the clinical terms and are described at the end.*

Etiology of ascites			
Nonperitoneal causes		***Peritoneal causes***	
Intrahepatic portal hypertension	• Cirrhosis • Fulminant hepatic failure • Veno-occlusive disease	**Granulomatous peritonitis**	• Tuberculous peritonitis • Fungal and parasitic infections • Sarcoidosis • Foreign bodies (cotton, starch, barium)
Extrahepatic portal hypertension	• Hepatic vein obstruction (i.e. Budd–Chiari syndrome) • Congestive heart failure	**Malignant ascites**	• Primary peritoneal mesothelioma • Secondary peritoneal carcinomatosis
Hypoalbuminemia	• Nephrotic syndrome • Protein-losing enteropathy • Malnutrition	**Vasculitis**	• Systemic lupus erythematosus • Henoch-Schönlein purpura
Miscellaneous disorders	• Myxedema • Ovarian tumors • Pancreatic and biliary ascites	**Miscellaneous disorders**	• Eosinophilic gastroenteritis • Whipple disease • Endometriosis
Chylous	• Secondary to malignancy, trauma		

Serum-ascites albumin gradient (SAAG):

- SAAG = (serum albumin)–(albumin level of ascitic fluid)
- The serum-ascites albumin gradient (SAAG) is a better discriminant than older measures (transudate versus exudate) for the causes of ascites.
- The presence of a gradient ≥1.1 g/dL (≥11 g/L) predicts that the patient has portal hypertension with 97% accuracy.

High albumin gradient (SAAG ≥1.1 g/dL)	Low albumin gradient (SAAG <1.1 g/dL)
• Cirrhosis • Alcoholic hepatitis • Heart failure • Massive hepatic metastases • Heart failure/constrictive pericarditis • Budd–Chiari syndrome • Portal vein thrombosis • Idiopathic portal fibrosis	• Peritoneal carcinomatosis • Peritoneal tuberculosis • Pancreatitis • Serositis • Nephrotic syndrome • Biliary ascites • Bowel obstruction • Bowel infarction

Ascites praecox:

It is defined as appearance of **ascites** before the generalized edema. It is usually associated with chronic constrictive pericarditis.

Causes of ascites without significant edema:

- Chronic constrictive pericarditis
- Tuberculous peritonitis
- Malignant peritonitis
- Pancreatic ascites
- Acute Budd–Chiari syndrome.

Grading systems of ascites		
	The International Ascites Club grading (2003)	***Traditional system***
Grade 1	Mild ascites detectable only by ultrasonography	**1+** is minimal and barely detectable **2+** is moderate **3+** is massive but not tense **4+** is massive and tense
Grade 2	Moderate ascites manifested by moderate symmetrical abdominal distension	
Grade 3	Large or gross ascites with marked abdominal distension	

Following methods have been discussed of demonstration of ascites:

1. Fullness of flank
2. Horseshoe dullness
3. Shifting dullness
4. Fluid wave/fluid thrill
5. Puddle sign
6. Auscultatory percussion sign of Guarino.

1. Bulging flanks/fullness of flanks/horseshoe dullness

- Occurs when the weight of abdominal free fluid is sufficient to push the flanks outward **(Fig. 5D.52)**.
- On inspection, it can be seen as fullness of flanks or bulging of flanks.
- Bulging of flanks can be caused by ascites or by obesity.
- One method for discriminating between the two is to test for flank dullness.
- With the patient recumbent, gas-filled loops of bowel will characteristically float on top of ascites, making the percussion note tympanic at the umbilicus and dull beyond the fluid meniscus into the flanks—horseshoe dullness.

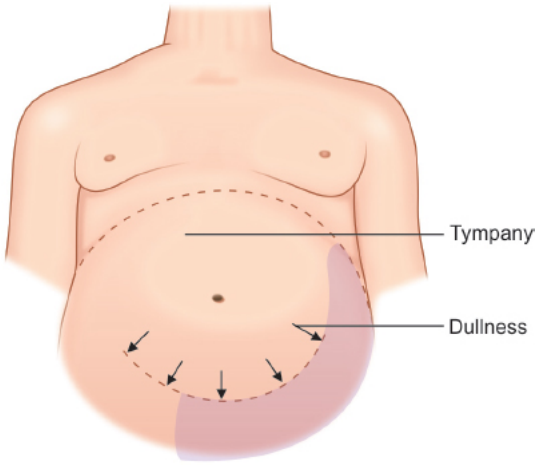


Fig. 5D.52: Horseshoe dullness.

2. Shifting dullness (Fig. 5D.53):

- Presence of shifting dullness indicates at least 1.5 liters of free fluid in the peritoneal space.

Examination (Figs. 5D.54A to K):

- Patient in supine position, start percussion from above downwards in the midline, till below the umbilicus you get dullness.
- This dullness could be due to distended urinary bladder, hence repeat this after making the patient empty the bladder.

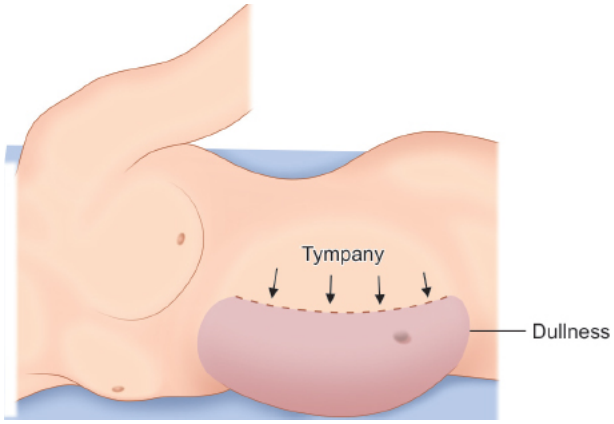
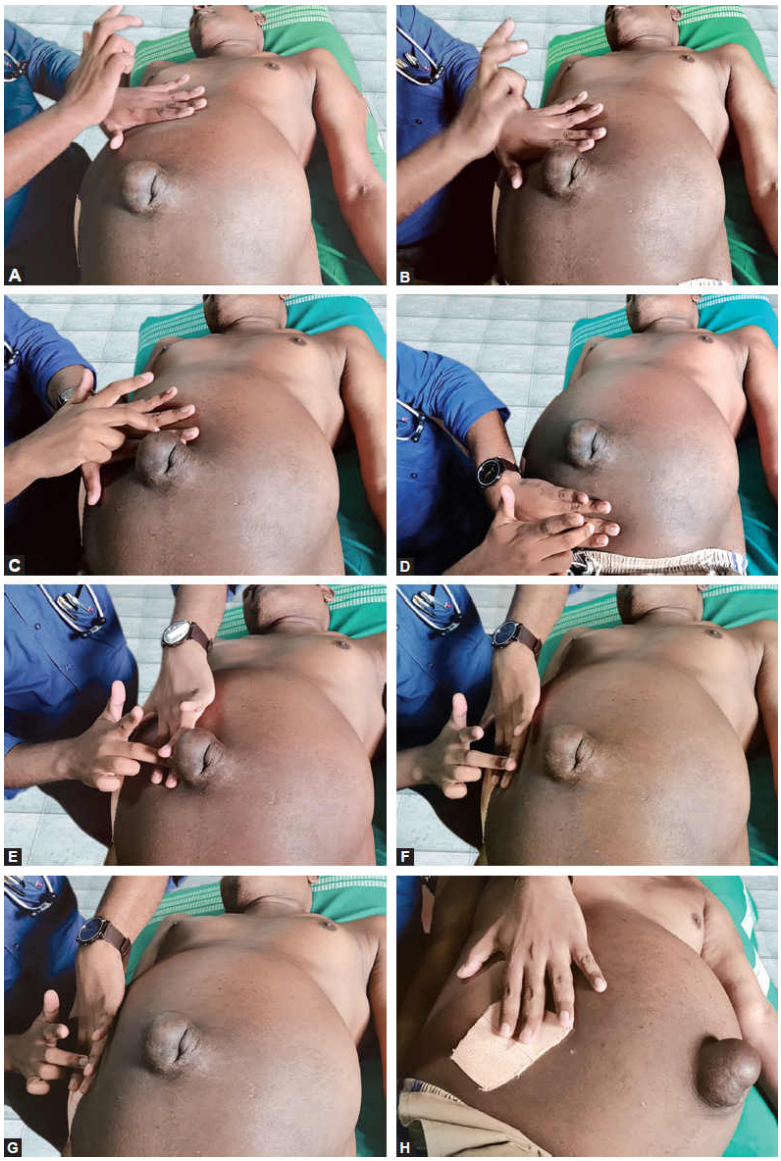
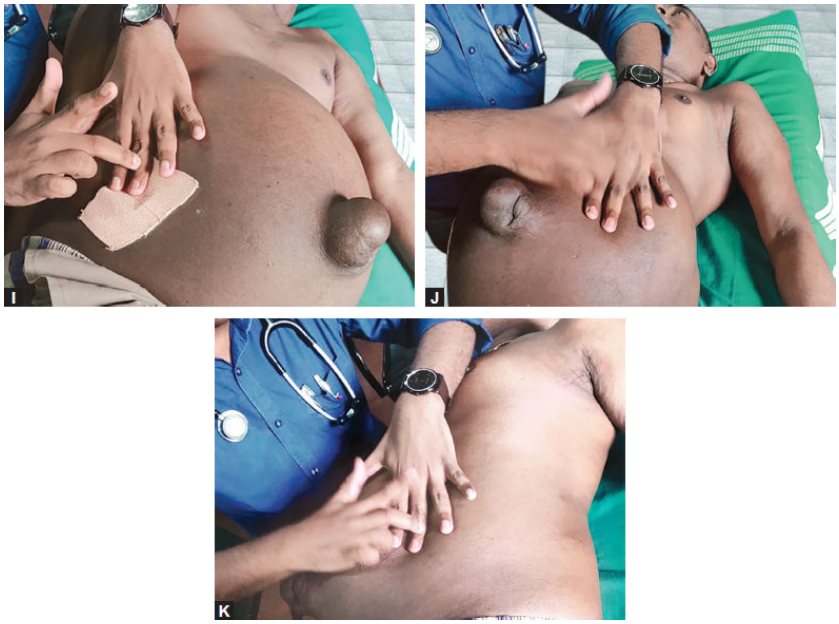


Fig. 5D.53: Shift of dullness on lying in lateral decubitus position.



Figs. 5D.54A to H



Figs. 5D.54I to K

Figs. 5D.54A to K: Demonstration of shifting dullness.

- Now, begin by percussing at the umbilicus and moving toward the flanks.
- The transition from air to fluid can be identified when the percussion note changes from tympanic to dull.
- Mark the dullness-tympany transition point.
- Turn the patient to opposite lateral side and wait for 30–60 seconds.
- Now percuss the area again.
- The area of tympany will shift towards the top and the area of dullness shifts towards the bottom.
- Repeat the same maneuver on the opposite side.

Causes of ascites without shifting dullness:

- Massive ascites
- Loculated ascites

3. Fluid thrill (fluid wave) assessment for ascites:

- In supine position, ask the patient or an assistant to place the ulnar surface of one hand above the umbilicus, pressing firmly (so the subcutaneous tissue and fat does not jiggle) with the hand pointing towards the patient's toes **(Fig. 5D.55)**.
- Use one hand to palpate and one hand to percuss.
- Place a hand on the lateral aspect of the patient's abdomen between the costal margin and the ilium in the anterior axillary line.

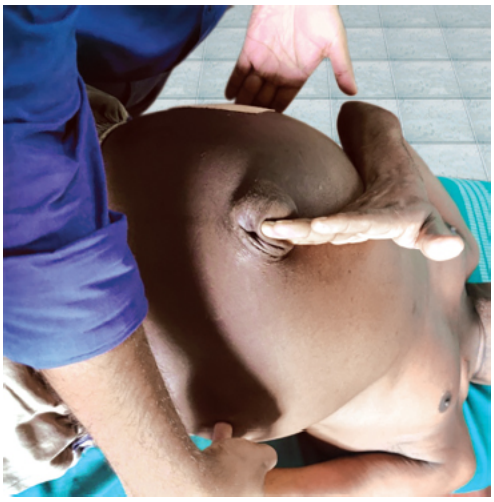


Fig. 5D.55: Demonstration of fluid thrill.

- Tap one side of the patients flank sharply with your fingertips.
- Feel on the opposite flank for an impulse transmitted through the fluid.
- Repeat procedure by flicking on the other side.
- Results:
  - » **Positive**: An easily palpable impulse is felt on the opposite side of tapping suggesting ascites of around more than 2 liters.
  - » **Negative**: No impulse is felt.
  - » **False positive**: Can be felt over large ovarian cyst or large hydatid cyst or large hydronephrosis.

4. Puddle sign (Fig. 5D.57):

- It is a sign of mild ascites of around 250 mL.
- Not frequently done.
- Patient is prone for 3–5 minutes and then examined in knee-elbow position as shown in the **Figure 5D.57**.
- Diaphragm of the stethoscope is placed over the most dependent area of the abdomen. Place diaphragm of the stethoscope over the umbilical region and scratch the abdominal wall from periphery to umbilicus.
- Sudden change in the note is a positive sign.
- Sign can be false positive in case of massive splenomegaly or distended urinary bladder.

5. Auscultatory percussion (described by Guarino)

- After voiding, the patient sits or stands so that free fluid gravitates to the pelvis, and the examiner places a stethoscope in the midline, immediately above the pubic crest.
- Finger-flicking percussion is performed along radial spokes from the subcostal margin downward toward the pelvis.
- The percussion note is initially dull but changes sharply to a loud note at the border of increased pelvic density.
- In the absence of ascites, the border is approximately 4.5 cm above the pelvic crest (the pelvic baseline).
- In patients with ascites, free fluid raises the demarcating border clearly above the pelvic baseline.
- When the patient is supine, this clear line of demarcation is obliterated because the free fluid gravitates to the flanks.

The sensitivity, specificity, and likelihood ratio of different methods of examination of ascites:

Method	Amount of fluid	LR+	LR-	Sn	Sp
Fullness of flanks		2.0	0.3	0.81	0.59
Horseshoe dullness		2.0	0.3	0.84	0.59
Shifting dullness	1.5 liters	2.7	0.3	0.77	0.72
Fluid thrill	> 2 liters	6.0	0.4	0.62	0.9
Puddle sign	250 mL	1.6	0.8	0.45	0.73

What is tense ascites and massive ascites?

- The earliest clinical sign of ascites is puddle sign which is positive with as low as 250 mL of ascitic fluid.
- Shifting dullness is a specific sign of ascites which occurs due to the floating of the bowel loops in ascitic fluid. This appears when the fluid accumulation is around 1.2 liters.
- As the fluids accumulate further, fluid thrill appears (at around 2 liters). Appearance of fluid thrill makes the ascites tense.
- As the ascitic fluid fills, the mesentery is stretched and bowel loops float in the ascitic fluid. As the mesentery can only stretch up to a limit, further fluid accumulation results in the submersion of bowel loops. At this stage, shifting dullness disappears; however, fluid thrill persists **(Fig. 5D.56)**. This condition is called as massive ascites.

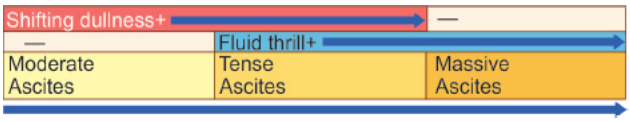


Fig. 5D.56: Schematic representation showing relationship between shifting dullness and fluid thrill with respect to increasing ascites.

Diagrammatic representation of signs of ascites (Fig. 5D.57):

Examination of Dilated veins

Position of Patient

Make the patient stand and examine the anterior abdominal wall, the flanks, and back for dilated veins. Dilated tortuous veins are significant.

Steps of examination (Harvey's sign) (Figs. 5D.58A to D):

- The direction of blood flow in the veins is examined by placing the tips of the index fingers together and compressing the vein.
- Then, the finger tips are slid apart producing an empty segment of the vein between the fingers **(Fig. 5D.59A)**.
- Then, one finger is removed and filling of the vein is observed **(Fig. 5D.59B).**
- The procedure is repeated but, now the opposite finger is removed and filling is observed **(Fig. 5D.59C)**.
- The direction of flow of the veins is the direction in which the filling was rapid and more.

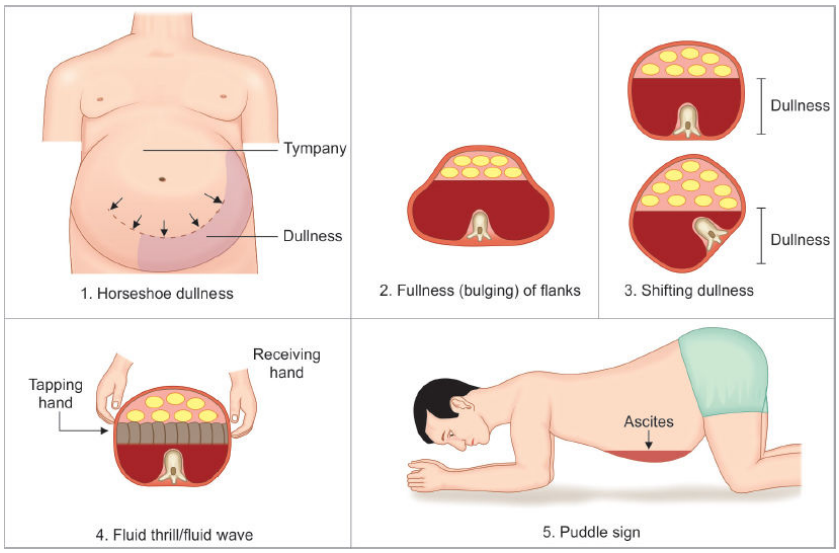
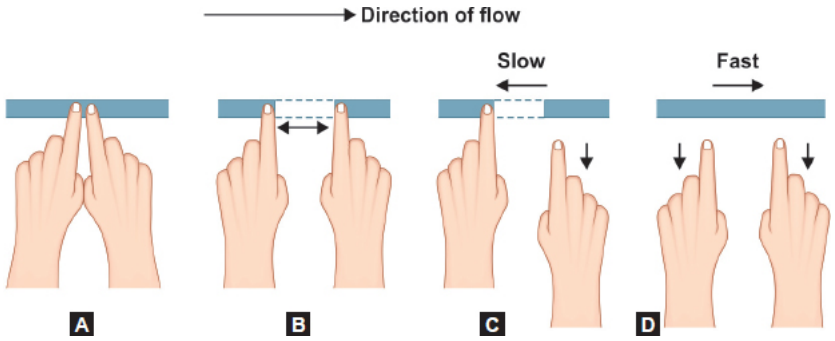


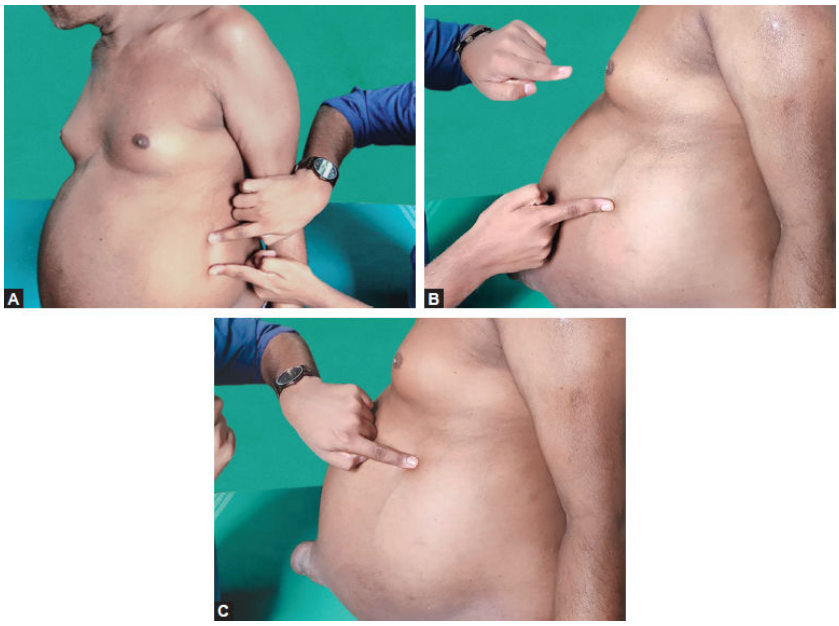
Fig. 5D.57: Signs of ascites.



Figs. 5D.58A to D: Harvey's sign.

Condition (Fig. 5D.60)	Direction of flow in veins above umbilicus	Direction of flow in veins below umbilicus
Normal (veins not visible)	Upwards	Downwards
Portal hypertension (veins are visible and tortuous)	Upwards	Downwards
Portal vein thrombosis	Downwards	Upwards
Superior vena cava (SVC) obstruction	Downwards	Downwards
Inferior vena cava (IVC) obstruction	Upwards	Upwards

Note: Caput medusa: Dilated tortuous veins around the umbilicus resembling the head of medusa.



Figs. 5D.59A to C: (A) The finger tips are slid apart producing an empty segment of the vein between the fingers; (B) One finger is removed and filling of the vein is observed; (C) Procedure is repeated but, now the opposite finger is removed and filling is observed.

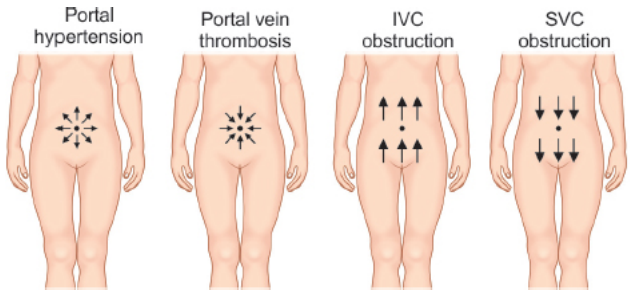


Fig. 5D.60: Direction of flow of veins.

Per-Rectal Examination

Rectal examination consists of:

- Visual inspection of the perianal skin
- Digital palpation of the rectum
- Assessment of neuromuscular function of the perineum.

Preferred position of examination:

The *lateral decubitus*, or *Sims position*, provides optimal examination. The patient lies on the left side with the buttocks near the edge of the examining table or bedside with the right knee and hip in slight flexion.

The rectal examination involves both inspection and palpation. First, using a gloved hand, the examiner inspects the buttocks for fistulous tracts, the skin tags, excoriations, blood, fissures in patients with inflammatory bowel disease, rectal prolapse, and superficial ulcers.

Palpation of the rectum can reveal ulcers, masses.

Tenderness may be felt with prostatitis, pelvic inflammatory disease, tubo-ovarian abscesses, ovarian cysts, ectopic pregnancy, and inflammatory bowel disease.

Also note the consistency, color, and presence of frank or occult blood in the stool (melena). Black stools result from degraded blood (melena), iron, licorice, bismuth, rhubarb, or overindulgence in chocolate cookies. Red-colored stools may be due to brisk bleeding known as hematochezia (usually distal to the ligament of Treitz).

Hemorrhoids are usually not felt unless thrombosed. Proctoscopy is the best way to look for hemorrhoids.

Others

Per vaginal/per speculum examination—

- In female patients with ascites, ovarian neoplasms, pelvic tumor, per vaginal mass/bleeding can be detected.
- GIT examination is incomplete without examination of the **three S's; Scrotum, Spine, and Supraclavicular Fossa**
- **Scrotum**—hydrocele, hernia, testicular atrophy, and testicular tumors
- **Spine**—metastasis and Pott's spine
- **Supraclavicular fossa**—metastasis to left scalene node.

COMPLICATIONS OF CIRRHOSIS

Table 5D.1 represents complications of cirrhosis.

Table 5D.1: Complications of cirrhosis.		
Portal hypertension and its sequelae	Hepatic encephalopathy	Hepatocellular carcinoma
Ascites	Portal gastropathy	Bleeding manifestations and coagulopathy
Spontaneous bacterial peritonitis	Hepatorenal syndrome	Cirrhotic cardiomyopathy
Portopulmonary hypertension	Hepatopulmonary syndrome	Hepatic hydrothorax
Coagulopathy, thrombocytopenia, Hyponatremia	Endocrine dysfunction—adrenal insufficiency, gonadal dysfunction, and thyroid dysfunction	Cirrhotic osteodystrophy

Hepatic Encephalopathy

Types of Hepatic Encephalopathy (Fig. 5D.61):

West Haven criteria clinical grade of hepatic encephalopathy		
Grade	***Description***	***Asterixis***
Grade 0/Minimal HE	Lack of detectable changes in personality or behavior	Absent
Grade 1	Trivial lack of awareness, euphoria or anxiety, shortened attention span, impaired performance of addition	May be present
Grade 2	Lethargy or apathy, minimal disorientation for time or place, subtle personality change, inappropriate behavior, slurred speech, impaired performance of subtraction	Present
Grade 3	Somnolence to semi-stupor, but responsive to verbal stimuli, confusion, gross disorientation	Usually absent
Grade 4	Coma (unresponsive to verbal or noxious stimuli)	–

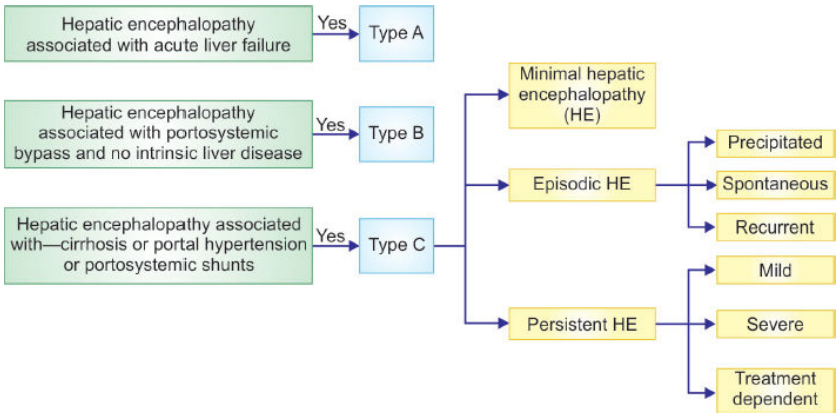


Fig. 5D.61: Types of hepatic encephalopathy.

Asterixis:

Described earlier in signs of liver cell failure.

Diagnosis of Minimal Hepatic Encephalopathy

It is currently based on neuropsychometric tests, including the number connection test, digit symbol test, and the block design test.

Reitan's number-connection test (Fig. 5D.62):

There are 25 numbered circles which can normally be joined together within 30 seconds.

Hepatorenal Syndrome

Diagnostic criteria for hepatorenal syndrome
All of the following must be present for the diagnosis of hepatorenal syndrome (HRS)
• Cirrhosis with ascites • Serum creatinine >1.5 mg/dL • No improvement of serum creatinine (decrease to a level of 1.5 mg/dL or less) after at least 2 days of diuretic withdrawal and volume expansion withalbumin • Absence of shock • No current or recent treatment with nephrotoxic drugs • Absence of parenchymal kidney disease as indicated by proteinuria >500 mg/day, microhematuria (>50 red blood cells per high power field), and/or abnormal renal ultrasonography

Time required in seconds	HE grade
≤30	None-minimal
31–50	Minimal-1
51–80	I-II
81–120	II-III
Forced termination	III

Fig. 5D.62: Reitan's number-connection test.

Types of hepatorenal syndromes (HRS)	
Type 1 hepatorenal syndrome	***Type 2 hepatorenal syndrome***
• It is characterized by progressive oliguria, a rapid rise of the serum creatinine to above 2.5 mg/dL and has a very poor prognosis • Usually precipitated by spontaneous bacterial peritonitis • Without treatment, median survival is less than 1 month and almost all patients die within 10 weeks after the onset of renal failure	• It is characterized by a reduction in glomerular filtration, moderate and stable increase in serum creatinine (>1.5 mg/dL), but it is fairly stable and has a better prognosis than Type 1 HRS • Usually occurs in patients with refractory ascites (resistant to diuretics) • Median survival is 3–6 months

Precipitating factors for hepatorenal syndrome
• Gastrointestinal bleeding • Aggressive paracentesis • Diuretic therapy • Sepsis including spontaneous bacterial peritonitis • Diarrhea

SITES OF PORTOSYSTEMIC ANASTOMOSIS (FIG. 5D.63)

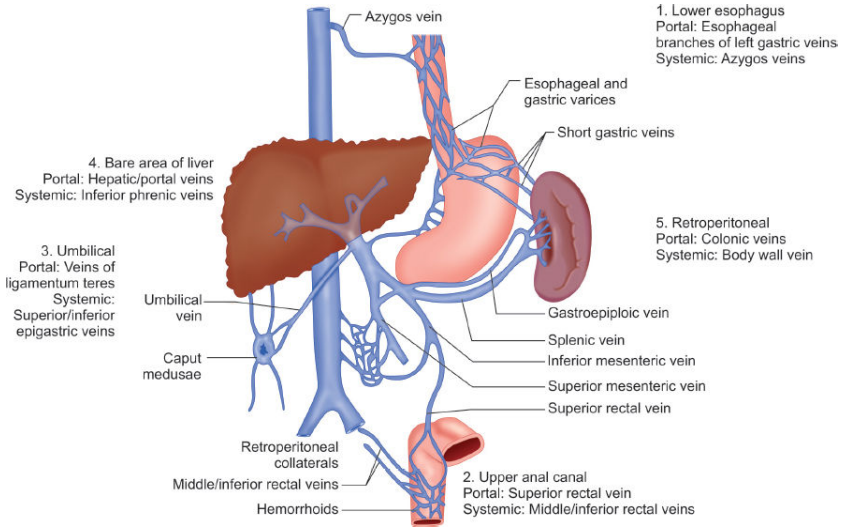


Fig. 5D.63: Sites of portosystemic anastomosis in cirrhosis.

CLASSIFICATION OF PORTAL HYPERTENSION (FIG. 5D.64)

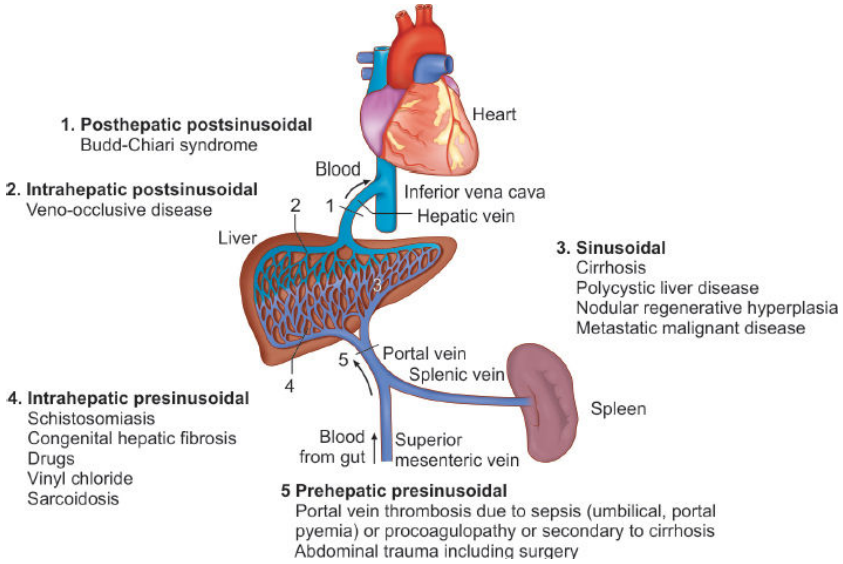


Fig. 5D.64: Classification of portal hypertension according to site of vascular obstruction.

NOTES

CHAPTER 6

Nervous System

A. CASE SHEET FORMAT

HISTORY TAKING

Name:

Age:

Sex:

Residence:

Occupation:

Chief complaints:

1. _____ × days
2. _____ × days
3. _____ × days

History of presenting illness:

HIGHER MENTAL FUNCTION

Altered state of consciousness:

- Onset
- Any seizures and blackouts
- Any fall/injuries
- Any ear or nose bleed
- Fever
- Any ear pain or discharge
- Drug history
- Any addictions.

Mental state and cognition:

- Changes in the memory
- State of alertness and drowsiness
- Changes in the mood and affect (loss of spontaneity)
- Language changes
- Loss of spatial orientation
- Diminished ability to carry out routine activities of daily living.

Other higher mental functions:

- Speech difficulty
- Difficulty to recognize people or objects
- Inappropriate crying or laughter
- Lack of interest
- Social disinhibition
- Delusions/hallucinations.

CRANIAL NERVE DYSFUNCTION

Ask about:

- Loss of vision, smell, and taste
- Alteration in facial feeling
- Double vision/visual symptoms
- Problems with swallowing and chewing
- Speech alterations
- Vertigo/hearing abnormalities
- Hoarseness of voice, dysphagia, nasal regurgitation, and nasal intonation of speech
- Pain/difficulty in neck movements.

Example

Left lower motor neuron (LMN) 7th nerve palsy: History of retroauricular pain followed by abrupt onset deviation of angle of mouth to right with slurring of speech and difficulty in left eye closure with history of hyperacusis.

MOTOR DYSFUNCTION

Weakness

Distribution of weakness:

- Is it symmetrical/asymmetric:
- Paresis or plegia:
- Limbs involved:
- Ipsilateral or contralateral:
- Patterned weakness.

Example

Right middle cerebral artery (MCA) territory embolic infarct: History of sudden onset, complete loss of power in left upper limb and lower limb. Weakness maximum at onset and nonprogressive.

Onset and progression:

- *Acute, subacute, or chronic*

Progression of the weakness:

- *Ascending weakness or descending weakness*
- *Ellsberg phenomenon*
- *Variation throughout the day*
- *Muscles/limb(s) involved.*

Proximal upper limb—shoulder/arm:	Difficulties in combing hair, reaching for high objects, winging of scapula
Distal upper limb—forearm/hand:	Finger/wrist drop, poor hand grip, cannot open jar, difficulty in buttoning/unbuttoning
Proximal lower limb—pelvic/thigh:	Cannot rise from chair or squatting position, waddling gait
Distal upper limbs—leg/foot:	Difficulty in gripping *chappals*, cannot walk on heels/toes foot drop
Neck muscles	Dropped head/broken neck

Trunk	Inability to roll on the bed

Example

Guillain–Barré syndrome (GBS): History of preceding gastrointestinal (GI) infection followed by acute onset difficulty in getting up from squatting position, difficulty walking, progressing to involve upper limbs (difficulty combing hair), and neck muscle weakness. No sensory symptoms.

Wasting/Loss of Muscle Bulk

- Wasting—present/absent
- Fasciculations—present/absent

Stiffness of Limbs

- Stiffness—present/absent
- Heaviness—present/absent

Gait Abnormalities

- Limp or dragging foot
- Scissoring/circumduction.

Involuntary Movements

- Type
- Symmetrical/asymmetrical
- Part of the body involved
- Present at rest
- Functional disability.

SENSORY DYSFUNCTION

- Numbness/loss of feeling
- Altered feeling:
  - Paresthesia
  - Dysesthesias (tingling and pin-needles)
  - Spontaneous pain
- Pattern of sensory loss.

CEREBELLAR HISTORY

- Swaying to one side
- Tremors while reaching objects
- Lack of coordination of activities
- Overshooting acts
- Abnormal involuntary eye movements (oscillopsia/nystagmus).

HISTORY SUGGESTING MENINGITIS/RAISED INTRACRANIAL PRESSURE

- Headache
- Neck pain
- Projectile vomiting
- Blurring of vision
- Seizures
- Photophobia.

HISTORY SUGGESTING AUTONOMIC DYSFUNCTION

- Dryness of skin
- Palpitations
- Perspiration
- Syncopal attacks/postural giddiness
- Bladder dysfunction:
  - Urinary retention
  - Loss of awareness of bladder control
  - Frequency, urgency
  - Urge/overflow incontenence.

REVIEW OF COMMON NEUROLOGICAL SYMPTOMS

Headaches

- Onset and duration of headache
- Location of headache, unilateral versus bilateral
- Severity
- Frequency
- Radiation
- Quality of headache (dull and diffuse)
- Types:
  a. Continuous
  b. Pulsating
  c. Stabbing
  d. Sharp
  e. Throbbing
  f. Dull
  g. Thunderclap
- Alleviating factors
- Triggers for the headache/aggravating factors
- Temporal association (headache not worse in mornings)
- Association with nausea/vomiting/tearing of eyes/redness of eyes
- Vision changes before or during headache
- Precipitating factors:
  - Stress
  - Menses
  - Allergens

  - Sleep deprivation
  - Coughing
  - Straining
  - Bending forwards
- Associated motor/sensory symptoms: Weakness, numbness, and tingling in upper or lower extremities
- Photophobia/phonophobia
- Systemic symptoms—weight loss, low energy, and anorexia
- Fever and neck stiffness
- History of head trauma
- History of migraine
- Family history of migraines
- Effect on daily activities
- Use of oral contraceptive pills
- Caffeine intake
- Smoking and alcohol history.

Example

Classical migraine: Visual aura followed by insidious onset, unilateral, severe pulsating type of heading lasting for >4 hours associated with nausea and photophobia. Repeated such attacks every month with history of some identifiable precipitating factors and a positive family history of migraine.

Seizures

- Onset and duration
- Frequency
- Factors which precipitate these episodes
- Injury sustained as a result of the seizure
- Postictal symptoms: Confusion
- Associated sensory deficits
- Associated motor deficits
- Associated cognitive deficits
- Muscle spasms
- Anatomical progression of motor involvement (e.g. Jacksonian March)
- Symptoms suggesting aura
- Associated incontinence
- Tongue biting and salivation
- Automatisms associated with these episodes
- History of head trauma
- Perinatal infection
- Drug history
- History of seizure disorder
- Family history of seizure disorders
- Effect on daily activities.

Example

Generalized tonic clonic seizure (GTCS): Abrupt onset tonic clonic contraction of muscle associated with tongue bite and urinary incontinence. Patients generally regain consciousness within few minutes with postictal confusion and headache.

Past history:
- Asthma
- Chronic obstructive airway disease
- Tuberculosis
- History of contact with tuberculosis
- Diabetes mellitus (DM)
- Hypertension (HTN)
- Ischemic heart disease (IHD)
- Seizure disorder and drugs used (in detail).

Family history:
(draw pedigree chart representing three generations)

Personal history:
- Bowel habits
- Bladder habits
- Appetite
- Loss of weight
- Occupational exposure
- Sleep
- Dietary habits and taboo
- Food allergies
- Smoking (in smoking Index or Pack years)
- Alcohol history (__ grams of alcohol/day or ___ units of alcohol/week).

Menstrual and obstetric history:
- G__P__L__A__
- Age of menarche __
- Menopause at __
- Flow—amenorrhea/oligorrhea/menorrhagia.

Summarize:

Differential diagnosis:
1.
2.
3.

GENERAL EXAMINATION

Patient
- Conscious
- Cooperative
- Obeying commands

Body Mass Index (BMI)

- Wt (kg)/Ht^2 (meters)
- Grading according to WHO for Southeast Asian countries

Vitals

- **Pulse**
  - Rate
  - Rhythm
  - Volume
  - Character
  - Vessel wall thickening
  - Radio-radial delay and radio-femoral delay
  - Peripheral pulses
- **Carotid and vertebral bruit**
- **Blood pressure**
  - Right arm
  - Left arm
  - Leg—right/left
- **Respiratory rate**
  - Regular
  - Abdominothoracic (male) or thoracoabdominal (female)
  - Usage of accessory muscles
- **Jugular venous pulse**
  - Waveform
- **Jugular venous pressure**
  - __ cm of blood above sternal angle (+ 5 cm water)

On Physical Examination

- Pallor
- Icterus
- Cyanosis
- Clubbing
- Lymphadenopathy
- Edema

Others Head to Toe

- Nerve thickening
- Neurocutaneous markers
- External markers of atherosclerosis
- Signs of nutritional deficiency, alcoholism, etc.
- Any other general examination finding

NERVOUS SYSTEM EXAMINATION

- Right/left handed person
- Education

HIGHER MENTAL FUNCTIONS

- Consciousness—if impaired document using Glasgow coma scale
- Orientation to time/place/person
- Memory:
  - Immediate (repetition—30 seconds)
  - Recent (up to 5 minutes—recall)
  - Remote (> 5 minutes)
- Intelligence
- Mood/emotion
- Concentration and calculation (subtract seven from 100)
- Speech:
  - Spontaneous speech—comprehension
  - Fluency
  - Repetition
  - Reading
  - Writing
  - Naming objects
  - Phonation
  - Aphasia
  - Dysarthria
- Apraxias—present/absent
- Hemineglect—present/absent
- Hallucinations and delusions—present/absent

Cranial nerves	R	L
Olfactory—I nerve: Sense of smell (peppermint, soap, coffee, lemon peel or vanilla) *Both eyes shut, one nostril checked at a time Appreciate smell ± identify it		
Optic—II nerve: Visual acuity (perception of light/hand movements and finger counting/Snellen's chart at 6 meters/Jaeger's chart at 14 inches) Visual field (confrontation method/menace reflex)—mention defects, if any Color vision (Ishihara's test) Fundus		
Oculomotor, trochlear, abducens—III, IV, VI nerves: Eyelids (any ptosis) Position of eyeballs at rest (any deviation, exophthalmos, enophthalmos) Extraocular movements: I. Binocular movements – Saccadic: – Pursuit: – Reflex (doll's eye, caloric stimulation) II. Uniocular movements (#Comment on ophthalmoplegia, if present—supranuclear, internuclear, individual nerves, or muscles) Pupil • Size (in mm) • Shape		

• Reaction • Direct light reflex • Consensual light reflex • Accommodation reflex Nystagmus (Describe whether spontaneous or provoked/type—horizontal, vertical, rotatory, pendular)		
Trigeminal nerve—V nerve: • **Sensory:** – Touch – Pain – Temperature (To be checked on all three divisions around the jawline, on the cheek, and on the forehead) • **Motor:** – Jaw deviation – Hollowing above and below zygoma – Clenching teeth (feel temporalis and masseter) – Open mouth against resistance – Side to side movement of jaw (pterygoid) • **Reflexes:** – Corneal—present/absent (superficial reflex, 5th nerve afferent, 7th nerve efferent) – Jaw jerk—present/absent/exaggerated (deep reflex, afferent and efferent, both 5th nerve, center mid-pons)		
Facial nerve—VII nerve: Facial asymmetry (look for absence of wrinkling, drooping of corner of mouth, obliteration of nasolabial fold, widened palpebral fissures) • **Motor:** – Frontalis (raise the eyebrows) – Orbicularis oculi (shut the eyes tight) – Buccinator (show teeth, smile, blow check, whistle) – Orbicularis oris (close lips, pronounce labials “p”,”b”,’’m”) – Platysma (pull down the corners of mouth) (## Look for Bell’s phenomenon) • **Sensory:** – Anterior 2/3rd tongue taste (sugar, lime, salt, quinine) **Lacrimation** **Hyperacusis**—present/absent **Emotional fibers checking**—emotions preserved or not		
Vestibulocochlear nerve—VIII nerve: The ability to hear the sound produced by rubbing the thumb and forefinger together is then tested for each ear at distances up to a few centimeters • Rinne’s test—air conduction/bone conduction (AC/BC) • Weber’s test—lateralized/centralized • Caloric test [Irrigates one external auditory canal with cool (about 30°C) or warm (40°C) water. Normally, cool water in one ear produces nystagmus on the opposite side. Warm water produces it on the same side]		
Glossopharyngeal, vagus IX, X nerve: Note the patient’s ability to drink water and eat solid food and also see the character, volume and sound of the patient’s voice. • Position of uvula • Movement of uvula on saying “ah”—any deviation • Gag reflex—present/absent/exaggerated (taste over the posterior third of the tongue and can be tested)		
Spinal accessory—XI nerve: • Sternocleidomastoid (instruct the patient to rotate head against resistance applied to the side of the chin to tests the function of the opposite sternocleidomastoid muscle. To test both sternocleidomastoid muscles together, the patient flexes the head forward against resistance placed under the chin)		

• Trapezius (shrugging a shoulder against resistance)		
Hypoglossal nerve—XII: Inspection (inside the mouth): • Size of tongue • Symmetry/any wasting • Fasciculation (on protrusion) • Deviation—side • Tremors Palpation: • Tone • Power • Speech		

MOTOR SYSTEM

Attitude

- Upper limb
- Lower limb

Bulk

Inspection: Symmetry, generalized wasting comment on small muscle wasting, deformities, claw hand, foot drop, if any.

Measurement in cm	*R*	*L*
Arm (10 cm above olecranon)		
Forearm (10 cm below olecranon)		
Thigh (18 cm above the superior border of patella)		
Leg (10 cm below the tibial tuberosity)		

Note: Bilateral similar distance from fixed bony points till the maximum bulk of muscle.

Tone

	R	*L*
Upper limb		
Lower limb		

Note: Comment whether normal, hypotonia or hypertonia (spasticity/rigidity).

Power

Checked both isometric (resistance against movement) and isotonic (resistance at end of movement).

0	Complete paralysis
1	A flicker of contraction only
2	Power detectable only when gravity is excluded by postural adjustment
3	Limb can be held against gravity but not resistance
4	Limb can be held against gravity and some resistance

5	Normal power

Muscle	R	L
Neck • **Flexors** (SCM, platysma, scalene, suprahyoid, infrahyoid, longus colli and capitis, rectus capitis) • **Extensors** (trapezius and paravertebral muscles—splenius, erector spinae, transversospinalis, interspinal intertransverse) *Note:* Avoid active movement checking if cervical cord injury suspected		
Shoulder • **Abduction** (0–15°—supraspinatus, 15–90°—middle fibers of deltoid, above 90°—trapezius and serratus anterior) • **Adduction** (pectoralis major, latissimus dorsi and teres major) • **Flexion** (biceps brachii (both heads), pectoralis major, anterior deltoid, and coracobrachialis) • **Extension** (posterior deltoid, latissimus dorsi, and teres major)		
Elbow • Flexion (biceps brachii) • Extension (triceps brachii)		
Wrist • Flexion (FCR, FCU) • Extension (ECRL, ECRB, ECU)		
Hand grip (long flexors)		
Small muscles of hand		
Trunk (rectus abdominis, transversus abdominis, oblique, pyramidalis) • Elevation of head or leg in supine position • Beevor's sign if present • Abdominal binding to check for intercostal muscle weakness • Intercostal binding to check for diaphragmatic weakness		
Hip • Flexion (iliopsoas) • Extension (gluteus maximus) • Abduction (gluteus medius and minimus, tensor fascia lata) • Adduction (adductor longus, brevis, and magnus)		
Knee • Flexion (hamstrings) • Extension (quadriceps)		
Ankle • Plantar flexion (gastrocnemius, soleus) • Dorsiflexion (tibialis anterior)		
Small muscles of foot, EHL if needed		

REFLEXES

Superficial reflexes	R	L
Corneal (cranial nerve V and VII)		
Abdominal: • Epigastric (T6–T9) • Mid-abdominal (T9–T11)		

• Hypogastric (T11–L1)		
Cremasteric (L1, L2)		
Anal reflex (S2, S3)		
Plantar: • Reflexogenic zone—S1 • Afferent nerve—tibial nerve • SC segments—L4, L5, S1, S2		
Chaddock's (lateral aspect of foot from below up), Gordon's (calf), Oppenheim's (anterior tibia), Schaffer's (Achilles tendon), Gonda's (press down 4th toe), Stransky's (adduct little toe), Bing's (pinprick on dorsolateral foot)		

Deep tendon reflexes	R	L
Jaw jerk (afferent and efferent both 5th nerve and center mid pons)		
Biceps (C5, C6)		
Brachioradial/supinator/radial periosteal (C5, C6)		
Triceps (C6, C7, C8)		
Knee jerk/quadriceps/patellar reflex (L2, L3, L4)		
Ankle jerk (L5, S1, S2)		
Clonus—present/absent • Patellar • Ankle		
Latent reflexes (suggest pyramidal lesion if present unilaterally) Tromner's/finger flexor reflex/Hoffmann's sign Wartenberg's sign		
By convention the deep tendon reflexes are graded as follows: • 0 = no response; always abnormal • 1+ = a slight but definitely present response; may or may not be normal • 2+ = a brisk response; normal • 3+ = a very brisk response; may or may not be normal • 4+ = a tap elicits a repeating reflex (clonus); always abnormal		
Please do reinforcement maneuvers before saying DTR's are absent		
Primitive reflexes • Glabellar tap • Palmomental (both sides) • Sucking • Rooting • Pout and snout • Grasp		

Involuntary movements (describe in detail)

Coordination (described later under cerebellum)

SENSORY SYSTEM

Primary sensation	R	L
Touch		

Pain		
Temperature		
Vibration		
Joint position sense Any sensory level Pattern of sensory loss (graded/dissociative/crossed/hemi)		

Cortical sensation (to be tested only in the presence of primary sensation intact)	R	L
Tactile localization (topognosis)		
Two point discrimination		
Stereognosis		
Graphesthesia (figure identification)		
Sensory extinction		

Romberg's test:

CEREBELLAR SIGNS

Upper extremity	*R*	*L*
Limb ataxia: • Outstretched arm test • Finger nose test • Nose-finger-nose test • Finger-finger test		
Rapid alternating movements: • Rapid hand tapping • Pronation-supination • Thigh slapping		
Pointing and past pointing		
Writing (macrographia)		
Rebound phenomenon (arm)		
Tremors (intention)		

Lower limbs	*R*	*L*
Heel knee test		
Pendular knee jerk		
Finger toe test		
Rapid alternating movements—foot tapping		
General		
Titubation		
Nystagmus		

Tremors
Hypotonia
Truncal ataxia
Tandem walking
Gait

GAIT

- Base—wide or narrow
- Slow/rapid
- Falling to sides
- Look which part of foot touches ground first (toe/heel)
- How high foot lifted above ground?
- Hand swing
- Turning around
- Position of hip, sound produced while foot touches ground.

Signs of Involvement of Autonomic Nervous System

- Dryness of skin/excessive sweating/spoon test
- Postural hypotension
- Heart rate—baseline, on respiration, on standing
- Palpable bladder
- Pupillary reactions
- Valsalva maneuver.

Signs of Meningeal Irritation

- Neck stiffness
- Kernig's sign
- Brudzinski's sign—neck, leg, and pubis.

Skull and Spine

- Deformities
- Tenderness
- Short neck.

SOFT NEUROLOGICAL SIGNS

- ***Pyramidal drift*** describes a tendency for the hand to move upward and supinate if the hands are held outstretched in a pronated position (palms downward), or to pronate downward if the hands are held in supination.
- ***Cerebellar drift*** is generally upward with excessive rebound movements if the hand is suddenly displaced downward by the examiner.
- ***Parietal drift*** is an outward movement on displacing the ulnar border of the supinated hand.

OTHER SYSTEMS

Respiratory system:

- Inspection:
- Palpation:
- Percussion:
- Auscultation:

Cardiovascular system:

- Inspection:
- Palpation:
- Percussion:
- Auscultation:

Gastrointestinal system:

- Inspection:
- Palpation:
- Percussion:
- Auscultation:

B. DIAGNOSIS FORMAT

GENERAL FORMAT

Nature of Disease

- Onset: Sudden/acute/subacute/chronic (sudden—vascular, acute—demyelinating, subacute—infections/space occupying lesions, chronic—degenerative)
- Deficit: Monoplegia/hemiplegia/quadriplegia/paraplegia/nerve palsies/ataxia/sensory disturbance/movement disorders.

Site of Involvement of Nervous System

- Upper motor neuron disease—intracranial (brain or cerebellum) or extracranial (spinal cord)
- Lower motor neuron disease—anterior horn cell disease, radiculopathies, neuropathies, neuromuscular junction diseases, and myopathies.

FOR CEREBROVASCULAR ACCIDENT

Sudden onset, right-sided dense hemiplegia with right upper motor neuron (UMN) facial palsy due to cerebrovascular accident possible thrombotic in etiology with site of lesion being left internal capsule, possible involving the lenticulostriate branch of middle cerebral artery (MCA). Patient is in state of neuronal shock. Patient has following risk factors _____.

FOR NEUROPATHY

Acute onset of symmetrical flaccid quadriplegia (ascending) with no evidence of sensory, bowel, bladder involvement with bilateral lower motor neuron (LMN) facial palsy, possible site of lesion in the peripheral nerve, pathology being demyelination—acute inflammatory demyelinating polyneuropathy (AIDP).

FOR SPINAL CORD DISEASE

Subacute onset of symmetrical spastic paraplegia with involvement of sensory, bladder, and bowel; with no involvement of cranial nerves with vertebral tenderness at T4-5, possible site of lesion is spinal cord, the disease being compressive myelopathy.

- Horizontal level
  - Extradural extramedullary
- Vertical level
  - Motor level: Above T10
  - Sensory level: At T8
  - Autonomic level: Above T12
  - Reflex level: Above T10
  - Spinal level: T8
  - Vertebral level: T5.

Possible etiology: Tuberculosis—Pott's spine.

FOR EXTRAPYRAMIDAL (PARKINSON'S DISEASE)

Insidious onset, slowly progressive, degenerative disease involving the motor system (in the form of rigidity and tremors) with no evidence of sensory, cranial nerves or bowel, bladder, we would consider involvement of extrapyramidal system probably parkinsonism with no evidence of secondary causes, no signs or symptoms of Parkinson's plus syndromes, functional status—Stage III (Hoehn and Yahr staging system).

FOR ATAXIA

Insidious onset, slowly progressive, symmetrical ataxia and cerebellar signs of trunk and limbs with no evidence of sensory, cranial nerve or autonomic involvement. I would like to consider the possibility of degenerative cerebellar ataxia possibly inherited (family history +ve).

C. CENTRAL NERVOUS SYSTEM: DISCUSSION ON CARDINAL SYMPTOMS

DISCUSSION ON CARDINAL SYMPTOMS

Taking a Neurological History

The neurological history should be a focused, goal-directed exercise that seeks to answer the following questions:

1. Which part of the nervous system is affected by "a pathological process" and is causing the symptoms (where is the lesion)? Is it a single lesion or are there multiple diffuse lesions? Alternatively, is there a diffuse problem affecting many neurological systems?
2. What is the underlying pathological process (e.g. vascular, inflammatory, degenerative)?
3. Is this a purely neurological problem or a neurological manifestation of a systemic disease?

Note:

- Ask the patient to tell their story in their own words
- Explore each symptom in detail, evaluating the evolution and the way the symptoms affect the ability to function
- Ask for an eyewitness account when cognition or consciousness is involved
- If you cannot make a neurological diagnosis, take the history again before arranging investigations.

Pathology of neurological diseases		
Acute	***Subacute***	***Chronic***
Vascular—stroke Demyelination Metabolic	Infection Space occupying lesions Metabolic	Degeneration

HIGHER MENTAL FUNCTION

Altered State of Consciousness

- Onset
- Any seizures, blackouts
- Any fall/injuries
- Any ear or nose bleed
- Fever
- Any ear pain or discharge
- Drug history
- Any addictions.

Other Higher Mental Functions

- Speech difficulty
- Difficulty to recognize people or objects
- Memory defects
- Inappropriate crying or laughter

- Lack of interest
- Social disinhibition
- Delusions/hallucinations.

Mental State and Cognition

- Changes in the memory
- State of alertness and drowsiness
- Changes in the mood and affect (loss of spontaneity)
- Language changes
- Loss of spatial orientation
- Diminished ability to carry out routine activities of daily living.

CRANIAL NERVE DYSFUNCTION

Ask about:

CN	Symptoms
1	Smell disturbance
2, 3, 4, 6	Diplopia, blurred vision, blindness, difficulty in opening eyelid (CN3)
5	Difficulty in chewing, loss of sensations over face
7	Deviation of angle of mouth, accumulation of food at one side of the mouth, dribbling of saliva, loss of taste sensation, hyperacusis
8	Tinnitus, hearing loss, dizziness, loss of balance
9, 10	Nasal intonation, nasal regurgitation of food, dysphagia, difficulty in speech, hoarseness of voice
11	Difficulty in neck/shoulder movements
12	Difficulty in mixing food in the mouth, difficulty in speech

For example: *Left LMN 7th nerve palsy—history of retroauricular pain followed by abrupt onset deviation of angle of mouth to right with slurring of speech and difficulty in left eye closure with history of hyperacusis.*

MOTOR DYSFUNCTION

Weakness

Distribution of Weakness

- Is it symmetrical/asymmetric?
- Plegia—complete loss of power—0/5 vs paresis—incomplete loss of power
- One limb: Monoparesis.
- Two limbs, same side: Hemiparesis.
- Both lower limbs: Paraparesis.
- All four limbs: Quadriparesis (or tetraparesis).
- **Pentaplegia** is a spinal cord injury at or above C4 level, resulting in complete loss of motor functions below the injury level and paralysis of respiratory muscles.

- Two (contralateral to each other) or three limbs (upper and lower limbs), e.g. right upper limb and left lower limb or left arm and both legs, both arms and one leg.
- Patterned weakness:
  - The pattern of pyramidal weakness is weakness of upper limbs extensors and lower limbs flexors.

For example: *Right MCA territory embolic infarct—history of sudden onset, complete loss of power in left upper limb, lower limb associated with left UMN facial palsy. Weakness—maximum at onset, nonprogressive.*

Causes of monoplegia affecting the lower limb	Causes of monoplegia affecting the upper limb
1. Stroke, affecting anterior cerebral artery territory. 2. Cerebral venous sinus thrombosis affecting superior sagittal sinus. 3. Trauma, head injury, with contusion in the frontal lobe. 4. Infection, such as granuloma affecting frontal lobe. 5. Trauma to the lumbosacral plexus, diabetic lumbosacral plexopathy. 6. Functional or psychogenic.	1. Stroke, affecting superior division of contralateral middle cerebral artery territory, affecting parietal lobe, or unpaired anterior cerebral artery. 2. Head injury, with contusion in the parietal lobe. 3. Trauma to the brachial plexus. 4. Injury to multiple cervical nerve roots. 5. Functional or psychogenic.

Causes of hemiplegia
1. Ischemic or hemorrhagic stroke, affecting contralateral cerebral hemisphere, internal capsule, brainstem or ipsilateral upper cervical cord. 2. Cerebral venous sinus thrombosis with venous infarction of contralateral cerebral hemisphere. 3. Acute central nervous system infection, such as meningitis or encephalitis, brain abscess, granulomatous infections. 4. Head injury causing contusion/bleeding in the contralateral cerebral hemisphere, internal capsule, basal ganglia, or brainstem. 5. Tumor affecting cerebral hemisphere, internal capsule, basal ganglia, brainstem or cervical cord. 6. Bleeding into a brain tumor on the contralateral side. 7. Demyelinating illness, such as acute disseminated encephalomyelitis (ADEM) or multiple sclerosis (MS). 8. Todd's paresis. 9. Mill's hemiplegic variant of motor neuron disease (MND).

Causes of Quadriplegia (Table 6C.1)

Table 6C.1: Causes of quadriplegia.

UMN causes	*LMN causes*
• Cerebral palsy • Bilateral brainstem lesion (glioma) • Craniovertebral junction anomaly • High cervical cord compression • Multiple sclerosis • Motor neuron disease	• Acute anterior poliomyelitis • GB syndrome • Peripheral neuropathy • Myopathy or polymyositis • Myasthenia gravis • Periodic paralysis • Snake bite, organophosphorous poisoning, etc.

Causes of Paraplegia

Causes of Flaccid Paraplegia (LMN type)

- **UMN lesion in shock stage,** i.e. sudden onset or history of long duration as in extradural transverse myelitis and spinal injury
- **Lesion involving anterior horn cells:**
  - Acute anterior poliomyelitis

  - Progressive muscular atrophy (a variety of motor neuron disease)
- **Diseases affecting nerve root:** tabes dorsalis, radiculitis, GB syndrome
- **Diseases affecting peripheral nerves:**
  - Acute infective polyneuropathy (GB syndrome)
  - High cauda equina syndrome
  - Disease of peripheral nerves involving both the lower limbs
  - Lumbar plexus injury (psoas abscess or hematoma)
- **Diseases affecting myoneural junction:**
  - Myasthenia gravis, Lambert-Eaton syndrome
  - Periodic paralysis due to hypo- or hyperkalemia
- **Diseases affecting muscles:** Myopathy.

Onset and Progression

- Acute, subacute, or chronic.
- Reversible, stable nonreversible, fluctuating, stuttering or step-ladder, or progressive.
- **Ascending weakness**—first lower limbs→upper limbs→GB syndrome, extramedullary compressive myelopathy
- **Descending weakness**—first upper limbs→lower limbs→Miller Fisher variant of GB syndrome, intramedullary compressive myelopathy.
- **Ellsberg phenomenon**—compressive lesions near the high cervical cord produce weakness of the ipsilateral shoulder and arm followed by weakness of the ipsilateral leg, then the contralateral leg, and finally the contralateral arm, an "anticlock-wise" pattern that may begin in any of the four limbs.

Table 6C.2: Causes of spastic paraplegia [upper motor neuron (UMN) type lesion].

A. Gradual onset	*B. Sudden onset*
Cerebral causes	
• Parasagittal meningioma • Hydrocephalus	Thrombosis of unpaired anterior cerebral artery or superior sagittal sinus
Spinal causes	
Compressive or transverse lesion in the spinal cord: Cord compression **Noncompressive or longitudinal lesion** or systemic disease of the spinal cord • Motor neuron disease (MND), e.g. amyotrophic lateral sclerosis • Multiple sclerosis, Friedreich's ataxia • Subacute combined degeneration (i.e. from vitamin B_{12} deficiency) • Lathyrism, Syringomyelia, Erb's spastic paraplegia, Tropical spastic paraplegia • Radiation myelopathy	**Compressive causes** • Injury to the spinal cord (fracture-dislocation or collapse of the vertebra) • Intervertebral disc prolapse • Spinal epidural abscess or hematoma **Noncompressive causes** • Acute transverse myelitis • Thrombosis of anterior spinal artery • Hematomyelia (from arteriovenous malformation, angiomas, or endarteritis)

Muscles/Limb(s) Involved

Proximal upper limb—shoulder/arm:	Difficulties combing hair, reaching for high objects, winging of scapula
Distal upper limb—forearm/hand:	Finger/wrist drop, poor hand grip, cannot open jar, difficulty in buttoning/unbuttoning
Proximal lower limb—pelvic/thigh:	Cannot rise from chair or squatting position, waddling gait
Distal lower limbs—leg/foot:	Difficulty in gripping chappals, cannot walk on heels/toes, foot drop

Neck muscles	Dropped head/broken neck
Trunk	Inability to roll on the bed

- **Variation throughout day—fatigability:** In postsynaptic neuromuscular junction disorders like myasthenia gravis the weakness worsens on exertion.
- **Wasting/loss of muscle bulk**—wasting is a feature of LMN disease. Florid wasting is seen in motor neuron disease. Usually associated with fasciculations. In late stages of UMN disease disuse atrophy may be seen.

  Wasting of muscles also results in undue prominence of underlying bones.
- **Stiffness of limbs**—increased tone of the limbs resulting in stiffness and heaviness of limbs is a characteristic feature of UMN disease. Patients may complain that the limbs are heavy as log of wood in spasticity, while they may say that the limbs are floppy in LMN diseases.
- **Gait abnormalities:** It may aid in the diagnosis.
  - Limp or dragging foot—might suggest LMN disease/foot drop
  - Scissoring/circumduction may suggest UMN disease.
- **Involuntary movements:**
  - Type
  - Symmetrical/asymmetrical
  - Part of the body involved
  - Present at rest
  - Functional disability.

SENSORY DYSFUNCTION

- Numbness/loss of feeling
- Altered feeling:
  - Paresthesia
  - Dysesthesias (tingling, pin-needles)
  - Spontaneous pain
- Pattern of sensory loss:

Pattern of sensory loss	Site of the lesion
Hemisensory loss—same side face and body	Internal capsule/thalamus
Crossed sensory—one side face, opposite side body	Lateral medulla
Ascending sensory loss—lower limbs → upper limb	Extramedullary compressive myelopathy
Descending sensory loss—upper limbs → lower limb	Intramedullary compressive myelopathy
Dissociative sensory loss (only pain and temperature lost, posterior column sensations preserved)	Intramedullary compressive myelopathy Lateral medullary syndrome Anterior cord syndrome
Definite sensory level (below which all sensations lost)	Suggestive of spinal cord disease
Graded sensory loss—glove and stocking	Suggestive of peripheral neuropathy

Positive and Negative Symptoms

Abnormal sensory symptoms can be divided into two categories: positive and negative.

Positive Symptoms

- Altered sensation that are described as pricking, bandlike, lightning-like shooting feelings (lancinations), aching, knifelike, burning, scarring, electrical. Such symptoms are often painful.
- Positive phenomena usually result from trains of impulses generated at sites of lowered threshold or heightened excitability along a peripheral or central sensory pathway.
- Because positive phenomena represent excessive activity in sensory pathways, they may or may not be associated with a sensory deficit (loss) on examination.

Negative Symptoms

- Represent loss of sensory function and are characterized by diminished or absent feeling that often is experienced as numbness and by abnormal findings on sensory examination.
- It is estimated that at least one-half of the afferent axons innervating a particular site are lost or functionless before a sensory deficit can be demonstrated by clinical examinations.
- Subclinical degrees of sensory dysfunction may be revealed by sensory nerve conduction studies.
- Whereas sensory symptoms may be either positive or negative, sensory signs on examination are always a measure of negative phenomena.

Sense	Test device	Endings activated	Fiber size mediating
Pain	Pin prick	Cutaneous nociceptors	Small
Temperature (heat)	Warm metal object	Cutaneous thermoreceptors for hot	Small
Temperature (cold)	Cold metal object	Cutaneous thermoreceptors for cold	Small
Touch	Cotton wisp, fine brush	Cutaneous mechanoreceptors, also naked endings	Large and small
Vibration	Tuning fork, 128 Hz	Mechanoreceptors, especially Pacinian corpuscles	Large
Joint position	Passive movements of specific joints	Joint capsule tendon endings, muscle spindles	Large

CEREBELLAR EXAMINATION

Coordination and Balance

1. Difficulty in walking
2. Unsteadiness
3. Falls
4. Staggering
5. Loss of balance in dark.

AUTONOMIC DYSFUNCTION

Bladder Dysfunction (Table 6C.3)

- History of:

– Urinary retention
– Loss of awareness of bladder control
– Frequency, urgency, urge and overflow maintenance.

MENINGEAL SIGNS

- Headache
- Projectile vomiting
- Photophobia
- Neck pain

OTHERS

Dizziness, vertigo, blackouts, and fatigue

Dizziness: It covers many complaints, from a vague feeling of unsteadiness to severe, acute vertigo. It is frequently used to describe lightheadedness felt in panic and anxiety, during palpitations, and in syncope or chronic ill-health. The real nature of this symptom must be determined.

Vertigo: An illusion of movement—is more definite. It is a sensation of rotation, or tipping. The patient feels that the surroundings are spinning or moving. It is distinctly unpleasant and often accompanied by nausea or vomiting.

Blackout like dizziness, is a descriptive term implying either altered consciousness, visual disturbance or falling. Epilepsy, syncope, hypoglycemia, anemia must be considered. However, commonly no sinister cause is found. A careful history from an eyewitness is essential.

Fatigue is another common symptom of neurological disorders.

Table 6C.3: Various causes of neurogenic bladder.

Type	*Uninhibited bladder/detrusor hyperreflexia*	*Automatic bladder/detrusor sphincteric dyssynergia*	*Autonomous bladder/detrusor areflexia*	*Sensory atonic bladder*	*Motor atonic bladder*
Site of lesion	Suprapontine neurologic disorder, mostly frontal lobe	UMN disorder of the suprasacral spinal cord	LMN lesion at the sacral cord	LMN lesion—peripheral nerve	
Causes	Frontal tumors, parasagittal meningioma, ACA aneurysm, NPH	Spinal cord trauma, compressive myelopathy, myelitis	Cauda equina syndrome, conus medullaris lesion, spinal shock	Diabetes mellitus, amyloidosis, tabes dorsalis	Lumbosacral meningomyelocele, tethered cord syndrome, lumbar canal stenosis
Bladder sensation	Preserved	Interrupted	Absent	Absent	Intact
Size of bladder	Normal	Small	Large	Large	Large
Ability to initiate voiding	Present	Absent	Absent	Present	Lost
Type of incontinence	Urge/social disinhibition	Urge	Overflow	Overflow	Overflow
Residual urine	Nil	Small	Large amount	Large	Large

Anal sphincter tone	Normal	Normal	Lost	Normal	Lost
Perianal sensation	Normal	Normal	Absent	Absent	Preserved
Bulbocavernous/anal reflex	Normal	Normal	Absent	Absent	Preserved
Treatment	Anticholinergic medication	Self-intermittent catheterization	Continuous catheterization		

NECK PAIN

Deformities: Infantile torticollis	**Infections of bone:** TB of cervical spine. Pyogenic infection of cervical spine	**Tumors:** Benign and malignant tumors in relation to cervical spine and nerve roots
Arthritis of spinal joints: Rheumatoid arthritis-ankylosing spondylitis (RA-AS) Cervical spondylosis	**Mechanical derangement:** • Prolapsed cervical disc • Cervical spondylolisthesis • Whiplash injury • Cervical spine fracture • Neck muscle strain • Neck sprain	**Referred pain:** • Ear • Throat • Brachial plexus • Angina (pain extends to neck) • Aortic aneurysm • Meningismus

BACKACHE

Musculoskeletal	***Infectious***
• Nonspecific musculoskeletal backpain • Spondylolysis/spondylolisthesis • Scoliosis • Scheuermann disease • Disc degeneration and/or prolapsed	• Discitis • Vertebral osteomyelitis including tuberculosis (Pott disease) • Epidural abscess • Sacroiliac joint infection
Others	***Nonspinal infection***
• Intervertebral disc calcification • Congenital absence of pedicle • Vertebral apophyseal fracture • Aneurysmal bone cyst • Sacroiliac joint stress reaction • Idiopathic juvenile osteoporosis	• Paraspinous muscle abscess • Pyelonephritis • Pneumonia • Pelvic inflammatory disease • Endocarditis • Viral myalgias
Inflammatory	***Neoplastic***
• Ankylosing spondylitis • Psoriatic arthritis • Inflammatory bowel disease-associated arthritis • Reactive arthritis	• Osteoid osteoma • Leukemia or lymphoma • Solid malignancy, primary or metastatic • Other benign tumor: Neurofibroma, vascular malformation
Others	
• Appendicitis • Sickle cell pain crisis • Syringomyelia • Cholecystitis • Pancreatitis	• Chronic recurrent multifocal osteomyelitis • Psychosomatic illness • Nephrolithiasis • Ureteropelvic junction obstruction

RED FLAGS FOR ACUTE LOW BACK PAIN

History
• Cancer • Unexplained weight loss • Immunosuppression • Prolonged use of steroids • Intravenous drug use • Urinary tract infection • Pain worse at night or when supine • Fever • Significant trauma related to age • Bladder or bowel incontinence • Urinary retention (with overflow incontinence)
Physical examination
• Saddle anesthesia • Loss of anal sphincter tone • Major motor weakness in lower extremities • Fever • Vertebral tenderness • Limited spinal range of motion • Neurologic findings persisting beyond 1 month

NOTES

GENERAL PHYSICAL EXAMINATION IN NERVOUS SYSTEM

Pulse

- Decreased pulse rate—increased intracranial pressure (ICP)—Cushing reflex
- Resting tachycardia autonomic dysfunction
- Irregularly irregular—atrial fibrillation (AF)
- Feeble pulse, carotid bruit–atherosclerosis.

Blood pressure

- Increased BP—intracranial (IC) bleed—reactionary hypertension
- Cushing's reflex.
- Orthostatic hypotension

Jugular Venous Pressure

Increased in high output states.

Fever

- Meningitis
- Encephalitis
- CVA
- Brain abscess
- Epidural abscess
- Vasculitis
- ADEM
- Complex partial seizures
- Normal pressure hydrocephalus
- Myotonic dystrophy
- Hypothalamic dysfunction.

Pallor

- Vitamin B_{12} deficiency
- Pica, restless leg syndrome—iron deficiency
- Chronic liver disease (CLD), chronic kidney disease (CKD)—encephalopathy.

Icterus

- Hepatic encephalopathy
- Kernicterus.

Clubbing

- Syringomyelia
- Chronic hemiplegia
- Median nerve injury.

Lymphadenopathy

- Lymphoma—neuropathy, cerebellar ataxia, intracranial metastasis
- Paraneoplastic syndrome:
  - Lung carcinoma—Lambert–Eaton Myasthenic syndrome
  - Lymphoma.
- Drug induced—phenytoin.

Pedal Edema

- Chronic liver disease
- Chronic kidney disease
- Autonomic dysfunction.

Signs of Nutritional Deficiency

Discussed earlier.

NEUROCUTANEOUS SYNDROMES/PHAKOMATOSES

The neurocutaneous syndromes include a heterogeneous group of disorders characterized by abnormalities of both the integument and central nervous system (CNS).

Most disorders are familial and believed to arise from a defect in differentiation of the primitive ectoderm.

Common neurocutaneous syndromes	
• Neurofibromatosis I and II • Tuberous sclerosis • Von Hippel–Lindau disease • Sturge–Weber syndrome • Klippel–Trenaunay–Weber syndrome • Osler–Weber–Rendu syndrome • PHACE syndrome • Wyburn–Mason syndrome • Linear nevus sebaceous syndrome • Neurocutaneous melanosis • Waardenburg syndrome type 1 and 2 • Fabry's disease	• Lentiginosis, deafness, cardiopathy syndrome • Hypomelanosis of Ito • Ataxia-telangiectasia (Louis–Bar syndrome) • Xeroderma pigmentosum • Cockayne's syndrome • Rothmund-Thomson syndrome • Sjögren-Larsson syndrome • Neuroichthyosis • Werner syndrome and progeria • Incontinentia pigmenti • Neurocutaneous melanosis • Retinal—neurocutaneous cavernous hemangioma syndrome (Weskamp-Cotlier syndrome)

NEUROFIBROMATOSIS [FIG. 6D(I).1]

Two types of neurofibromatosis (type 1 and type 2).

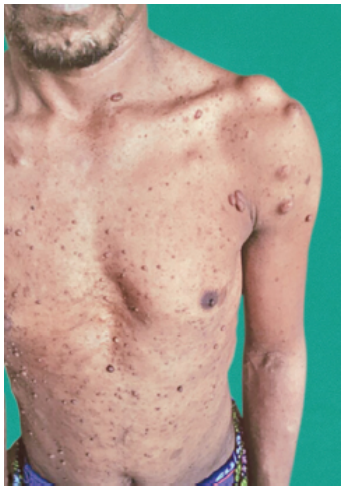


Fig. 6D(i).1: Neurofibromas.

Neurofibromatosis 1

Synonyms: von Recklinghausen disease and Watson disease.

Most prevalent neurocutaneous syndrome.

- Autosomal dominant
- The *NF1* gene on chromosome region 17q11.2 encodes a protein also known as neurofibromin. Neurofibromin acts as an inhibitor of the oncogene Ras.

Diagnostic Criteria

Two out of the following seven signs
1. Six or more café-au-lait macules over 5 mm in greatest diameter in prepubertal individuals and over 15 mm in greatest diameter in postpubertal individuals.
2. Axillary or inguinal freckling.
3. Two or more Iris Lisch nodules **[Fig. 6D(i).2]**.
4. Two or more neurofibromas or one plexiform neurofibroma.
5. A distinctive osseous lesion, such as sphenoid dysplasia (which may cause pulsating exophthalmos) Or cortical thinning of long bones with or without pseudarthrosis.
6. Optic gliomas.
7. A first-degree relative with NF1 whose diagnosis was based on aforementioned criteria.

Conditions with Café-au-lait Macules [Fig. 6D(i).3]

- Neurofibromatosis type 1 and 2
- McCune–Albright syndrome
- Ataxia telangiectasia
- Bloom's syndrome
- Familial Café-au-lait macules.

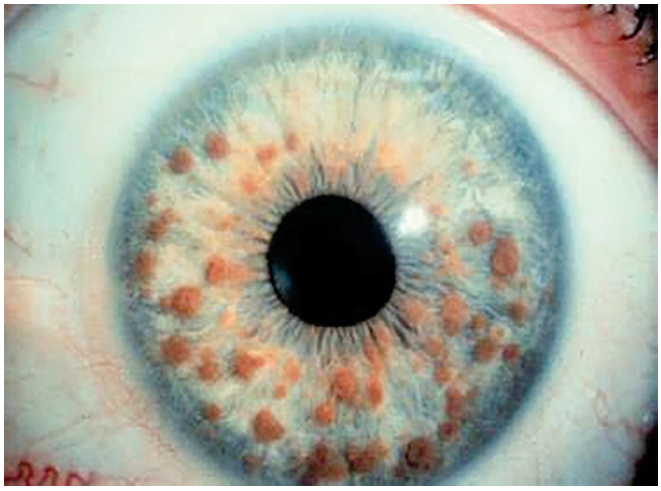


Fig. 6D(i).2: Iris nodules (Lisch nodules).

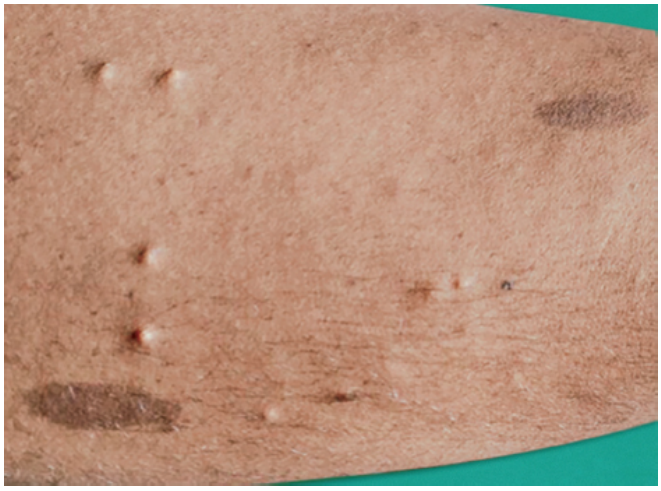


Fig. 6D(i).3: Café-au-lait macules (CALM).

Neurofibromatosis 2

The *NF2* gene (also known as merlin or schwannomin) is located on chromosome 22q1.11.

Diagnostic Criteria for Neurofibromatosis 2

One of the following three features is present
1. Bilateral vestibular schwannomas 2. A parent, sibling, or child with NF2 and either unilateral vestibular schwannoma or any two of the following: meningioma, schwannoma, glioma, neurofibroma, or posterior subcapsular lenticular opacities 3. Multiple meningiomas (two or more) and unilateral vestibular schwannoma or any two of the following: schwannoma, glioma, neurofibroma, or cataract.

TUBEROUS SCLEROSIS [TABLE 6D.(I).1)]

- Also called Bourneville disease
- Autosomal dominant

– Widespread hamartomas—brain, eyes, skin, kidneys, liver, heart, and lungs.
– Clinical triad described by Vogt:
EPI-LOI-A
» Epilepsy
» Low intelligence
» Adenoma sebaceum **[Figs. 6D(i).4A to C]**.

STURGE–WEBER SYNDROME [FIG. 6D(I).5]

- Results from anomalous development of the primordial vascular bed in the early stages of cerebral vascularization.
- As a result, brain becomes atrophic and calcified, particularly in the molecular layer of the cortex.

Clinical Manifestations

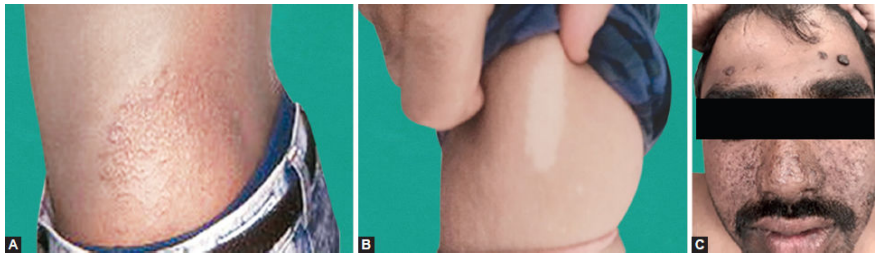
- Facial capillary malformation—Port-wine stain
- Unilateral facial nevus
- Buphthalmos and glaucoma of the ipsilateral eye
- Seizures in the 1st year of life in most patients.

Skull Radiograph

Serpentine or railroad track intracranial calcification in the occipitoparietal region.

Table 6D(i).1: Diagnostic criteria for tuberous sclerosis complex (TSC).

Major features	*Minor features*
• Facial angiofibromas or forehead plaque • Nontraumatic ungual or periungual fibroma (Koenen's tumour) • Shagreen patch (connective tissue nevus) **(Fig. 6D(i).4A)** • Hypomelanotic macules (more than three) **(Fig. 6D(i).4B)** • Multiple retinal nodular hamartomas • Cortical tuber • Subependymal nodule • Subependymal giant cell astrocytoma • Cardiac rhabdomyoma, single or multiple • Lymphangiomyomatosis • Renal angiomyolipoma	• Multiple randomly distributed pits in dental enamel • Hamartomatous rectal polyps • Bone cysts • Cerebral white matter migration lines • Gingival fibromas • Non-renal hamartoma • Retinal achronic patch • "Confetti" skin lesions • Multiple renal cysts
Definite TSC: Either two major features or one major feature with two minor features **Probable TSC:** One major feature and one minor feature **Possible TSC:** Either one major feature or two or more minor features	



Figs. 6D(i).4A to C: (A) Shagreen patch; (B) Ash leaf-shaped macule is a hypopigmented macule oval at one end and pointed at the opposite end; (C) Adenoma sebaceum.

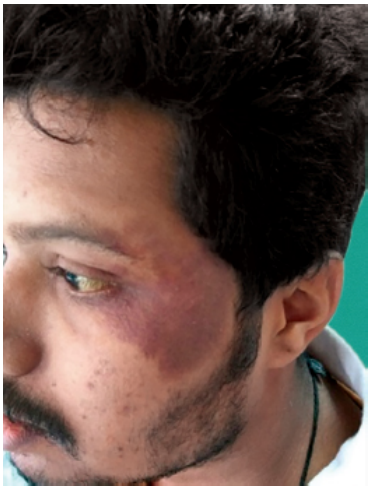


Fig. 6D(i).5: Sturge–weber syndrome.

VON HIPPEL–LINDAU DISEASE

- Autosomal dominant trait
- von Hippel-Lindau (VHL) tumor suppressor gene located on 3p25-26.

Clinical Features

- Cerebellar hemangioblastoma
- Retinal angioma
- Cystic lesions of the kidneys, pancreas, liver, and epididymis
- Pheochromocytoma.

PHACE SYNDROME

- Posterior fossa malformation
- Hemangiomas ipsilateral to the aortic arch
- Arterial anomalies
- Coarctation of the aorta, aplasia or hypoplasia of carotid arteries, aneurysmal carotid dilatation, aberrant left subclavian artery
- Eye abnormalities—glaucoma, cataracts, microphthalmia, and optic nerve hypoplasia.

ATAXIA TELANGIECTASIA

- Autosomal recessive
- Chromosome 11
- Cerebellar atrophy
- Telangiectasia appears on bulbar conjunctiva and skin
- Sinopulmonary infections
- Lymphoreticular malignancies
- Immune deficiency.

NERVE THICKENING

Detecting enlargement of accessible nerves is very helpful in assessing patients with peripheral nerve disorders, as only a few types of neuropathy lead to nerve thickening. Clinical landmarks and sites of palpable nerves are given in **Table 6D(i).2** and **Figure 6D(i).6**.

Table 6D(i).2: Clinical landmarks of palpable nerves.

Nerve	*Anatomical site*	*Palpated against*
Supraorbital [Fig. 6D(i).7]	Forehead	Orbital ridge of frontal bone
Infraorbital	Cheek	Zygomatic bone
Greater auricular [Figs. 6D(i).8 and 6D(i).9]	Neck, anterior branch across the sternocleidomastoid, posterior branch over the sternocleidomastoid	Sternocleidomastoid
Ulnar [Fig. 6D(i).10]	Elbow joint	Behind medial epicondyle in olecranon groove
Superficial radial	Above wrist joint	Against lateral border of radius
Median	Near wrist joint, proximal to the flexor retinaculum	Against carpal bones
Common peroneal [Fig. 6.D(i).11]	Knee joint	Against fibular head
Posterior tibial	Ankle joint, below and behind medial malleolus	Against calcaneus
Sural	Lateral side of lower third of leg	Fibula

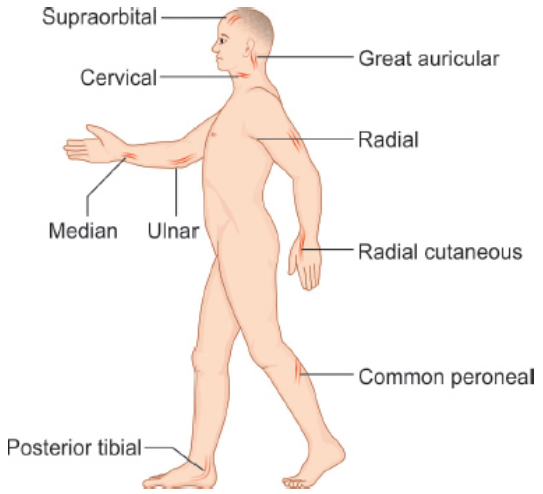


Fig. 6D(i).6: Sites of palpable nerves.

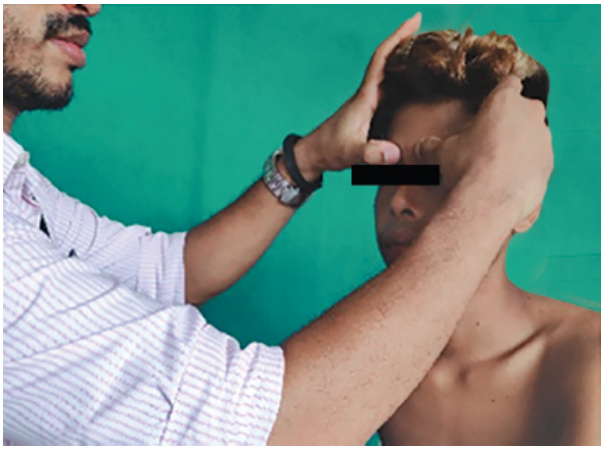


Fig. 6D(i).7: Supraorbital nerve.

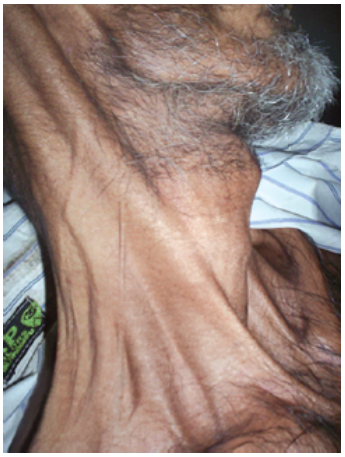


Fig. 6D(i).8: Greater auricular nerve.

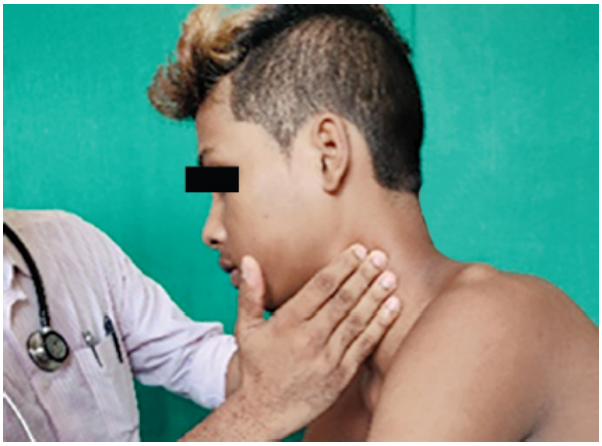


Fig. 6D(i).9: Greater auricular nerve of neck.

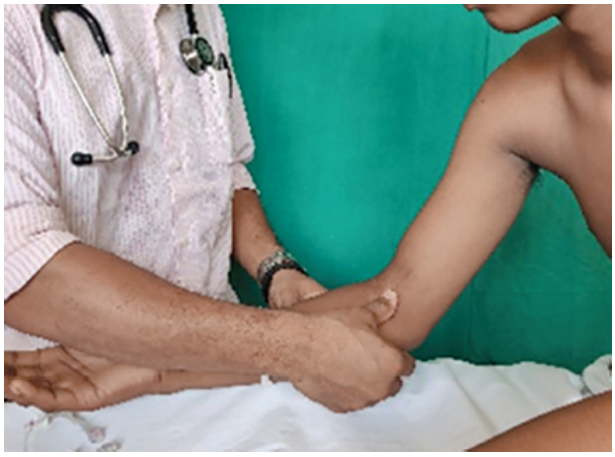


Fig. 6D(i).10: Ulnar nerve.

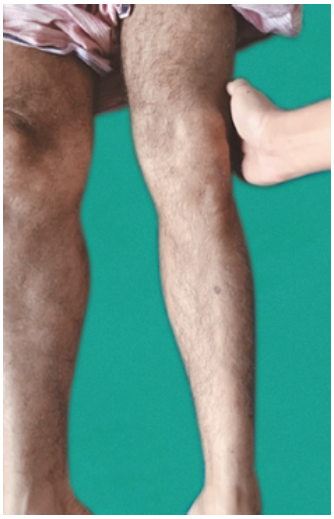


Fig. 6D(i).11: Common peroneal nerve.

Causes of Nerve Thickening

Infective

Leprosy

Hereditary

- Hereditary motor and sensory neuropathy types 1 and 3 (Charcot–Marie–Tooth neuropathy, Dejerine–Sottas syndrome)
- Refsum's disease.

Acquired immune mediated

- Chronic inflammatory demyelinating polyradiculoneuropathy (CIDP)
- Chronic inflammatory sensory polyradiculopathy (CISP)
- Multifocal acquired demyelinating sensory and motor polyneuropathy (MADSAM)
- Relapsing Guillian-Barre syndrome (GBS).

Tumors of nerves or nerve sheath

- Localized hypertrophic neuropathy
- Schwannoma
- Neurofibromatosis 1 and 2.

Nerve infiltrations

- Neurolymphomatosis
- Acromegaly
- Amyloidosis
- Sarcoidosis.

NOTES

NERVOUS SYSTEM EXAMINATION

Handedness

Handedness	
Right handed (90–95%)	***Left handed (5–10%)***
99% have—left dominant hemisphere 1% have—right dominant hemisphere	60–70% have—left dominant hemisphere 15–20% have—right dominant hemisphere 15–20% have—mixed dominance

Examination

Any of the following methods can be adopted:

- Ask the patient to kick a football, normally the dominant side leg is used.
- Ask the patient to peep through a keyhole, normally the dominant side eye is used.
- Ask the patient to fold the arms in front one over the other, the dominant hand is the one which lies anteriorly.
- Ask the patient to "stand at ease" position, the dominant hand is the one which lies posteriorly.

Clinical Implications

1. Handedness is important for rehabilitation of the patient (right-handed individuals—dominant left hemisphere needs to be aggressively rehabilitated so as to have minimal residual deficit).
2. Degenerative diseases like Huntington's disease have been postulated to be more common in individuals with right dominant cortex.
3. Failure to develop clear hemispheric dominance has been implicated in dyslexia, stuttering, mirror writing, learning disability, and general clumsiness.

Education

- Formal education up to standard _____.
- It is important for testing components of higher mental functions like calculation, reading, and writing.

CONSCIOUSNESS

The ascending reticular activating system (RAS) arising from the reticular formation of the brainstem, primarily the paramedian tegmentum of the upper pons and midbrain, and projects to the paramedian, parafascicular, centromedian, and intralaminar nuclei of the thalamus. This is the primary control of consciousness.

The hypothalamus is also important for consciousness; arousal can be produced by stimulation of the posterior hypothalamic region.

Coma	• It is a state of complete loss of consciousness from which the patient cannot be aroused by ordinary stimuli. • There is complete unresponsiveness to self and the environment. • The patient in coma has no awareness of themselves, makes no voluntary movements, and has no sleep-wake cycles.

Stupor	• It is a state of partial or relative loss of response to the environment in which the patient's consciousness may be impaired to varying degrees. • The patient can be aroused only with vigorous or unpleasant stimuli (e.g. sharp pressure or pinch, or rolling a pencil across the nail bed). • No significant voluntary verbal or motor responses. • Mass movement responses may be observed in response to painful stimuli or loud noises. **For example:** • Bilateral cerebral hemisphere disease • Upper brainstem diseases
Lethargy/drowsiness	Patient can usually be aroused or awakened and may then appear to be in complete possession of their senses, but promptly falls asleep when left alone. It resembles normal sleepiness. **For example:** High brainstem disturbances
Obtundation	Refers to moderate reduction in the patient's level of awareness such that stimuli of mild-to-moderate intensity fail to arouse; when arousal does occur, the patient is slow to respond.
Minimally conscious (vegetative) state	• Return of irregular sleep-wake cycles and normalization of the so-called vegetative functions—respiration, digestion, and blood pressure control. • The patient may be aroused, but remains unaware of his or her environment. • There is no purposeful attention or cognitive responsiveness.
Persistent vegetative state	Individuals who remain in a vegetative state 1 year or longer after traumatic brain injury (TBI) and 3 months or more after anoxic brain injury.
Confusional state	Patients may appear alert, but are confused and disoriented. It is usually tested in three dimensions: 1. Time 2. Place 3. Person.
Delirium	It is an acute organic mental disorder characterized by confusion, restlessness, incoherence, inattention, anxiety, or hallucinations which may be reversible with treatment. **For example:** • Toxicity (alcohol) • Infections
Catatonia	• Symptom of psychotic state in which the patient is otherwise normal. • He does not follow movements, does not appear to pay attention to surroundings and will often have aplastic rigidity of limbs which may remain in any position in which they are placed (however bizarre the position may be).

It is preferable to describe the patient's state of responsiveness or use an objective and well-defined scheme, such as the Glasgow Coma Scale (GCS).

Glascow Coma Scale (GCS)					
Eye opening		***Best verbal response***		***Best motor response***	
				Obeys commands	**6**
		Oriented and converses	**5**	Localizes pain	**5**
Open spontaneously	**4**	Converses, but disoriented, confused	**4**	Exhibits flexion withdrawal	**4**
Open only to verbal stimuli	**3**	Uses inappropriate words	**3**	Decorticate rigidity	**3**
Open only to pain	**2**	Makes incomprehensible sounds	**2**	Decerebrate rigidity	**2**
Never open	**1**	No verbal response	**1**	No motor response	**1**
Maximum score = **15** Minimum score = **3** Coma is equal to GCS of **8 or less**.					

Mnemonic (GCS → EVM = 4, 5, and 6)

Note: In intubated patients, verbal response is denoted as V_T.

Glasgow coma scale–pupils score

- The Glasgow coma scale-pupils score (GCS-P) was described in 2018 as a strategy to combine the two key indicators of the severity of traumatic brain injury into a single simple index
- Calculation of the GCS-P is by subtracting the pupil reactivity score (PRS) from the Glasgow coma scale (GCS) total score:

GCS-P = GCS – PRS

- The pupil reactivity score is calculated as follows:

Pupils unreactive to light	*Pupil reactivity score*
Both pupils	2
One pupil	1
Neither pupil	0

- The GCS-P score can range from 1 and 15 and extends the range over which early severity can be shown to relate to outcomes of either mortality or independent recovery.

ORIENTATION

Time	Ask for year, season, month, date, and time
Place	Ask for country, state, city, hospital name, and floor/ward
Person	• What is your name? • How old are you? • Where were you born? • What is the name of your wife/husband?

Findings are documented in the medical record as follows: Patient is alert and oriented × 3 (time, person, and place) or × 2 (person, place) depending on the domains correctly identified.

An additional domain that can be examined is **circumstance.**

(What happened to you? What kind of a place is this? Why do people come here?)

APPEARANCE/BEHAVIOR

- Mood and affect
- Thought and perception

These have been discussed under Chapter 9—Approach to Psychiatric Illness.

MEMORY

Classification of Memory

Explicit memory (declarative memory)	Implicit memory
Involves conscious recall and requires integrity of various cortical regions	Does not require conscious recall. Involves basal ganglia and cerebellum
Can be tested bedside	Cannot be tested bedside
It includes: • Immediate (prefrontal cortex) • Recent (medial temporal structures) • Remote (widespread neocortical areas).	It includes: • Procedural memory (basal ganglia)—like riding a car • Classical conditioning (cerebellum) • Probabilistic classification learning (basal ganglia).

Examination of Explicit Memory

Types of memory	Description and testing	Areas in brain
Immediate (working memory)	• Digit span is a test of immediate memory, a very short-term function in which the material is not actually committed to memory • Ask patient to repeat series of random digits forward and backward • Normal digit span is 7 ± 2	Dorsolateral frontal lobe, prefrontal cortex, and perisylvian cortex
Recent (short-term)	• Recent, or short-term memory is tested by giving the patient items (pen, phone, and bottle) to recall • After ensuring the patient has registered the items, proceed with other testing. After approximately 5 minutes, ask the patient to recall the items	• Mammillothalamic tract • Hippocampus • Parahippocamal cortex (spatial memory) • Amygdala (emotional aspects) • Perirhinal cortex (for visual) • Medial temporal structures and connections
Remote (long-term)	• A patient's fund of information reflects their remote memory. The fund of information includes schooling details, famous personalities, major events in history, etc.	• Widespread • Neocortical areas
Episodic memory refers to the system involved in remembering particular episodes or experiences, such as the movie you saw last weekend or the meeting you attended yesterday. **Semantic memory** refers to the type of long-term memory concerned with factual details outside of personal details		
Budson and Price concept of memory systems: The frontal lobe can be considered as filing clerk, deciding which information has to be filed or retrieved. The medial temporal lobes are the actual filing cabinets for recent memories and the neocortical regions are filing cabinets for remote memories		
Wernicke's encephalopathy—g lobal confusion, **o**phthalmoplegia and **a**taxia (mneumonic—goa). **Korsakoff's psychosis**: Recent memory loss + confabulation (anteromedial thalamus)		

Amnesia

Anterograde amnesia	Impaired registration and recall of new information
Retrograde amnesia	Impaired recall of information registered within a certain interval before the disease onset

ATTENTION

- Attention is the directing of consciousness to a person, thing, perception, or thought.
- It depends on the capacity of the brain to process information from the environment or from long-term memory.
- An individual with intact selective attention is able to screen and process relevant sensory information about both the task and the environment while screening out irrelevant information.

- Selective attention can be examined by asking the patient to attend to a particular task.
- For example, the doctor asks the patient to repeat a short list of numbers forward or backward (digit span test).
- Normally, individuals can recall seven forward and five backward numbers.
- **Sustained attention (or vigilance)** is examined by determining how long the patient is able to maintain attention on a particular task (time on task).
- **Alternating attention (attention flexibility)** is examined by requesting the patient to alternate back and forth between two different tasks (e.g. add the first two pairs of numbers, then subtract the next two pairs of numbers).
- Requesting the patient to perform two tasks simultaneously determines divided attention.
- For example, the patient talks while walking (Walkie–Talkie test).

INTELLIGENCE/CALCULATION

Serial sevens, or spelling of any word backward.

COGNITION ASSESSMENT TOOLS

- Mini Mental Status Examination (MMSE)—Folstein's

O	**Orientation**	Place Time	10
R	**Registration**	Name 3 objects	3
A	**Attention and calculation**	Serial 7/word backward	5
R	**Registration recall**	Recall previously named 3 objects	3
L	**Language**	3 stage command Name two objects Read and follow Draw a pentagon Repetition Write a sentence	9

- MMSE total score:
  - 21–24: Mild cognitive dysfunction
  - 10–20: Moderate
  - Less than 10: Severe.
- Montreal cognitive assessment (MoCA)
- Cognitive state test (COST)
- Addenbrooke's cognitive examination (ACE)
- Cambridge cognitive examination (CAMCOG)
- Brief cognitive assessment tool (BCAT), and
- Short test of mental status (STMS).

SPEECH

Definitions

Phonation	It is defined as the production of vocal sounds without word formation; it is entirely a function of the larynx

Vocalization	It is the sound made by the vibration of the vocal folds, modified by working of the vocal tract
Speech	It consists of words which are articulate vocal sounds that symbolize and communicate ideas
Articulation	It is the enunciation of words and phrases; it is a function of organs and muscles innervated by the brainstem
Language (Fig. 6D(ii).1)	• It is a mechanism for expressing thoughts and ideas as follows: • By speech (auditory symbols) • By writing (graphic symbols), or • By gestures and pantomime (motor symbols) • Language may be regarded as any means of expressing or communicating feeling or thought using a system of symbols. • It is a function of the cerebral cortex
Aphasia	Aphasia is an acquired disorder with loss or defective language content of speech resulting from damage to the speech centers within the dominant (usually left in 97%) hemisphere
Paraphasia	Substitution in the components of speech, e.g. foon for spoon
Neologism	Use of words which are nonexistent. Classically seen with Wernicke's aphasia
Jargon	Completely meaningless speech containing neologisms and paraphasias. Described in Wernicke's aphasia
Echolalia	Continuous repetition of heard words or sentences. Seen with transcortical sensory and transcortical mixed aphasias.
Alexia	It is the impairment of visual word recognition, in the context of intact auditory word recognition and writing ability
Agraphia	It is the inability to write, as a language disorder resulting from brain damage
Anomia	In this, word approximates the correct answer but it phonetically inaccurate (plentil for pencil)—phonemic paraphasia. When the patient cannot say the appropriate name when an object is shown but can point the object when the name is provided, it is known as one way or retrieval-based naming deficit
Mutism	Unable to speak or make sound
Aphonia	Unable to produce sound
Aphemia	Loss of speech

Slurred speech can be because of aphasia or dysarthria.

Aphasia	***Dysarthria***
Aphasia is a disorder of language	Dysarthria is a disorder of the motor production or articulation of speech
Usually due to cerebral dysfunction/lesions	Dysarthria is defective articulation of sounds or words of neurologic origin (usually brainstem)
Aphasia usually affects other language functions, such as reading and writing	In dysarthria, there are often other accompanying bulbar abnormalities, such as dysphagia

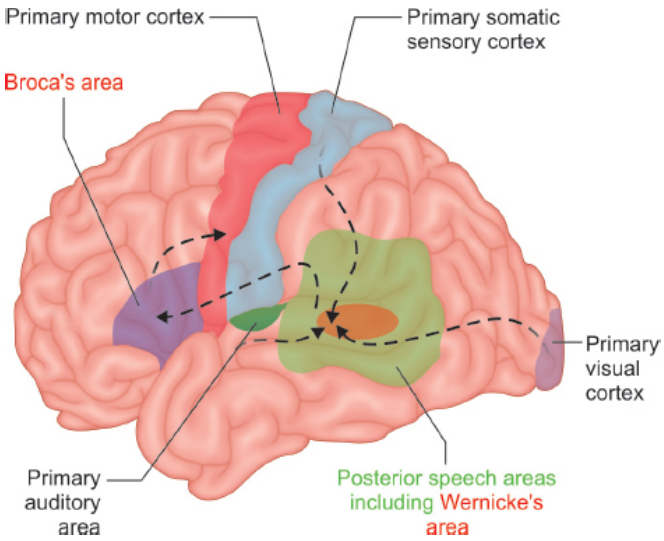


Fig. 6D(ii).1: Language and the brain.

Wernicke's area (area 22)	***Arcuate fasciculus***	***Broca's area (area 44)***
Decoding of sounds into language information (comprehension)	Communication between the Broca's and Wernicke's area. Needed for speech repetition	Responsible for spontaneous speech output (i.e.) fluency. Approximate number words produced per minute is 100/min for males and 150/min for females

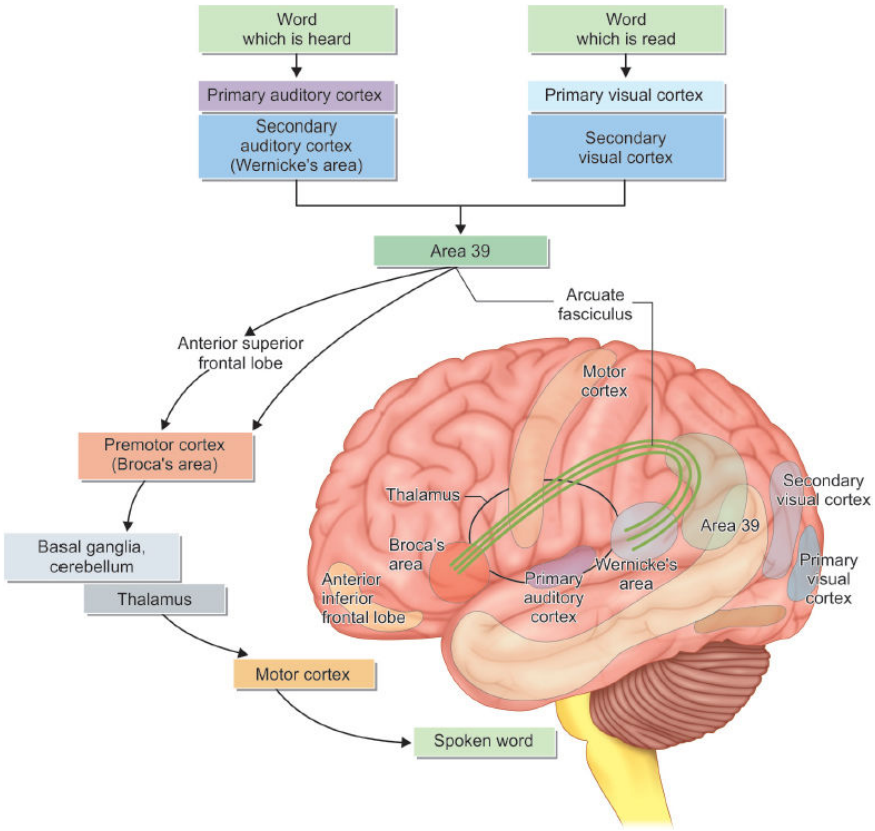


Fig. 6D(ii).2: Genesis of speech.

APHASIAS

- Aphasia is an acquired disorder with loss or defective language content of speech resulting from damage to the speech centers within the dominant (usually left in 97%) hemisphere.
- A language disturbance occurring after a right hemisphere lesion in a right hander is known as crossed aphasia.
- It includes defect in or loss of the power of expression by speech, writing, or gestures or a defect in or loss of the ability to comprehend spoken or written language or to interpret gestures.
- Aphasia may be categorized according to whether the speech output is fluent or nonfluent.
  - **Fluent aphasias** (receptive aphasias) are impairments mostly due to the input or reception of language with difficulties either in auditory verbal comprehension or in the repetition of words, phrases, or sentences spoken by others. For example, Wernicke's aphasia.
  - **Nonfluent aphasias** (expressive aphasias) are difficulties in articulating with relatively good auditory, verbal comprehension. For example, Broca's aphasia **[Fig. 6D(ii).3]**.
- **Normal fluency** 100–150 words/min, sentence length >7 words.
- Reduced fluency in Broca's aphasia, transcortical motor, global aphasia, and primary progressive aphasia.

Domains of Language

1. Spontaneous speech/fluency
2. Comprehension
3. Repetition

4. Reading
5. Writing
6. Naming.

C—Comprehension (requires intact Wernicke's and transcortical sensory area)
R—Repetition (requires intact Wernicke's, arcuate fibers, and Broca's area)
F—Fluency (requires intact Broca's and transcortical motor area) **[Flowchart 6D(ii).1]**.

	Aphasia	Site of lesion	C	R	F
1	**Wernicke's—sensory/receptive/posterior**	Infarction of inferior division of middle cerebral artery	–	–	+
2	**Broca's—motor/expressive/anterior**	Infarction of superior frontal branch of middle cerebral artery	+	–	–
3	**Conduction/arcuate**	Arcuate fasciculus	+	–	+
4	**Transcortical sensory**	Posterior watershed zone	–	+	+
5	**Transcortical motor**	Anterior watershed zone	+	+	–
6	**Isolation aphasia (mixed transcortical aphasia)**	Both anterior and posterior watershed areas	-	+	–
7	**Global aphasia**	Dominant frontal, parietal and superior temporal lobe	-	–	–

Note:
C—Comprehension
R—Repetition
F—Fluency

Once the comprehension, repetition, and fluency are intact, we look for reading, writing, and naming disorders associated with reading, writing, and naming.

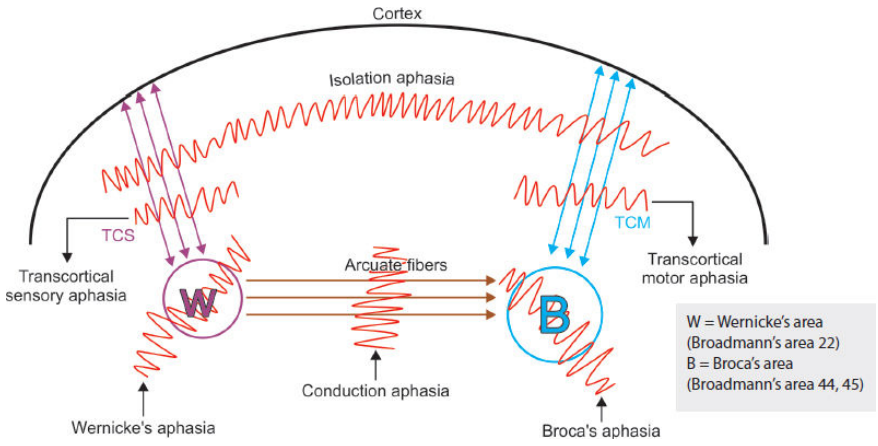
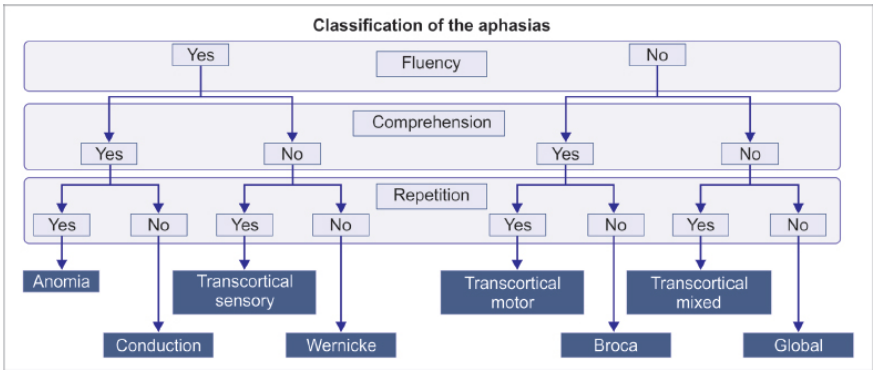


Fig. 6D(ii).3: Schematic representation of aphasias and associated lesions.

Flowchart 6D(ii).1: Approach for aphasias.



			R	W	N
8	**Alexia without agraphia**	Occipitotemporal region	–	+	+
9	**Alexia with agraphia**	Left angular gyrus	–	–	+
10	**Nominal/anomic/amnesic**	Temporoparietal	+	+	–

- Lesions in the anterior limb of internal capsule/basal ganglia can produce Broca's like aphasia.
- Lesions in the thalamus can produce Wernicke's like aphasia.
- Most common type of aphasia seen in stroke: Broca's aphasia.
- Overall most common type of aphasia is anomic aphasia.

DYSARTHRIAS

Production of sounds requires:

1. Normal respiration
2. Muscles of articulation (labial, lingual, and palatal muscles)
3. Phonation (by larynx)
4. Resonance (by nasopharynx).

Articulated Sounds

Articulated labials (b, p, m, and w) are formed principally by the lips.

Modified labials (o and u, and to a lesser extent i, e, and a) are altered by lip contraction.

Labiodentals (f and v) are formed by placing the teeth against the lower lip.

Linguals are sounds formed with tongue action.

T, d, l, r, and n are tongue point, or alveolar sounds formed by touching the tip of the tongue to the upper alveolar ridge.

S, z, sh, zh, ch, and j are dentals, or tongue blade sounds.

To hear distorted linguals, place the tip of your tongue against the back of your bottom teeth, hold it there and say "top dog," "go jump", and "train".

To hear distorted labials, hold your upper lip between the thumb and forefinger of one hand and your bottom lip similarly with the other and say "my baby".

Gutturals (velars, or tongue back sounds, such as k, g, and ng) are articulated between the back of the tongue and the soft palate.

Palatals (German ch and g, and the French gn) are formed when the dorsum of the tongue approximates the hard palate.

Types of dysarthrias		
Types	***Description***	***Cause***
Flaccid (lingual, buccal, and guttural)	LMN weakness of facial, lingual, or pharyngeal muscles. • **Facial paralysis** causes difficulty with labials, such as b, p, m, and w. • **Tongue paralysis** affects a large number of sounds, particularly l, d, n, s, t, and x. • **Palatal paralysis** produces a nasal twang in speech.	Cerebrovascular accidents (especially brainstem lesions)
Spastic (hot potato voice)	Strained, slurred hot potato-like voice	UMN weakness (bilateral), e.g. pseudobulbar palsy
Ataxic speech	***Scanning speech:*** Undue separation of syllables (monosyllable speech)	Cerebellar diseases
	Staccato speech: Explosive type of speech with emphasis on syllables	
Hypokinetic	Slow monotonous, low voice with inappropriate silence	Extrapyramidal (parkinsonism)
Hyperkinetic dysarthria	Distorted speech with continuous change in articulation	Chorea, athetosis, and dyskinesias
Myasthenic dysarthria	Voice is normal in the beginning but becomes weak as sentences progress	Myasthenia gravis

APRAXIA

Definition

Apraxia is impaired ability (inability) to carry out (perform) skilled, complex, and organized motor activities in the presence of normal basic motor, sensory, and cerebellar functions.

Examples of complex motor activities: Dressing, using cutlery, and geographical orientation.

Types	
Ideomotor apraxia	Most common. It is the inability to perform a specific motor command/act (e.g. cough, lighting a cigarette with a matchstick) in the absence of motor weakness, incoordination, and sensory loss or aphasia. Site of lesion is bilateral parietal lobe. Buccofacial apraxia involves apraxic deficits in movements of the face and mouth. Limb apraxia encompasses apraxic deficits in movements of the arms and legs
Dressing apraxia	Site of lesion is nondominant parietal lobe. It is inability to wear his/her dress
Constructional apraxia	It is inability to copy simple diagrams or build simple blocks. Site of lesion is nondominant parietal lobe
Ideational apraxia	It is a deficit in the execution of a goal-directed sequence of movements even with real object (e.g. asked to pick up a pen and write, the sequence of uncapping the pen, and placing the cap at the opposite end). This is commonly associated with confusion and dementia rather than focal lesions associated with aphasic conditions
Gait apraxia (Bruns ataxia)	Seen in normal pressure hydrocephalus (NPH)
Gaze apraxia	Part of Balint syndrome
Other apraxias	Speech apraxia, conceptual apraxia, and conduction apraxia

AGNOSIA

Definition

Agnosia is failure to recognize objects (e.g. places, clothing, persons, sounds, shapes, or smells), despite the presence of intact sensory system.

Site of lesion: Contralateral parietal lobe.

Types of agnosias	
Visual agnosia	Failure to recognize what is seen with eyes despite the presence of intact visual pathways. The individual can describe the shape, color, and size without naming it. Site of lesion is in the posterior occipital or temporal lobes
Prosopagnosia	A type of visual agnosia in which patient cannot identify familiar faces, sometimes the reflection of his or her own face in the mirror even including their own. Site of lesion is parieto-occipital lobe
Simultanagnosia	It is inability to perceive more than one object at a time
Autotopagnosia	It is a form of agnosia, characterized by an inability to localize and orient different parts of the body
Pseudopolymelia	The feeling of false—the feeling of false extremities. More frequent, the patients feel the extremities. More frequent, the patients feel the third hand
Anosognosia	It is an inability or refusal to recognize a defect or disorder that is clinically evident
Auditory agnosia	It consists of the loss of ability to know objects on sounds characteristic for them (clock—on ticking)

DELUSIONS

Definition

Delusion is a belief held with strong conviction despite superior evidence to the contrary (strongly held false beliefs).

It is a disorder of content of thought.

Types of delusion (based on their content)	
Persecutory delusions	Conviction that others are out to get me
Grandiose delusions	Belief that one has special powers or status
Nihilistic delusions	Conviction that “my head is missing/rotting”, “i have no body”, and “I am dead”
Erotomanic delusions	Believing a movie star loves them
Somatic delusions	Believing head is filled with air/worms
Delusions of reference	Believing story in a book is referring to them
Delusions of control/passivity	Believing one’s thoughts and movements are controlled by aliens
Other delusions are	Delusions of misinterpretation, hypochondrial delusions, fantastic/bizarre delusions, delusions of passivity, delusions of jealousy

HALLUCINATIONS

Definition

Hallucinations are perceptions without external stimuli ***(wakeful sensory experiences of content that is not actually present)***. They can occur in any sensory modality, most common being ***visual or auditory***.

For example, hearing voices when no one else is present, or seeing “visions”. Other types include tactile (cocaine bug), olfactory, gustatory, command kinesthetic/psychomotor, and lilliputian and complex hallucinations.

Pseudohallucinations

These are hallucinations that are perceived as originating in the external world, not in the patient’s own mind.

Hypnagogic and Hypnopompic Hallucinations

In narcolepsy 2, specific hallucinations are seen. **Hypnagogic:** They occur when falling asleep. **Hypnopompic:** They occur on waking up from sleep.

(mnemonic—hypno**GO**gic hallucinations are perceived while **GO**ing to sleep).

Hallucinations	***Illusions***
Perceptions without external stimuli	Misperceptions of real external stimuli
For example, hallucinating that someone is talking to them when there is no actual stimulus	For example, mistaking a rope for snake

Functions and effects of damage to various lobes of cerebral hemispheres are listed in **Table 6D(ii).1** and **Figure 6D(ii).4**.

Table 6D(ii).1: Functions and effects of damage to various lobes of cerebral hemispheres.

Lobe	***Function***	***Cognitive/behavioral effects of damage***
Frontal **P**lease **SMILE** (MNEMONIC)	**P**ersonality	
	Social behavior	Antisocial behavior
	Micturition	Incontinence
	Intelligence	
	Language	Expressive dysphasia
	Emotional response	Disinhibition
Parietal: Dominant side	Language	Dysphasia, dyslexia
	Calculation	Acalculia
	Others	Apraxia, agnosia
Parietal: Nondominant side	Spatial orientation	Spatial disorientation, neglect of contralateral side
	Constructional skills	Constructional apraxia, dressing apraxia
Temporal: Dominant side	Auditory perception	Receptive aphasia
	Language	Dyslexia
	Verbal memory	Impaired verbal memory
	Smell	

	Balance	
Temporal: Nondominant side	Auditory perception	Impaired nonverbal memory
	Melody/pitch perception	Impaired musical skills (tonal perception)
	Nonverbal memory	
	Smell	
	Balance	
Occipital	Visual processing	Visual inattention, visual loss, visual agnosia (Anton–Babinski syndrome)

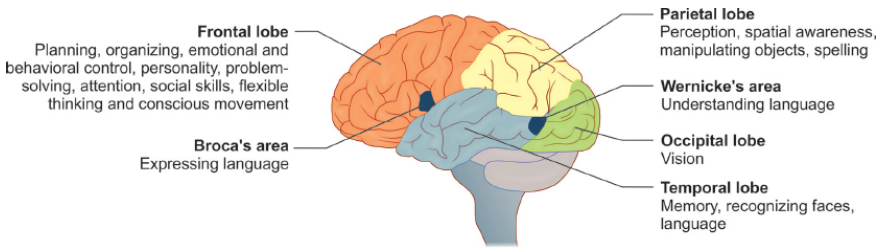


Fig. 6D(ii).4: Various lobes of cerebral hemispheres.

LESIONS OF NONDOMINANT (RIGHT) HEMISPHERE

Neglect

Definition → directed inattention, or a relative lack of attention, paid to one hemisphere; patients are less aware (or completely unaware) of objections or actions in one side of the world (usually the left).

Diagnosis

- *Severe forms* → patients completely ignore left side, denying that, such as side even exists; they may leave their left side ungroomed, unshaven, and undressed; may leave food on left side of plate uneaten; may deny they have a left hand, and when confronted with it, may claim that it is actually the examiner's.
- *Milder forms* → may perform actions with their left side only with encouragement or after repeated prodding.
- *Most sensitive sign* → extinction to double simultaneous stimulation; sensory stimuli applied singly to either side are properly felt, but when both sides are stimulated simultaneously, only the non-neglected side is felt; extinction may exist with tactile, visual, or auditory stimulation.
- *Etiology* → lesions in right hemisphere (frontal or parietal lobe), most commonly an acute finding after stroke.
  - *Frontal lobe* lesion → more of a motor neglect in which patient has tendency to not use left side for motor actions
  - *Parietal lobe* lesion → more of a sensory neglect in which stimuli from the left side tend to be ignored.

Others

- **Prosody** → while semantic elements of language (pure meaning) reside in dominant hemisphere, some other elements of successful oral communication (e.g. proper voice inflection) reside in nondominant hemisphere
- **Anosognosia** → tendency to be unaware of one's deficits in some patient's w/right hemispheric lesions
  - For example, patient with complete left hemiplegia may insist on immediate discharge from hospital because he feels nothing is wrong
  - For example, patient with dense left hemianopia may wonder why she keeps bumping into others since she notices nothing wrong with her vision.

NOTES

CRANIAL NERVE I—OLFACTORY NERVE

Prerequisites for Examination

- Rule out nose blocks
- Close eyes while examining
- Test each nostril separately.

Substances Which Can be Used for Testing

- Peppermint
- Soap
- Coffee beans
- Lemon peel
- Vanilla.

Note: ***Avoid*** *irritants like ammonia as they directly stimulate the trigeminal nerve endings.*

Method of Examination

- Examine each nostril separately while occluding the other **[Fig. 6D(iii).1]**.
- With the patient's eyes closed and one nostril occluded, bring the test substance near the open one.
- Instruct the patient to sniff repetitively and to tell you when an odor is detected, identifying the odor, if recognized.
- Bring the test odor up to within 30 cm or less of the nose.
- Repeat for the other nostril and compare the two sides.

Note: The side that might be abnormal should be examined first.

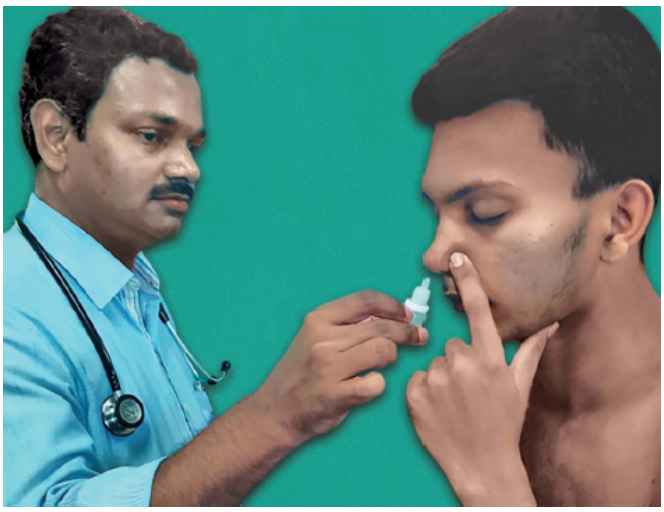


Fig. 6D(iii).1: Method of examination of olfactory nerve.

Interpretation

- Patient able to detect smell, recognize, and name
- Patient able to detect smell, recognize but not name
- Patient able to detect, but not recognize or name.

Olfactory pathway	
The **Ist order neurons** of the olfactory system are bipolar sensory cells that lie in the olfactory epithelium, which occupies a small area on the superior nasal concha, upper nasal septum, and roof of the nose	
↓	
Peripheral and central processes	
↓	
Olfactory axons	
↓	
Pierce the cribriform plate	
↓	
Olfactory bulb	
↓	
Within the olfactory bulbs, axons of incoming fibers synapse on dendrites of mitral and tufted cells in the olfactory glomeruli. The mitral and tufted cells are the output cells of the olfactory bulb	
↓	
2nd order neurons (predominantly mitral cells)	
↓	
Pass through the anterior perforating substance	
↓	↓
Medial striae	***Lateral striae***
Carry axons across the medial plane of anterior commissure where they meet the olfactory bulb of opposite side	Primary olfactory cortex (pyriform cortex, amygdala, olfactory tubules, and secondary olfactory cortex)

Note:

- The olfactory nerves are the ***unmyelinated filaments*** that pass through the cribriform plate.
- The bulbs and tracts are part of the rhinencephalon.

Disturbances in olfaction
Anosmia
Local causes: • Acute rhinitis (most common cause) • Heavy smoking • Atrophy of bulb **Systemic causes:** • Parkinsonism • Meningitis • Head trauma • Intracranial tumors • Endocrine diseases:

  - Diabetes mellitus
  - Hypothyroidism
  - Kallmann syndrome
  - Turner syndrome
- Vitamin B_{12} deficiency
- Chronic kidney diseases.
- Refsum's disease

Syndromes associated:

- **Foster–Kennedy syndrome** (anosmia, optic atrophy of one eye, and contralateral eye papilledema due to tumor in brain)
- **Pseudo-Foster–Kennedy syndrome** (above features in absence of tumor)

Impaired smell
K: Korsakoff **B**: Basilar meningitis **C**: Chorea huntington's **A**: Anterior cerebral artery diseases **S**: Spinocerebellar ataxia **H:** Hydrocephalus

Other miscellaneous points
• Anosmia is commonly associated with hypogeusia/ageusia • Olfactory hallucinations: Usually of unpleasant odors like burned rubber, can occur in temporal lobe epilepsy, migraine and schizophrenia • Hyperosmia: May be seen with Addison's disease, cystic fibrosis or pituitary tumors • Merciful anosmia—atrophic rhinitis.

Note:

- Olfactory is the only nerve which does not process through thalamus.
- Olfactory and optic are the two nerves which do not pass through brainstem.
- Loss of smell is usually associated with loss of taste sensation (Aguesia/hypogeusia).

CRANIAL NERVE II—OPTIC NERVE

1. Visual acuity
2. Visual field
3. Color vision
4. Fundus examination.

Visual Acuity

Assessment of visual acuity is usually done by asking the patient to read the specific charts as described below. The least possible distance with best vision is considered as the viewing distance.

Visual acuity	
For far vision	***For near vision***
Snellen chart **[Fig. 6D(iii).2]**	Jaeger chart **[Fig. 6D(iii).3]**
Examined at 6 m	Examined at 30 cm
Described as x/y → × (numerator—suggests the viewing distance of patient) and y (denominator—viewing distance of normal person)	• Describes as J_1, J_2, etc. • Normal range of near vision is J_1 to J_4

Note: In absence of Snellen's chart finger counting can be done.

Defects in visual acuity may be due to:

- Refractive errors
- Cataract
- Vitreous opacity, etc.

Fig. 6D(iii).2: Snellen's chart for far vision.

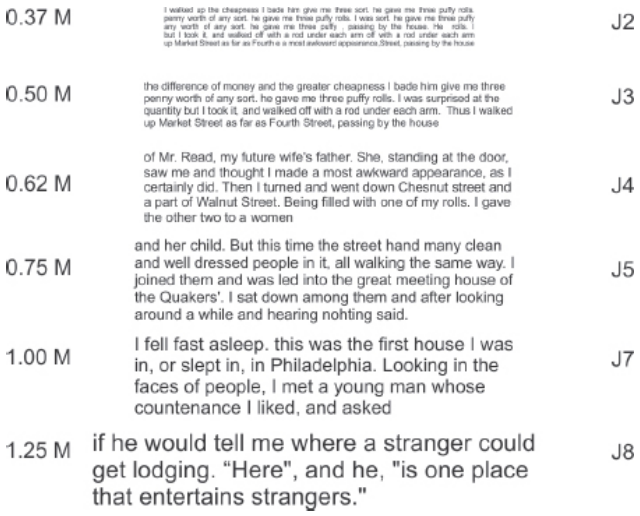


Fig. 6D(iii).3: Jaeger's chart for near vision.

Visual Field Testing

Confrontation Method

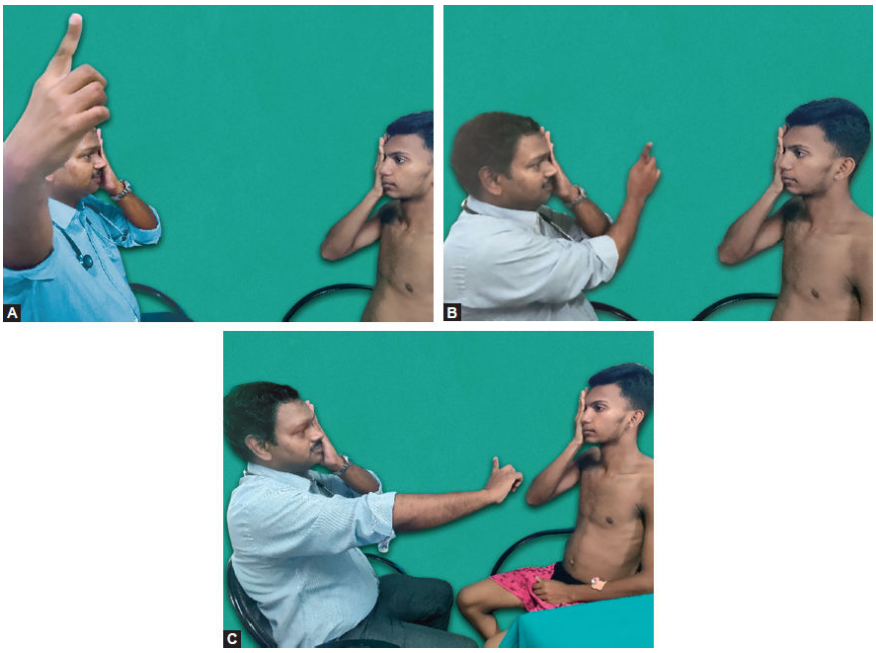
Testing distance: 1 m or one full hands distance **[Figs. 6D(iii).4A to C]**

Instructions:

- Subject and examiner should be sitting at the same height with each one looking into each other's eye separated by distance of 1 m.
- For checking the visual field of right eye of the subject, he is instructed to close his left eye with his left hand while the examiner closes his right eye with right hand. Now, the examiner brings in the flickering index finger of left hand from extremes of all four directions/quadrants diagonally toward the center of the visual field.
- The subject is instructed to give the signal at the first instance of perceiving the flickering finger movement.
- Normal extent of visual field of individual eye:
  - Vertically up 60°
  - Vertically down 75°
  - Medially 60°
  - Laterally 100°
- Normal extent of visual field in binocular vision:
  - Horizontally = 200°
  - Vertically = 140°

Shortcomings of Confrontation Method

1. **Field and defects [Figs. 6D(iii).5 and 6D(iii).6]:**



Figs. 6D(iii).4A to C: Method of examination (confrontation method).

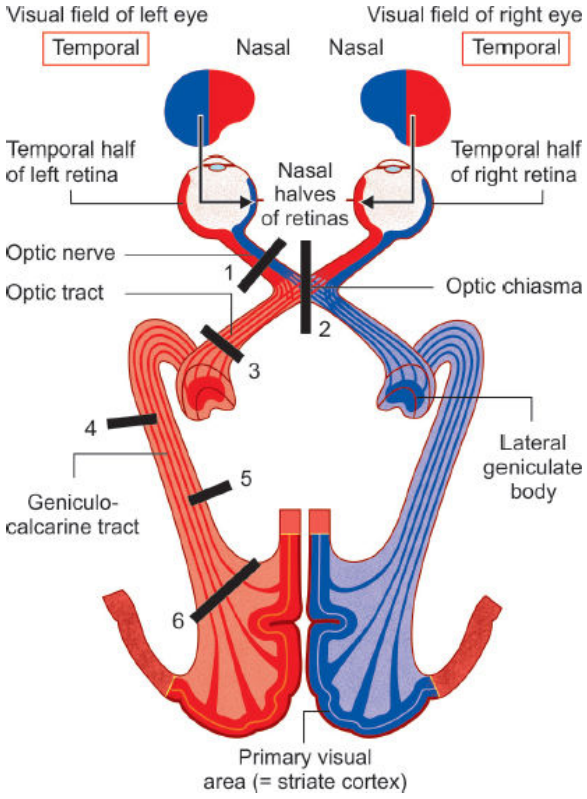


Fig. 6D(iii).5: Sites of lesions causing visual field defects.

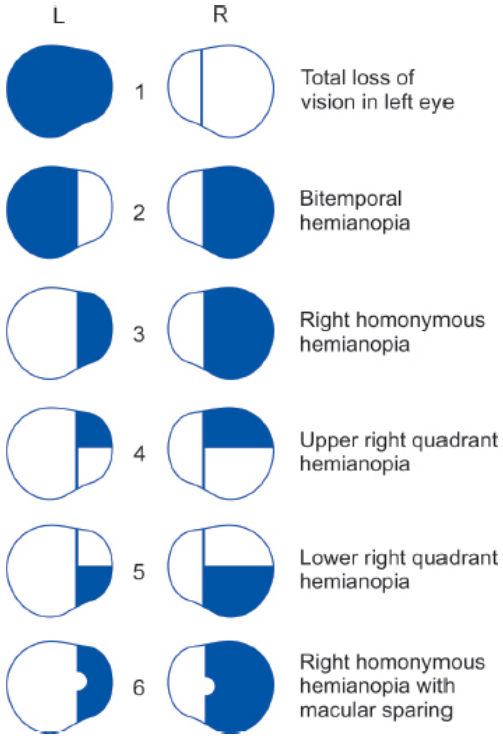


Fig. 6D(iii).6: Visual field defects.

Visual field defect		
	Site of lesion	***Type of defect***
1	**Optic nerve**	Total loss of vision in left eye
2	**Optic chiasma**	Bitemporal hemianopia
3	**Optic tract**	Right homonymous hemianopia
4	**Geniculocalcarine tract**	Upper right quadrantanopia
5	**Geniculocalcarine tract**	Lower right quadrantanopia
6	**Macula**	Right homonymous hemianopia with macular sparing

Note : Visual field defect produced by papilledema—enlarged blind spot

Color Vision (Red/Green/Blue)

Chart used: Ishihara chart **[Figs. 6D(iii).7 and 6D(iii).8]**

Congenital anomalies:

- **Red and green** = chromosome X (mnemonic: remember Red, Green and Symbol X all are traffic symbols)
- **Blue** = chromosome 7 (mnemonic: remember sky is **blue** which has rainbow containing **7** colors).

Acquired defects: Color vision occur in macular and optic nerve diseases, and due to certain **drugs (e.g. ethambutol, chloroquine, digitalis, and sildenafil).**

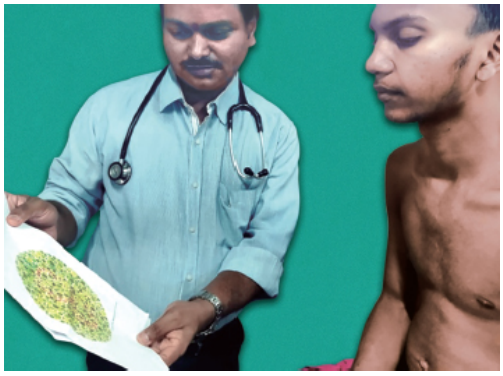


Fig. 6D(iii).7: Method of examining color vision.

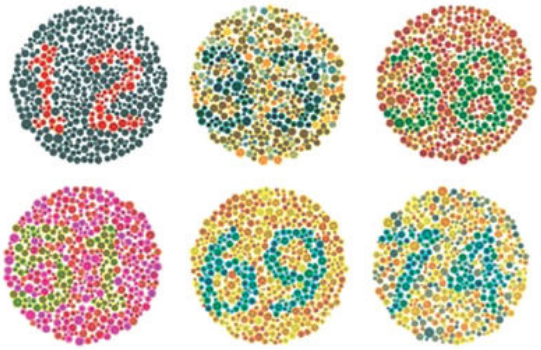


Fig. 6D(iii).8: Ishihara chart for color vision.

Fundus Examination

Instrument used: Direct ophthalmoscope.

How to use:

- The subject should be examined in sitting or lying down position.
- Examination room should be semidark.
- Keep the eye as still as possible.
- Hold ophthalmoscope in same hand as eye you are looking at, and looking through (e.g. hold ophthalmoscope in the left hand for examining patients left eye, through your left eye) **[Figs. 6D(iii).9 and 6D(iii).10]**.
- Hold head steady with thumb above eyebrow, or hold shoulder.
- At about 30 cm distance with light on eye, locate red reflex (seen as an orange glow in the pupil).
- Follow red reflex into the eye as this will get you directly into the optic disc.
- If you cannot find the disc, trace any blood vessels back to it.
- Examine vessels in all four quadrants of eye (upper and lower, nasal and temporal quadrants).
- Identify macula—slightly darker pigmented area, two optic disc widths lateral away from the optic disc.

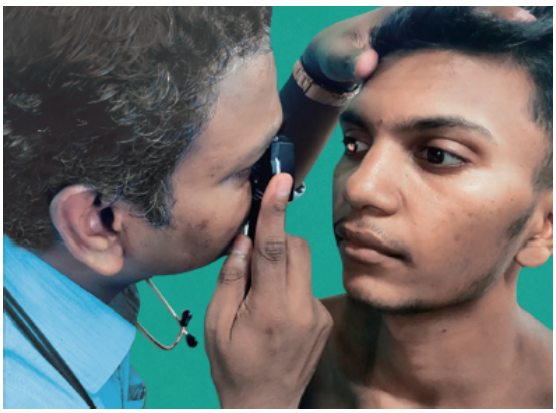


Fig. 6D(iii).9: Fundus examination of right eye.

Fig. 6D(iii).10: Fundus examination of left eye.

Look for optic atrophy and papilledema.

Also watch for feature of retinopathy like hemorrhages, exudates, cotton wool spots, and arteriolar changes.

Fundoscopic Finding

Papilledema is a disease entity which refers to the swelling of the optic disc due to elevated intracranial pressure (ICP) **[Fig. 6D(iii).11]**.

Grade	***Description***
1	Disruption of the normal radial arrangement of nerve fiber bundles with a blurring of the nasal border of the optic disc and normal temporal margin
2	Nasal and temporal (circumferential) blurring of the optic disc with more pronounced changes from grade 1
3	The elevated and blurred disc margin borders obscure one or more major retinal vessel segments
4	More pronounced changes than from grade 3 and with total obscuration of a segment of the central retinal artery or vein
5	More pronounced changes than from grade 4 and with total obscuration of all disc vessels

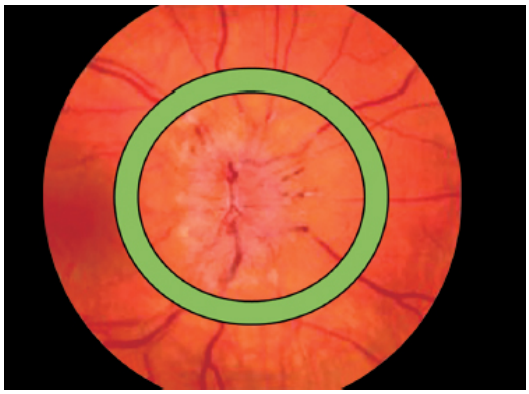


Fig. 6D(iii).11: Papilledema.

Causes of Papilledema

Space-occupying lesions:

- Intracranial mass
- Abscess
- Hemorrhage
- Arteriovenous malformation

Focal or diffuse cerebral edema:

- Trauma
- Toxic
- Anoxia

Blockage of CSF flow: Noncommunicating hydrocephalus

Reduction in CSF reabsorption:

- Meningitis
- Elevated cerebral venous sinus pressure
- Elevated CSF protein—Guillain–Barré syndrome

Pseudotumor cerebri

Systemic causes:

- Hypercarbia
- Hypertension
- Hypercalcemia
- Hypoparathyroidism.

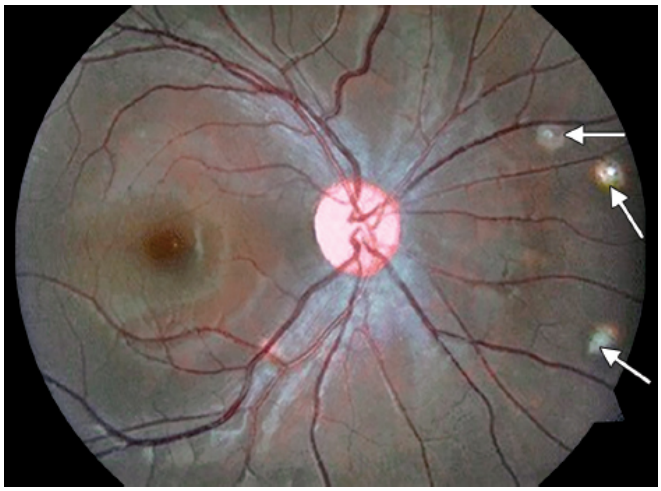


Fig. 6D(iii).12: Choroid tubercles in tuberculosis.

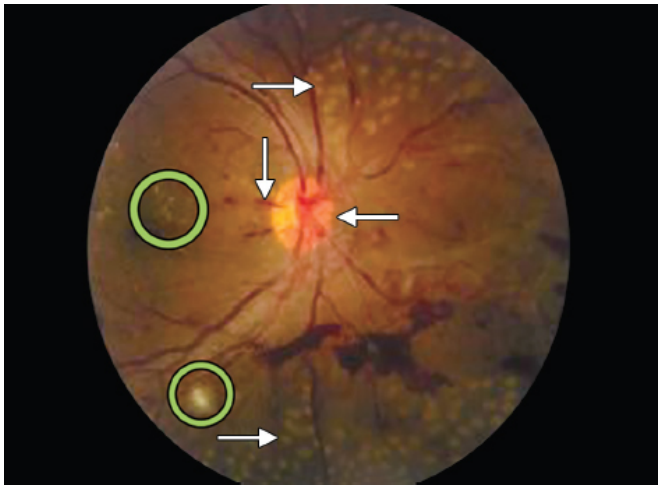


Fig. 6D(iii).13: Proliferative diabetic retinopathy with panretinal photocoagulation.

STAGES OF HYPERTENSIVE RETINOPATHY [FIGS. 6D(III).14 TO 6D(III).17]

Keith-Wagener-Barker Classification

- Group 1: Slight constriction of retinal arterioles
- Group 2: Group 1 + focal narrowing of retinal arterioles + AV nicking
- Group 3: Group 2 + flame-shaped hemorrhages + cotton-wool spots + hard exudates and copper wiring
- Group 4: Group 3 + optic disc swelling and silver wiring.

STAGES OF DIABETIC RETINOPATHY

Nonproliferative Diabetic Retinopathy

Very mild: Microaneurysms only.

Mild
Any or all of: Microaneurysms, retinal hemorrhages, cotton wool spots.

Moderate
- Severe retinal hemorrhages in 1–3 quadrants or mild IRMA
- Significant venous beading in no more than 1 quadrant
- Cotton wool spots.

Severe
The 4-2-1 rule:
- Severe retinal hemorrhages in all 4 quadrants
- Significant venous beading in ≥2 quadrants
- Moderate IRMA in ≥1 quadrants.

Very severe: ≥2 of the criteria for severe.

Proliferative Diabetic Retinopathy [Fig. 6D(iii).13]

Mild-moderate
- New vessels on the disc (NVD) < 1/3 disc area
- New vessels elsewhere (NVE) <1/2 disc area.

High-risk
- NVD >1/3 disc area
- Any NVD with vitreous or preretinal hemorrhage
- NVE >1/2 disc area with vitreous or preretinal hemorrhage.

Advanced diabetic eye disease
- Preretinal (retrohyaloid) and/or intragel hemorrhage
- Tractional retinal detachment
- Tractional retinoschisis
- Rubeosis iridis (iris neovascularization).

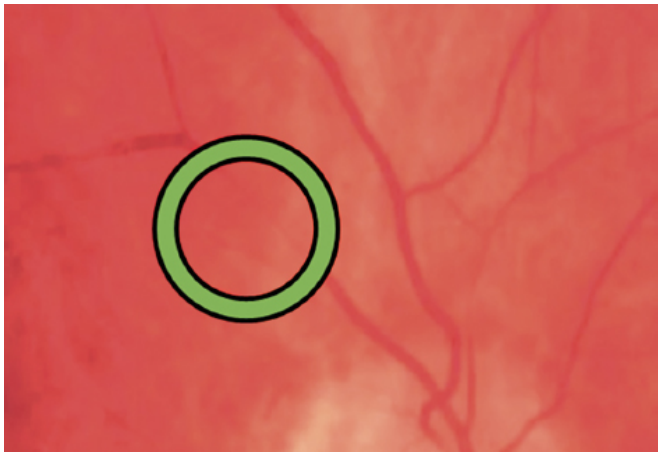


Fig. 6D(iii).14: Focal arteriolar narrowing.

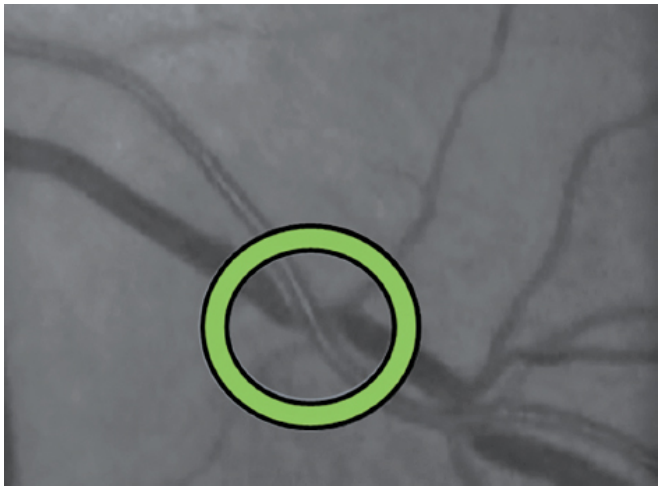


Fig. 6D(iii).15: AV nipping.

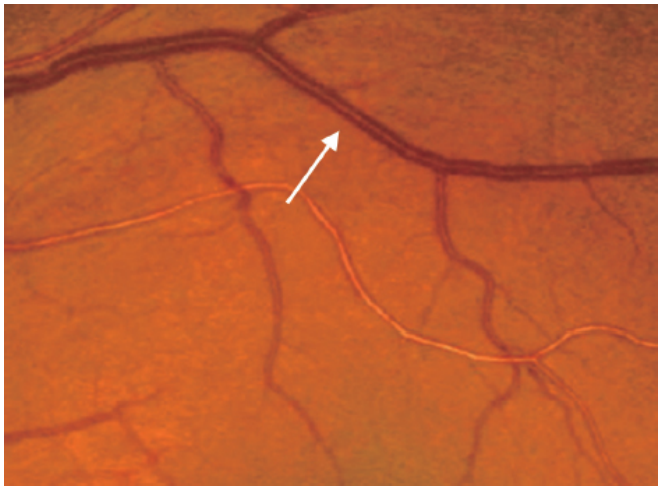


Fig. 6D(iii).16: Copper wiring.

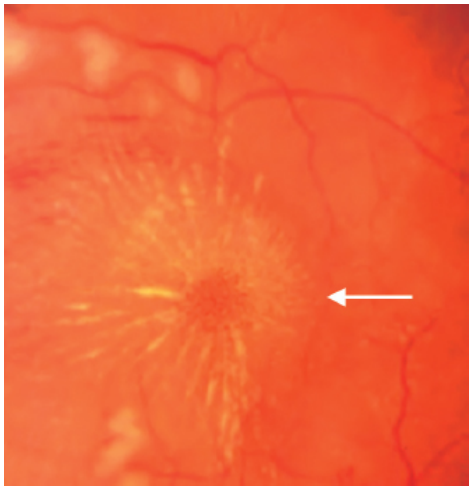


Fig. 6D(iii).17: Cotton wool spots and exudates forming macular star.

Background diabetic retinopathy (bdr)

- It is the earliest phase of diabetic retinopathy (DR).
- Characterized by microaneurysms, dot and blot hemorrhages and exudates.

Diabetic maculopathy: Refers to presence of any retinopathy at the macula.

Preproliferative diabetic retinopathy (PPDR): Cotton wool spots, venous changes, intraretinal microvascular abnormality (IRMA) and deep retinal hemorrhages.

Diabetic papillopathy: It is a form of optic neuropathy seen in young type I diabetics. It is unrelated to glycemic control or any other known feature of diabetes.

CAUSES OF OPTIC ATROPHY

1. Inflammation
2. Ischemia

3. Compression, including raised ICP
4. Nutritional deficiencies/effect of toxins
5. Trauma
6. Hereditary conditions and childhood optic atrophy.

CRANIAL NERVES III, IV AND VI—OCULOMOTOR, TROCHLEAR AND ABDUCENS

Anatomy:

Nuclei	Location	Additional points
III	Upper midbrain	• Four paired nuclei (SR, IR, MR, and IO muscles) • One unpaired nuclei (LPS muscles of both sides)
IV	Midbrain	At level of inferior colliculus (SO muscle)
VI	Mid to lower pons	LR muscle

(SR: superior rectus; IR: inferior rectus; SO: superior oblique; IO: inferior oblique; MR: medial rectus; LR: lateral rectus; LPS: levator palpabrae superioris)

Examined under following headings:
1. Eyelids
2. Eyeballs at rest
3. Extraocular muscles
4. Pupils
5. Nystagmus.

Eyelids

Ptosis: The narrowing of the palpebral fissures due to inability to open an upper eyelid is called ptosis.

Ptosis can be due to	
↓	↓
Paralysis of levator palpebrae superioris (LPS)	***Paralysis of tarsal muscle***
LPS supplied by III cranial nerve	Tarsal muscle supplied by sympathetic system
LPS is paralyzed and the patient cannot voluntarily rise the eyelid, he compensates by contracting frontalis muscle and thus there is wrinkling of forehead seen in long-standing cases	Here since the III nerve is intact and LPS is not paralyzed, ptosis disappears on voluntary contraction of LPS

Cause of ptosis

1. Congenital ptosis	
2. Acquired ptosis	
Neurogenic	• Horner's syndrome • III nerve palsy
Neuromuscular disorder	• Myasthenia gravis (fatigable ptosis)

	• Poisoning (snake bite/botulism)
Myogenic	• Mitochondrial myopathy • Oculopharyngeal muscle dystrophy • Myotonic dystrophy
Mechanical ptosis	Due to eyelid edema or tumors

Unilateral and bilateral ptosis

Unilateral ptosis	***Bilateral ptosis***
• Third cranial nerve lesion • Lesion of cervical sympathetic pathway (Horner's syndrome) • Lesions of the upper eyelid	• Myopathies • Myasthenia gravis • Bilateral Horner's syndrome • Snake bite • Botulism

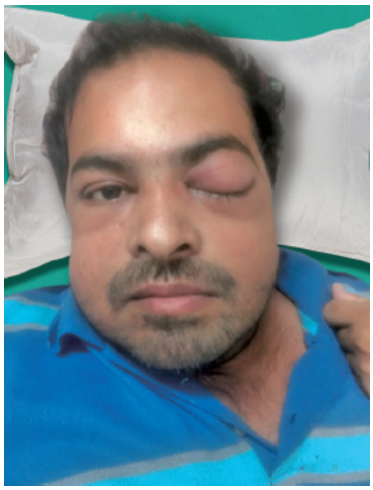


Fig. 6D(iii).18: Neurogenic ptosis.

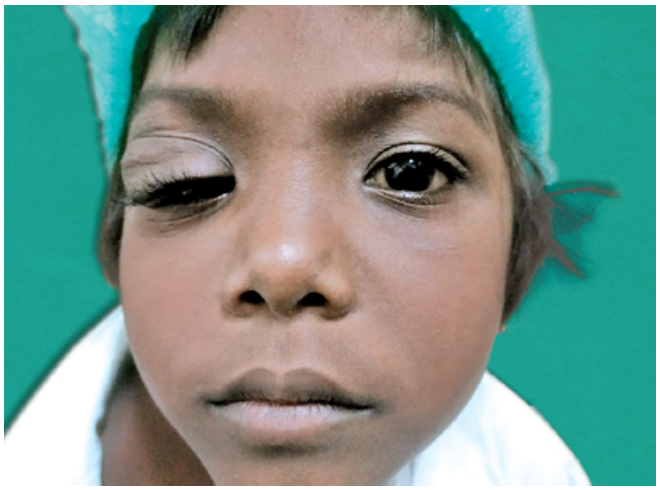


Fig. 6D(iii).19: Mechanical ptosis secondary to edema.

Ptosis and pupil size

Ptosis with	
Small pupil	Horner's syndrome
Large pupil	IIIrd nerve palsy (compressive lesions)
Normal pupillary size	Infarction of IIIrd nerve, myasthenia gravis, myopathies or Guillain–Barré syndrome

Lid retraction:

- Lid is buried under the brow
- Sclera clearly visible above iris
- Example—hyperthyroidism, large doses of anticholinesterases
- Collier's sign: Seen in Parinaud's syndrome. Produces retraction nystagmus.

Reversible ptosis: Myasthenia gravis—**ice pack test [Figs. 6D(iii).20A to C]**

- The ice pack test is cheap, safe, and very quick to perform as it can be carried out at the bedside in approximately 3–5 minutes
- Positive test is the improvement of ptosis by >2 mm or more. This transient improvement in ptosis is due to the**cold** decreasing the acetylcholinesterase breakdown of acetylcholine at the neuromuscular junction.

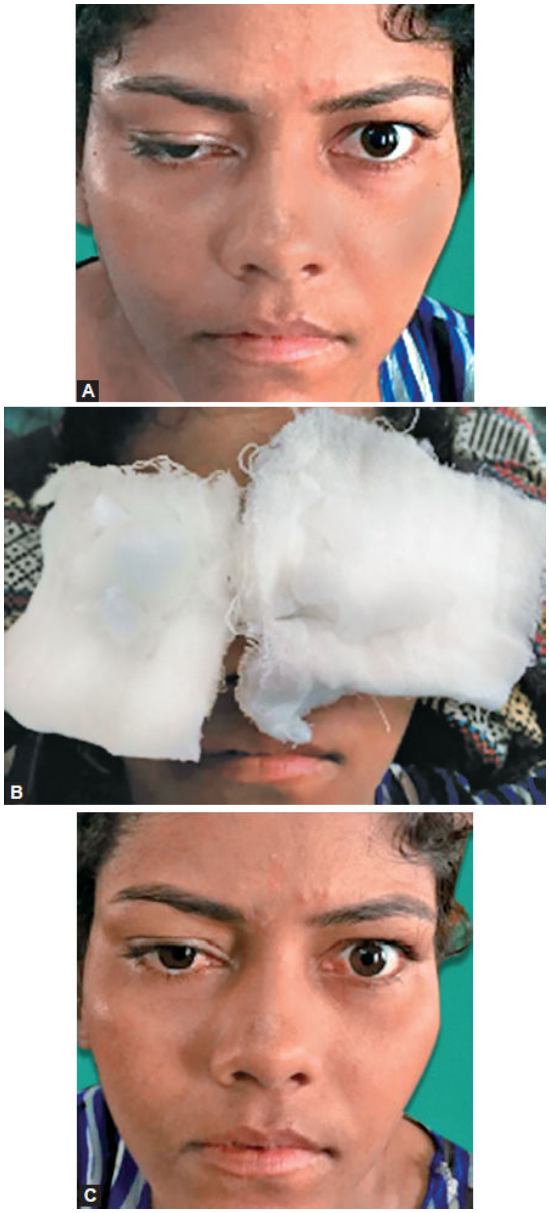
Position of Eyeballs at Rest

Exophthalmos:

- Proptosis of eye
- Most commonly seen in hyperthyroidism

Unilateral exophthalmos:

- Carotid-cavernous fistula (pulsatile exophthalmos)
- Thyroid disorder—hyperthyroidism
- Orbital mass lesion
- Cavernous sinus thrombosis
- Sphenoid wing meningioma



Figs. 6D(iii).20A to C: Reversible ptosis (ice pack test).

- Meningocele
- Mucormycosis.

Enophthalmos: Enophthalmos can be defined as a relative, posterior displacement of a normal-sized globe in relation to the bony orbital margin. Causes are trauma, microphthalmia, post radiation, Horner's syndrome (apparent enophthalmos), Marfan syndrome, Duane's syndrome, or phthisis bulbi.

Extraocular Muscles

Functions of extraocular muscles [Fig. 6D(iii).21]:

	Primary function	Secondary function	Tertiary function

SR	Elevation	Intorsion	Adduction
IR	Depression	Extorsion	Adduction
SO	Intorsion	Depression	Abduction
IO	Extorsion	Elevation	Abduction
MR	Adduction		
LR	Abduction		

(SR: superior rectus; IR: inferior rectus; SO: superior oblique; IO: inferior oblique; MR: medial rectus; LR: lateral rectus)

Mnemonic: **S** in **R** ad

- All **S**uperiors are **IN**tortors
- All **R**ecti are **AD**ductors except lateral rectus
- Function of **R**ecti is **r**egular (superior rectus is for elevation)
- Function of **O**blique is **o**pposite (superior oblique is for depression
- In adducted eye—elevation is by inferior oblique and depression is by superior oblique
- In abducted eye—elevation is by superior rectus and depression is by inferior rectus.

Note: position of testing the muscle and actual action of the muscle usually is opposite with respect to horizontal gaze.

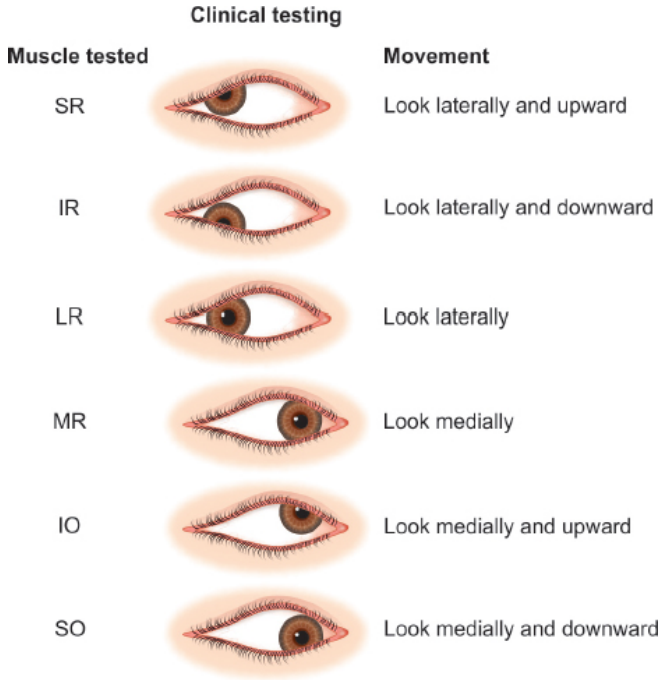


Fig. 6D(iii).21: Extraocular movements.

(SR: superior rectus; IR: inferior rectus; SO: superior oblique; IO: inferior oblique; MR: medial rectus; LR: lateral rectus)

Binocular Movements

Center for conjugate eye movements: Frontal eye field area number 8.

Saccades:

- Conjugate rapid eye movements
- Frontal lobe (premotor area number 6) controls saccadic movements.

Pursuits:

- Slow and smooth movement of eye following a moving target
- Occipital lobe is connected to the PPRF which is responsible for the horizontal pursuit movements.

Reflexes:

- Dolls eye reflex (oculocephalic reflex)
- Caloric stimulation test (vestibuloocular reflex).

Uniocular Movements

Nerve involved and features

Nerve involved	Clinical features
III cranial nerve	• Down and out eye • Divergent squint • Ptosis • Dilatation of pupil
IV cranial nerve	• Defective downward eye movement • Outward rotation of eyeball by unopposed action of inferior rectus • Compensated by head tilt to opposite side
VI cranial nerve	• Defective lateral gaze • Medial squint • Patient may have diplopia on lateral gaze • Compensated by head turn to same side

In the oculomotor nerve **[Fig. 6D(iii).22]**, the parasympathetic fibers lying on the peripheral part have dual blood supply via vasa nervosum and vessels on the sheet. In compressive lesions from outside (tumor and hematoma), pupils are involved early.

In ischemic lesions, pupils are spared since the center of the nerve is affected early.

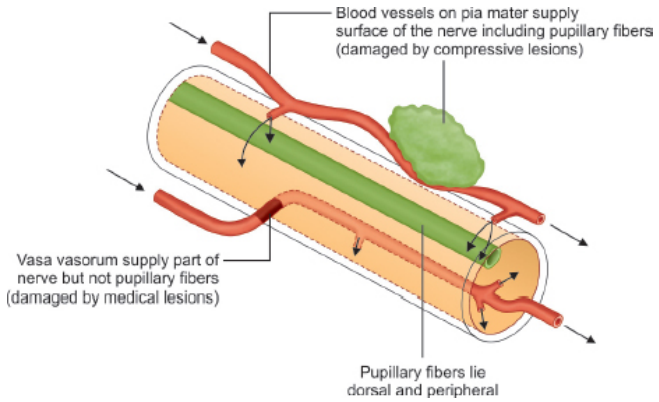


Fig. 6D(iii).22: Oculomotor nerve.

OCULAR MOVEMENT TESTING

Ask the patient to follow the examiner's finger or a red topped hat pin which is kept 60 cm away from the patient's face in all directions **[Figs. 6D(iii).23 and 6D(iii).24]**.

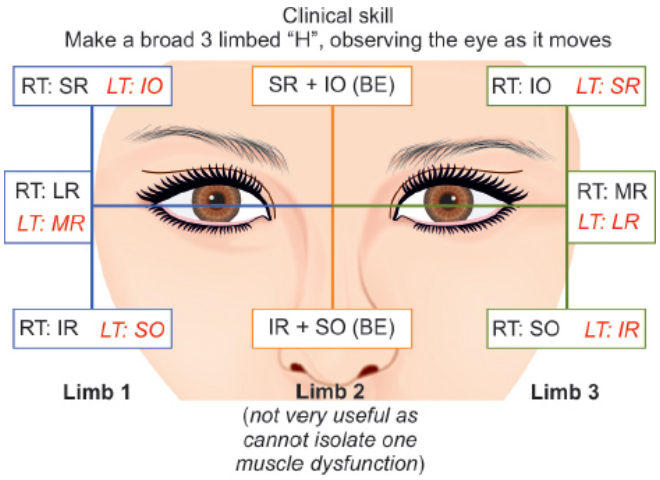


Fig. 6D(iii).23: Ocular movements testing method.

(RT: right; SR: superior rectus; IO: inferior oblique; LT: left; LR: lateral rectus; MR: medial rectus; IR: inferior rectus; SO: superior oblique; IO: inferior oblique)

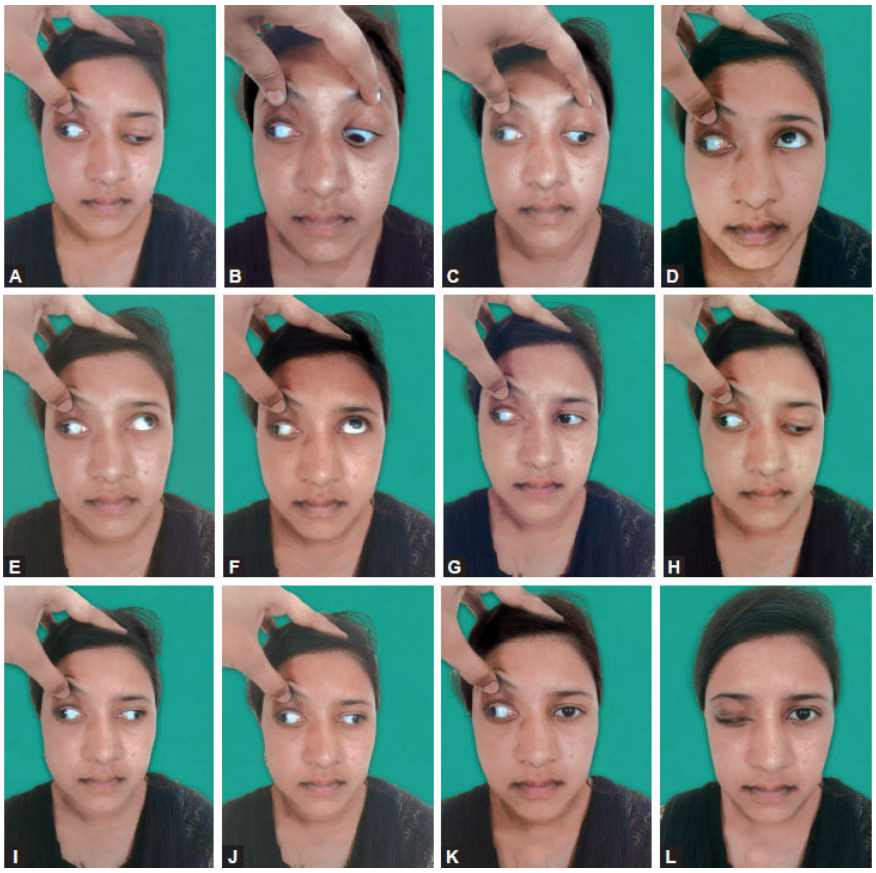
Etiology of III, IV, and VI nerve palsies		
	Medical palsy	***Surgical palsy***
III nerve ophthalmoplegia	Pupil sparing	Pupil involving
	Due to vascular causes where in the central part of nerve is involved (as visualized from the cut section)	Due to compression from the outside on the peripheral part of nerve (as visualized from the cut section)
	• Diabetes • Vasculitis • Myasthenia gravis • Myopathy	• Posterior communicating aneurysm • Tumors of base of skull
IV nerve palsy	Nuclear lesion	
VI nerve palsy	• Pontine lesions • False localizing sign wherein raised ICT is the cause for palsy	

DIPLOPIA

Diplopia means double vision. Most common subjective complaint elicited by lesions in the oculomotor system. Occurs more frequently with lesions of the extraocular muscles or oculomotor nerves than with supranuclear lesions which result in gaze palsies.

Monocular Diplopia

- The first point to clarify is whether diplopia persists in either eye after covering the fellow eye. If it does, the diagnosis is monocular diplopia.



Figs. 6D(iii).24A to L: Ocular movements testing in a patient with right complete ophthalmoplegia.

- The cause is usually intrinsic to the eye. For example, corneal aberrations, uncorrected refractive error, cataract, foveal traction or foreign body in the aqueous or vitreous may give rise to monocular diplopia.

Binocular Diplopia

Diplopia improved by covering one eye is binocular diplopia and is caused by disruption of ocular alignment. Occurs only if both eyes are open.

Binocular diplopia occurs from a wide range or processes: For example, infectious, neoplastic, metabolic, degenerative, inflammatory, and vascular.

Assessment of Diplopia

- Cover one of the patient's eye with a transparent red shield. Move a point of light in the direction of action of each muscle.
- Ask the patient if he sees one object or two.
- If double, do the images lie side by side or one above the other?
  - Side by side—medial rectus (MR)/lateral rectus (LR)
  - One above the other—superior rectus (SR)/inferior rectus (IR) and superior oblique (SO)/inferior oblique (IO)
- Which is the red image?
- In which position the images are the farthest.

Points to note:

- In diplopia two images, one real and one false are formed. The real image is closer to the eye and distinct; the false image is farther away from eye and indistinct.
- Separation of images is maximum in the direction of action of weak muscle.

Muscle	Movement affected	Squint	Diplopia	
LR	Abduction	Convergent	Uncrossed	Maximum on looking laterally
MR	Adduction	Divergent	Crossed	Maximum on looking medially
SO	Downward movement in adduction	Convergent—in elevation and extorsion	Uncrossed	Maximum on looking down and medially
IO	Upward movement in adduction	Convergent—in depression and intorsion	Uncrossed	Maximum on looking up and medially
SR	Upward movement in abduction	Divergent—in depression and extorsion	Crossed	Maximum on looking up and laterally
IR	Downward movement in abduction	Divergent—in elevation and intorsion	Crossed	Maximum on looking down and laterally

(LR: lateral rectus; MR: medial rectus; SO: superior oblique; IO: inferior oblique; SR: superior rectus; IR: inferior rectus)

STRABISMUS/SQUINT

- Loss of parallelism of eyeball resulting in abnormal position of eyes.
- Primary deviation—deviation in the paralyzed eye
- Secondary deviation—deviation in the normal eye.

Types of Squint

Paralytic	Nonparalytic/concomitant
Secondary deviation > primary deviation	Secondary deviation = primary deviation
Acquired	• Usually congenital • Starts in childhood
Diplopia present	No diplopia
Ocular movements affected	Ocular movements are full in all directions

Pupils

Miosis and mydriasis

Large pupils	Small pupils
Unilateral: • Physiological • Pharmacological • Oculomotor nerve palsy • Adie's pupil • Uncal herniation • Traumatic sphincter paralysis • Iris ischemia • Ocular siderosis	**Unilateral:** • Physiological • Horner's syndrome • Anterior uveitis • Long standing Adie's pupil • Pharmacological
Bilateral:	**Bilateral:**

• Pharmacological • Parinaud's dorsal midbrain syndrome • Benign periodic mydriasis • Brainstem death	• Physiological senile miosis • Pharmacological • Argyll Robertson pupil • Lepromatous miosis • Congenital microcoria • Myotonic dystrophy

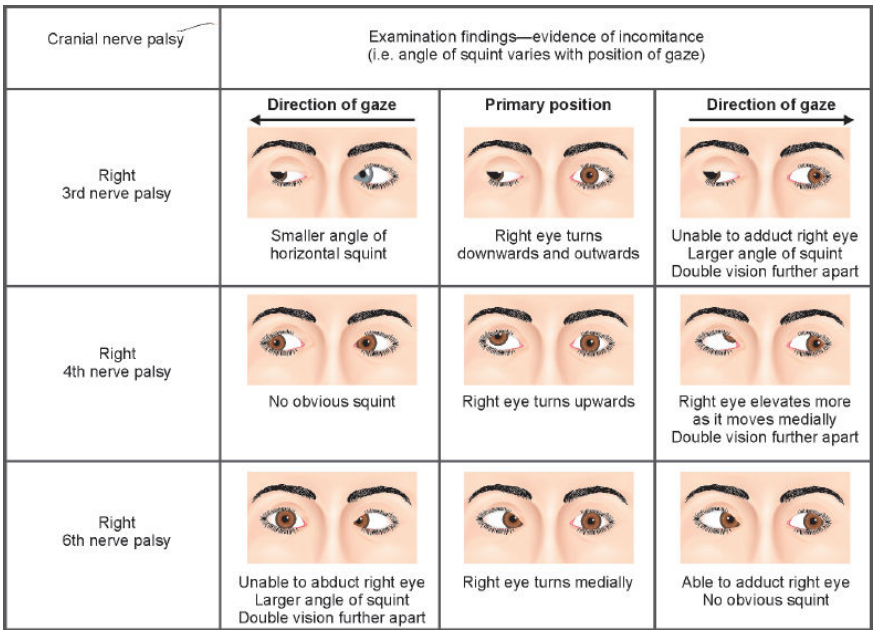


Fig. 6D(iii).25: Cranial nerve 3, 4, and 6 palsy.

Light reflex

- Mediated by retinal photoreceptors.
  - Subserved by four neurons **[Fig. 6D(iii).26]**
    1. First (sensory)—connects each retina with both pretectal nuclei, nasal fibers decussate, and temporal fibers uncrossed
    2. Second (internuncial)—connects each pretectal nucleus to both Edinger–Westphal nuclei —indirect reflex
    3. Third (preganglionic motor)—connects Edinger–Westphal nucleus to ciliary ganglion.
       - Parasympathetic fibers pass through III nerve inferior division and reach the ciliary ganglion via the nerve to the inferior oblique muscle.
    4. Fourth (postganglionic motor) leaves the ciliary ganglion and passes in the short ciliary nerves to innervate the sphincter pupillae.

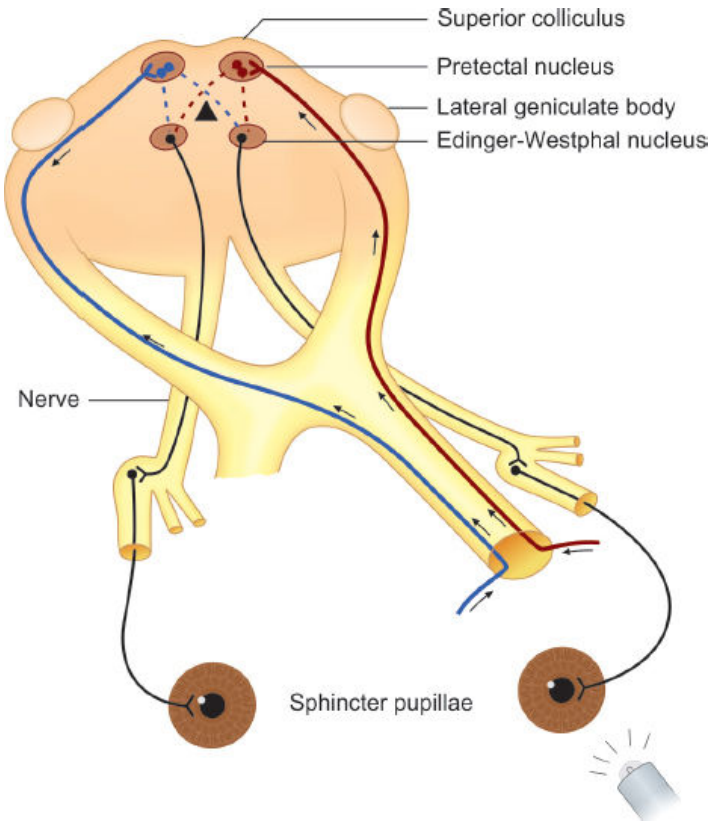


Fig. 6D(iii).26: Light reflex pathway.

- Tested in each eye individually
- Patient fixing at a distance
- Light shown to the eye obliquely.
- Cover uncover technique—uses ambient light
- Normal response: Brisk constriction—slight dilatation back to an intermediate state.
- Can be recorded: prompt, sluggish, and absent—graded 0–4+

The accommodation reflex

- Relax accommodation by gazing at a distant object.
- Shifting gaze to some near object.
- The primary stimulus for accommodation is blurring.
- Response: Accommodation, convergence, and miosis.

Pathway similar to light reflex till **[Fig. 6D(iii).27]**

- Fibers of Edinger–Westphal nucleus when entering the eye will cause constriction of the pupil and stimulation of ciliary muscle, so the parasympathetic causes the two changes (constriction of the pupil and contraction of ciliary muscle that increases the thickness of the lens thus increasing its power).
- The third change is convergence (adduction of both eyes by stimulating medial rectus on both sides); this is achieved by the Vergence center that affects the oculomotor nucleus in

the midbrain on this side and the other. Fibers coming from the oculomotor nucleus will enter and stimulate the medial rectus on both sides, when both eyes are adducted, the image will be on the same area (focus) of the retina.

PUPILLARY ABNORMALITIES

Argyll Robertson Pupil

- Small irregular pupil having light near dissociation **[Fig. 6D(iii).28]**
  Characteristic feature:
  - In dim light, both pupils are small and may be irregular.
  - In bright light, neither pupil constricts.
  - On accommodation both pupils constrict (light near dissociation).
  - After instillation of pilocarpine 0.1% into both eyes, neither pupil constricts.
- Described for neurosyphilis.
- Lesion in periaqueductal region, pretectal, and rostral midbrain.

Other causes: Diabetes mellitus, chronic alcoholism, multiple sclerosis, and sarcoidosis.

Reverse Argyll Robertson Pupil

In this accommodation, reflex on the pupil is absent.
Cause: Diphtheria and tumors at corpora quadrigemina.

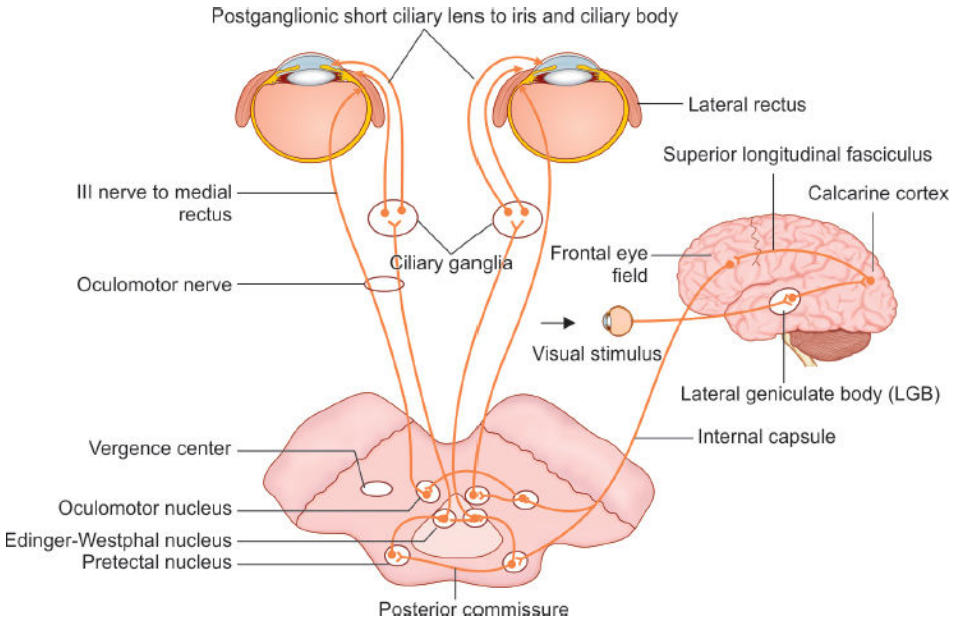


Fig. 6D(iii).27: Accommodation of reflex pathway.

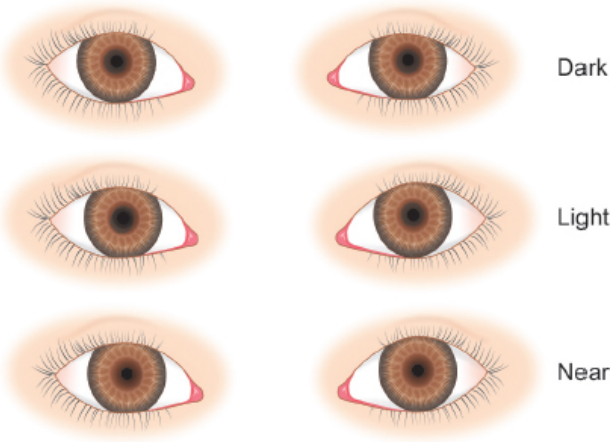


Fig. 6D(iii).28: Argyll Robertson pupil.

Wernicke's Hemianopic Pupil

- It indicates lesion of the optic tract.
- In this condition, light reflex (ipsilateral direct and contralateral consensual) is absent when light is thrown on the temporal half of the retina of the affected side and nasal half of the opposite side; while it is present when the light is thrown on the nasal half of the affected side and temporal half of the opposite side.

The Adie's Tonic Pupil

In this condition, reaction to light is absent and to near reflex is very slow and tonic.

- The affected pupil is larger (anisocoria).
- Its exact cause is not known.
- It is usually unilateral, associated with absent knee jerk and occurs more often in young women.
- Adie's pupil constricts with weak pilocarpine (0.125%) drops, while normal pupil does not.
- In long-standing cases, the pupil may become small ("little old Adie").
- In some cases, are diminished deep tendon reflexes (Holmes-Adie syndrome).

Afferent Pupillary Defect or Marcus Gunn Pupil

- The status of the light reflex must be judged by comparing the two eyes **[Fig. 6D(iii).29]**
- Indicator of optic nerve function
- Swinging flashlight test: Light is held about 1 inch from the eye and just below the visual axis; the light is rapidly alternated.
  - The examiner attends only to the stimulated eye.
  - Comparing the amplitude and velocity of the initial constriction in the two eyes.
- The reaction is relatively weaker when the bad eye is illuminated.

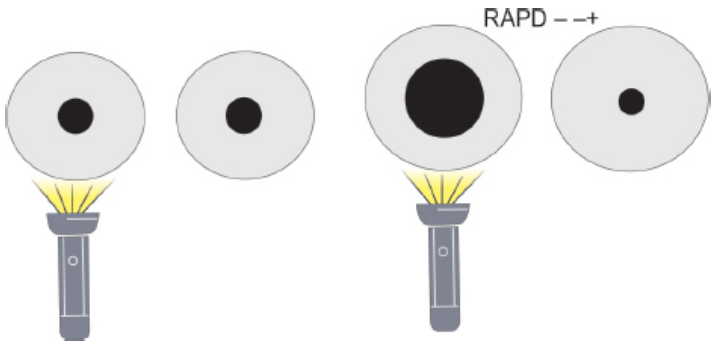


Fig. 6D(iii).29: Relative afferent pupillary defect (RAPD)/Marcus Gunn pupil.

- The brain detects a relative diminution in light intensity and the pupil may dilate a bit in response.
- Bring out the dynamic anisocoria.
- The weaker direct response or the paradoxical dilation of the light-stimulated pupil is termed as an afferent pupillary defect (APD).

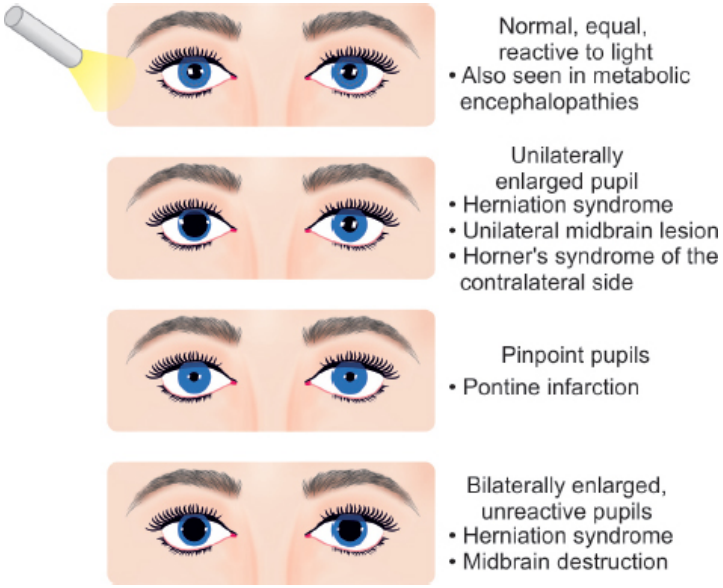


Fig. 6D(iii).30: Pupillary abnormalities in coma.

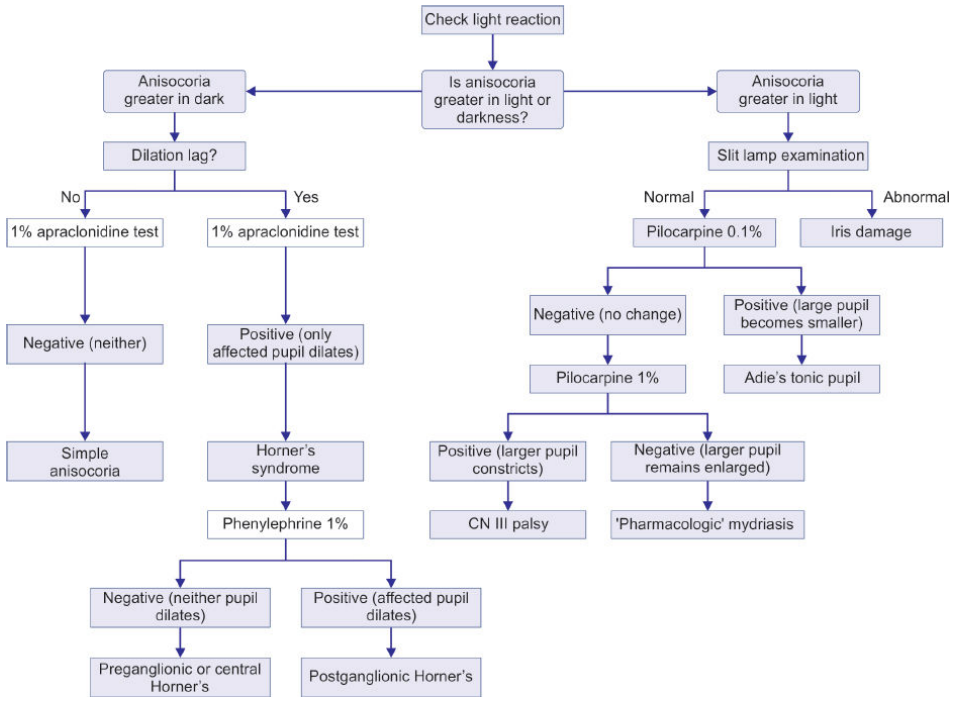


Fig. 6D(iii).31: Approach to pupillary abnormalities.

- Trace APD: Pupil that has an initial constriction, but then it escapes to a larger intermediate position than in the other eye.
- 1 to 2+ APD: No change in pupil size initially, then dilation.
- 3 to 4+ APD: Immediate dilation of the affected pupil.

Hutchinson's Pupil

- Seen in comatose patients
- Dilated poorly reactive pupil
- Due to expanding intracranial supratentorial mass causing uncal herniation and III nerve compression.

Hippus

- Irregular rhythmic visible pupillary oscillations 2 mm/more in amplitude irregular dilating and constricting movements are observed
- Also called as pupillary athetosis
- **Cause:** Myasthenia gravis.

Tectal Pupils

Large pupils with light near dissociation: Seen in lesions affecting the upper midbrain.

Horner's Syndrome: Oculosympathetic Palsy

- **P**tosis: Denervation of Müller's muscles
- **M**iosis: Denervation of dilators
- **E**nophthalmos: Narrowing of palpebral fissure

- **A**nhydrosis: Sympathetic denervation
- **L**oss of ciliospinal reflex.

Mnemonic—**P**rotein **MEAL [Fig. 6D(iii).33]**.

Usually unilateral: The smooth muscle fibers of the lower eyelid retractors also lose their sympathetic supply in patients with Horner's syndrome and, thus, the lower eyelid appears slightly elevated. This appearance has been termed "**upside-down ptosis" or "reverse ptosis".**

- Hypochromic heterochromia (iris of different color—Horner is lighter) may be seen if congenital or long-standing. Sympathetic innervation is thought to be required for the formation of melanin by stromal melanocytes.
- Reduced ipsilateral sweating if the lesion is below the superior cervical ganglion, because the sudomotor fibers supplying the skin of the face run along the external carotid artery.
- Horner's syndrome is usually characterized by "**partial ptosis**" and "**apparent enophthalmos**"

Unilateral (dilated)		Reaction to light (direct)	Associated signs
Third nerve palsy		None	Ptosis (partial or complete), externalophthalmoplegia
Holmes-Adie syndrome		Slow	Better response to accommodation, lower limb areflexia
Marcus Gunn pupil		Slow and incomplete	Normal consensual response, optic atrophy, central scotoma, impaired color vision
Local lesion of the iris		Variable depending on extent of local damage	Irregular pupil
Unilateral (constricted)			
Horner's syndrome		Reduced dilatation to shade	Ptosis (partial), ipsilateral facial anhidrosis, "enophthalmos"
Bilateral (dilated)			
Midbrain lession		None	Mid-position pupils; impaired vertical gaze
Iatrogenic/atropine, tricyclic antidepressants		None or reduced	
Bilateral (constricted)			
Senile		None or reduced	
Iatrogenic/ pilocarpine drops		None or reduced	
Pontine lesion		None	Pin-point pupils, coma, Cheyne-Stokes respiration
Argyll-Robertson		None	Irregular pupils, normal accommodation

Fig. 6D(iii).32: Summary of pupillary abnormalities.

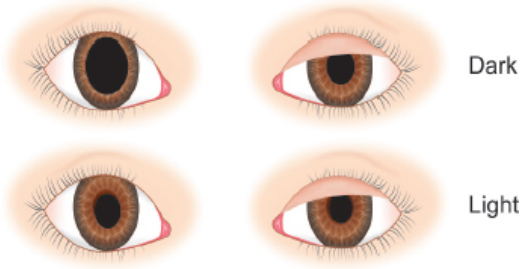


Fig. 6D(iii).33: Horner's syndrome.

Causes of Horner's Syndrome [Fig. 6D(iii).34]

Unilateral	*Bilateral*
Central (1st order neurons): Brainstem disease (tumor, vascular, and demyelination), syringomyelia, lateral medullary (Wallenberg) syndrome, spinal cord tumor, and base of skull tumors/injury **Preganglionic (2nd order neuron):** • Pancoast tumor, carotid and aortic aneurysm and dissection, neck lesions (glands, trauma, and postsurgical) • Birth trauma with lower brachial plexus injury and cervical rib **Postganglionic (3rd order neuron):** Cluster headaches (migrainous neuralgia), internal carotid artery dissection, nasopharyngeal tumor, otitis media, cavernous sinus mass, Raeder syndrome (paratrigeminal syndrome), and carotid cavernous fistula	Diabetic autonomic neuropathy, amyloidosis, pure autonomic failure, Anderson–Fabry disease, familial dysautonomia, and paraneoplastic syndrome

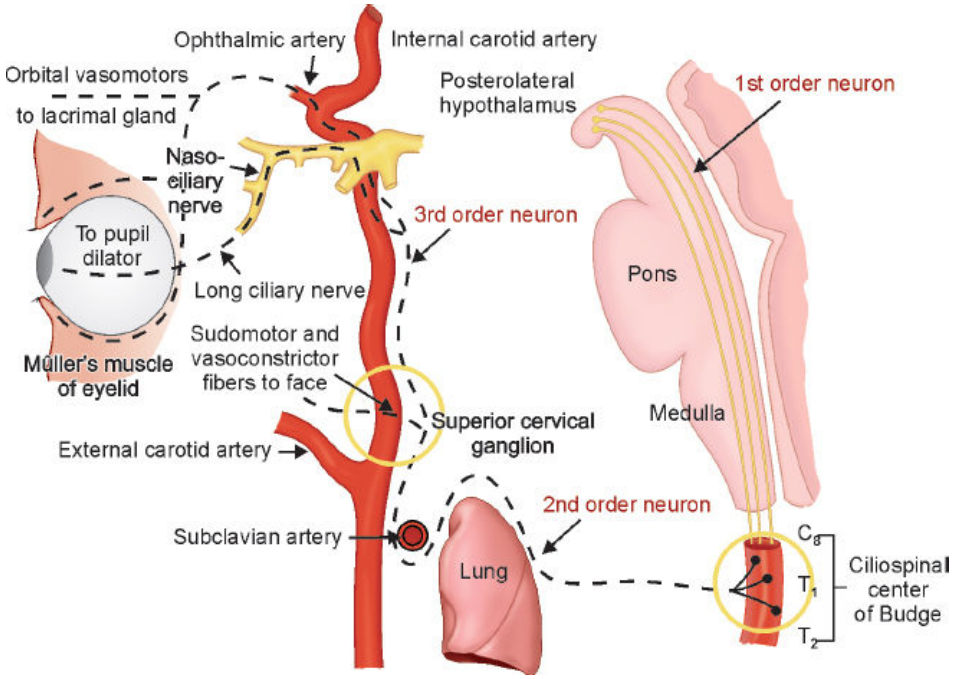


Fig. 6D(iii).34: Diagrammatic representation of sites of involvement of Horner's syndrome.

OPHTHALMOPLEGIA

Definitions:

- **Supranuclear ophthalmoplegia:** Also called as gaze palsies. It is due to involvement of corticonuclear fibers of the III, IV, and VI cranial nerves.
- **Internuclear ophthalmoplegia:** It is due to involvement of MLF and PPRF which connect the III nerve to the contralateral VI nerve.
- **Nuclear/infranuclear ophthalmoplegia:** Involvement of individual cranial nerves (CN III, IV, and VI).

1st neuron: Associated symptoms of brainstem involvement, such as dizziness, vertigo, transient ischemic attacks suggestive of hemianopia with/without long tract signs
- Hydroxyamphetamine—dilates both pupils
- Phenylephrine—dilates both pupils
- Cocaine—Horner's pupil dilates more poorly than normal pupil

2nd neuron: Chest mass with arm pain, phrenic nerve paralysis, supraclavicular nodes, neck mass, thyroid enlargement, neck surgery, neck injury, cervical osteoarthritis with bone spurs
- Hydroxyamphetamine—dilates both pupils
- Phenylephrine—dilates both pupils
- Cocaine—Horner's pupil dilates more poorly than normal pupil

3rd neuron: History of vascular headache (migraine, Raeder's, cluster), carotid artery disease with ipsilateral visual loss and contralateral motor and sensory signs. Sweating present if above bifurcation of carotid artery and absent if below bifurcation
- Hydroxyamphetamine—Horner's pupil dilates less or not at all
- Phenylephrine—Horner's pupil dilates more
- Cocaine—Horner's pupil dilates more poorly or not at all

Fig. 6D(iii).35: Differentiating features of 1st order, 2nd order, and 3rd order Horner's syndrome.

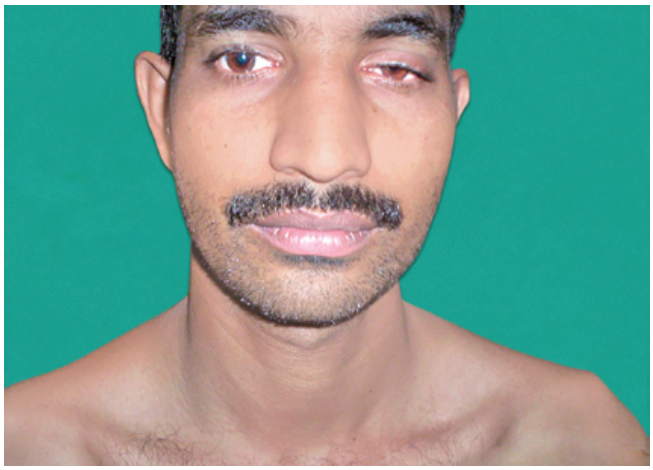


Fig. 6D(iii).36: Horner's syndrome.

- **Internal ophthalmoplegia:** Paralysis of constrictor pupillae and ciliary muscle
- **External ophthalmoplegia:** Paralysis of extraocular muscles
- **Total ophthalmoplegia:** Combination of external and internal ophthalmoplegia.

Gaze Palsies/Supranuclear Ophthalmoplegia

Vertical Gaze Palsies

Upward gaze palsy:

- Lesions at the superior colliculus—Parinaud's syndrome
- Progressive supranuclear palsy
- Parkinson's disease
- Wernicke's encephalopathy
- Thalamic hemorrhage (Sunset sign).

Downward gaze palsy:

- Huntington's chorea
- Niemann–Pick disease
- Olivopontocerebellar ataxia
- Progressive supranuclear palsy
- Parkinson's disease.

Combined upward and downward gaze palsy:

- Bilateral frontal lobe lesions
- Progressive supranuclear palsy
- Parkinson's disease.

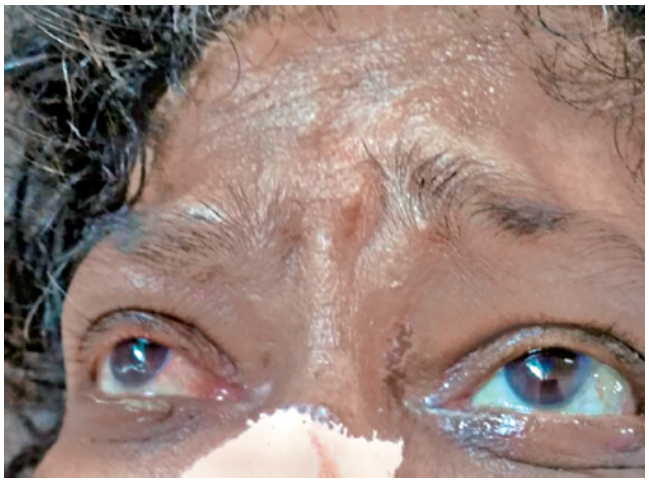


Fig. 6D(iii).37: Reptilian stare in progressive supranuclear palsy.

Horizontal Gaze Palsies

• Frontal eye field (Area number 8) • Destructive lesion—both eyes will turn toward the side of lesion (Vulpian sign) • Irritative lesion—both eyes will turn to opposite side	• Pontine lateral gaze center • Destructive lesion—loss of lateral gaze to the same side • Irritative lesion—eyes deviate to the same side as lesion

Internuclear Ophthalmoplegia

- Caused by a lesion of the medial longitudinal fascicle (MLF), which carries signals from the abducens nucleus to the contralateral medial rectus oculomotor subnucleus **[Fig. 6D(iii).38]**.

- The abducens nerve and MLF coordinate conjugate horizontal eye movements with co-contraction of ipsilateral lateral rectus and contralateral medial rectus muscles.
- Classic signs of unilateral internuclear ophthalmoplegia include impaired adduction of the ipsilesional eye and abducting nystagmus of the contralateral eye.
- Despite ipsilateral adduction weakness with direct motility testing, adduction is often intact with convergence because convergence signals to the medial rectus nucleus are distinct from the MLF.
- Multiple sclerosis and microvascular brainstem ischemia are the most common causes.

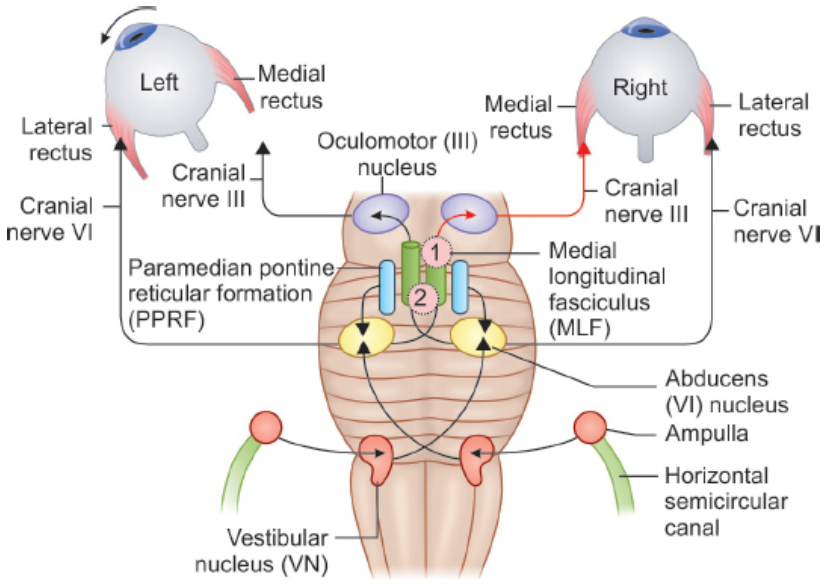


Fig. 6D(iii).38: Internuclear ophthalmoplegia.

Superior INO (Lhermitte's syndrome)	Lesions in the brainstem
Inferior INO (Lutz syndrome)	Lesions in the pontine lateral gaze center → to the abducens nucleus
Pseudo-INO	• Myasthenia Gravis • Miller Fisher syndrome
Webino syndrome (wall eyed bilateral INO)	Bilateral MLF and bilateral medial rectus nucleus
Wemino syndrome (wall eyed mono-ocular INO)	Unilateral MLF and unilateral medial rectus nucleus
One and a half syndrome	Involvement of pontine PPRF and adjacent MLF
Eight and a half syndrome	One and a half syndrome + 7th nerve palsy

(INO: internuclear ophthalmoplegia; MLF: medial longitudinal fasciculus; PPRF: paramedian pontine reticular formation; WEBINO: wall-eyed bilateral internuclear ophthalmoplegia; WEMINO: wall-eyed mono-ocular internuclear ophthalmoplegia)

Etiology of Nuclear or Infranuclear Palsy

Site	Occulomotor nerve	Trochlear nerve	Abducens nerve palsy

	palsy	palsy	
Brainstem	• Weber's syndrome • Nothnagel syndrome • Benedict's syndrome • Claude's syndrome	Midbrain syndromes	• Millard–Gubler syndrome • Raymond-Céstan syndrome • Foville's syndrome • Möbius syndrome
Subarachnoid space	+	+	–
Petrous apex—Dorello's canal	–	–	+ (Gradenigo's syndrome)
Cavernous sinus	+	+	+
Superior orbital fissure	+	+	+
Orbit	+/–	+	–

Painful Ophthalmoplegia

- Cavernous sinus thrombosis
- Superior orbital fissure syndrome—Tolosa–Hunt syndrome
- Ophthalmoplegic migraine
- Pituitary apoplexy
- Orbital cellulitis
- Orbital tumors.

	Supranuclear ophthalmoplegia	Nuclear/infranuclear ophthalmoplegia
Movements affected	Gaze	Individual muscle movements
Diplopia and squint	Absent	Present
Pupils	Normal	May or may not be involved
Vestibulo-ocular reflex (cold caloric)	+	–

NYSTAGMUS

Definition: Nystagmus is involuntary, conjugate, repetitive, and rhythmic movements of eyeball.

Method of examination: Eyes should be deviated in all four directions for at least 5 seconds and deviation should not be of extremes.

Grading/degrees of nystagmus	
I	Nystagmus only on deviation of eyes
II	Nystagmus on looking forward
III	Direction of nystagmus opposite to the fast beating component

Types of Nystagmus

Pendular nystagmus	Jerk nystagmus			Nystagmus of dissociated rhythm
In this time of amplitude of nystagmus is equal in either directions	In this type of nystagmus, there is slow component followed by fast (jerk) component due to cortical correction			Usually gaze evoked nystagmus
	Horizontal	***Vertical***	***Rotatory***	
These are predominantly seen in congenital conditions especially due to visual defects from earlier years	• Labyrinthine disorders • Cerebellar disorders • Uppermost cervical lesion	• Never labyrinthine • Cerebellar disorders • Brainstem lesions • Drugs like benzodiazepines and barbiturate	• Labyrinthine disorders • Brainstem lesions	• MLF lesions • Multiple sclerosis

Other Common Types of Nystagmus

	Description	Condition seen
Seesaw nystagmus	Upward deflection of one eyeball with downward deflection on the contralateral eyeball	Suprasellar region anterior to III ventricle
Up beat nystagmus	Fast movement upward	Lesions in the vermis of the cerebellum
Down beat nystagmus	Fast component is down	Foramen magnum lesions
Optokinetic nystagmus	Railway track nystagmus	Deep parietal lobe lesions
Convergence retraction nystagmus	Attempted upgaze provokes jerk nystagmus with fast component in inward convergent manner	Lesion at superior colliculus—Parinaud's syndrome

Non-nystagmus Oscillations of Eyeball

Ocular flutter	Periodic horizontal saccades	Cerebellar and PPRF lesions
Opsoclonus	Irregular oscillations with different amplitude and directions	• Toxins • Encephalitis
Ocular bobbing	Rapid downstroke followed by slow uprise of eyeball	Pontine destruction
Ocular dipping	Slow downstroke followed by rapid uprise of the eyeball	Toxic encephalopathy

(PPRF: paramedian pontine reticular formmation)

	Central nystagmus	Peripheral nystagmus
Fast component	Fast component is toward same side of pathology	Fast component is to the opposite of the pathology

Duration of episode	Long lasting	Acute and transient
Vertigo	Less prominent	Usually associated
Suppression on fixation using Frensel lens	Not suppressed	Suppressed
Pursuits and saccades	Usually present	Absent
Other clinical finding	CNS involvement is seen	Hardness of hearing and tinnitus is seen

(CNS: central nervous system)

CRANIAL NERVE V—TRIGEMINAL NERVE

- Largest among cranial nerves
- Most complex of the cranial nerves

We shall discuss trigeminal nerve under:

1. Sensory component and motor components
2. Reflexes
3. Disorders of trigeminal nerve dysfunction

Sensory and Motor Component

Component	Sensory part	Motor part
Size	Larger	Smaller
Nuclei	Three nuclei	One nuclei
Distribution	• Face (except angle of mandible) • Teeth • Oral cavity • Nasal cavity • Scalp to vertex • Intracranial dura • Cerebral vasculature • Proprioception to muscles of mastication	Muscles of mastication

Distribution [Fig. 6D(iii).39]: The distribution of CN V3 does not extend to the jaw line; there is a large “notch” at the angle of the jaw innervated by the greater auricular nerve (C2-3).

Nuclei and functions:

Nuclei	Location	Function
Motor nuclei	Pons	• **M**uscles of mastication • **M**ylohyoid • **A**nterior belly of digastric • **T**ensor veli palatini • **T**ensor tympani

Principle sensory nucleus	Pons	• Pressure • Touch • Vibration
Mesencephalic nuclei	Extends to midbrain	Proprioception of muscles of mastication, EOM, facial expression
Spinal nucleus	Extends to spinal nucleus (C3, 4) via medulla — quintothalamic tract	• Pain • Temperature

Note:

- All the sensory supply relay via trigeminal ganglion which is also called as Gasserian ganglion or semilunar ganglion.
- It is largest ganglion located at Meckel's cave, lateral to ICA and posterior to cavernous sinus.
- It is analogous to dorsal root ganglion.

Testing of sensory component:

- Test the sensation of the face for touch, pain, and temperature in each of the divisions.
- Sensation should be compared in each trigeminal division, and the perioral region compared to the posterior face to exclude an onion skin pattern **(Figs. 6D(iii).40 to 6D(iii).43)**
- Pain or temperature should be compared with touch to exclude dissociated sensory loss (a common finding in lateral medullary syndrome).

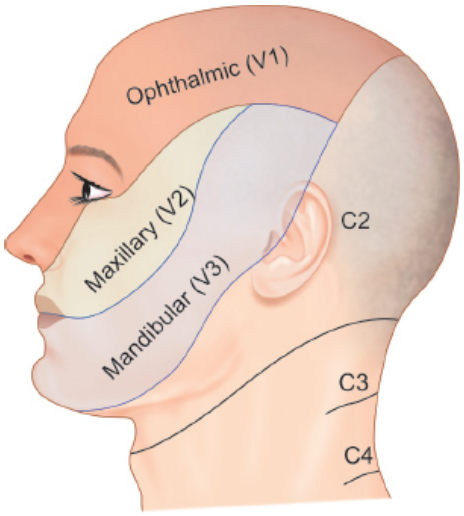


Fig. 6D(iii).39: Image showing sensory distribution of three divisions of trigeminal nerve.

- On the trunk, organic sensory loss typically stops short of midline because of the overlap from the opposite side, and crossing of the midline suggests nonorganic nature of the symptoms. However, this finding is not reliable on the face because there is less midline overlap, so organic facial sensory loss may extend to the midline.

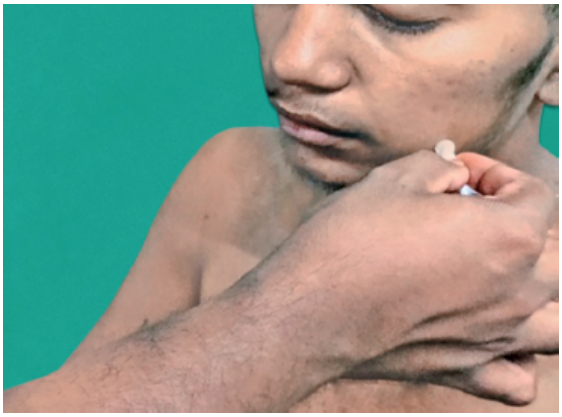


Fig. 6D(iii).40: Examination of sensory component of trigeminal nerve.

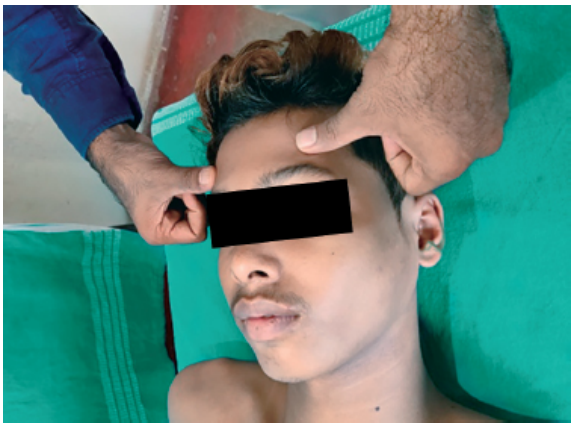


Fig. 6D(iii).41: Examination of ophthalmic division of trigeminal nerve.

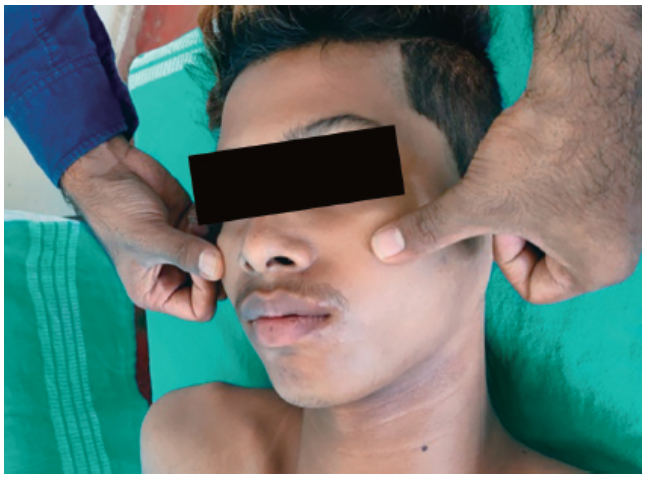


Fig. 6D(iii).42: Examination of maxillary division of trigeminal nerve.

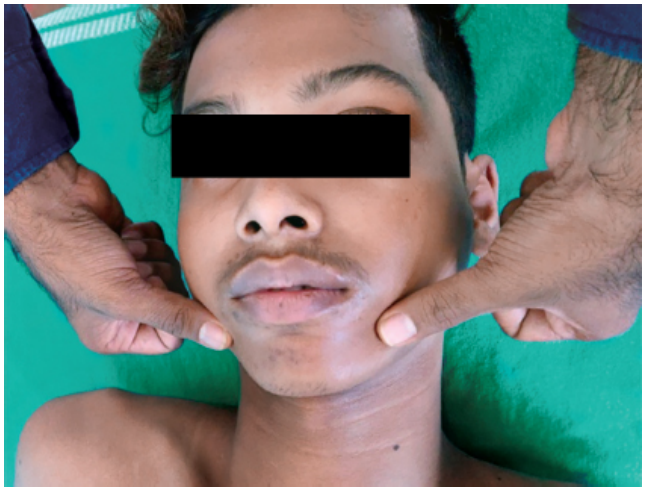


Fig. 6D(iii).43: Examination of mandibular division of trigeminal nerve.

Testing of motor component [Figs. 6D(iii).44 and 6D(iii).45]:

- Motor component can be gauged by palpating these muscles as the patient clinches the jaw. An effective technique is to place the examining fingers along the anterior or lateral border of the masseters bilaterally.
- When the jaw is clenched, the fingers will move forward (when fingers placed anteriorly) or sideward (when fingers placed laterally); this movement should be symmetric on the two sides.
- Unilateral trigeminal motor weakness causes deviation of the jaw toward the weak side on opening, due to the unopposed action of the contralateral lateral pterygoid. Careful observation of jaw opening is often the earliest clue to the presence of an abnormality.
- It is occasionally difficult to be certain whether the jaw is deviating or not. Note the relationship of the midline notch between the upper and lower incisor teeth; it is a reliable indicator.

Unilateral weakness of CN V innervated muscles	Bilateral weakness of the muscles of mastication with inability to close the mouth (dangling jaw)
Suggests: • The brainstem • Gasserian ganglion • The motor root of CN V at the base of the skull	**Suggests:** • Motor neuron disease • Neuromuscular transmission disorder • Myopathy

Fig. 6D(iii).44: Examination of motor component of trigeminal nerve (masseter muscle).

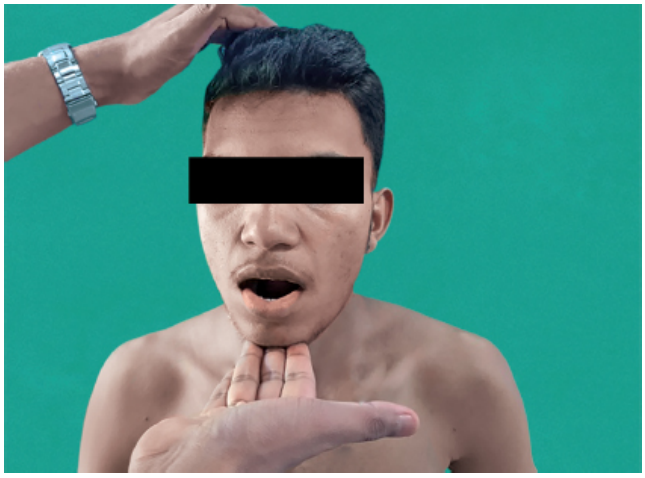


Fig. 6d(iii).45: Examination of motor component of trigeminal nerve (pterygoid muscle).

Rule of 17 (10 + 7 and 12 + 5)
• **10 + 7** → In facial nerve weakness and vagus nerve involvement, the deviation will be toward the normal side • The levator anguli oris (in CN 7) and palatopharyngeus (in CN 10) are 'pulling' muscles. Hence, the normal side 'pulls' the angle of mouth/uvula toward the normal side
• **12 + 5** → In trigeminal nerve and hypoglossal nerve weakness, the deviation will be toward the affected side • The lateral pterygoid (CN 5) and the genioglossus (CN 12) are 'pushing' muscles. Hence, the normal side 'pushes' the angle of jaw/tongue toward the affected side

Reflexes

Reflexes associated with V nerve:

1. Jaw jerk **[Fig. 6D(iii).46]**
2. Sternutatory reflex
3. Corneal reflex
4. Conjunctival reflex

Jaw jerk or Masseter or Mandibular reflex

Theory: Sensory fibers → mesencephalic nucleus → reflex center in pons → motor nucleus → motor fibers

Normal	Minimal or absent response
Limb hyperreflexia due to cervical spinal lesion	Normal jaw reflex
Generalized hyperreflexia	Exaggerated jaw reflex

Note: Exaggerated reflex is due to lesion in the bilateral corticobulbar tracts above motor nucleus, e.g. pseudobulbar palsy or amyotrophic lateral sclerosis.

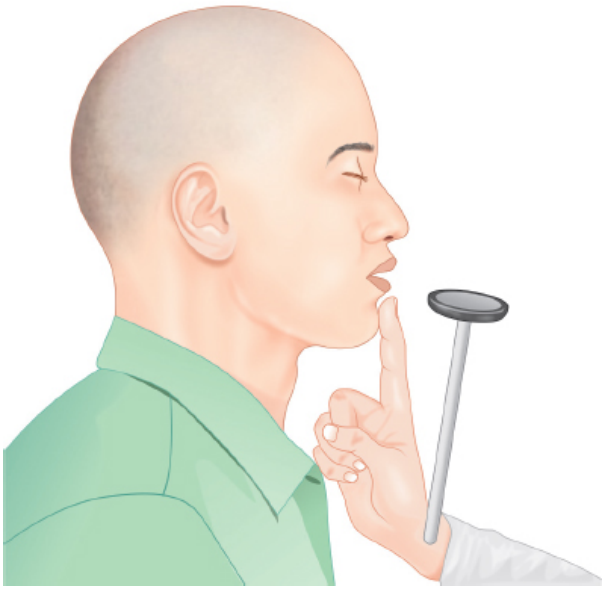


Fig. 6D(iii).46: Illustration showing examination of jaw jerk.

Testing [Fig. 6D(iii).47]:

- Examiner places the index finger or thumb over the middle of patient's chin, holding the mouth open about midway with jaw relaxed and then taps the finger with reflex hammer.
- The response is upward jerk of mandible.

Other methods:

- For bilateral response:
  - Tapping chin directly
  - Placing the tongue blade over the tongue or lower incisor and tapping the protruding end.
- For unilateral response:
  - Tapping the angle of the jaw
  - Placing the tongue blade over the lower molar teeth of one side and tapping the protruding end.

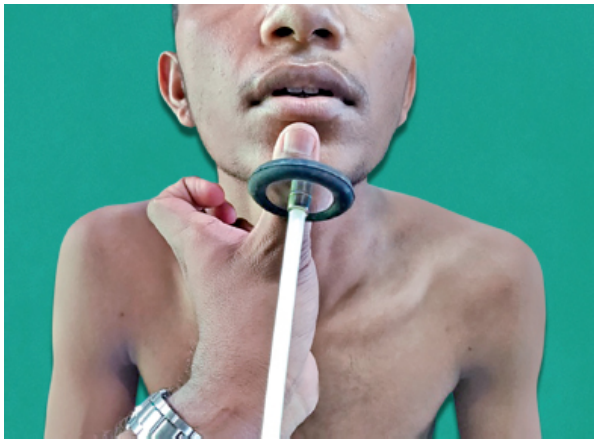


Fig. 6D(iii).47: Examination of jaw jerk.

Sternutatory/Nasal/Sneeze Reflex

Primary clinical use is to cross check the corneal reflex.

Method: Stimulation of nasal mucous membrane with cotton, a spear of tissue or similar object → wrinkling of nose, eye closure, and often a forceful exhalation resembling a feeble sneeze.

Theory: The ophthalmic division of trigeminal innervates the nasal septum and anterior nasal passages.

Afferent limb	Center	Efferent limb
V1	Brainstem and upper spinal cord	V VII IX X

Corneal Reflex

- Elicited by lightly touching the cornea with wisp of cotton or tissue **[Fig. 6D(iii).48]**.
- Stimulus is ideally delivered to upper cornea because the lower cornea may be innervated by CN V2 in some individuals.
- Stimulus should be ideally brought in from the side so that patient cannot see it.
- Stimulus must be delivered to cornea but not sclera

Afferent limb	Efferent limb
V1	VII

Conjunctival Reflex

- Same as corneal reflex **[Fig. 6D(iii).48]**
- However, the sensitivity of corneal reflex is more.

Trigeminal lesion (complete)		
	Direct reflex	***Consensual (indirect) reflex***

Stimulus to involved eye	Absent	Absent
Stimulus to opposite eye	Present	Present
Facial nerve lesion (complete)		
	Direct reflex	***Consensual (indirect) reflex***
Stimulus to involved eye	Absent	Present
Stimulus to opposite side	Present	Absent

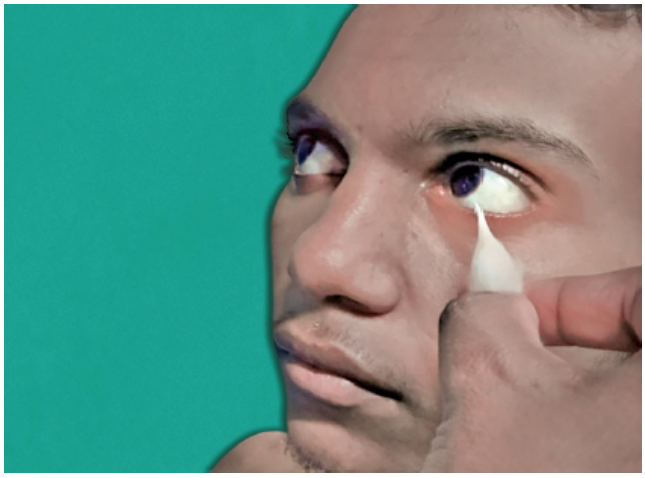


Fig. 6D(iii).48: Demonstration of corneal/conjunctival reflex.

Disorders of V Nerve Dysfunction

1. Motor Dysfunction

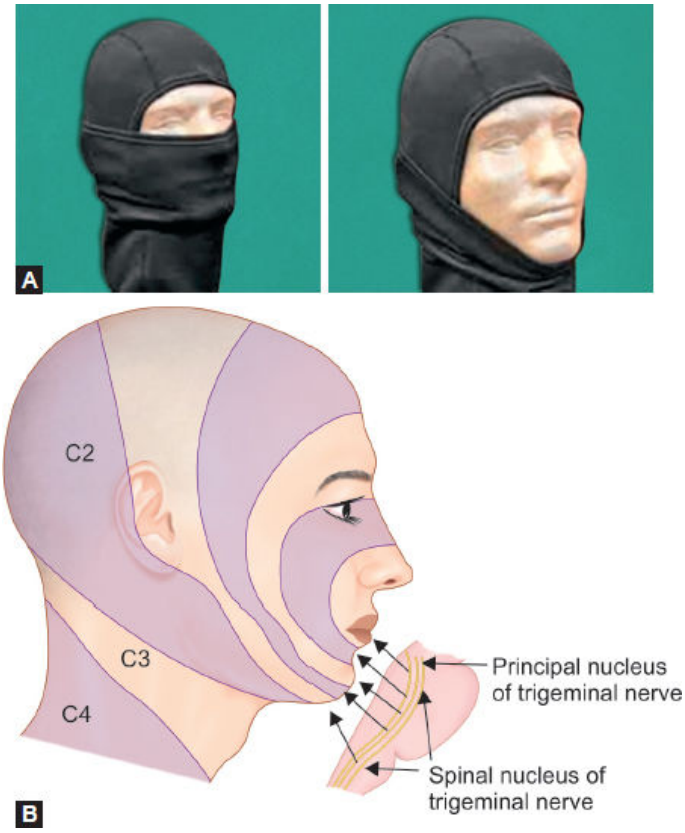
- Unilateral UMN lesion—generally no weakness observed.
- Bilateral UMN lesion—pseudobulbar palsy—marked weakness seen with exaggerated jaw jerk.
- Myasthenia gravis—masticatory fatigue (not to be confused with claudication pain of giant cell arteritis)
- ALS: Jaw drop with diminished jaw jerk—dysphagia and difficulty in swallowing their own saliva.
- Involuntary movements include—dystonia (extrapyramidal symptoms of antipsychotic drugs), Meige syndrome (oromandibular dystonia with blepharospasm), and trismus.

Causes of trigeminal nerve involvement
• Supranuclear—bilateral (pseudobulbar) palsy • Nuclear—syringobulbia • Nerve root—cerebellopontine angle tumor • Gasserian ganglion—Gradenigo syndrome, otitis media, meningitis, and aneurysms of internal carotid artery • Cavernous sinus—thrombosis/tumor • Superior orbital fissure—Tolosa–Hunt

- Individual branches involvement

2. Sensory Dysfunction

Site of lesion	Disease	Manifestation
Parietal lobe or sensory radiation (supranuclear lesion)	Stroke/tumors	May raise the sensory threshold of contralateral face
Thalamic lesion	Stroke/tumors	Facial hypoesthesia with hyperpathia or allodynia
Principal sensory nucleus: • **Pressure** • **Touch** • **Vibration**	Stroke/tumors	Diminished tactile sensation of skin and mucous membrane of that side
Spinal nucleus	Lateral medullary or pontine lesion/tumors	Pain and temperature loss
Intramedullary lesion	Syringomyelia/syringobulbia/tumors	Dissociative loss of sensation



Figs. 6D(iii).49A and B: (A) Balaclava helmet and (B) Dejerine onion skin distribution seen in syringobulbia.

Trigeminal Neuralgia (Also known as Fothergill's disease Tic douloureux)

- Most common disorder to involve trigeminal sensory function.

- Paroxysms of fleeting but excruciation unilateral facial pain—usually involves II and III division and rarely I division.
- Pain lasts for few seconds but may occur many times per day.
- Trigger for pain may be talking, chewing, brushing, exposure to cold or by wind on face.
- Most common cause for compression of sensory root by ecstatic arterial loop of the basilar artery (AICA or superior cerebellar artery)
- Other causes include MS, tumors of CP angle—bilateral is suggestive of MS.

3. Postherpetic Neuralgia

Acute herpes zoster is extremely painful.

- Usually in CN V1—pain in vesicles in forehead, eyelid, and cornea but may affect other division also.
- Persistent neuralgic pain syndrome after 1 month of acute eruption is appropriately labeled as postherpetic neuralgia. It is a dysesthetic with burning component, constant but with superimposed paroxysm of lancinating pain that may be provoked by touching certain spots with affected area.
- There may be hypo- or hyperesthesia.

4. Facial Numbness

- *Numb chin syndrome:* In distribution of mental nerve—due to metastatic process in mental foramen.
- *Numb cheek syndrome:* Involvement of infraorbital nerve.

5. Other Trigeminal Nerve Disorders

Marcus-gunn phenomenon or jaw winking phenomenon	Seen in congenital ptosis: Opening the mouth, chewing or lateral jaw movements cause an exaggerated reflex elevation of the ptotic lid due to proprioceptive impulses form the pterygoid muscles being misdirected to the oculomotor nucleus
Reversed Gunn phenomenon or inverse jaw winking or Marin-Amat sign	Synkinesis due to aberrant regeneration of facial nerve where there is involuntary closure of one eye on mouth opening
Frey syndrome	Flushing, warmness, and excessive perspiration over the cheek and pinna on one side following ingestion of spicy food—due to misdirection of secretory fibers to parotid gland to the sweat glands and vasodilator ending in the auriculotemporal nerve distribution—usually follows trauma or infection of parotid gland or local nerve injury
Sturge–Weber or Weber–Dimitri disease	Congenital nevi or angiomas over the side of face in the trigeminal distribution with associated ipsilateral leptomeningeal angiomas and intracortical calcification with attendant neurologic complications
Raeder's paratrigeminal syndrome	• Unilateral oculosympathetic paresis (differential diagnosis with Horner) • Ipsilateral trigeminal involvement
Gradenigo's syndrome	• Damage to V1 division of trigeminal nerve • Ipsilateral 6th nerve palsy
Cavernous sinus syndrome	3, 4, 6 nerves with V1 and V2 (less often)

Superior orbital fissure syndrome	Never involving V2, other than that similar to cavernous sinus syndrome. Exophthalmos and blindness can be present
V1: Bilateral corneal anesthesia	Diabetic neuropathy
V2: Numb cheek syndrome	• Infraorbital nerve • Distribution: Squamous cell carcinoma, skin and LASIK
V2: Trumpet player's neuropathy	Anterior superior alveolar nerve
V3: Tongue numbness	• Lingual nerve in temporal • Arteritis
V3: Numb chin syndrome/roger's sign	Mental neuropathy: Cancer of breast and lung, giant cell arteritis, Burkitt lymphoma, and sickle cell disease

FACIAL NERVE

Motor (70%)	Sensory	Parasympathetic
• Muscles of facial expression • Scalp • Ear • Buccinators • Platysma • Stapedius • Stylohyoid • Posterior belly of digastrics	**Taste:** Anterior 2/3 **Exteroceptive:** • Eardrum • EAC **Proprioception:** From the muscles supplied by it **GVS:** • Salivary glands • Mucosa of nose and pharynx	• Submandibular • Sublingual • Lacrimal • Mucous membrane of oral and nasal mucosa

(EAC: external auditory canal)

Note:

- There is anatomical segregation of motor component from sensory and autonomic fibers.
- Sensory root (nervus intermedius of Wrisberg)—contains both sensory and autonomic fibers.

Examination of Motor Function

Inspection: • Facial asymmetry, nasolabial fold with forehead wrinkles, and movements during spontaneous facial expression • Tone of the muscles of facial expression • Atrophy and fasciculations • Abnormal muscle contractions and involuntary movements • Spontaneous blinking for frequency and symmetry
Testing the temporal branches of the facial nerve: Patient is asked to frown and wrinkle his or her forehead
Testing the zygomatic branches of the facial nerve: Patient is asked to close their eyes tightly

Testing the buccal branches of the facial nerve:

- Puff up cheeks (buccinator)
- Smile and show teeth (orbicularis oris)
- Tap with finger over each cheek to detect ease of air expulsion on the affected side

Muscle tested	Instruction	Response in palsy
Frontal belly of occipitofrontalis (Fig. 6D(iii).50)	Ask the patient to wrinkle his/her forehead	Asymmetry as he/she cannot wrinkle his forehead on the side of palsy in lower motor neuron (LMN) palsy
Orbicularis oculi [Fig. 6D(iii).51]	Ask the patient to close his/her eyes forcibly while you try to open the eyelids with your fingers	In LMN palsy, eyelids do not close completely. Instead the eyeball rolls up. This is known as Bell's phenomenon. In healthy individuals, eyelids cannot be opened with mild force against patient's resistance
Levator anguli oris, zygomatic major and minor, depressor anguli oris, buccinator, and risorius [Fig. 6D(iii).52]	Ask the patient to show his/her teeth or smile	Angle of mouth deviates toward normal side
Orbicularis oris and buccinators [Fig. 6D(iii).53]	Ask the patient to blowout cheeks with mouth closed, i.e. puff the cheeks and assess power by your attempt to deflate the cheek. Ask the patient to whistle	Patient cannot blowout his cheek as air escapes from affected side
Platysma [Fig. 6D(iii).54]	Ask the patient to clench his/her teeth and simultaneously depress the angles of mouth	Folds of platysma is seen in the neck as flat

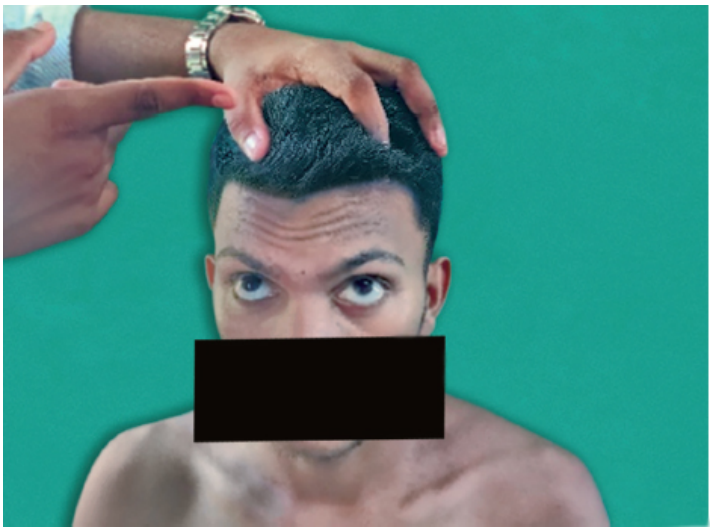


Fig. 6D(iii).50: Examination of frontal belly of occipitofrontalis.

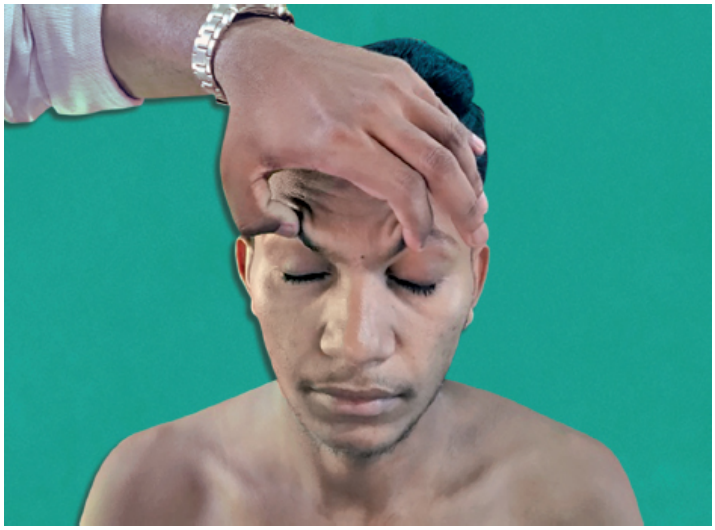


Fig. 6D(iii).51: Examination of orbicularis oculi.

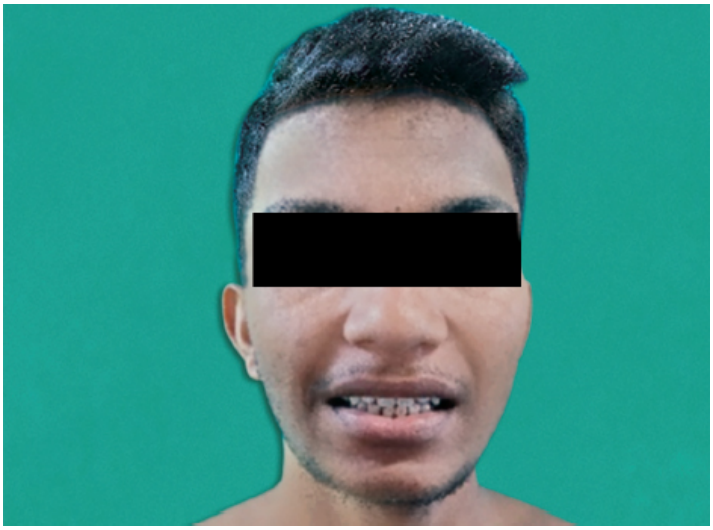


Fig. 6D(iii).52: Examination of levator anguli oris.

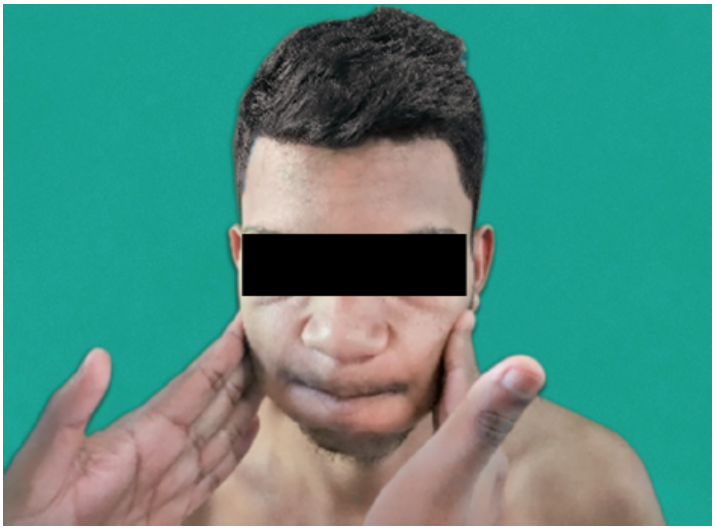


Fig. 6D(iii).53: Examination of buccinator.

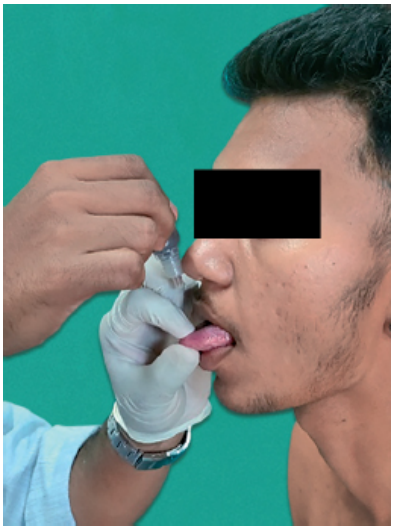


Fig. 6D(iii).55: Examination of taste sensation.

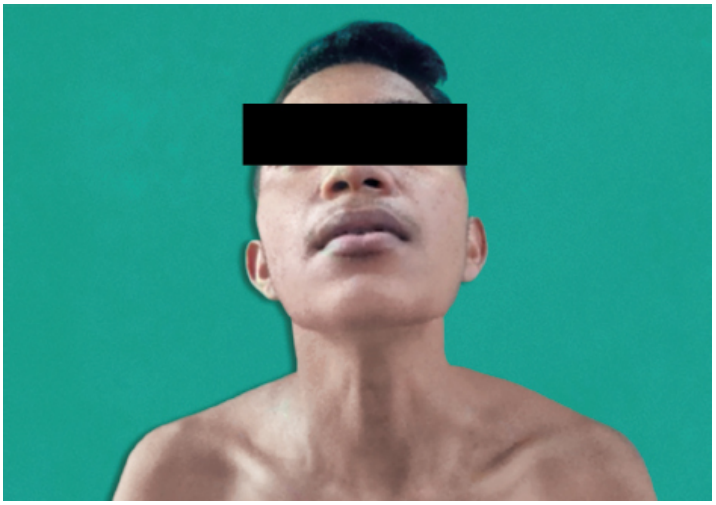


Fig. 6D(iii).54: Examination of platysma.

Examination of Sensory System

Anterior two-thirds of tongue [Fig. 6D(iii).55]

- Tongue protruded
- Hold with soft gauze
- With applicator's tip apply over the dorsum of the tongue
- Rinse after each test with water
- Sensations from the tip to deep—follow sweet → salt → sour → bitter (last)
- Fifth modality—umami appreciated with compounds of some amino acids
- Normally taste is appreciated within 10 seconds
- Artificial sweeteners make better test substances than ordinary sugar.

Aguesia	Complete inability to perceive taste

Hypogeusia	Blunted or delayed taste
Parageusia	Perversions of taste
Impaired taste	Lesion is proximal to junction with chorda tympani
Not affected	Lesion is at or distal to stylomastoid foramen

Secretory Function

1. Lacrimation: Schirmer's test→10 mm is normal
2. Nasolacrimal test: By diluted solution of ammonium and formaldehyde—trigeminal nerve → greater superficial petrosal nerve.

Reflexes
Orbicularis oculi reflex
Percussion causes reflex contraction of the eye muscle. The reflex is known as the supraorbital, glabellar, or nasopalpebral reflex, depending upon the site of the stimulus. Both eyes usually close, with the contralateral response being weaker. The trigeminal nerve is the afferent side and the facial nerve the efferent side of the reflex. Light and sound can also produce the reflex, with the optic and acoustic nerves providing the afferent side
The response is weak or abolished in nuclear and peripheral lesions, and present or exaggerated in supranuclear lesions. It is exaggerated in Parkinsonism and cannot be voluntarily inhibited
Palpebral oculogyric reflex
The eyeballs deviate upward when the eyes are closed, both when awake and asleep. The afferent arc is proprioceptive impulses carried through the facial nerve to the medial longitudinal fasciculus. The oculomotor nerve to the superior rectus muscles forms the efferent side
In peripheral and nuclear lesions, an exaggeration of this reflex is known as **Bell's phenomenon**
Orbicularis oris reflex
Percussion on the side of the nose or the upper lip causes ipsilateral elevation of the angle of the mouth and upper lip. The reflex arc is composed of the fifth and seventh nerves. *Synonyms:* nasomental, buccal, oral, or perioral reflex
This reflex disappears after about the first year of life, recurring with supranuclear facial nerve lesions and with extrapyramidal diseases, such as Parkinsonism
Snout reflex
Tapping the upper lip lightly with a reflex hammer, tongue blade, or finger causes bilateral contraction of the muscles around the mouth and base of the nose. The mouth resembles a snout
This is an exaggeration of the orbicularis oris reflex. It is present with bilateral supranuclear lesions and in diffuse cerebral diseases, such as various causes of dementia
Sucking reflex
Sucking movements of lips, tongue, and mouth are brought about by lightly touching or tapping on the lips. At times, merely bringing an object near the lips produces the reflex
Occurs in patients with diffuse cerebral lesions. The snout reflex occurs in similar circumstances
Palmomental reflex
A stimulus of the thenar area of the hand causes a reflex contraction ipsilaterally of the orbicularis oris and mentalis muscles

A number of normal individuals have this reflex, and also patients with diffuse cerebral disease. It is significant when other similar reflexes are also present
Corneal reflex
Stimulation of the cornea with a wisp of cotton produces reflex closure of both ipsilateral (strongest) and contralateral eyelids. The fifth nerve carries the afferent impulses, and the facial nerve the efferent impulses

Site of cranial nerve 7 lesion and associated manifestatio n

Lesion location	Manifestations
Above the facial nucleus (supranuclear lesion)	Contralateral paralysis of lower facial muscles with relative preservation of upper muscles. Lesion located cortex, internal capsule or midbrain
Pons (nuclear or fascicular lesion)	Ventral pontine lesion (of Millard–Gubler): Ipsilateral facial monoplegia, lateral rectus palsy (VI), and contralateral hemiplegia (corticospinal fibers). Pontine tegmentum lesion (of Foville): Ipsilateral facial monoplegia; contralateral hemiplegia (corticospinal fibers); paralysis of conjugate gaze to side of lesion (pontine paramedian reticular formation)
Cerebellopontine angle (peripheral nerve lesion)	Ipsilateral facial monoplegia, loss of taste to anterior two-thirds of tongue, impairment of salivary and tear secretion, hyperacusis (if VIII is not affected). Additional cranial nerves may be involved: deafness, tinnitus, and vertigo (VIII): sensory loss over face and absence of corneal reflex (V); ipsilateral ataxia (cerebellar peduncle)
Facial canal between internal auditory meatus and geniculate ganglion (peripheral nerve type lesion here and subsequently)	Same as above except cranial nerves other than VII are not involved
Facial canal between geniculate ganglion and nerve to stapedius muscle	Facial monoplegia; impaired salivary secretion; loss of taste; and hyperacusis
Facial canal between nerve to stapedius and leaving of chorda tympani	Facial monoplegia; impaired salivary secretion; and loss of taste
After branching of chorda tympani	Facial paralysis, distribution related to site of lesion

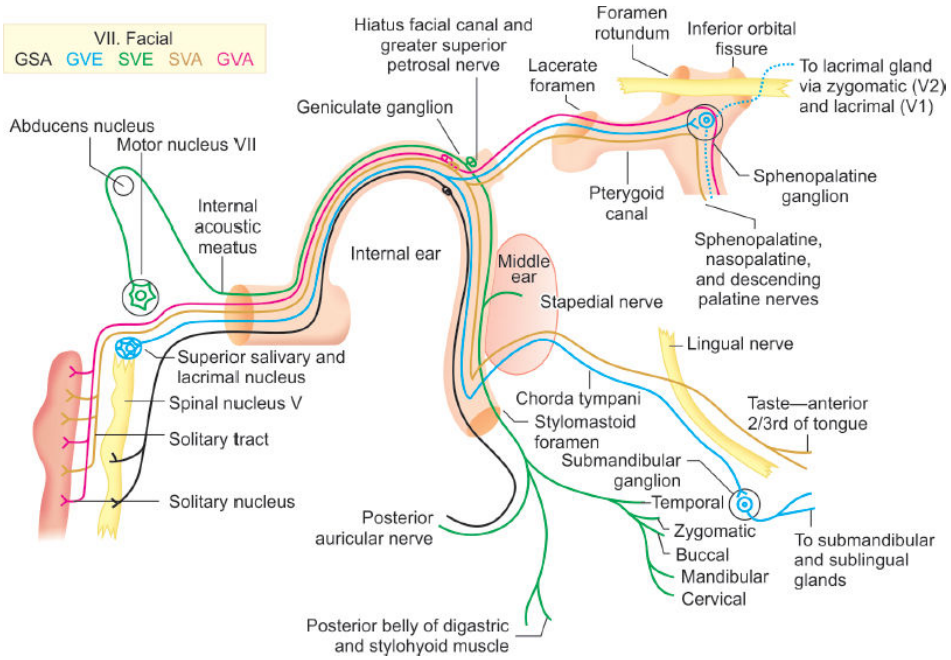


Fig. 6D(iii).56: Facial nerve pathway.

FACIAL NERVE PALSY

Peripheral Facial Palsy

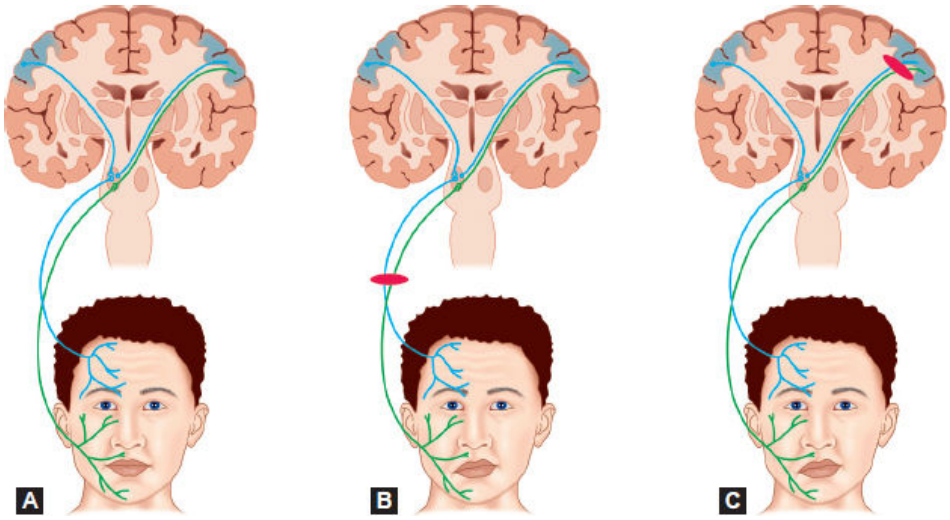
There is flaccid weakness of all the muscles of facial expression on the involved side, both upper and lower face, and the paralysis is usually complete.

Signs in Lmn Facial Palsy

Bell's phenomenon	Attempting to close involved eye causes a reflex upturning of the eyeball
Levator sign of Dutemps and Céstan	Patient look down, then close the eyes slowly; because the function of levator palpebrae superioris is no longer counteracted by orbicularis oculi, upper lid on the paralyzed side moves upward slightly
Negro's sign	Eyeball on the paralyzed side deviates outward and elevates more than the normal one when the patient raises her eyes
Bergara-Wartenberg sign	Loss of the fine vibrations palpable with the thumbs or fingertips resting lightly on the lids as the patient tries to close the eyes as tightly as possible
Platysma sign of Babinski	Asymmetric contraction of the platysma, less on the involved side, when the mouth is opened

House–Brackmann grading system of LMN facial palsy	
Grade I	Normal
Grade II	Mild dysfunction, slight weakness on close inspection, and normal symmetry at rest

Grade III	Moderate dysfunction, obvious but not disfiguring difference between sides, eye can be completely closed with effort
Grade IV	Moderately severe, normal tone at rest, obvious weakness or asymmetry with movement, incomplete closure of eye
Grade V	Severe dysfunction, only barely perceptible motion, and asymmetry at rest
Grade VI	No movement



Figs. 6D(iii).57A to C: Innervation by facial nerve.

Causes of LMN Facial Palsy

Congenital:
- Möbius syndrome
- Goldenhar syndrome
- Melkersson–Rosenthal syndrome

Birth related: Forceps delivery

Idiopathic: Bell's palsy

Infection:
- Viral infection, i.e. varicella zoster (Ramsay Hunt), herpes zoster, herpes simplex, and HIV
- Otitis media
- Cholesteatoma
- Necrotizing otitis externa
- Skull base osteomyelitis
- Lyme disease
- Leprosy

Trauma:
- Temporal bone fracture
- Gunshot or penetrating injury
- Laceration

Neoplastic:
- Schwannoma
- Meningioma
- Hemangioma
- Parotid malignancy

Iatrogenic: Brain, middle ear, mastoid, parotid or facial surgery

Neurological:
- Lacunar or brainstem infarct
- Guillain–Barré syndrome
- Myasthenia gravis
- Multiple sclerosis

Metabolic:
- Diabetes mellitus
- Hypertension
- Pregnancy
- Vitamin A deficiency

Central Facial Nerve Palsy (UMN Facial Nerve Palsy)

Facial weakness of central origin/UMN facial palsy	
• Weakness of the lower face, with relative sparing of upper face • Upper face is not necessarily completely spared, but it is always involved to a lesser degree than the lower face	
Volitional or voluntary	***Emotional or mimetic***
Lesion of the cortical center in the lower third of the precentral gyrus that controls facial movements, or the corticobulbar tract	Thalamic or striatocapsular lesions, usually infarction
Weakness more marked on voluntary contraction, when patient is asked to smile or bare her teeth	Facial asymmetry more apparent with spontaneous expression, as when laughing

Differences between UMN and LMN type of facial nerve palsy

	UMN type	LMN type
Facial motor function	Wrinkling of forehead preserved (frontalis unaffected)	Total face is involved
Bell's phenomenon [Fig. 6D(iii).60A to C]	Absent	Present
Facial muscles	Not atrophied	Fasciculations, Atrophied
Taste sensation	Preserved	May be lost

Corneal reflex	Preserved	Lost
Hemiplegia	Contralateral	Ipsilateral
Babinski reflex	Present	Absent

(UMN: upper motor neuron; LMN: lower motor neuron)

Fig. 6D(iii).58: Image showing deviation of angle of mouth.

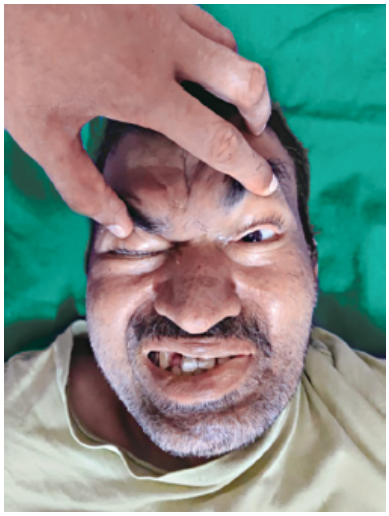


Fig. 6D(iii).59: Weakness of orbicularis oculi.

Bilateral VII Nerve Palsy

Bilateral UMN palsy	Bilateral LMN palsy
• Emotional fibers—spared • Emotional incontinence—present • Associated with bilateral long	• Bell's phenomenon present • Emotional fibers—affected • Long tract signs—absent

• tract signs • Jaw jerk—exaggerated • Corneal reflex—present • Taste sensation—spared • Gag reflex—exaggerated	• Jaw jerk—normal • Corneal reflex—absent • Taste sensation—absent

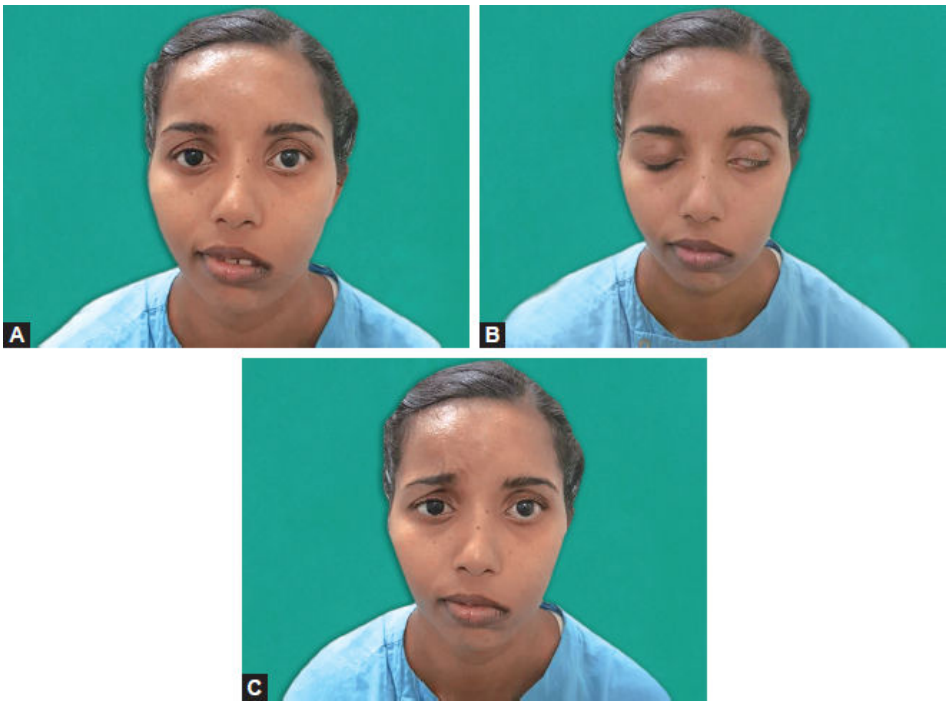
(UMN: upper motor neuron; LMN: lower motor neuron)

- **Causes of bilateral facial nerve palsy:**
- Diabetes
- Bilateral Bell's palsy
- Borreliosis
- *Mycoplasma pneumoniae* infection
- Guillain-Barré syndrome* and Miller–Fisher syndrome
- Sarcoidosis
- Möbius syndrome
- Leukemia
- Viral infections (Herpes simplex)
- Syphilis
- Basal skull fractures
- Pontine gliomas
- Leprosy
- Mononucleosis
- Brainstem encephalitis
- Hansen's disease
- Cryptococcal meningitis
- Pontine tegmental hemorrhage

*Most common cause

Syndromes of Facial Palsy

Syndromes with facial nerve palsy
• Foville's syndrome • Millard–gubler syndrome • Möbius syndrome • Ramsay hunt syndrome • Melkersson-rosenthal syndrome [triad of recurrent infranuclear facial paralysis, orofacial edema (predominately of the lips), and lingua plicata] • Guillain–barré syndrome • Progressive hemifacial atrophy (parry–romberg syndrome) • Meige syndrome (blepharospasm oromandibular dystonia, orofacial cervical dystonia, and brueghel's syndrome) • Uveoparotid fever (heerfordt's disease) • Goldenhar syndrome • Crocodile tear syndrome • Frey's syndrome



Figs. 6D(iii).60A to C: Bell's phenomenon.

CRANIAL NERVE VIII—VESTIBULOCOCHLEAR NERVE

Contains two components	
Vestibular component	***Cochlear component***
↓	↓
Responsible for equilibrium	Responsible for hearing
Pathway	
For linear accelerations Macula Utricle Saccule **For angular acceleration** Ampulla	Organ of corti
	↓
	Cochlear nuclei
	↓
	Inferior colliculus
	↓
	Lateral lemnisci
↓	↓
Vestibular ganglia	Medial geniculate body
↓	↓

Vestibular nerve	Brodmann areas 41 and 42 (transverse temporal gyrus of Heschl)

Examination	
Vestibular component	***Cochlear component***
Rotational test	Rubbing fingers
Calorie test **(Fig. 6D(iii).61)**	Rinne's test and Weber's test
Electronystagmography	Audiometric tests: • Pure tone audiometry • Tone decay • Bekesy audiometry

Testing for vertigo and nystagmus	
In sitting position, turn the head to one side by 45°	
↓	
Make the patient to lie down abruptly with the head handing down from the edge of cot	
↓	
This position is maintained for at least a minute	
↓	
Watch for nystagmus	
↓	
Fast component is toward the lower ear suggests following possibilities	
↓	↓
Benign paroxysmal positional vertigo	***Central cause***
Starts after short latency (3–10 sec), patient will have nystagmus associated with vertigo	Immediate nystagmus
Rapid adaptation	No adaptation

Testing the vestibular component of VIII nerve
Rotational test
Patient is seated in a chair that can be rotated with his head well supported and fixed in head rest
↓
To test Horizontal canal—head in flexed at 30° Vertical canal—head is flexed at 120°
↓
Chair is rotated 10 times in 20 seconds
↓
Normally when the rotation to the right has stopped, there is nystagmus with its slow phase to the right and vice

versa

Calorie test
The patient is placed supine with the head tilted up by 30°. In this way, the horizontal semicircular canal is oriented in a vertical plane
↓
250 mL of water (or air at controlled temperature) is irrigated through the external auditory meatus over period of 40 seconds, first using 30°C and later using 44°C
↓
Patient fixes his eyes on the given point immediately above his head
↓
After ceasing the irrigation, the time in seconds is measured during which nystagmus on the forward gaze persist
↓
Now the test is repeated on the other ear
↓
Normal response is cold water produces fast component toward the opposite side and warm water produces a fast component toward the same side (mnemonic—**COWS**)

Interpretation	
No response (canal paresis)	• Meniere's disease • Acoustic nerve tumor • Vestibular neuronitis • Lesions of vestibular nuclei
Directional preponderance	• Lesions of peripheral or central vestibular apparatus • Cerebellum • Corticofugal fibers deep in the temporal lobe
Combination of above two	Vestibular nerve or labyrinth lesions

Testing the Cochlear Component of VIII Nerve

Rinne's and Weber's test [Figs. 6D(iii).62 to 6D(iii).65]

- Done with 256/512 Hz tuning fork
- The prongs should be put equidistant on either ears while examining
- Examination should be done in quite room

Rinne's test	Weber test
By two methods: 1. An activated fork may be place first on the mastoid process, then immediately beside the ear and patient asked which is louder 2. Traditional method where—place the tuning fork on the mastoid and when no longer heard there move it beside the ear, where it should still be audible	A vibrating tuning fork is place in the midline on the vertex of the skull. Normally the sound is heard equally in both ears

Interpretation	
In conductive hearing loss	
BC > AC (Rinne negative)	Lateralized to abnormal side
In sensorineural hearing loss	
AC > BC (Rinne positive)	Lateralized to normal side

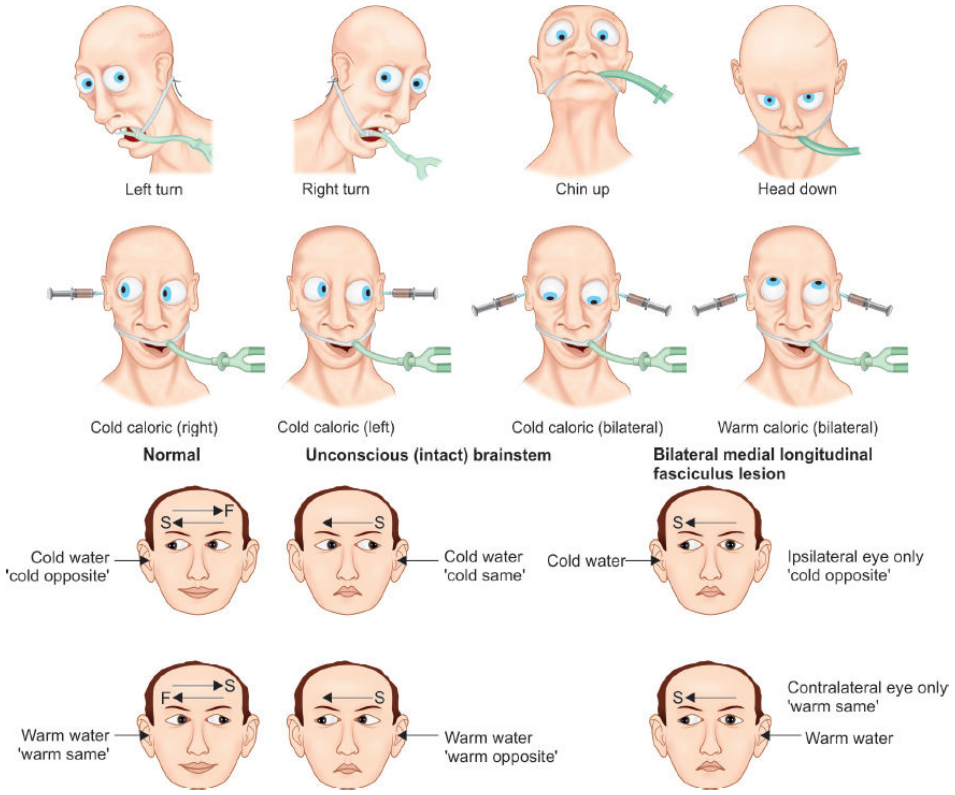


Fig. 6D(iii).61: Illustration demonstrating calorie test.

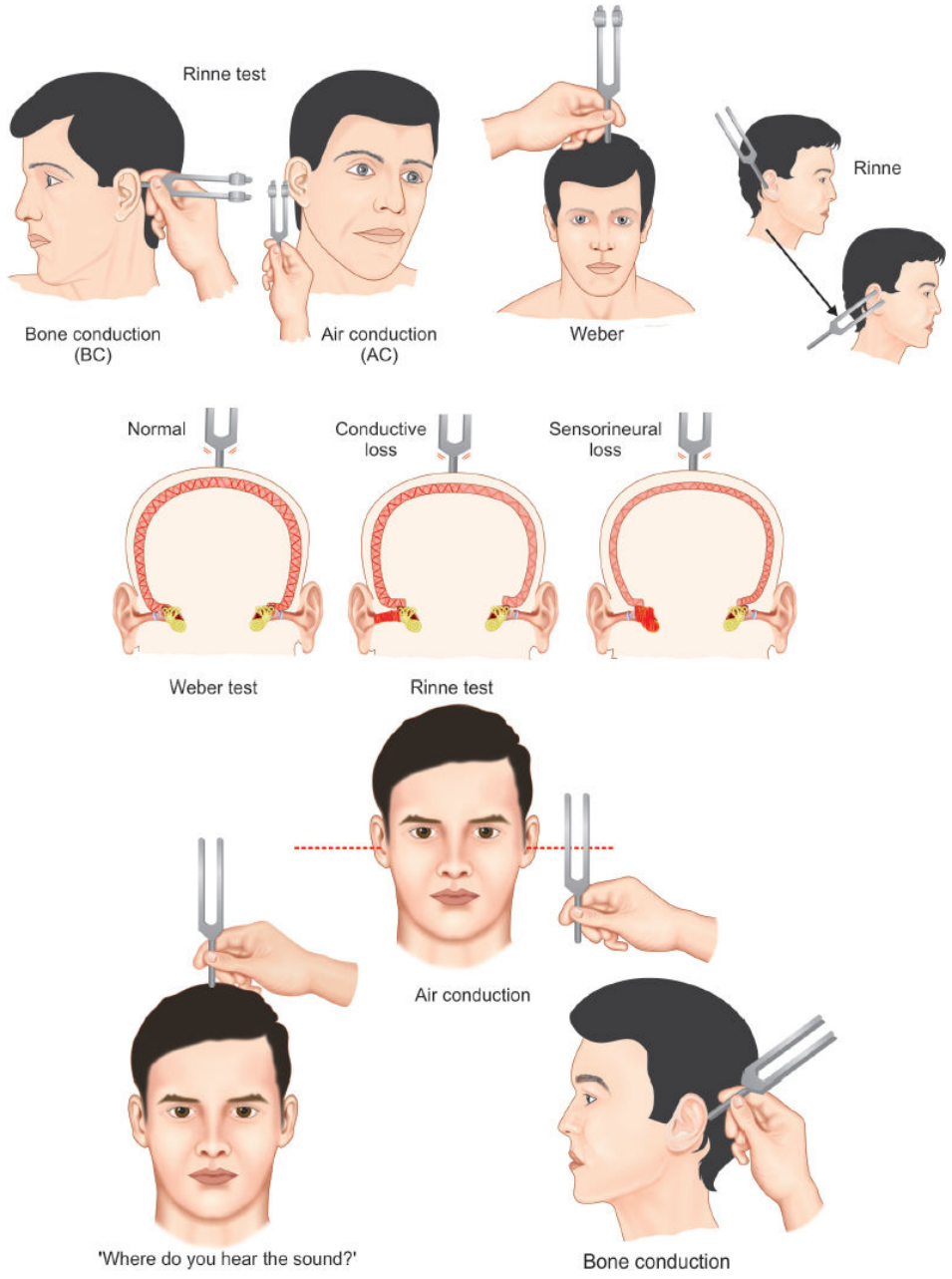


Fig. 6D(iii).62: Illustration showing demonstration of Rinne's test and Weber's test.

Fig. 6D(iii).63: Rinne's test: Placement of tuning fork on the mastoid process.

Fig. 6D(iii).64: Rinne's test: Placement of tuning fork beside the ear parallel to tympanic membrane.

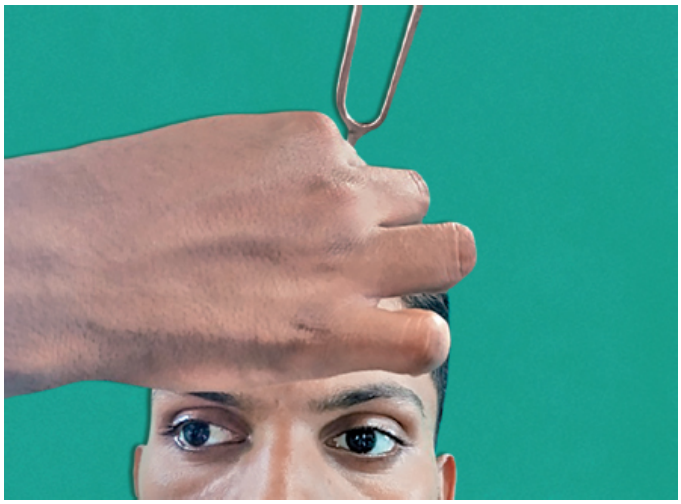


Fig. 6D(iii).65: Weber's test: Placement of tuning fork in midline on the vertex.

Causes of VIII Nerve Dysfunction Based on Site of Involvement

Vestibular component	Cochlear component
At level of labyrinth: • Meniere's disease • Motion sickness • Drug toxicity • Migraine **Vestibular nerve:** Vestibular neuronitis **Brainstem:** • Vascular insufficiency • Cerebellar tumors • IV ventricle tumors • Acute demyelinating diseases **Temporal lobe:** As epileptic manifestation	**Conduction defects:** • External meatus obstruction • Middle ear pathology • Eustachian tube block • Intracranial infection • Middle ear infection **Cochlear pathology:** • Meniere's disease • Osteosclerosis • Internal auditory meatus occlusion **Nerve trunk:** • Old age • Meningitis • Cerebellopontine angle tumors **Brainstem:** • Vascular pathology • Demyelination disease **Cerebrum:** Temporal disease

Unilateral and Bilateral Causes of VIII Nerve Dysfunction

Vestibular component		Cochlear component	
Unilateral	***Bilateral***	***Unilateral***	***Bilateral***
• Tumor (cerebellopontine angle and acoustic neuroma) • Fracture of the petrous temporal bone • Vascular disease of the internal auditory artery	• Industrial deafness • Presbycusis • Drug toxicity (gentamicin, salicylate, etc.) • Meniere's disease	• Tumor (cerebellopontine angle and acoustic neuroma) • Fracture of the petrous temporal bone • Vascular disease of the internal auditory artery	• Demyelinating illness, e.g. multiple sclerosis • Migraine

	• Brainstem lesion (e.g. stroke)	• Vestibular neuritis	

The "doll's eye" oculocephalic reflex

- Tests the vestibulocochlear nerve, the brainstem nuclei of the vestibulocochlear nerve, the fibers to the cerebellum, the fibers from the cerebellum, the medial longitudinal fasciculus (MLF), and the 3rd and 6th cranial nerves.
- The cause of the unconsciousness in a patient with a negative oculocephalic reflex is some sort of destructive brainstem pathology or brain death. Conversely, an intact oculocephalic reflex suggests that the coma is of a nonstructural cause, because much of the brainstem must be intact.

CRANIAL NERVE IX AND X—GLOSSOPHARYNGEAL AND VAGUS

The two nerves:

- Have motor and autonomic branches with nuclei of origin in the medulla.
- Both conduct general somatic afferent (GSA) as well as general visceral afferent (GVA) fibers to related or identical fiber tracts and nuclei in the brainstem.
- Both have a parasympathetic, or general visceral efferent, and a branchiomotor, or special visceral efferent (SVE), component
- Both leave the skull together
- Remain close in their course through the neck
- Both supply some of the same structures.
- They are often involved in the same disease processes
- Involvement of one may be difficult to differentiate from involvement of the other.

For these reasons, the two nerves are discussed together.

Muscles innervated by cranial nerve IX and X

IX nerve	
Muscular branch	Stylopharyngeus
X nerve	
Pharyngeal branch [Fig. 6D(iii).66]	• Musculus uvulae (azygos uvulae) • Levator veli palatini • Palatopharyngeus • Salpingopharyngeus • Palatoglossus • Superior, middle, and inferior • Constrictors of the pharynx
Superior laryngeal nerve	Cricothyroid
Recurrent laryngeal nerve	• Posterior cricoarytenoids • Lateral cricoarytenoids • Thyroarytenoids (vocalis) • Arytenoid

GLOSSOPHARYNGEAL NERVE IX

Functions:

Glossopharyngeal nerve: Sensory supply to posterior one-third of tongue, taste sensation, and pharyngeal mucosa.

Testing of IX Nerve

Cranial nerve IX is difficult to examine because most or all of its functions are shared by other nerves and because many of the structures it supplies are inaccessible.

Gag reflex [Fig. 6D(iii).67]

- The gag reflex is protective; it is designed to prevent noxious substances or foreign objects from going beyond the oral cavity.
- **Components of gag reflex:** There are three motor components: elevation of the soft palate to seal off the nasopharynx, closure of the glottis to protect the airway, and constriction of the pharynx to prevent entry of the substance.
- **Pathway:** The afferent limb of the reflex is mediated by CN IX and the efferent limb through CNs IX and X. The reflex center is in the medulla.
- **Testing of gag reflex:** The reflex is elicited by touching the lateral oropharynx in the region of the anterior faucial pillar with a tongue blade, applicator stick, or similar object (pharyngeal reflex), or by touching one side of the soft palate or uvula (palatal reflex). The reflex also occurs with touching the base of the tongue or posterior pharyngeal wall.
- **Clinical implication:** May be bilaterally absent in some normal individuals.

  Unilateral absence signifies a lower motor neuron lesion. Like most bulbar muscles, the pharynx receives bilateral supranuclear innervation, and a unilateral cerebral lesion does not cause detectable weakness. A hyperactive gag reflex may occur with bilateral cerebral lesions, as in pseudobulbar palsy and amyotrophic lateral sclerosis (ALS).

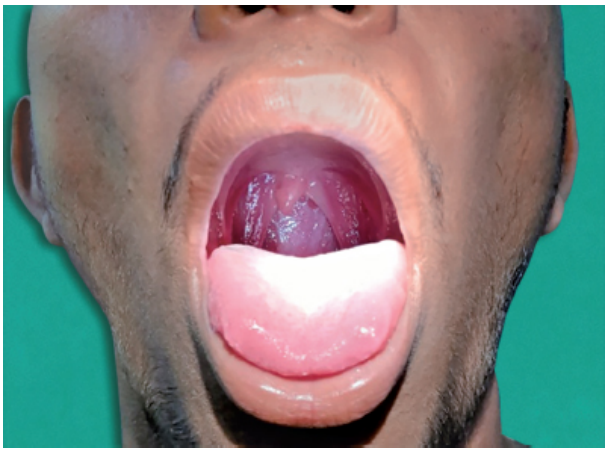


Fig. 6D(iii).66: Examination of deviation of uvula.

Fig. 6D(iii).67: Examination of gag reflex.

Disorders of IX Cranial Nerve

- **Unilateral supranuclear lesions** cause no deficit because of the bilateral corticobulbar innervation.
- **Bilateral supranuclear lesions** may cause pseudobulbar palsy.
- **Nuclear and infranuclear processes** that may affect CN IX include intramedullary and extramedullary neoplasms and other mass lesions (e.g. glomus jugulare tumor), trauma (e.g. basilar skull fracture or surgical dissection), motor neuron disease, syringobulbia, retropharyngeal abscess, demyelinating disease, birth injury, and brainstem ischemia.

The most important lesion of the ninth nerve is glossopharyngeal (or vagoglossopharyngeal) neuralgia or "tic douloureux of the ninth nerve". In this condition, the patient experiences attacks of severe lancinating pain originating in one side of the throat or tonsillar region and radiating along the course of the eustachian tube to the tympanic membrane, external auditory canal, behind the angle of the jaw, and adjacent portion of the ear. The pain may be brought on by talking, eating, swallowing, or coughing. It can lead to syncope, convulsions, and rarely to cardiac arrest because of stimulation of the carotid sinus reflex.

CRANIAL NERVE X—VAGUS

The vagus (in Latin means "wandering," because of its wide distribution) is the longest and most widely distributed.

The vagus emerges from the medulla as a series of rootlets just below those of the glossopharyngeal.

CN X leaves the skull through the jugular foramen in the same neural sheath as the cranial root of CN XI and behind CN IX. In the jugular foramen, the nerve lies close to the jugular bulb, a dilatation of the internal jugular vein that houses the glomus jugulare (tympanic body). The glomus jugulare has functions similar to the carotid body.

Branches of cranial nerves: There are 10 major terminal branches that arise at different levels: (a) meningeal, (b) auricular, (c) pharyngeal, (d) carotid, (e) superior laryngeal, (f) recurrent laryngeal, (g) cardiac, (h) esophageal, (i) pulmonary, and (j) gastrointestinal.

Motor: The vagus, with a contribution from the bulbar portion of CN XI, supplies all the striated muscles of the soft palate, pharynx, and larynx except for the stylopharyngeus (CN IX) and tensor veli palatini (CN V).

Parasympathetic: The vagus is the longest parasympathetic nerve in the body and a vagal discharge causes bradycardia, hypotension, bronchoconstriction, bronchorrhea, increased peristalsis, increased gastric secretion, and inhibition of adrenal function. The vagal centers in the medulla that control these functions are themselves under the control of higher centers in the cortex and hypothalamus. Inhibition of vagal function produces the opposite effects.

Sensory: Both vagal ganglia are sensory. The superior ganglion primarily conveys somatic sensation, and most of its communication is with the auricular nerve. The inferior ganglion relays general visceral sensation and taste.

Normal functions mediated by CNs IX and X include swallowing, phonation, and airway protection and modulation.

Examination

Motor function: The character of the voice and the ability to swallow provide information about the branchiomotor functions of the vagus.

Clinical implications:
A unilateral vagal lesion causes weakness of the soft palate, pharynx, and larynx. Acute lesions may produce difficulty swallowing both liquids and solids and hoarseness or a nasal quality to the voice. Sensory change is anesthesia of the larynx due to involvement of the superior laryngeal nerve. The gag reflex is absent on the involved side. Autonomic reflexes (vomiting, coughing, and sneezing) are not usually affected.

Bilateral complete vagal paralysis is incompatible with life. It causes complete paralysis of the palate, pharynx, and larynx, with marked dysphagia and dysarthria; tachycardia; slow, irregular, and respiration; vomiting; and gastrointestinal atonia.

Disorders of Cranial Nerve X

Unilateral supranuclear lesions generally cause no dysfunction because of bilateral innervation.

Bilateral supranuclear lesions, as from pseudobulbar palsy, cause dysphagia and dysarthria.

Extrapyramidal disorders may produce difficulty with swallowing and talking. Patients with Parkinson's disease typically have a hypokinetic dysarthria. Laryngeal spasm with stridor may occur in Parkinson's disease.

Nuclear lesions bulbar ALS, syringomyelia, and some neoplasms, may cause fasciculations in the palatal, pharyngeal, and laryngeal muscles.

Infranuclear Extramedullary and intracranial involvement can occur in processes involving the meninges, extramedullary tumors, aneurysms, trauma, sarcoidosis, and skull fractures.

Lesions at the jugular foramen or in the retroparotid space usually involve some combination of IX, X, XI, XII, and the cervical sympathetics.

Palatal myoclonus: Seen in lesions at Mollaret triangle.

Jacobson's neuralgia: Involvement of tympanic branch of CN 9.

Recurrent laryngeal nerve palsy:
Causes:
- Unilateral:
  - Mitral stenosis
  - Bronchogenic carcinoma
  - Aortic aneurysm
  - Hodgkin's disease
- Bilateral:

– Guillain–Barré syndrome
– Thyroidectomy
– Lymphomas

CRANIAL NERVE XI—SPINAL ACCESSORY

The spinal accessory (SA) nerve, cranial nerve XI (CN XI), is actually two nerves that run together in a common bundle for a short distance **[Fig. 6D(iii).68]**.

Cranial part (ramus internus): The smaller cranial portion is a special visceral efferent (SVE) accessory to the vagus. It emerges from the medulla laterally as four or five rootlets caudal to the vagal filaments. The cranial root runs to the jugular foramen and unites with the spinal portion, traveling with it for only a few millimeters to form the main trunk of CN XI. The cranial root communicates with the jugular ganglion of the vagus, and then exits through the jugular foramen separately from the spinal portion. It is distributed principally with the recurrent laryngeal nerve to sixth branchial arch muscles in the larynx.

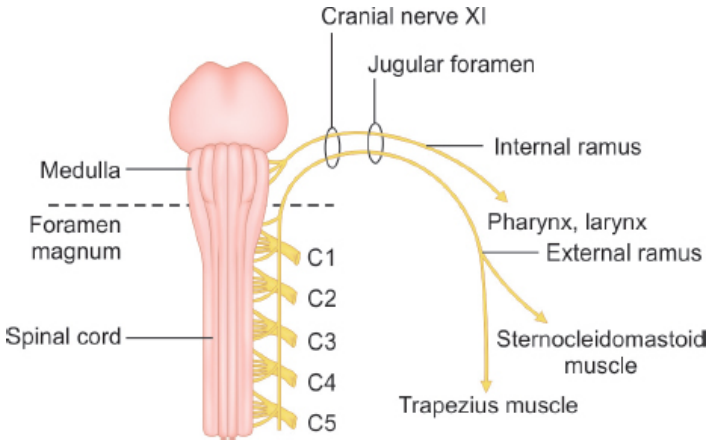


Fig. 6D(iii).68: Anatomy of spinal accessory nerve.

Spinal part (ramus externus): The major part of CN XI is the spinal portion. Its function is to innervate the sternocleidomastoid (SCM) and trapezius muscles. The fibers of the spinal root arise from SVE motor cells in the SA nuclei in the ventral horn from C2 to C5, or even C6. These unite into a single trunk, which ascends between the denticulate ligaments and the posterior roots. The nerve enters the skull through the foramen magnum, ascends the clivus for a short distance, and then curves laterally. The spinal root joins the cranial root for a short distance, probably receiving one or two filaments from it. It exits through the jugular foramen in company with CNs IX and X.

C1-2 supplies sternocleidomastoid.

C3-4 supplies trapezius.

Testing the Spinal Accessory Nerve

Cranial Part

The functions of the cranial portion of CN XI cannot be distinguished from those of CN X, and examination is limited to evaluation of the functions of the spinal portion.

Spinal Part

Testing SCM [Figs. 6D(iii).69 and 6D(iii).70]:

Testing one muscle at a time: To assess SCM power, have the patient turn the head fully to one side and hold it there, then try to turn the head back to midline, avoiding any tilting or leaning motion. The muscle usually stands out well, and its contraction can be seen and felt. Significant weakness of rotation can be detected if the patient tries to counteract firm resistance.

Testing two muscle at a time: The two SCM muscles can be examined simultaneously by having the patient flex his neck while the examiner exerts pressure on the forehead or by having the patient turn the head from side to side. Flexion of the head against resistance may cause deviation of the head toward the paralyzed side.

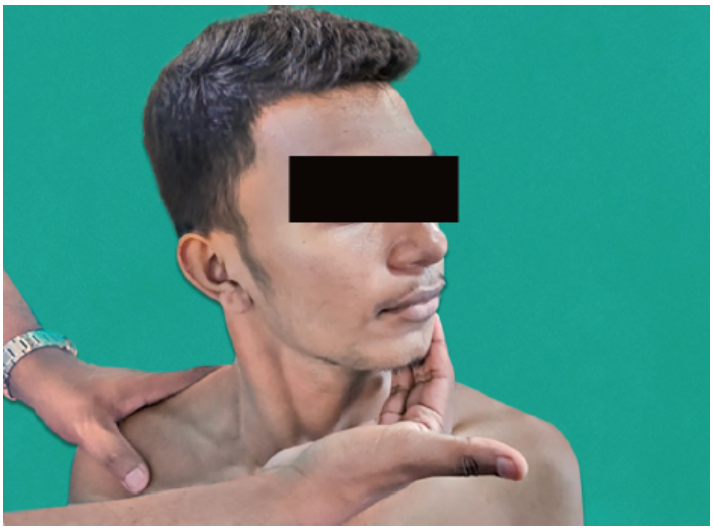


Fig. 6D(iii).69: Examination of sternocleidomastoid muscle (testing one muscle at a time).

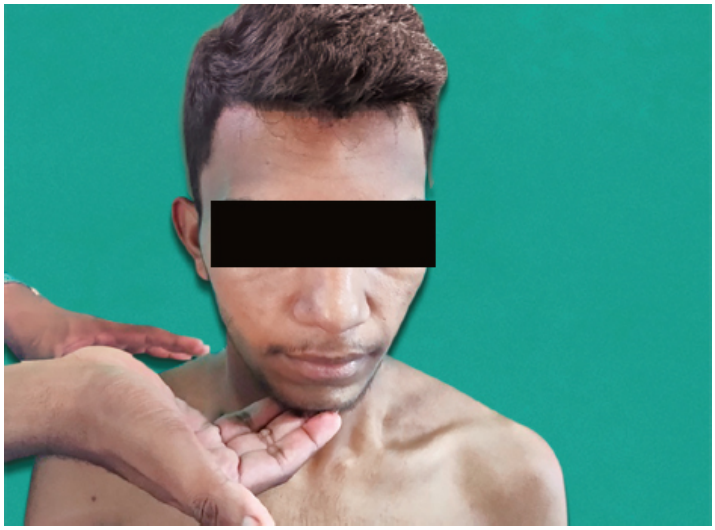


Fig. 6D(iii).70: Examination of sternocleidomastoid (testing both muscles at a time).

Interpretation: With unilateral paralysis, the involved muscle is flat and does not contract or become tense when attempting to turn the head contralaterally or to flex the neck against resistance. Weakness of both SCMs causes difficulty in anteroflexion of the neck, and the head may assume an extended position.

Testing trapezius muscle (Fig. 6D(iii).71):

Inspection: With trapezius atrophy, inspection findings include:

- Depression or drooping of the shoulder contour
- Flattening of the trapezius ridge
- Sagging of the shoulder

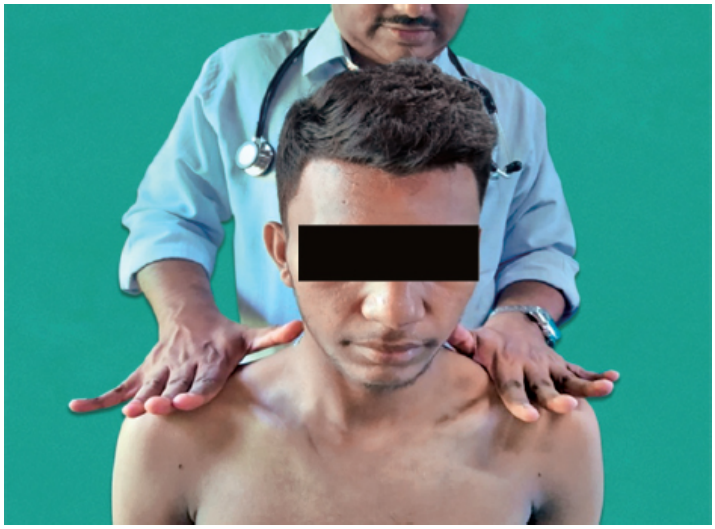


Fig. 6D(iii).71: Traditional method of assessing trapezius muscle (shrugging shoulders against resistance).

- The resting position of the scapula shifts downward
- The upper portion of the scapula tends to fall laterally while inferior angle moves inward (this scapular rotation and displacement are more obvious with arm abduction).

Palpation:

Traditional method: The strength of the trapezius is traditionally tested by having the patient shrug the shoulders against resistance. However, much of shoulder shrugging is due to the action of the levator scapulae.

Newer methods:

- **For upper trapezius:** Resisting the patient's attempt to approximate the occiput to the acromion. Impairment of upper trapezius function causes weakness of abduction beyond 90°.
- **For middle and lower trapezius:** Place the patient's abducted arm horizontally, palm up, and attempt to push the elbow forward. Muscle power should be compared on the two sides. Weakness of the middle trapezius muscle causes winging of the scapula.

Clinical implication: Weakness of the muscles supplied by CN XI may be caused by supranuclear, nuclear, or infranuclear lesions.

- **Supranuclear involvement:** Irritative supranuclear lesions may cause head turning away from the discharging hemisphere. This turning of the head (or head and eyes) may occur as part of a controversive, ipsiversive, or Jacksonian seizure and is often the first manifestation of the seizure. Extrapyramidal lesions may also involve the SCM and trapezius muscles, causing rigidity, akinesia, or hyperkinesis.
- **Nuclear involvement** of the SA nerve may occur in motor neuron disease, syringobulbia, and syringomyelia. In nuclear lesions, the weakness is frequently accompanied by atrophy and fasciculations.
- **Infranuclear or peripheral lesions**—either extramedullary but within the skull, in the jugular foramen, or in the neck—are the most common causes of impairment of function of the SA nerve. Tumors in the foramen magnum, lesions of the cerebellopontine angle, basal skull fractures, and meningitis.

"Dropped Head Syndrome"/Floppy Head Syndrome/Broken Neck Sign

This syndrome, characterized by weakness of the extensor muscles of neck with or without involvement of neck flexors, can be caused by:

- Myasthenia gravis
- Inflammatory myopathy—polymyositis
- Guillain–Barré syndrome
- Amyotrophic lateral sclerosis (ALS)/Bulbar polio
- Facio-scapulo-humeral dystrophy
- Neurotoxic snake bite/Organophosphorous compound poisoning.

CRANIAL NERVE XII—HYPOGLOSSAL NERVE

Function: CN XII supplies the intrinsic muscles, and all of the extrinsic muscles of the tongue except the palatoglossus.

Anatomy [Fig. 6D(iii).72]: Nucleus located in medial medulla. Distribution of fibers from rostral to caudal, the innervation is intrinsic tongue muscles, then genioglossus, hyoglossus, and styloglossus.

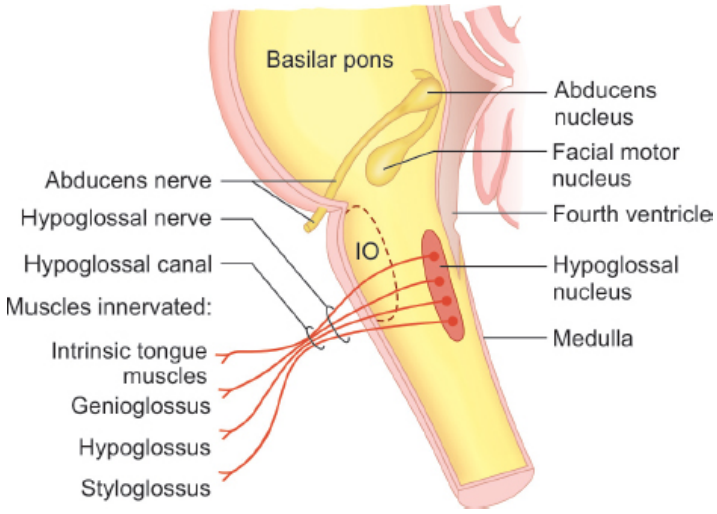


Fig. 6D(iii).72: Location of hypoglossal nerve.

Examination

The clinical examination of hypoglossal nerve function consists of evaluating the strength, bulk, and dexterity of the tongue—looking especially for weakness, atrophy, abnormal movements (particularly fasciculations), and impairment of rapid movements.

Inspection:

- **Tongue deviation:** To look for tongue deviation by asking the patient to protrude the tongue and also to move the tongue to either sides.
- **Fasciculations:** Ask the patient to open the mouth and with the tongue inside the mouth look for the fasciculations.

Palpation:

- Hold the tongue with gauze and palpate the tongue with gloved finger to examine the consistency of the tongue [Fig. 6D(iii).73].
- To examine the power of the tongue patient is instructed to push the tongue against the cheek while giving the counter resistance from outside **[Fig. 6D(iii).74]**.

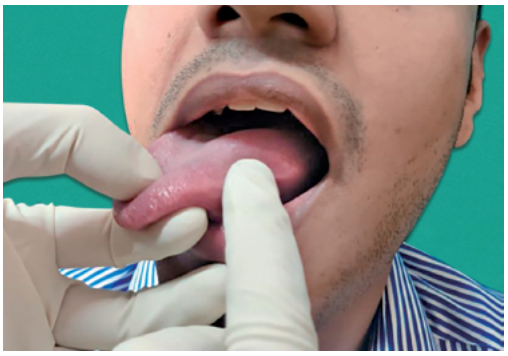


Fig. 6D(iii).73: Palpation of tongue.

Fig. 6D(iii).74: Examining the motor power of tongue.

Interpretation

On inspection:

- **Tongue deviation [Fig. 6D(iii).75]:** When unilateral weakness is present, the tongue deviates toward the weak side on protrusion because of the action of the normal genioglossus. And also there is impairment of the ability to deviate the protruded tongue toward the opposite side.
- Fasciculations: Presence of fasciculations suggests LMN paralysis of the 12th cranial nerve.

On palpation:

- **Small and stiff tongue:** Suggestive of UMN type of 12th nerve palsy.
- **Flabby tongue with fasciculations:** Suggestive of LMN type of 12th nerve palsy.

Other clinical aspects: The neck-tongue syndrome, consisting of pain in the neck and numbness or tingling in the ipsilateral half of the tongue on sharp rotation of the head, has been attributed to damage to lingual afferent fibers traveling in the hypoglossal nerve to the C2 spinal roots through the atlantoaxial space.

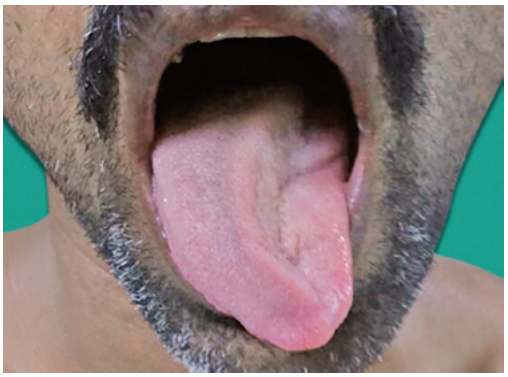


Fig. 6D(iii).75: Tongue deviation to the left suggestive of weakness of left hypoglossal muscle.

Bulbar palsy	Pseudobulbar palsy
Etiology: • Motor neuron disease • Syringobulbia • Guillain-Barré syndrome • Poliomyelitis • Subacute meningitis (carcinoma and lymphoma) • Neurosyphilis • Brainstem CVA	**Etiology:** The most common cause is bilateral CVAs affecting the internal capsule Other causes include: • Multiple sclerosis • Motor neuron disease • High brainstem tumors • Head injury
• Bilateral damage or injury of the nerve nuclei of cranial nerves IX, X, XI, and XII • Lower motor neuron palsy of the respective muscles • Gag reflex—absent • Tongue—wasted, fasciculations • "Wasted, wrinkled, thrown into folds, and increasingly motionless" • Palatal movement—absent • Jaw jerk—absent or normal • Speech—nasal "Indistinct (flaccid dysarthria), lacks modulation, and has a nasal twang" • Emotions – normal • Other—signs of the underlying cause, e.g. limb fasciculations	• Bilateral damage or injury of corticobulbar tracts to nerve nuclei of cranial nerves V, VII, X, XI, and XII • Upper motor neuron palsy of the respective muscles • Gag reflex—increased or normal • Tongue—spastic "It cannot be protruded, lies on the floor of the mouth and is small and tight" • Palatal movement—absent • Jaw jerk—increased • Speech—spastic: "A monotonous, slurred, high-pitched, 'Donald Duck', dysarthria" that "sounds as if the patient is trying to squeeze out words from tight lips". "Hot potato voice" • Emotions—labile • Other—bilateral upper motor neuron (long tract) limb signs. Bilateral extensor plantar and bilateral exaggerated reflexes

MULTIPLE CRANIAL NERVE PALSIES

Cranial nerve	Cavernous sinus thrombosis	Superior orbital fissure syndrome	Orbital apex syndrome	Jaccoud's (retro-sphenoid space)syndrome	Petrous apex gradinigo syndrome	Tolosa-Hunt, lateral cavernous sinus syndrome	CP angle tumor	Vernet jugular foramen syndrome	Villaret, post-retroparotid syndrome	sy
II			✓	✓						
III	✓	✓	✓	✓		✓				

IV	✓	✓	✓	✓		✓				
V1	✓			✓	✓	✓	✓			
V2	✓		✓	✓	✓					
V3				✓	✓					
VI	✓	✓	✓	✓	✓	✓	✓			
VII							✓			
VIII							✓			
IX								✓	✓	
X								✓	✓	
XI								✓	✓	
XII									✓	
Horner	✓								✓	

NOTES

D(iv). MOTOR SYSTEM EXAMINATION

Motor system examination includes examination of:

1. Attitude of the limbs
2. Bulk/nutrition
3. Assessment of tone
4. Examination of power
5. Reflexes
6. Coordination
7. Gait

Reflexes, coordination, and gait have been discussed separately in the successive sections.

ATTITUDE

Attitude is the position of the limbs which it adopts when the patient is in resting position.

In a patient with hemiplegia	
Upper limb	***Lower limb***
• Adduction at shoulder • Flexion at elbow • Semipronated • Thumb tucked into the palm	• Extended at hip and knee • Externally rotated at hip • Foot inverted • Plantar flexed

Few common attitudes

Paraplegia	Bilateral lower limbs are: • Extended at hip and knee • Externally rotated at hip • Foot inverted • Plantar flexed
Erb's palsy	On the affected side: • Arm: Adducted and internally rotated • Forearm: Extended and pronated • Wrist: flexed • "Waiter's tip deformity"

MUSCLE BULK/NUTRITION

- Muscle bulk is assessed by inspection as well as measurements at corresponding sites in the extremities.
- Symmetry is important with consideration given to handedness and overall body habitus.
- Wasting is considered if there is >1 cm reduction on the dominant extremity and >2 cm in the nondominant extremity. In some areas, just inspection is adequate (thenar eminence, hypothenar eminence, shoulder) whereas in other areas (thighs, legs, arms and forearms) measurement is required.
- Measurements of the circumferences of the limb are done at corresponding areas at fixed distances from bony landmarks, which are part of that limb. Example: 10 cm below the olecranon **[Fig.**

6D(iv).1], 10 cm above the medial humeral epicondyle **[Fig. 6D(iv).2]**, 18 cm above the patella, and 10 cm below the tibial tuberosity.

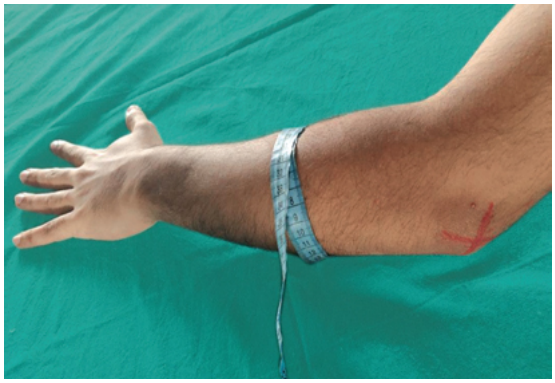


Fig. 6D(iv).1: Measurement of bulk in the forearm.

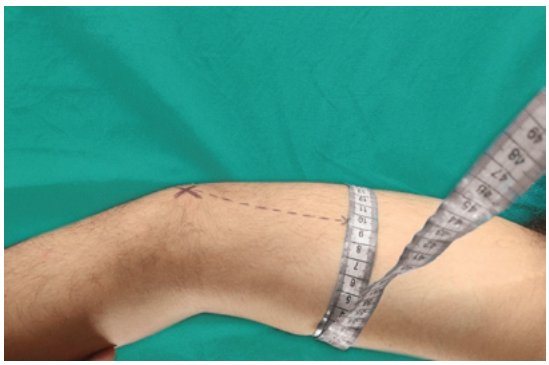


Fig. 6D(iv).2: Measurement of bulk in the arm.

Causes for Muscle Hypertrophy (Usually in the Calf) [Fig. 6D(iv).3]

True hypertrophy	Pseudohypertrophy (due to increased fat in muscle)
Exercise	• Duchene's muscular dystrophy • Becker's muscular dystrophy • Myotonia congenita—Thomson's disease • Kugelberg Welander spinal muscular atrophy • Hypothyroidism (infantile Hercules/Kocher–Debré–Semelaigne syndrome) • Storage disorders
Localized muscle swelling—muscle hemorrhage, myositis ossificans, abscess, tumor, muscle rupture or cysts (cysticercosis)	

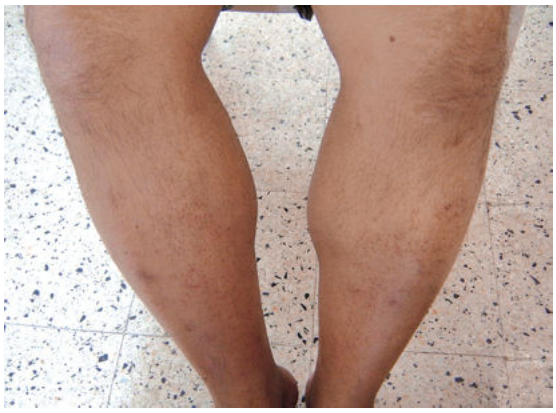


Fig. 6D(iv).3: Pseudohypertrophy of calf muscle.

Causes of Muscle Wasting

Generalized wasting	Proximal wasting	Distal wasting
• Malignancy • Cachexia • Tuberculosis • Thyrotoxicosis • Addison's disease • HIV/AIDS	• Motor neuron disease: Juvenile SMA (Kugelberg Welander) • Muscular dystrophy: FSHD **[Fig. 6D(iv).4]**, limb girdle dystrophy • Inflammatory myopathies • Brachial plexopathy • Axillary neuropathy	• Anterior horn cell disease—polio, motor neuron disease • Syringomyelia, intramedullary tumors • Peripheral neuropathies—leprosy, Carpal tunnel syndrome • Myotonic dystrophy • Plexopathies—lower brachial plexus • Arthritis—rheumatoid • Disuse atrophy

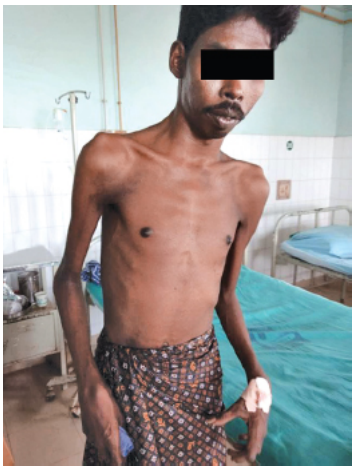


Fig. 6D(iv).4: Proximal muscle wasting seen in facioscapulohumeral dystrophy (FSHD).

Causes of hand muscle wasting [Fig. 6D(iv).5]

Anterior horn cell disease	• Motor neuron disease • Syringomyelia • Polio • Spinal muscular atrophy
Nerve root	• T1 compression by disc lesion. • Pachymeningitis

	• Cervical spondylosis • Syphilitic amyotrophy • C8–T1 tumors
Brachial plexus	• Pancoast tumor • Thoracic outlet obstruction, cervical rib • Trauma, Klumpke's paralysis • Other—infiltration, irradiation
Lesions of peripheral nerve (ulnar or median)	• Trauma • Acute compression (coma, anesthesia, deep sleep) • Chronic compression (entrapment) • Acute ischemia (collagen vascular disease, diabetes)
Muscle disease	• Myotonic dystrophy • Distal myopathy—Welander, Udd, Miyoshi, Nonaka, Markesbery
Others	• Rheumatoid arthritis • Disuse atrophy • Rarely—parietal lobe lesions

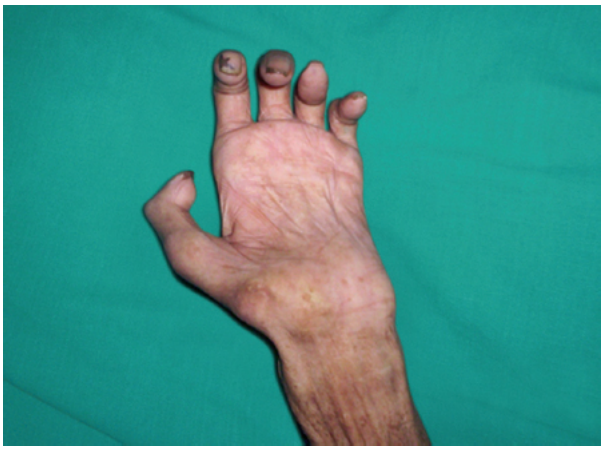


Fig. 6D(iv).5: Small muscle wasting of the hand.

The Split Hand Sign

- It is highly specific for amyotrophic lateral sclerosis (ALS).
- It is due to a lesion in the ulnar nerve or the lower trunk, which will cause predominant wasting of first dorsal interossei and hypothenar muscles with preserved thenar muscles (which are innervated by the median nerve).
- It is called split hand sign as it preferentially affects lateral part of the hand (abductor pollicis brevis and first dorsal interossei) and spares the medial part of the hand.
- This pattern of dissociated wasting does not correspond to a nerve or plexus or root distribution.
- This is in contrast to a C8-T1 root lesion, which will cause wasting of both thenar and hypothenar muscle as both median and ulnar nerves receive C8-T1 innervation.

MUSCLE TONE

Definition

Tone is defined as partial state of contraction of the muscle at rest which is demonstrated by resistance offered by the muscle to passive movement across the joint.

Tone is examined in the upper limb (wrist and elbow joint) and the lower limb (knee and ankle joint).

Testing for Tone in the Legs [Figs. 6D(iv).6 and 6D(iv).7]

- With the patient relaxed, place your hands on the thigh and roll the whole leg. Observe the movement of the foot
- With the patient in a supine position, place your hands behind the patient's knee, and lift the leg in a sudden motion. Observe if the heel drags along the bed. With normal muscle tone, the heel will drag along the surface of the bed. However, if there is an increased tone or spasticity, the foot may not make contact with the bed.
- Alternatively flex and extend the knee. Feel for the extensors during flexion and flexors during extension.

Testing for Tone in the Arms [Figs. 6D(iv).8 to 6D(iv).10]

- Lift the arm and let it drop. See the speed and smoothness.
- At the elbow, check for tone in biceps and triceps. Feel the biceps while extending the arm, and feel the triceps while flexing the arm.

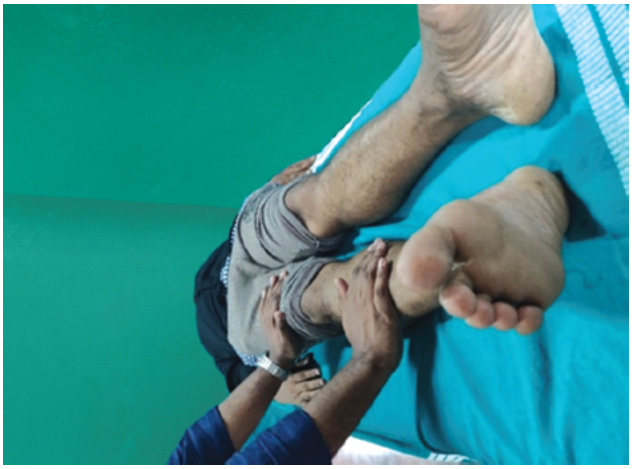


Fig. 6D(iv).6: Assessment of tone in the lower limbs.

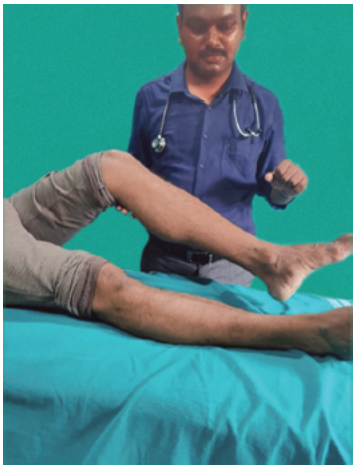


Fig. 6D(iv).7: Assessment of tone in the lower limbs.

- At the wrist, take the hand as if to shake it. First pronate and supinate the forearm. Then roll the hand around at the wrist. This demonstrates cog wheel rigidity **[Fig. 6D(iv).11]**.

Fig. 6D(iv).8: Examining tone of triceps.

Fig. 6D(iv).9: Examining the tone of biceps.

Fig. 6D(iv).10: Examining the tone in the upper limb.

Fig. 6D(iv).11: Examining for cog wheeling/rigidity.

Abnormalities of Tone

Hypotonia—decreased tone.
Causes:

- Lower motor neuron (LMN) disease
- Cerebellar disease
- Hypothyroidism
- Upper motor neuron (UMN) disease in a state of neuronal shock
- Chorea
- Hypermagnesemia
- Down syndrome
- Anesthesia and muscle relaxants.

Hypertonia—increased tone. Two principal types:

1. Spasticity
2. Rigidity

	Spasticity	Rigidity
Synonym	Clasp-knife	Lead-pipe/Cog-wheel
Diseases	Pyramidal	Extrapyramidal
Pathophysiology	Increased gamma activity	Increased gamma and alpha activity
Description	• Tone increased in the initial part of movement followed by sudden release—clasp-knife effect* • Supination-pronation of the forearm will reveal the so-called supinator catch	• Increased tone present continuously throughout the complete range of movement—lead-pipe • With associated tremors—cog-wheel**
Muscles involved	Anti-gravity muscles (flexors in the UL and extensors in the LL)	Both groups of muscles
Velocity	Velocity dependent (more with fast movements)	Velocity independent
Associated features	Hyperreflexia, extensor plantar	Tremors, bradykinesia

*__Clasp-knife phenomenon:__ The muscles at rest do not have excessive tone but a brisk stretch will produce a catch at about mid-length of the muscle followed by a sudden release of the catch and

relaxation of the muscle. The giving away or the release portion of the clasp-knife phenomenon is due to the increased firing of the inhibitory Golgi tendon organs. To elicit this phenomenon, the clinician extends the patient's knee using a constant velocity, but as the patient's knee nears full extension, the muscle tone of the quadriceps muscles increases dramatically and completes the movement, just as the blade of a pocket knife opens under the influence of its spring.

Cog-wheel rigidity: Lead pipe rigidity superimposed with tremors (Negro sign).

Causes of hypertonia:

- UMN disease—pyramidal and extrapyramidal
- Tetanus
- Tetany
- Strychnine poisoning
- Tonic phase of seizure
- Catatonia (seen in schizophrenia where there is increased tone for all movements)

Paratonia—altered tone seen in psychiatric diseases and frontal lobe dysfunction which is characterized by inability to relax the muscle during muscle tone assessment. Can be of two types:

1. Oppositional paratonia **(Gegenhalten)**—where the subjects involuntarily resist passive movements
2. Facilitatory paratonia **(Mitgehen)**—where the subject involuntarily assists passive movement.

Paratonia is present in bilateral frontal lobe dysfunction and diffuse cerebellar disorders.

Myotonia—Slow relaxation of muscle after voluntary contraction or contraction provoked by muscle percussion. Examples: myotonic dystrophy, congenital myotonia, hypothyroidism, neuromyotonia congenita, Issac syndrome **[Fig. 6D(iv).12]**.

Myoedema

Stationary muscle mounding after muscle percussion without electrical muscle activity is called myoedema. Myoedema is due to prolonged muscle contraction caused by delayed calcium reuptake by sarcoplasmic reticulum, following local calcium ion release brought out by percussion or pressure.

Can be seen in hypothyroidism, chronic debilitating diseases, severe cachexia as in TB.

MOTOR POWER

Prerequisites

- Explain the test and the movements you are planning to do clearly to the patient before performing the test.

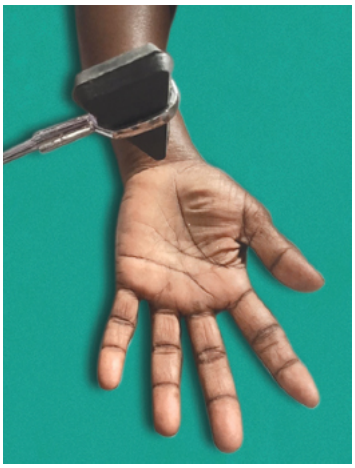


Fig. 6D(iv).12: Demonstration of myotonia.

- Position the patient according to the muscle which is being tested.

State of Muscle during Examination

- Fully contracted muscle
  - Muscle is at maximum advantage (small muscle)
- Fully relaxed muscle
  - Muscle at maximum disadvantage (may detect mild degrees of weakness)
- Mid-contracted muscle
  - Most feasible method
  - Used for most large muscles

Qualitative Assessment of Weakness (MRC Grading)

- Grade 0—no contraction
  Grade 1—Flicker or trace of contraction
  Grade 2—active movement, with gravity eliminated
  Grade 3—active movement against gravity
  Grade 4—active movement against gravity and resistance
  Grade 5—normal power
- Grades 4-, 4, and 4+ may be used to indicate movement against slight, moderate, and strong resistance, respectively.

Muscle of neck	
Flexion of neck (sternocleidomastoid/platysma)	The patient attempts to flex his neck against resistance while supporting the chest **[Fig. 6D(iv).13]**
Extensor of neck	The patient attempts to extend their neck against resistance; contraction of the trapezius and other extensor muscles can be seen and felt, and strength of movement can be judged **[Fig. 6D(iv).14]**
Upper limb	
Supraspinatus—C5	Patient initiates abduction of arm from side against resistance **[Fig. 6D(iv).15]**
Deltoid—C5	Patient holds his hand at 60° against resistance **[Fig. 6D(iv).16]**
Infraspinatus—C5	The patient flexes his elbow, examiner holds the elbow to his side, and then attempts external rotation of the forearm against resistance **[Fig. 6D(iv).17]**

Rhomboids—C5	With hands on hip ask the patient to force the elbow backward **[Fig. 6D(iv).18]**
Serratus anterior—C5, 6, 7	The patient pushes his arms forward against firm resistance **[Fig. 6D(iv).19]**
Pectoralis major—C6, 7, 8	• Placing hand on hip and pressing inward, sternocostal part of muscle can be seen and felt to contract **[Fig. 6D(iv).20]** • Raising the arm forward above 90° and attempting to adduct clavicular portion can be felt
Latissimus dorsi—C7	• While palpating muscles ask the patient to cough • Resist the patients attempt to adduct the arm when abducted to above 90° [Fig. 6D(iv).21]
Biceps—C5	Ask the patient to flex at the forearm with hand in supine position, against resistance **[Fig. 6D(iv).22]**
Brachioradialis—C5,6	The patient is asked to flex the elbow with the forearm midway between pronation and supination **[Fig. 6D(iv).23]**
Triceps—C7	The patient attempts to extend elbow against resistance **[Fig. 6D(iv).24]**
Extensor carpi radialis longus—C6, 7	The patient makes a fist and extends the wrist towards the radial side **[Fig. 6D(iv).25]**
Extensor carpi ulnaris—C7	The patient makes a fist and extends the wrist towards the ulnar side **[Fig. 6D(iv).26]**
Extensor digitorium—C7	The examiner attempts to flex the patient's extended fingers at the metacarpophalangeal joints **[Figs. 6D(iv).27A and B]**
Flexor carpi radialis—C6, 7	The examiner attempts to flex the wrist toward the radial side **[Fig. 6D(iv).28]**
Flexor carpi ulnaris—C8	Best seen while testing the abductor digiti minimi when it fixes its point of origin **[Figs. 6D(iv).29A and B]**
Abductor pollicis longus—C8	Patient maintains their thumb in the abduction against the examiner's resistance **[Fig. 6D(iv).30]**
Extensor pollicis brevis—C8	The patient attempts to extend the thumb while the examiner attempts to flex it at the **metacarpophalangeal joint [Fig. 6D(iv).31]**
Extensor pollicis longus—C8	The patient attempts to extend the thumb while the examiner attempts to flex it at the **interphalangeal joint**
Opponens pollicis—T1	The patient attempts to touch the little finger with the thumb **[Fig. 6D(iv).32]**
Abductor pollicis brevis—T1	Place an object between the thumb and base of forefinger to prevent full adduction Patient attempts to raise the edge of the thumb vertically against the resistance **[Fig. 6D(iv).33]**
Flexor pollicis longus—C8	Tested by attempting to extend the distal phalanx of the thumb against resistance, while holding the proximal phalanx **[Fig. 6D(iv).34]**
Adductor pollicis—T1	The patient attempts to hold a piece of paper between the thumb and the palmar aspect of forefinger and examiner tries to pull the paper **[Fig. 6D(iv).35]**
Lumbricals—C8, T1	The patient tries to flex the extended fingers at the metacarpophalangeal joints **[Fig. 6D(iv).36]**
Dorsal interossei	The patient attempts to keep the fingers abducted against resistance **[Fig. 6D(iv).37]**
First dorsal interossei and palmar interossei	Place the hand flat on table and the patient tries to abduct and adduct the forefinger against the resistance **[Figs. 6D(iv).38 and 6D(iv).39]**
Flexor digitorum sublimis—C8	The patient flexes the fingers at the proximal interphalangeal joint against resistance from the examiner's fingers placed on the middle phalanx **[Fig. 6D(iv).40]**
Flexor digitorum profundus—C8	The patient keeps his hand on a flat surface. The examiner holds the middle phalanx

	down; the patient flexes the distal phalanx against resistance **[Fig. 6D(iv).41]**
Flexor digiti minimi—T1	The back of hand is placed on the table and the little finger abducted against resistance. (often the only sign of an ulnar lesion)
Trunk muscles	
Abdominal muscles	The recumbent patient attempts to raise his head against resistance **[Fig. 6D(iv).43]**
Extensors of spine	The patient, lying prone, attempts to raise the head and upper part of the chest **[Fig. 6D(iv).44]**
Lower limb	
Iliopsoas—L1, 2, 3	The patient lies supine and attempts to flex the thigh against resistance **[Fig. 6D(iv).45]**
Adductor femoris—L5, S1 (Adductor magnus, longus and brevis)	The patient attempts to adduct the leg against resistance **[Fig. 6D(iv).46]**
Gluteus medius and minimus—L2, 3	Patient in prone, flexes the knee, and then forces the foot outward against resistance **[Fig. 6D(iv).47]**
Gluteus maximus—L5, S1	Patient in prone raises the thigh against resistance with the knee flexed to minimize the contribution from the hamstrings **[Fig. 6D(iv).48]**
Hamstrings—L4, 5, S1, 2 (biceps, semimembranosus, and semitendinosus)	Patient in prone and attempts to flex the knee against resistance **[Fig. 6D(iv).49]**
Quadriceps femoris—L3, 4	Patient is supine and extends the knee against resistance **[Fig. 6D(iv).50]**
Tibialis anterior— L4, 5	The patient dorsiflexes the foot against the resistance of examiner **[Fig. 6D(iv).51]**
Tibialis posterior—L4	The patient plantar flexes the foot slightly and then tries to invert it against resistance **[Fig. 6D(iv).52]**
Peronei—L5, S1	The patient everts the foot against resistance **[Fig. 6D(iv).53]**
Extensor digitorum longus—L5	Patient asked to dorsiflex the foot against resistance **[Fig. 6D(iv).54]**
Flexor digitorum longus—S1, 2	Patient asked to flex the terminal phalanges against resistance **[Fig. 6D(iv).55]**
Extensor hallucis longus—L5, S1	Patient asked to dorsiflex the great toe against resistance **[Fig. 6D(iv).56]**
Extensor digitorum brevis—S1	The patient dorsiflexes the toes against resistance **[Fig. 6D(iv).57]**

Fig. 6D(iv).13: Flexion of neck (sternocleidomastoid/platysma).

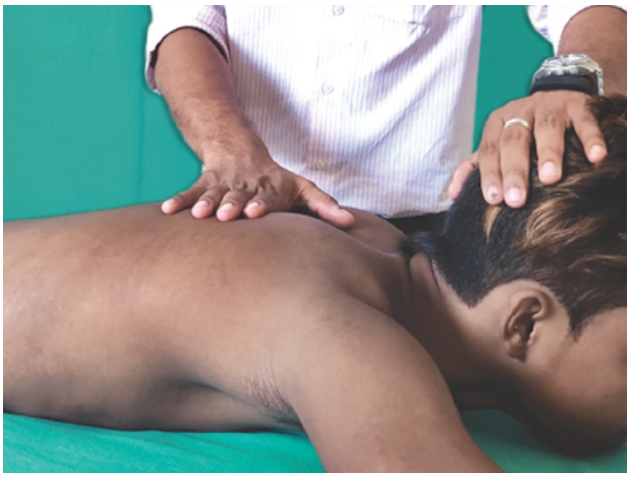


Fig. 6D(iv).14: Extensor of neck.

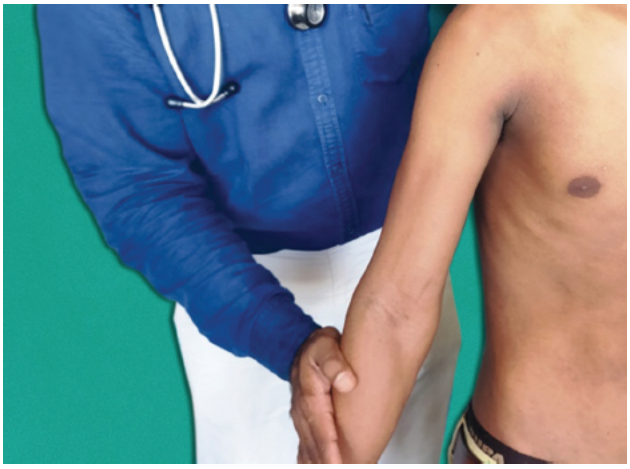


Fig. 6D(iv).15: Supraspinatus—C5. Patient initiates abduction of arm from side against resistance.

Fig. 6D(iv).16: Deltoid C5.

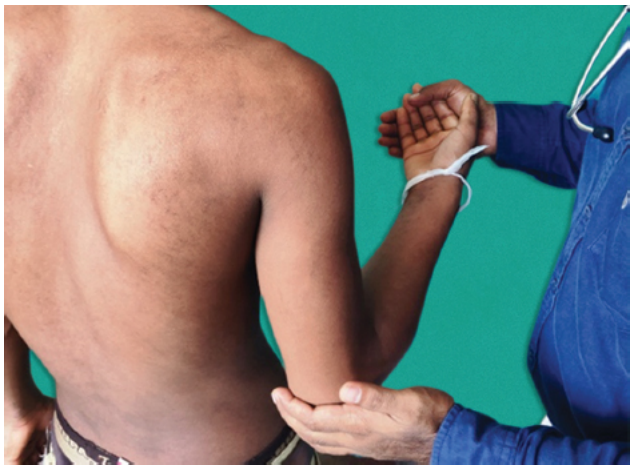


Fig. 6D(iv).17: Infraspinatus—C5.

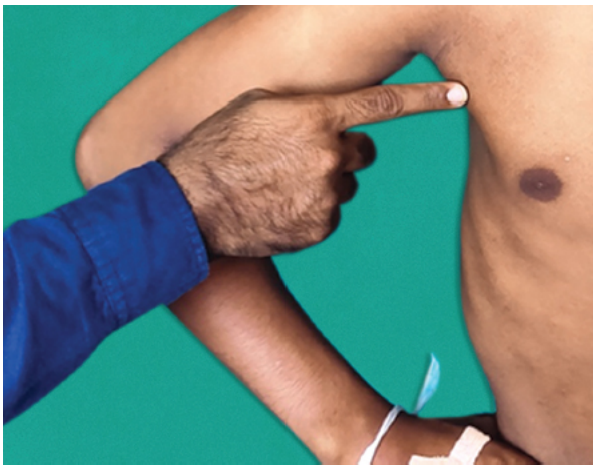


Fig. 6D(iv).20: Pectoralis major—C6, 7, 8.

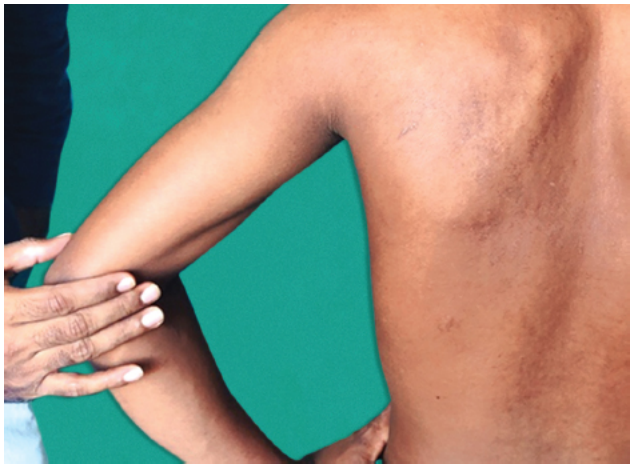


Fig. 6D(iv).18: Rhomboids—C5.

Fig. 6D(iv).21: Latissimus dorsi—C7.

Fig. 6D(iv).19: Serratus anterior—C5, 6, 7.

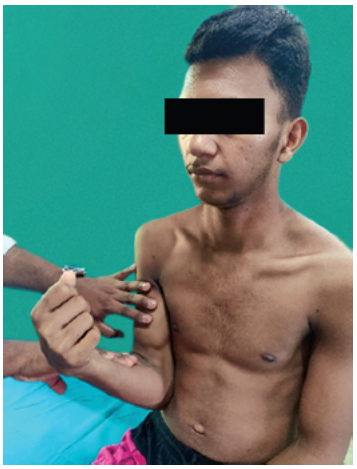


Fig. 6D(iv).22: Biceps—C5.

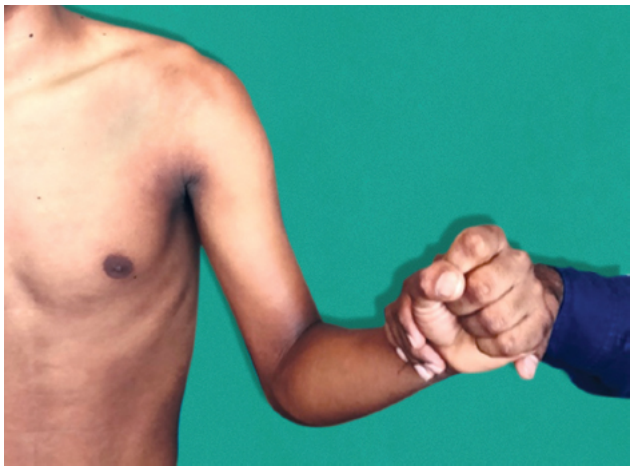
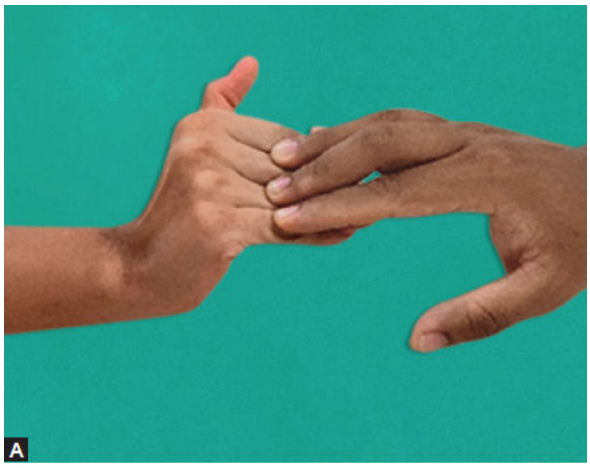


Fig. 6D(iv).23: Brachioradialis—C5, 6.



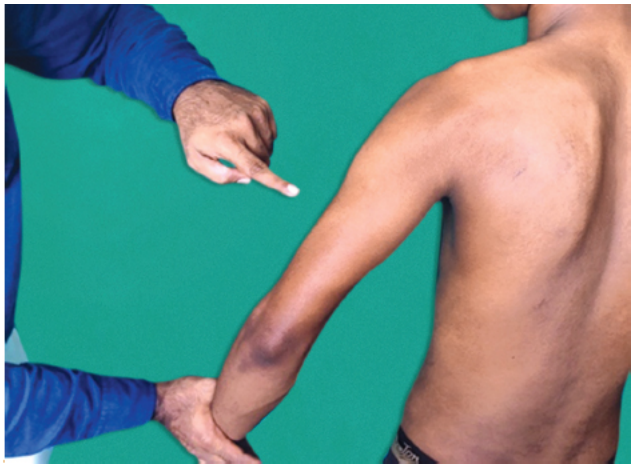
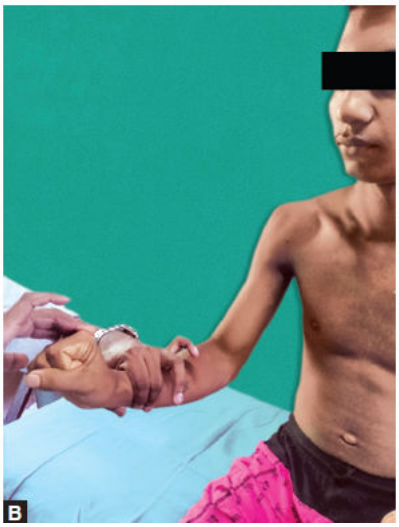


Fig. 6D(iv).24: Triceps—C7.



Figs. 6D(iv).27A and B: Extensor digitorum—C7.

Fig. 6D(iv).25: Extensor carpi radialis longus—C6, 7.

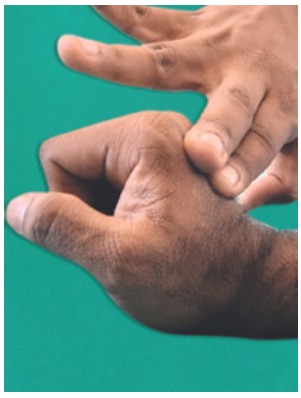


Fig. 6D(iv).26: Extensor carpi ulnaris—C7.

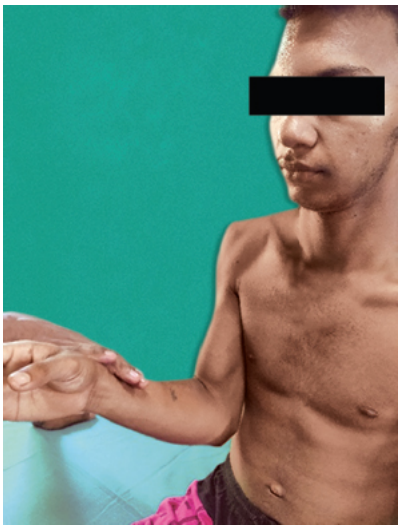
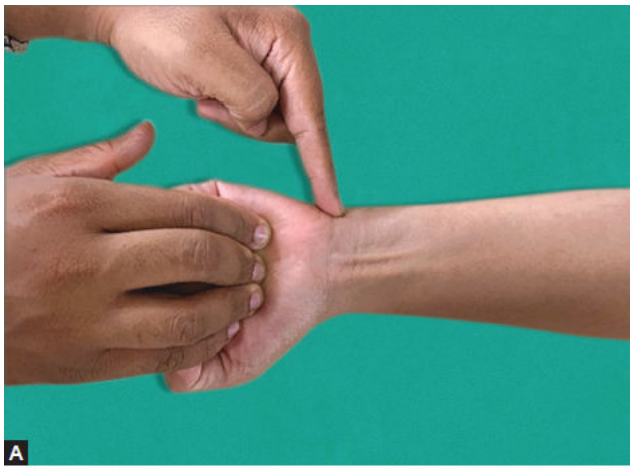


Fig. 6D(iv).28: Flexor carpi radialis—C6, 7.



Figs. 6D(iv).29A and B: Flexor carpi ulnaris—C8.

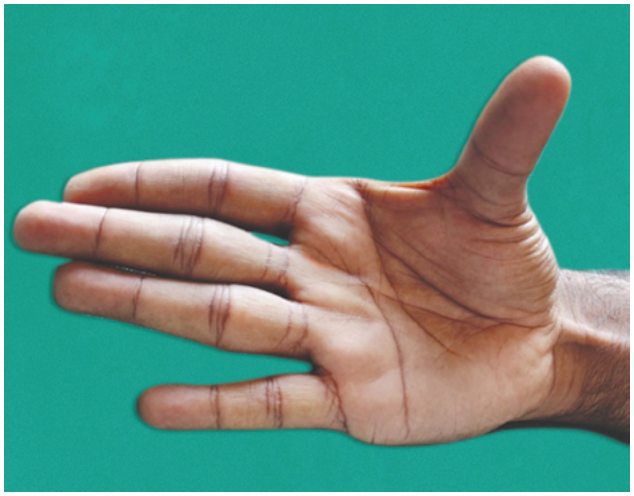


Fig. 6D(iv).30: Thumb abduction.

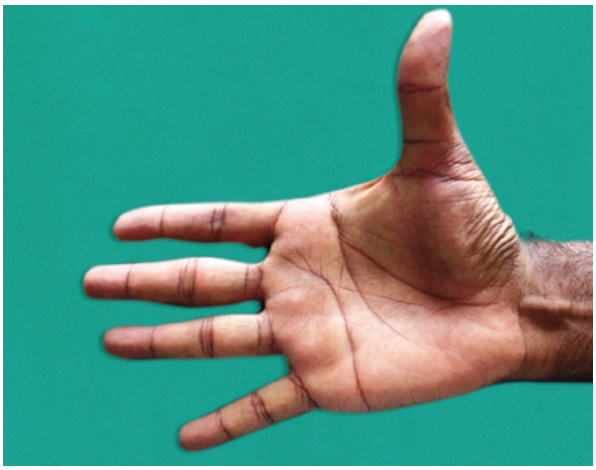


Fig. 6D(iv).31: Thumb extension.

Fig. 6D(iv).32: Opponens pollicis—T1.

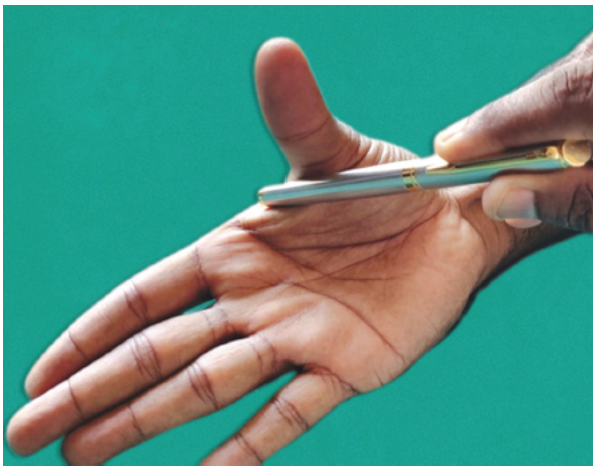


Fig. 6D(iv).33: Abductor pollicis brevis—T1.

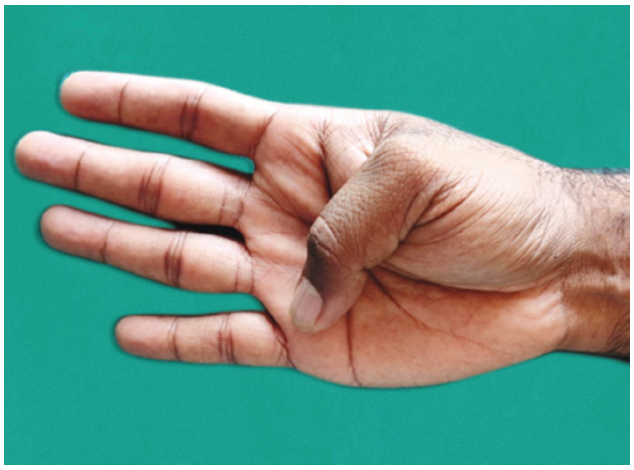


Fig. 6D(iv).34: Thumb flexion.

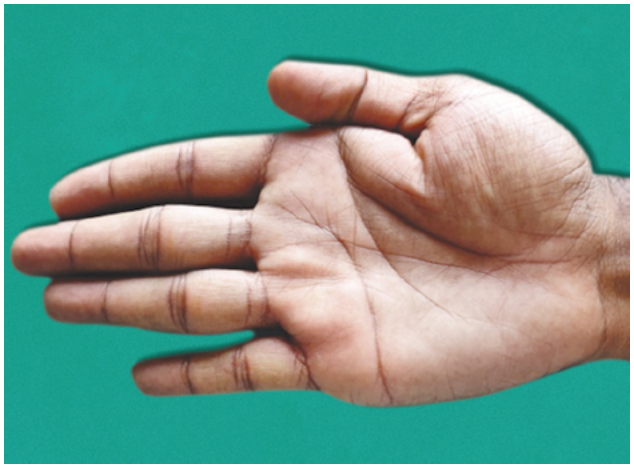


Fig. 6D(iv).35: Thumb adduction.

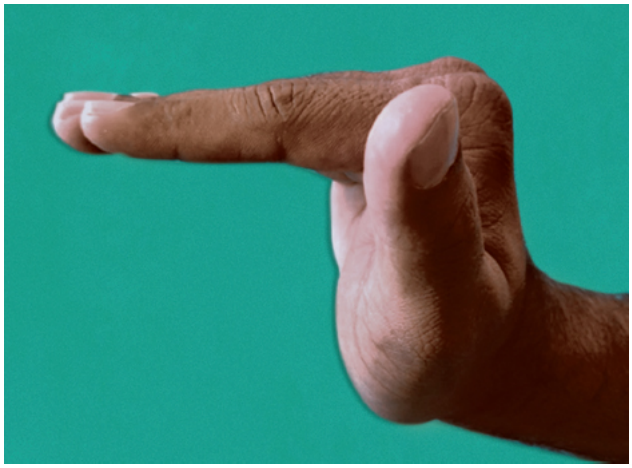


Fig. 6D(iv).36: Lumbricals—C8, T1.

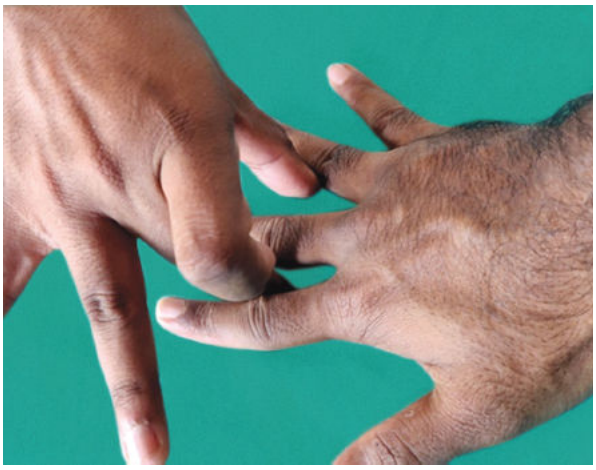


Fig. 6D(iv).37: Dorsal interossei.

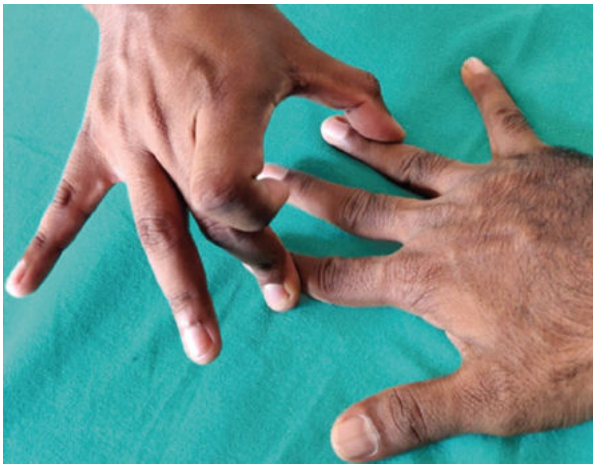


Fig. 6D(iv).38: Palmar interossei.

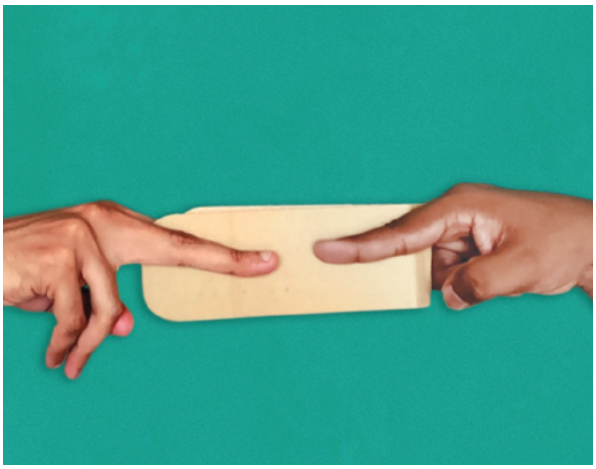


Fig. 6D(iv).39: Card test for palmar interossei.

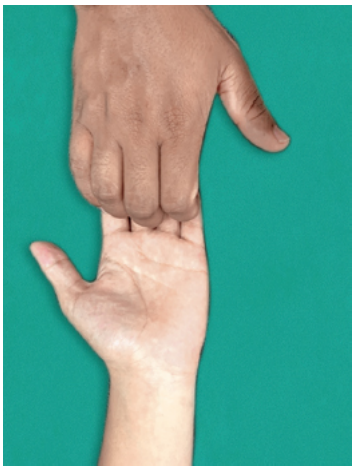


Fig. 6D(iv).40: Flexor digitorum sublimis.

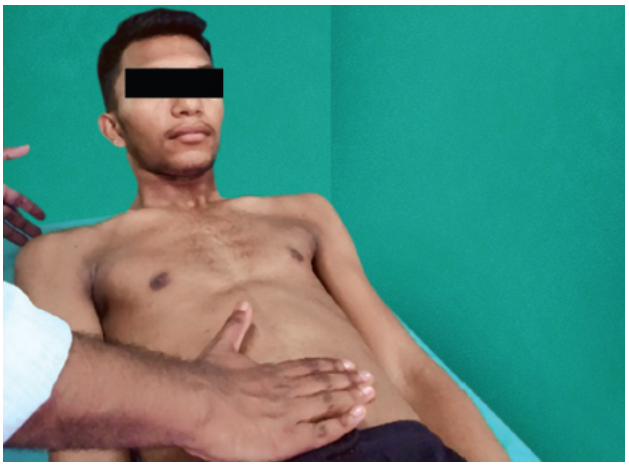


Fig. 6D(iv).43: Abdominal muscles T5–L1.

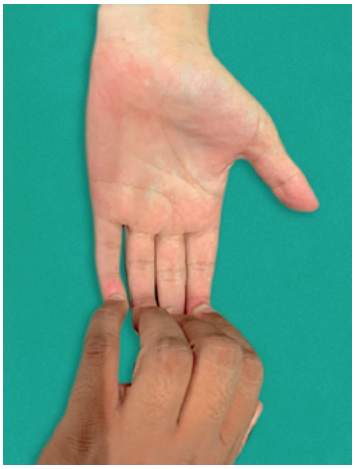


Fig. 6D(iv).41: Flexor digitorum profundus.

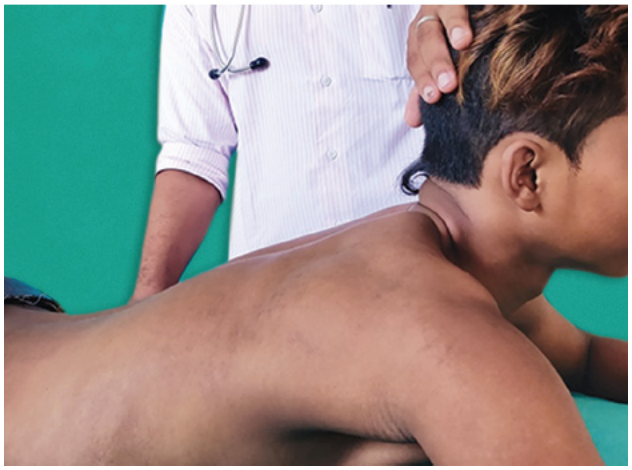


Fig. 6D(iv).44: Extensors of spine.

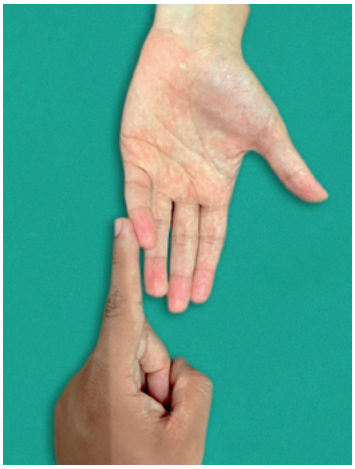


Fig. 6D(iv).42: Abductor digiti minimi.

Fig. 6D(iv).45: Iliopsoas—L1, 2, and 3.

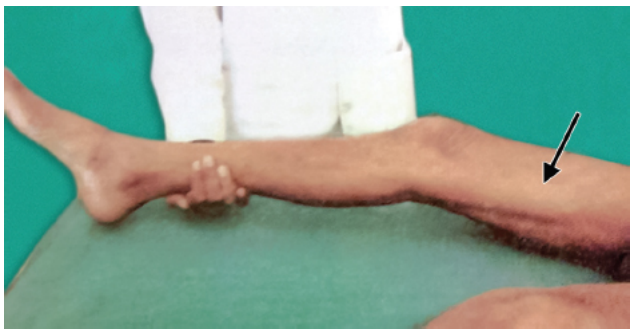


Fig. 6D(iv).46: Adductor femoris—L5, S1.

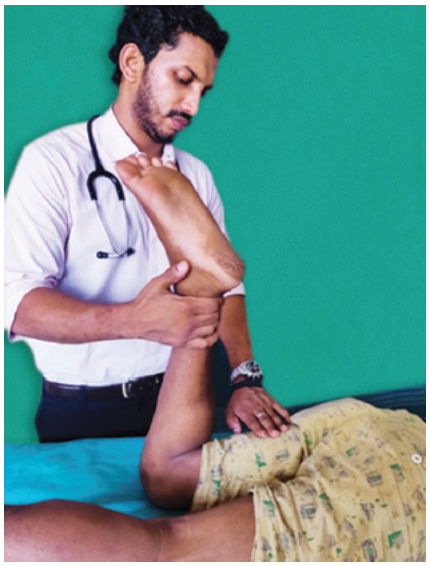


Fig. 6D(iv).47: Gluteus medius and minimus—L2, 3.

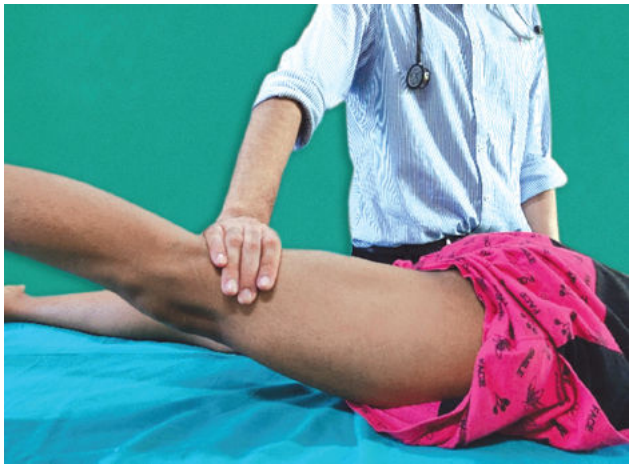


Fig. 6D(iv).48: Gluteus maximus—L5, S1.

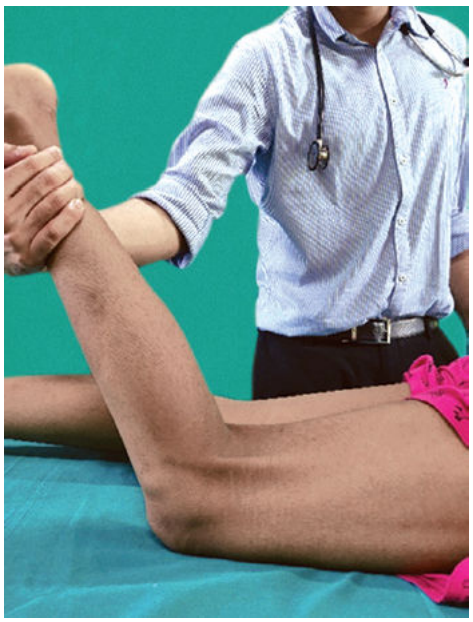


Fig. 6D(iv).49: Hamstrings—L4, 5, S1, 2 (biceps, semimembranosus, and semitendinosus).

Fig. 6D(iv).50: Quadriceps femoris—L3, 4.

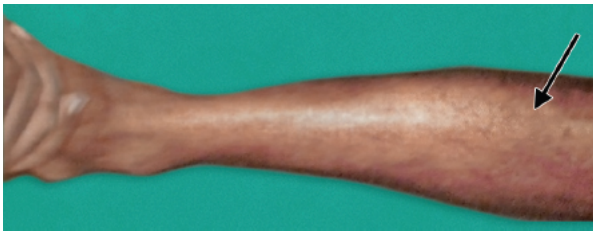


Fig. 6D(iv).51: Tibialis anticus—L4, 5.

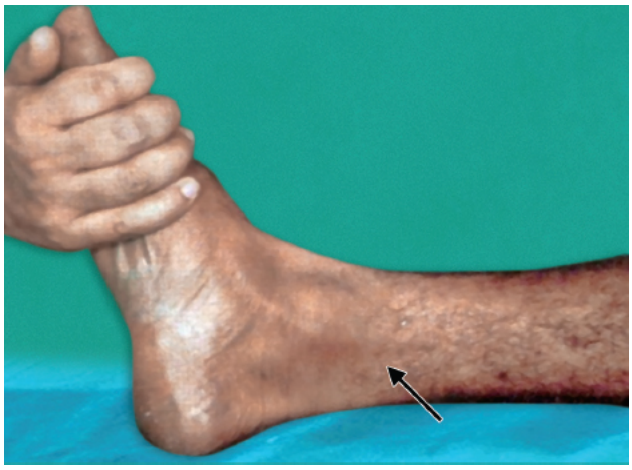


Fig. 6D(iv).52: Tibialis posticus—L4.

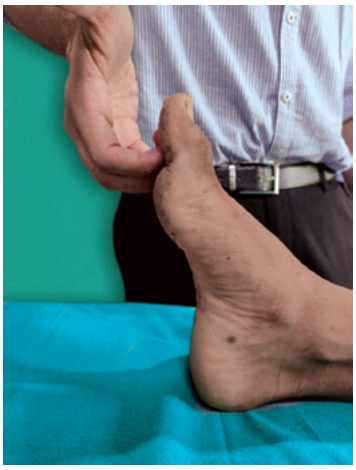


Fig. 6D(iv).55: Flexor digitorum longus—S1, 2.

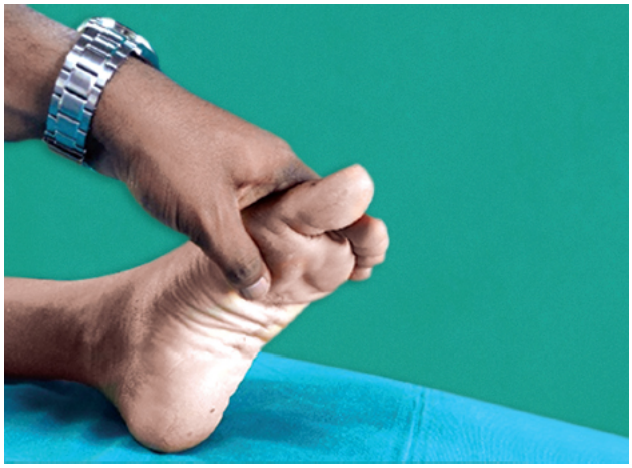


Fig. 6D(iv).53: Peronei—L5, S1.

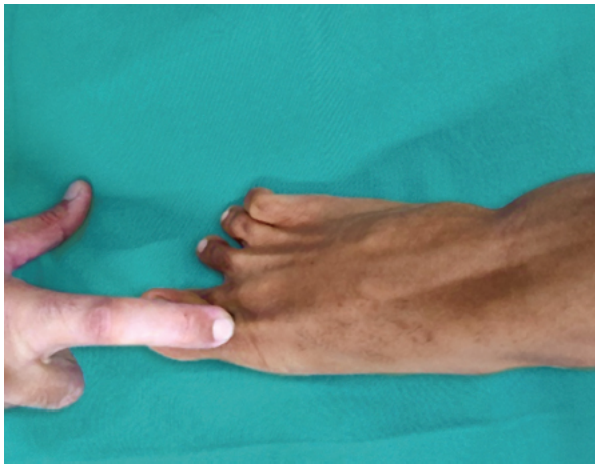


Fig. 6D(iv).56: Extensor hallucis longus—L5, S1.

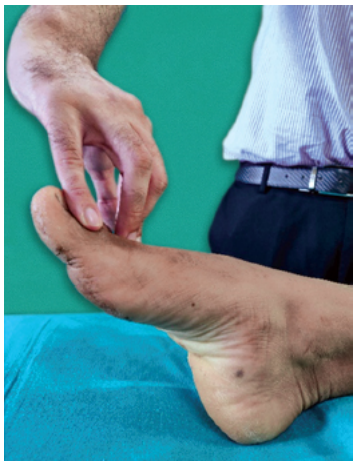


Fig. 6D(iv).54: Extensor digitorum longus—L5.

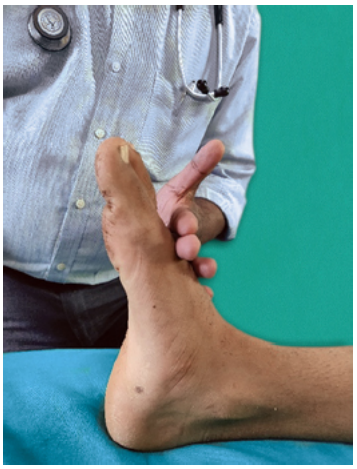


Fig. 6D(iv).57: Extensor digitorum brevis—S1.

EXAMINATION FOR SUBTLE HEMIPARESIS [FIG. 6D(IV).58]

1. **Pronator drift (Barre's sign)**
   - The patient stretches out both arms directly in front of him or her with palms upright (i.e. forearms supinated) and closes his or her eyes.
   - This position is held for 20–30 seconds.

Normal response: • Palm will remain flat, elbows straight and the limbs horizontal OR • Symmetrical deviation from this position (i.e. on both the sides—dominant hand may pronate slightly more than the non-dominant hand)
Positive pronator drift: Components of pronator drift as mentioned above are seen in the weaker side (asymmetric response) which indicates a lesion in contralateral cortex
• Positive with eyes open: Motor deficit • Positive with eyes closed: Sensory deficit (posterior column) • Outward and upward drift: Cerebellar drift • "Updrift" (involved arm rising overhead without patient awareness): Parietal lobe lesions (loss of position sense) • Drift without pronation: Functional upper limb paresis (conversion disorder)

2. **Forearm rolling test [Fig. 6D(iv).59]**
   - The patient bends each elbow and places both forearms parallel to each other.
   - He or she then rotates the forearms about each other, first in one direction and then the other.
   - In the abnormal response, the forearm contralateral to the lesion appears fixed while the other arm rotates around it.
3. **Rapid finger tapping test**
   - The patient rapidly taps the thumb and index finger repeatedly at a speed of about two taps per second.
   - Hemispheric lesions cause the contralateral finger and thumb to tap more slowly and with diminished amplitude.
4. **Foot tapping test**
   - The seated patient taps one forefoot at a time for 10 seconds on the floor, as fast as possible, while the heel maintains contact with the floor.
   - A discrepancy of more than five taps between the left and right foot indicates cerebral disease contralateral to the slower foot.

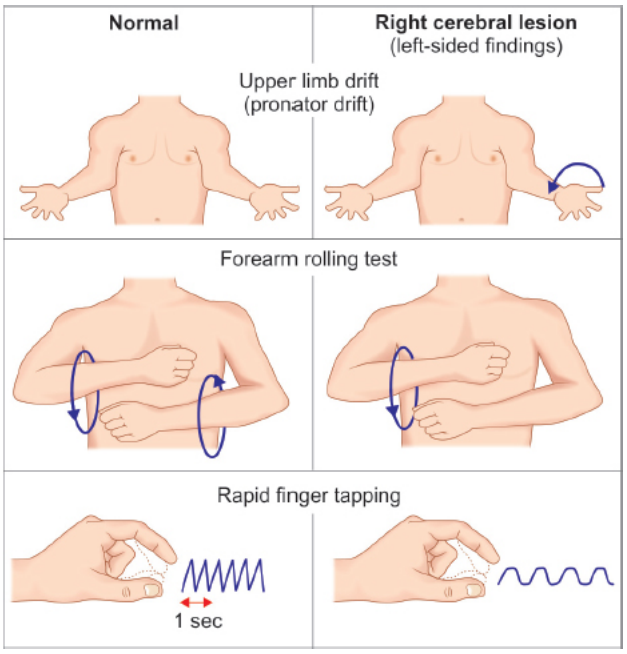


Fig. 6D(iv).58: Examination for subtle hemiparesis.

Fig. 6D(iv).59: Forearm rolling test.

D(v). REFLEXES

DEFINITION

A reflex is an involuntary response to a sensory stimulus.

MECHANISM OF REFLEX GENERATION [FIG. 6D(V).1]

Afferent impulses arising in a sensory organ produce a response in the effector organ. The response can be sensory, motor or autonomic.

It has two components:

Segmental component	Suprasegmental component
It consists of a local reflex center in the spinal cord or brainstem and its afferent and efferent connections	It is made up of descending central pathways that control, modulate, and regulate the segmental activity
	Diseases may increase the activity of some reflexes, decrease activity of others, and causes reflexes to appear that are not normally seen

TYPES OF REFLEXES

1. Deep tendon reflexes (monosynaptic reflex)
2. Superficial reflex (polysynaptic reflex)
3. Plantar reflex
4. Latent reflex
5. Primitive reflexes
6. Inverted and perverted reflexes.

GRADING OF REFLEXES (FOR DTR'S) NINDS SCALE

Absent reflex (even after reinforcement)	Grade 0
Present but diminished	Grade 1+
Normal	Grade 2+
Increased but not necessarily to pathologic degree	Grade 3+
Markedly hyperactive, pathologic, often with extrabeats or accompanying sustained clonus	Grade 4+

REINFORCEMENT MECHANISM AND METHODS

Mechanism

Normally, when a muscle spindle is stimulated two kinds of responses are seen via the following nerves:

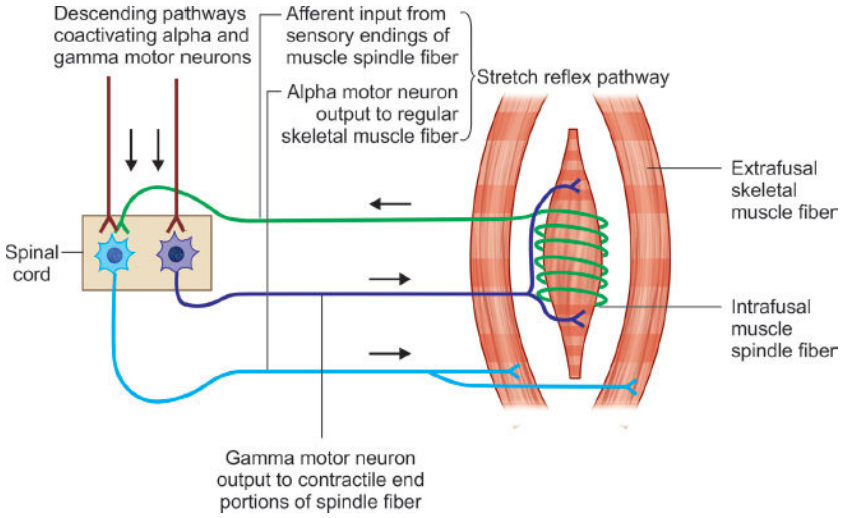


Fig. 6D(v).1: Schematic representation of innervation of muscle fiber and pathways.

Alpha motor neurons	Gamma motor neuron*	Inhibitory neuron
Causes: contraction of **E**xtrafusal fibers of muscle	Causes: contraction of **i**ntrafusal fibers of muscle	Causes: inhibition of reciprocal muscle contraction

*Normally gamma motor neurons are under the inhibitory control of upper motor neurons and reinforcement maneuvers remove the inhibitory effect on gamma motor neurons **[Fig. 6D(v).1]**.

Note: Mnemonic—**A**nti**E**pileptics cause **g**astro **i**ntestinal disturbance. (**A:** Alpha neuron, **E:** Extrafusal fibers), (**G:** Gamma neuron, **I:** Intrafusal fibers).

Reinforcement Maneuvers for Deep Tendon Reflexes (DTRs)

Distraction	Talk to the patient and cause diversion of thought process
Clenching the teeth or clenching the fist of the other arm [Fig. 6D(v).2]	Traditionally done for upper limb
Jendrassik maneuver (interlocking the flexed fingers of the two hands and pull one against each other) [Fig. 6D(v).3]	Preferably done for lower limb

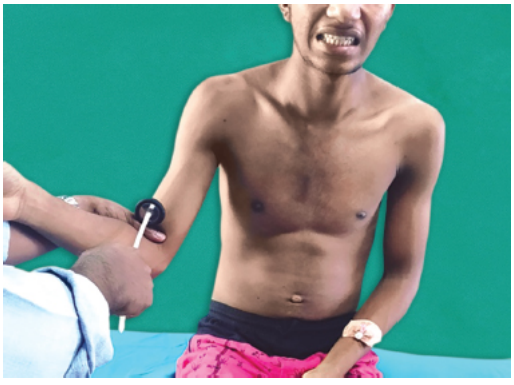


Fig. 6D(v).2: Clenching the teeth for reinforcement of upper limb reflexes.

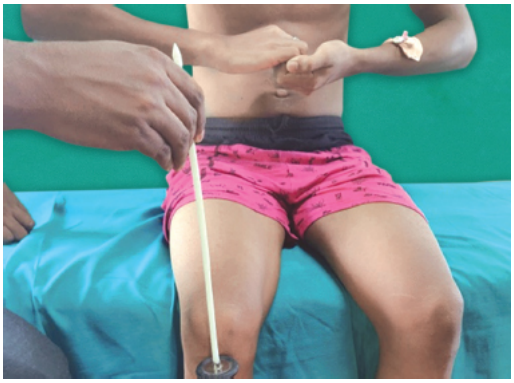


Fig. 6D(v).3: Jendrassik maneuver for reinforcement of lower limb reflexes.

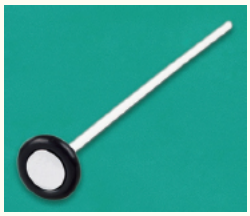
DEEP TENDON REFLEXES

These are monosynaptic reflexes.

Prerequisite for examination:

- Good knee hammer (preferably Queen Square reflex hammer)
- Expose adequately the muscle to be tested
- Make sure patient is not anxious
- The muscle should be placed in optimum position, slightly on stretch, but with plenty of room for contraction.

The most commonly used specialized reflex hammers are grouped into three types by the shape of the head: triangular/tomahawk shaped (Taylor), T-shaped (Tromner, Buck), or circular (Queen square, Babinski)	
Tromner neurological reflex hammer	
Taylor hammer	
Babinski neurological reflex hammer	

Queen square neurological reflex hammer	
Buck neurological reflex hammer	

Reflex	Root value
Biceps	C5C6 (musculocutaneous nerve)
Supinator (brachioradialis)	C5C6 (radial nerve)
Triceps	C7C8 (radial nerve)
Knee	L3L4 (femoral nerve)
Ankle	S1S2 (medial popliteal nerve)
Mnemonic*—*S1,2*: *L3,4*: *C5,6*:*C7,8 *(in sequence from below)*	
Few others	
Pectoral	C5-T1 (medial and lateral pectoral nerves)
Finger flexion	C6-T1 (median nerve)

Reflex	Method of elicitation	Normal response
Biceps [Figs. 6D(v).4A to C]	Press the forefinger gently on the biceps tendon in the antecubital fossa and then strike the finger with the hammer	Flexion of the elbow with visible contraction of the biceps muscle
Supinator [Figs. 6D(v).5A to C]	Strike the lower end of the radius about 5 cm above the wrist and watch for the movement of forearm and fingers	Contraction of brachioradialis and flexion of elbow
Triceps [Figs. 6D(v).6A to D]	By holding the patient's hand draw the arm across the trunk and allow it to lie loosely in the new position. Then strike the triceps tendon 5 cm above the elbow	Extension of elbow with visible contraction of triceps muscle
Knee [Figs. 6D(v).7A to C]	For right-handed examiner, the left arm is under both the knees in order to flex them together and tap the patellar tendon lightly on each side and compare the movements of lower leg and of quadriceps muscle	Extension of the knee and visible contraction of the quadriceps (in case of lower leg amputation keep finger just above the patella with legs extended and strike it in peripheral direction and look for upward pull of patella)
Ankle [Figs. 6D(v).8A to E]	• Patient's leg should be externally rotated and slightly flexed at the knee. Examiner uses the left hand to dorsiflex the foot. For the left leg move to the other side of the bed	Plantar flexion of foot and contraction of gastrocnemius

	• The Achilles tendon is then struck	
Few others		
Pectoral [Fig. 6D(v).9]	With patients arm in the mid position between adduction and abduction hook your index finger on the tendon of the pectoralis major muscle in the anterior fold of axilla and strike with hammer	Adduction of the arm and visible contraction of the pectoralis major
Finger flexion test [Fig. 6D(v).10]	Allow the patient's hand to rest palm upwards, the fingers slightly flexed. The examiner interlocks his fingers with patient's fingers and strikes them with the hammer	Slight flexion of all the fingers and of the interphalangeal joint of the thumb

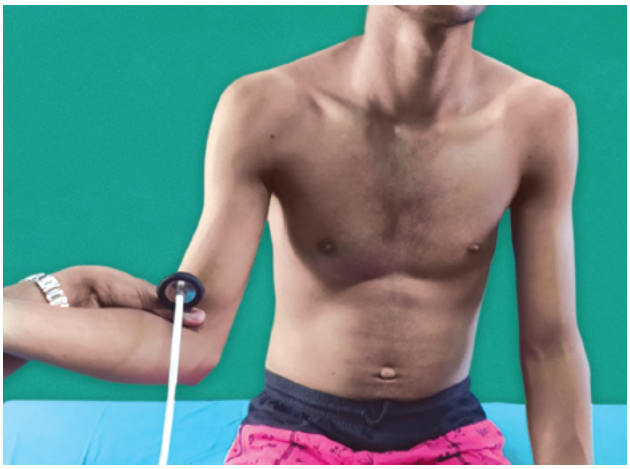


Fig. 6D(v).4A: Demonstration of biceps reflex (right hand).

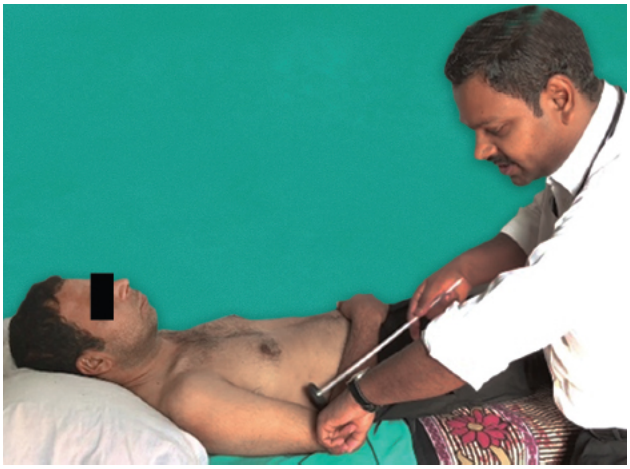


Fig. 6D(v).4B: Demonstration of biceps reflex supine position (right hand).

Fig. 6D(v).4C: Demonstration of biceps reflex (left hand).

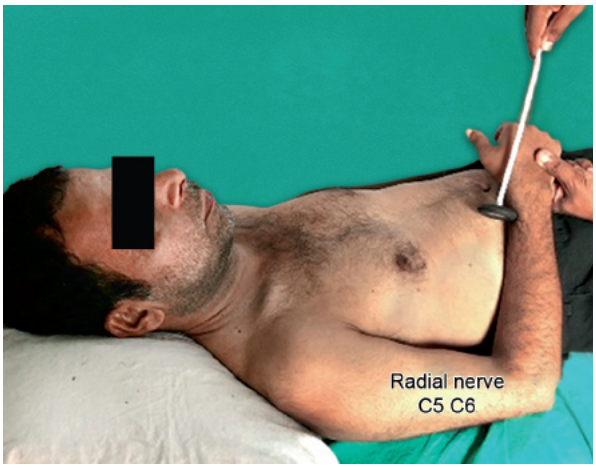


Fig. 6D(v).5C: Demonstration of supinator reflex in supine positi

Fig. 6D(v).5A: Demonstration of supinator reflex (right).

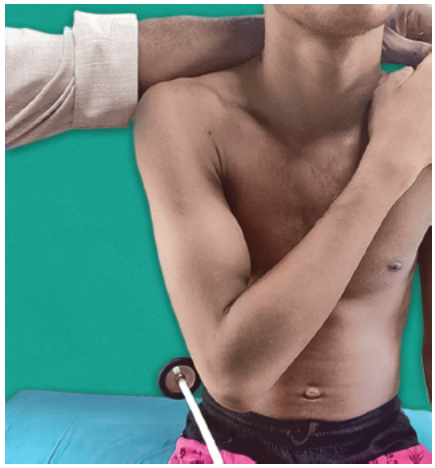


Fig. 6D(v).6A: Demonstration of triceps reflex (right hand).

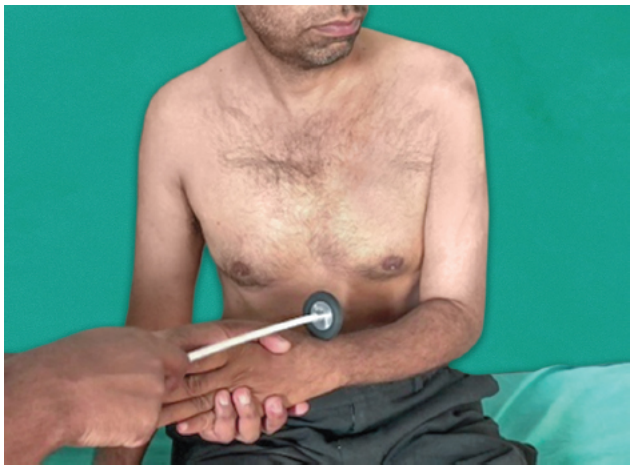


Fig. 6D(v).5B: Demonstration of supinator reflex (left).

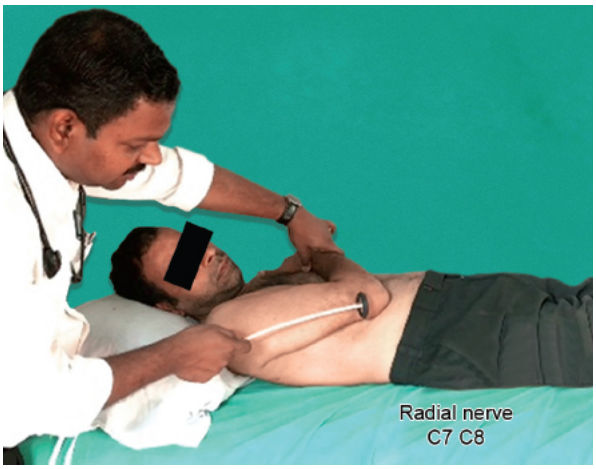


Fig. 6D(v).6B: Demonstration of triceps reflex (right hand) in sup position.

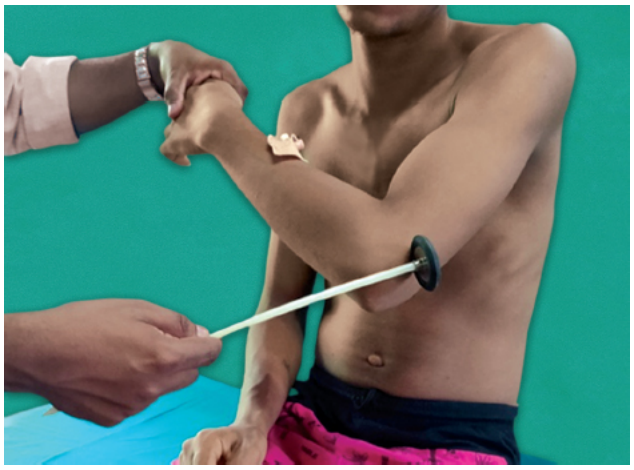


Fig. 6D(v).6C: Demonstration of triceps reflex (left hand).

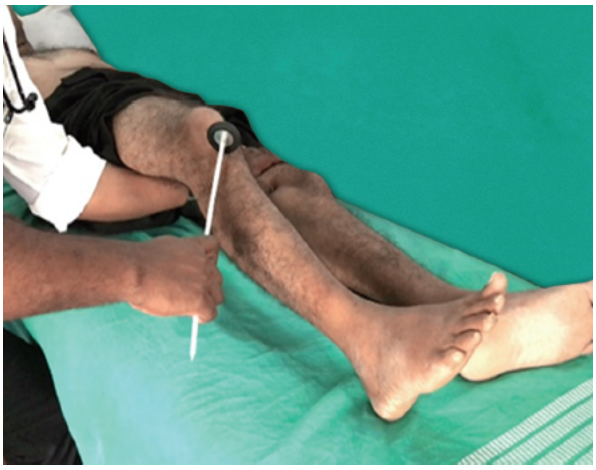


Fig. 6D(v).7B: Demonstration of right knee jerk in supine positi

Fig. 6D(v).6D: Demonstration of triceps reflex (left hand) in supine position.

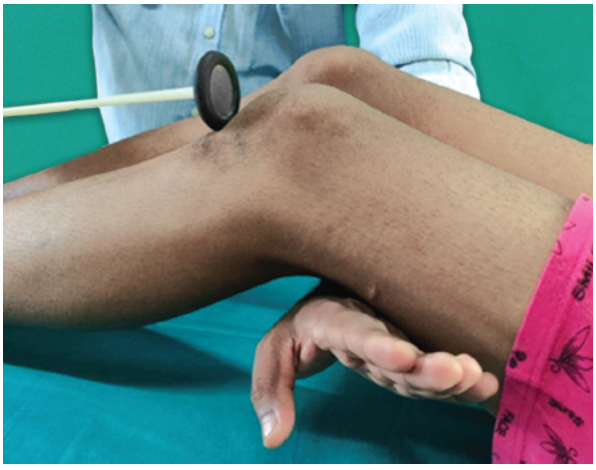
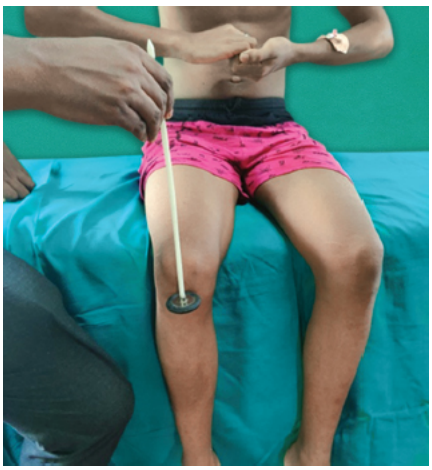


Fig. 6D(v).7C: Demonstration of knee jerk (for comparing bot sides).



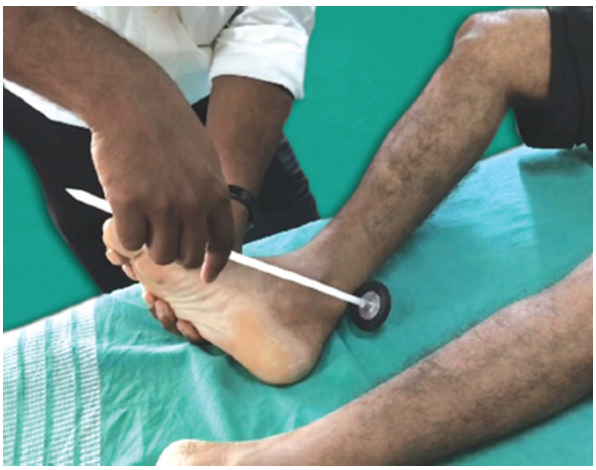


Fig. 6D(v).7A: Demonstration of knee jerk sitting position (for pendular movement).

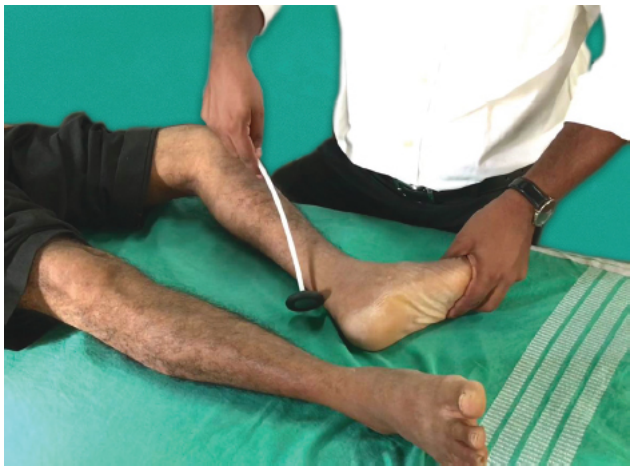


Fig. 6D(v).8B: Demonstration of ankle reflex of left leg.

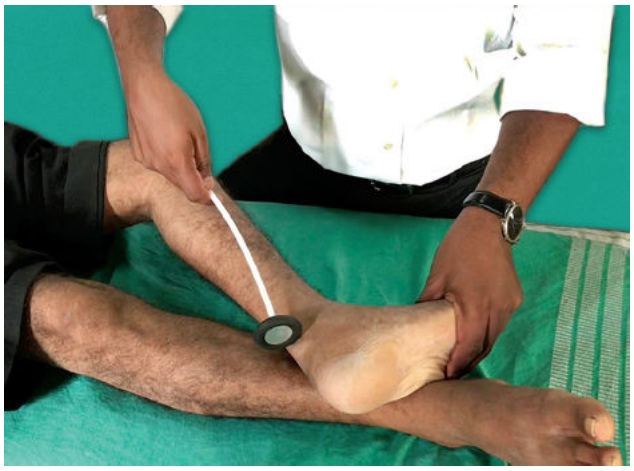


Fig. 6D(v).8C: Demonstration of ankle reflex of left leg.

Fig. 6D(v).8A: Demonstration of ankle reflex of right leg.

Fig. 6D(v).8E: Demonstration of ankle reflex with foot dangling c the edge of table.

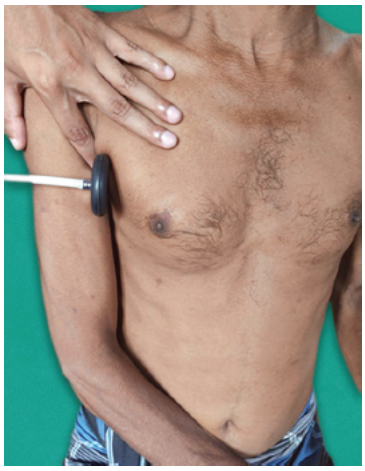


Fig. 6D(v).9: Demonstration of pectoral reflex.

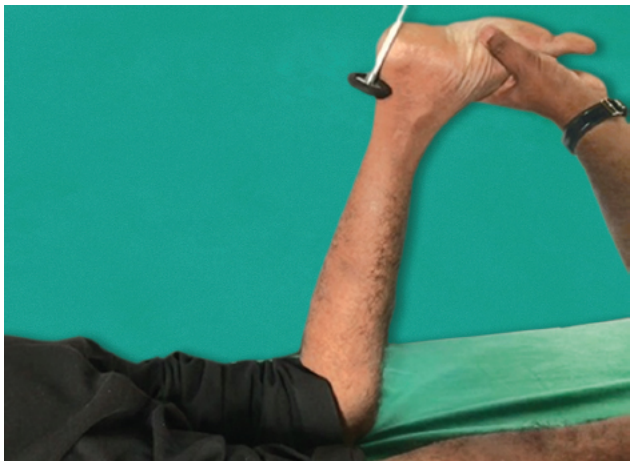


Fig. 6D(v).8D: Demonstration of ankle reflex in prone position.

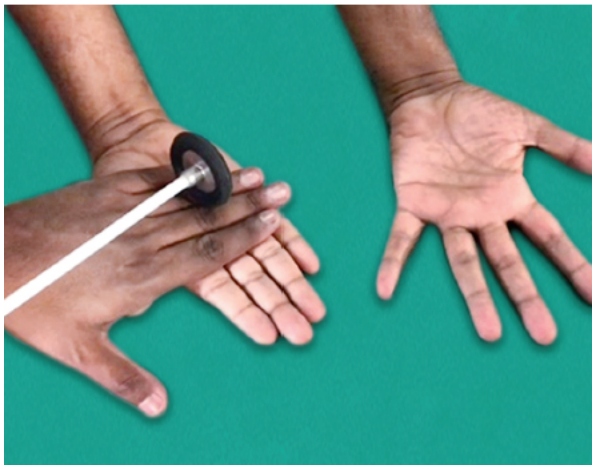


Fig. 6D(v).10: Demonstration of finger flexion reflex.

Clonus

Clonus is a series of rhythmic involuntary muscular contractions induced by the sudden passive stretching of a muscle or tendon.

Clonus	Demonstration
Ankle clonus [Figs. 6D(v).12A and B]	Examiner supports the leg, preferably with one hand under the knee, grasps the foot from below with the other hand, and quickly dorsiflexes the foot while maintaining slight pressure on the sole at the end of the dorsiflexion • The leg and foot should be well relaxed, the knee and ankle in moderate flexion, and the foot slightly everted • Right ankle clonus is examined by standing on the right side of the patient and left ankle clonus by standing on the left side • Unsustained clonus fades away after a few beats; sustained clonus persists as long as the examiner continues to hold slight dorsiflexion pressure on the foot
Patellar clonus [(Figs. 6D(v).11A and B]	Examiner grasps the patella between index finger and thumb and executes a sudden, sharp, downward thrust, holding downward pressure at the end of the movement
Wrist clonus	Sudden passive extension of the wrist produces wrist clonus

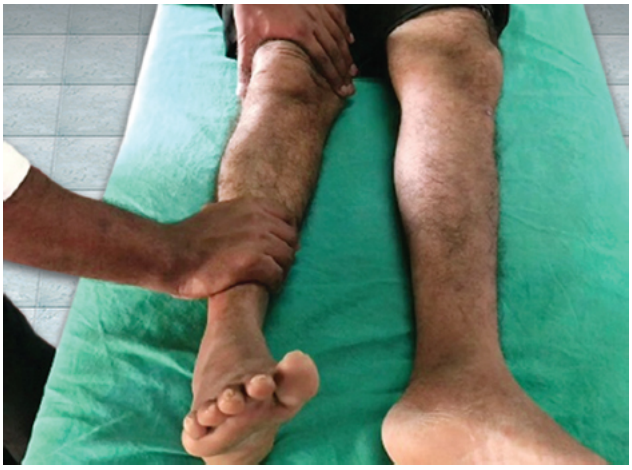


Fig. 6D(v).11A: Demonstration of right patellar clonus.

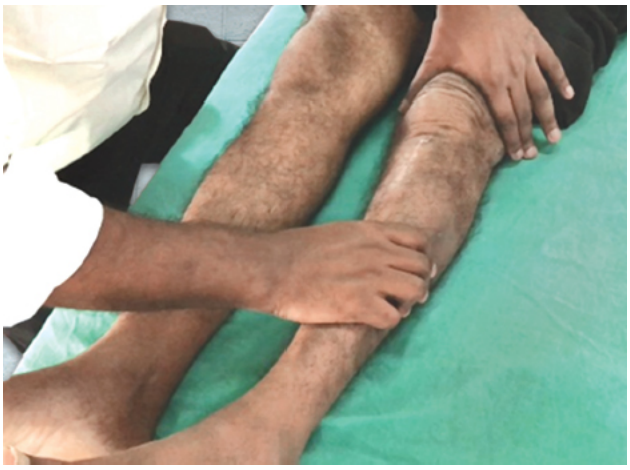


Fig. 6D(v).11B: Demonstration of left patellar clonus.

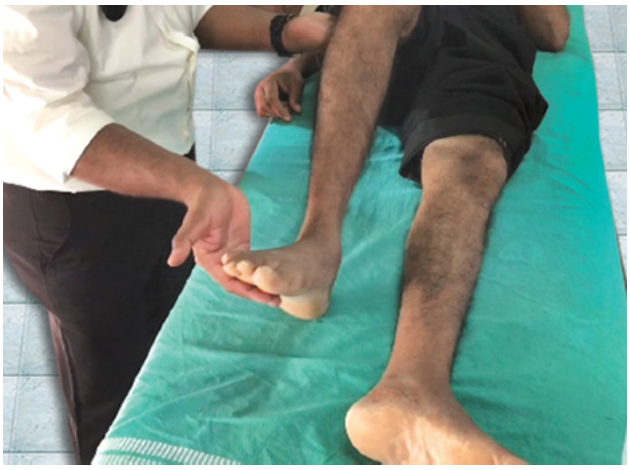


Fig. 6D(v).12A: Demonstration of right ankle clonus.

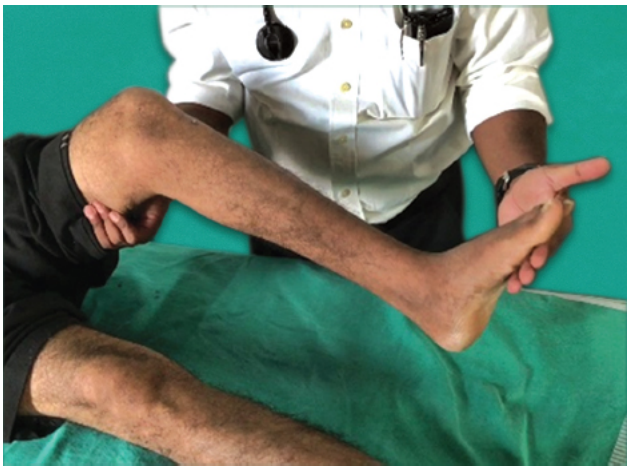


Fig. 6D(v).12B: Demonstration of left ankle clonus.

SUPERFICIAL REFLEXES

These are the responses to stimulation of either the skin or mucous membrane.

Clinical Significance

Superficial reflexes are abolished by pyramidal tract lesions.

Superficial reflex	Deep tendon reflex
Polysynaptic reflexes	Monosynaptic reflexes
Respond slowly	Faster response
Latency is longer	Latency is slower
Fatigue easily	Fatigue slowly
Not as consistently present as deep tendon reflexes	Consistently present
Abolished by pyramidal tract lesions	Exaggerated by pyramidal tract lesions

Superficial reflex	Elicitation
Corneal (cranial nerve V and VII)	Lightly touching the upper cornea with wisp of cotton or tissue, brought in from the side so the patient cannot see
Abdominal [Fig. 6D(v).13] • **Epigastric (T6-T9)** • **Mid abdominal (T9-T11)** • **Hypogastric (T11-L1)**	Stimulus is delivered by stroking the abdominal wall (preferably towards the umbilicus) and watch for contractions
Cremasteric [Fig. 6D(v).14] (L1, L2)	Stroking the skin in upper inner aspect of thigh and watch for the upward movement of testes in scrotum
Anal reflex (S2, S3)	Contraction of external sphincter in response to stroking the skin or mucous membrane in the perianal region
Bulbocavernosus reflex (S2, S3) [Fig. 6D(v).15]	Contraction of anal sphincter which is best appreciated by a gloved finger in the rectum on stimulation of glans penis or clitoris

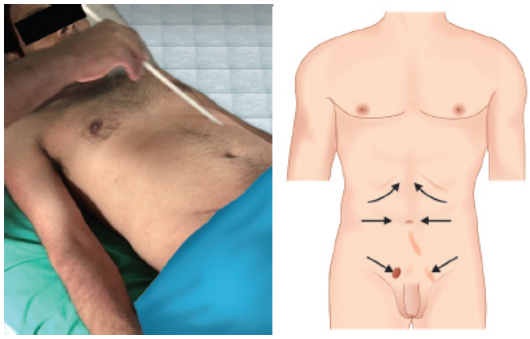


Fig. 6D(v).13: Demonstration of abdominal reflex.

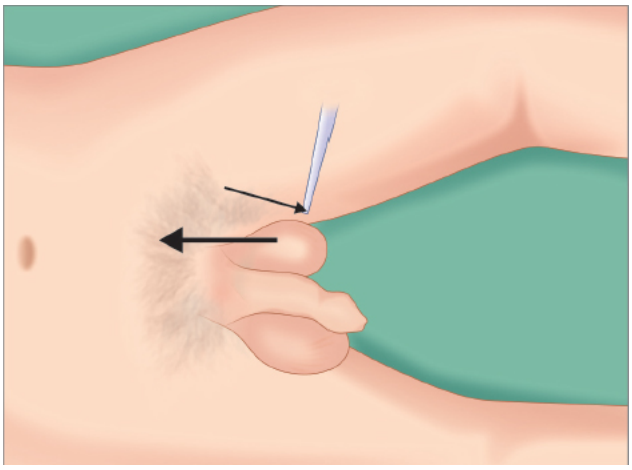


Fig. 6D(v).14: Direction of stimulus and movement of testes in cremasteric reflex.

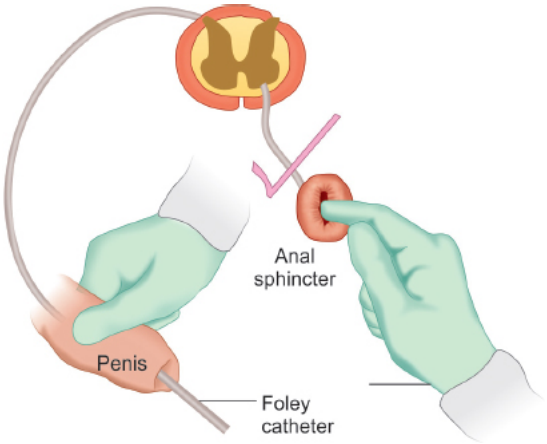


Fig. 6D(v).15: Pictorial representation of bulbocavernosus reflex.

PLANTAR REFLEX AND VARIATIONS

Plantar Reflex

Stroking the plantar surface of foot from the heel forward is normally followed by plantar flexion of foot and toes.

Babinski Sign

It is the pathologic variation of plantar reflex (i.e. extensor plantar response). It is part of primitive flexion reflex. In higher vertebrates, the flexion response includes flexion at hip, flexion at knee, and dorsiflexion of ankle (all of which help in removing the threatened part form danger). Normally the descending motor pathway suppresses the primitive flexion response.

Positioning of patient [Fig. 6D(v).16]	• Best position is supine • Knee must be extended • Heels should rest on the bed
Prerequisites	Rule out ankylosis of great toe
Stimulating agent	• Applicator stick • Blunt key

	• Hand of reflex hammer • Broken tongue blade • Thumb nail
Strength of stimulus	Variable strength with strong stimulus for thick soles and minimal stimulation when response is strongly extensor
Site of stimulus	• Reflexogenic area of S1 • Stimulus should begin near the heel on the lateral aspect of sole and carried up to metatarsophalangeal joint of little toe and then carried medially falling short of 1st metatarsophalangeal joint **[Fig. 6D(v).17]**
Normal response	Flexion of the great toe and other toes
Abnormal response (Babinski sign)	• Dorsiflexion of great toe and small toes • Fanning of toes • Dorsiflexion of ankle • Flexion of knee joint • Flexion at hip joint • Contraction of tensor fascia lata
Reinforcement of plantar reflex	By asking patient to rotate the head to opposite side

Fig. 6D(v).16: Position of leg for demonstration of plantar reflex.

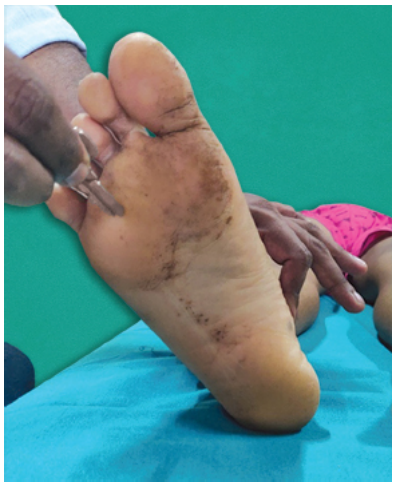


Fig. 6D(v).17: Direction of stimuli for demonstrating the plantar reflex.

Variants of Plantar Response

Equivocal response	• Rapid extension followed by flexion • Only great toe extension • Extension of great toe with flexion of fingers • No response to the plantar stimulus • Flexion at hip and knee, but no movement of toes
Minimal plantar response	• No toe movement • Contraction of tensor fascia lata with mild internal rotation and abduction of hip
Pseudo Babinski	• Voluntary extension of great toe due to hyperesthesia or strong painful stimulus • Dystonic posturing of great toe

Other method of obtaining plantar reflex

Method	Elicitation
Chaddock [Fig. 6D(v).18]	• Elicited by stimulating the lateral aspect of the foot, not the sole, beginning about under the lateral malleolus near the junction of the dorsal and plantar skin, drawing the stimulus from the heel forward to the small toe • The Chaddock is the only alternative toe sign that is truly useful • It may be more sensitive than the Babinski but is less specific • It produces less withdrawal than plantar stimulation
Reverse Chaddock	The stimulus moves from the small toe toward the heel
Oppenheim [Fig. 6D(v).19]	• Dragging the knuckles heavily down the anteromedial surface of the tibia from the infrapatellar region to the ankle. • The response is slow and often occurs toward the end of stimulation
Shaeffer's sign [Fig. 6D(v).20]	Deep pressure on Achilles tendon
Gordon's sign [Fig. 6D(v).21]	Squeezing of calf muscles
Bing's sign [Fig. 6D(v).22]	Pricking dorsum of foot with a pin
Moniz' sign [Fig. 6D(v).23]	Forceful passive plantar flexion at ankle

Throckmorton's sign	Percussing over dorsal aspect of metatarsophalangeal joint of great toe just medial to EHL tendon
Stransky	Small toe forcibly abducted, then released
Szapiro	Pressure against dorsum of second through fifth toes, causing firm passive plantar flexion while stimulating plantar surface of foot
Strümpell's phenomenon	Forceful pressure over anterior tibial region
Cornell response	Scratching dorsum of foot along inner side of EHL tendon
Combining two methods may elicit minimal reflexes **[Fig. 6D(v).24]**	

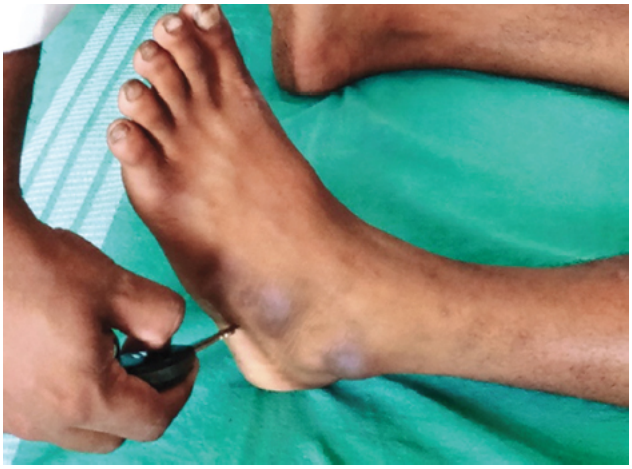


Fig. 6D(v).18: Chaddock's sign.

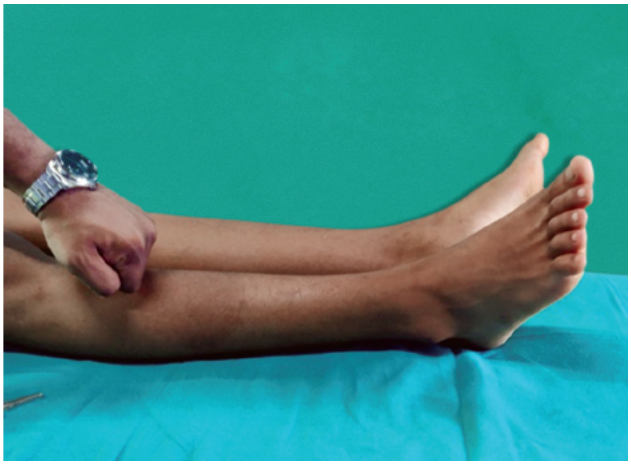


Fig. 6D(v).19: Openheim's technique.

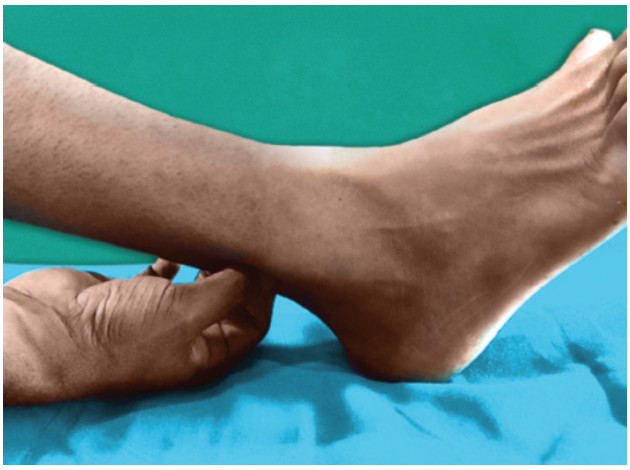


Fig. 6D(v).20: Shaeffer's technique.

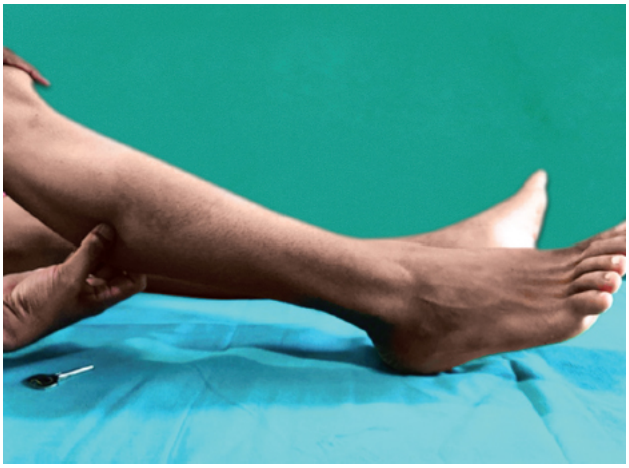


Fig. 6D(v).21: Gordon's technique.

Fig. 6D(v).22: Bing's sign.

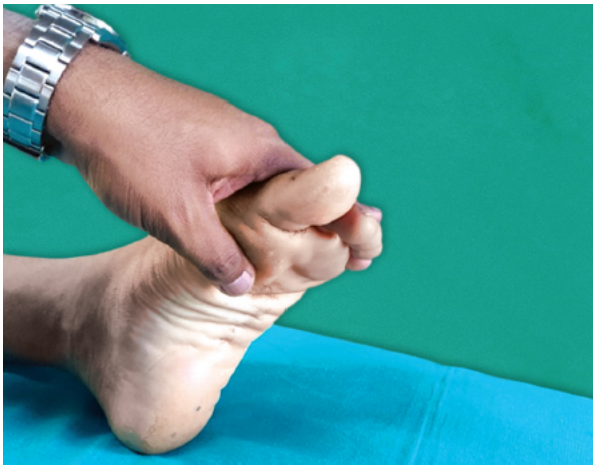


Fig. 6D(v).23: Moniz's sign.

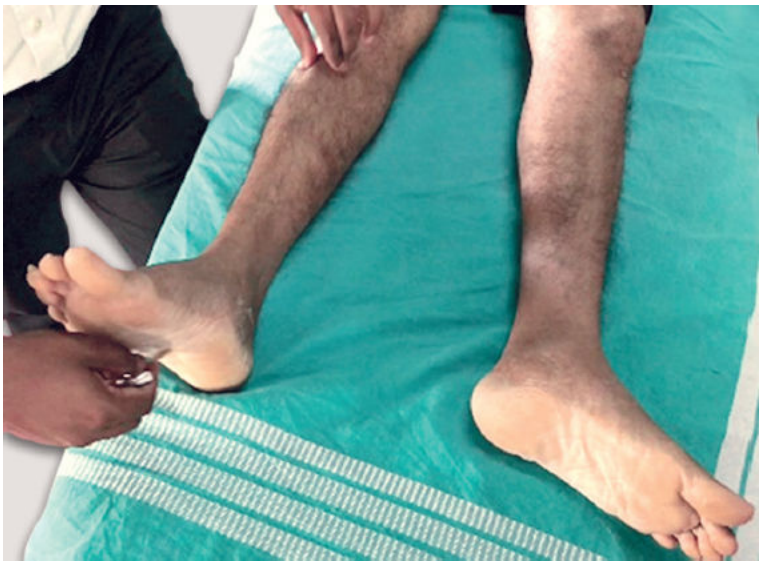


Fig. 6D(v).24: Eliciting plantar by simultaneous stimulus from Openheim's and plantar strike.

LATENT REFLEXES OF UPPER LIMB

Reflex	Elicitation
Wartenberg's reflex [Fig. 6D(v).25]	Patient's fingers are interlocked with examiner's fingers and pulled apart. Normally thumb extends. However in pyramidal lesions thumb is adducted and flexed. This sign is equivalent of Babinski of lower limb
Hoffman's reflex [Fig. 6D(v).26]	Flexion of the interphalangeal joint of middle finger of patient produces flexion response in other fingers along with adduction of thumb
Tromner's reflex [Fig. 6D(v).27]	Examiner holds the patient's partially extended middle finger, letting the hand dangle, then, with the other hand, thumps or flicks the finger pad. The response is the same as that in the Hoffmann test

Fig. 6D(v).25: Wartenberg's sign.

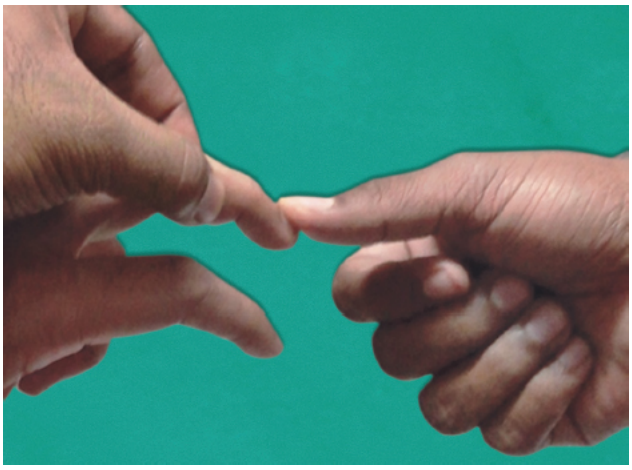


Fig. 6D(v).26: Hoffman's reflex.

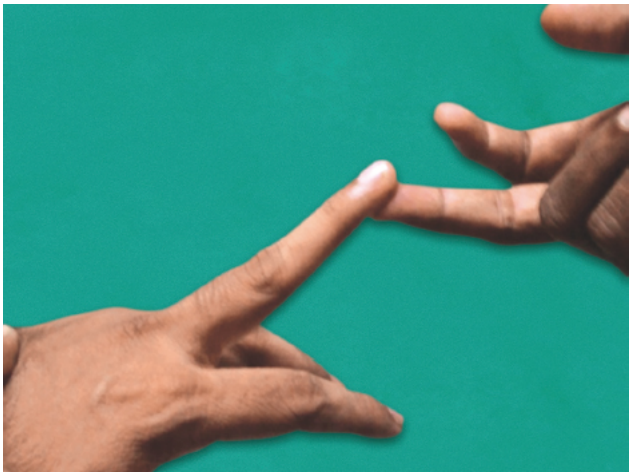


Fig. 6D(v).27: Tromner's reflex.

PRIMITIVE REFLEXES

Reflex	Elicitation
Glabellar tap (Myerson's sign) [Fig. 6D(v).28]	Repetitive tapping of the forehead between the eyebrows causing blinking, which usually stops within few taps. However if blinking persists, it suggests positive frontal release sign. *Note:* To avoid visual stimulus bring the hand from above and behind
Palmomental reflex of Marinesco–Radovici [Fig. 6D(v).29]	• Stroke the thenar eminence in a proximal to distal direction using a sharp object such as the pointed end of a reflex hammer, key, paper clip, or fingernail and watch for twitch of chin muscle • This reflex does not have any localizing value, and is commonly seen in elderly patients with degenerative disease of the cortex
Sucking reflex [Fig. 6D(v).30]	Sucking reflexes may be seen in response to tactile stimulation in the oral region, or in response to the insertion of an object (for example, a spatula) into the mouth
Rooting reflex [Fig. 6D(v).31]	Rooting responses are seen when the mouth turns towards an object gently stroking the cheek (tactile rooting), or towards an object (for example, tendon hammer) brought into the patient's field of view (visual rooting)
Pout and snout reflex	The snout reflex is present when the lips pucker in response to gentle pressure over the nasal philtrum

[Fig. 6D(v).32]	
Grasp reflex [Fig. 6D(v).33]	• If the examiner's fingers are placed in the patient's hand, especially between the thumb and forefinger, or if the palmar skin is stimulated gently, there is slow flexion of the digits • The patient's fingers may close around the examiner's fingers

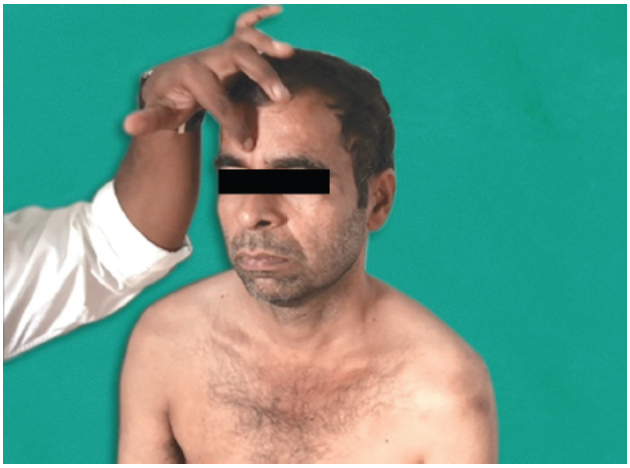


Fig. 6D(v).28: Glabellar tap.

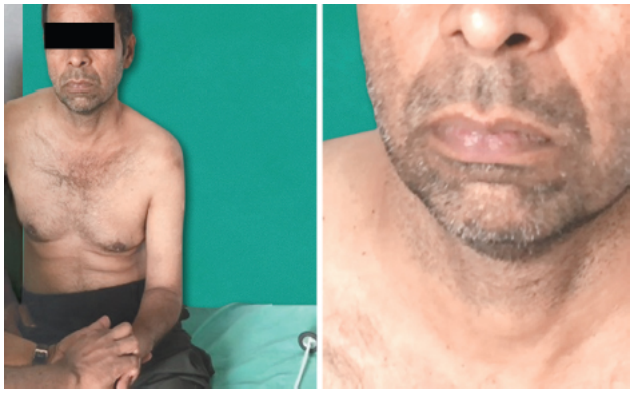


Fig. 6D(v).29: Palmomental reflex.

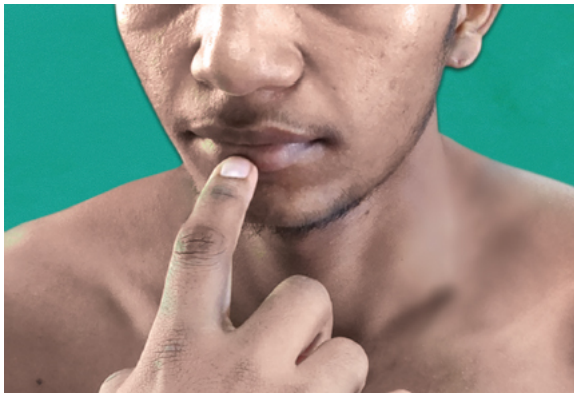


Fig. 6D(v).30: Sucking reflex.

Fig. 6D(v).31: Rooting reflex.

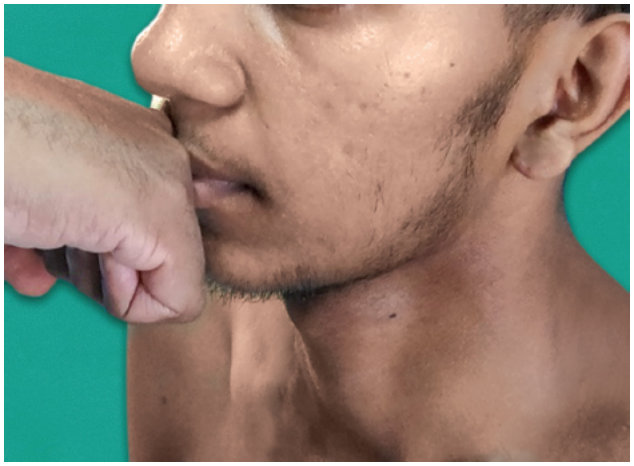


Fig. 6D(v).32: Pout reflex.

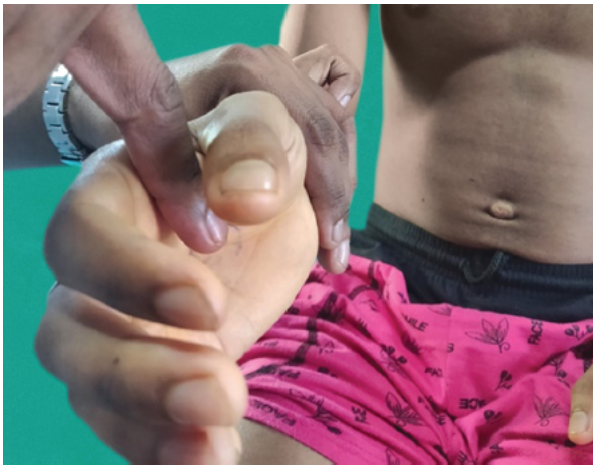


Fig. 6D(v).33: Grasp reflex.

INVERTED AND PERVERTED REFLEXES

Reflex	Description and example
Inverted reflex	Contractions opposite to that of expected For example: **An inverted brachioradialis reflex:** • When the supinator reflex elicits finger flexion and not elbow flexion • Is associated with an absent biceps jerk and an exaggerated triceps jerk • Is indicative of a spinal cord lesion at C5 or C6, e.g. due to trauma, syringomyelia, or disc prolapse **Inversion of biceps reflex** • On eliciting bicep reflex the following are noticed: – There is no flexion at the elbow – But instead there is extension at the elbow due contraction of the triceps muscle • Presence of this reflex indicates that the lesion is at the level of C5 segment **Inversion of triceps reflex** With disc protrusions at C6/7 there is a “paradoxical triceps reflex” with forearm muscles acting to flex the elbow against no triceps resistance

	Inversion of knee reflex • On eliciting the knee jerk • There is no extension of the knee joint • But instead there is flexion of the knee due to contraction of the hamstring muscles • Presence of this indicates that the lesion is at the level of L3, 4
Perverted reflex	It is false inverted reflex where there is an alteration in the response rather than true inversion For example: When supinator jerk is elicited there is a perverted response of finger flexion. (*Note:* In the presence of brachioradialis reflex this phenomenon is called as spread of reflex, while in the absent of brachioradialis reflex this is considered as pseudo inverted reflex or perverted reflex)

Other Causes of Altered Reflexes
Thyroid disease **Woltman's sign** of myxedema, is the delayed relaxation phase of the muscle stretch reflex. In hypothermia or β-blockade, the relaxation phase of the ankle jerk may be prolonged.
Chorea: "**Hung-up" knee jerk** is a specific but rarely appreciated clinical sign of Huntington disease (HD) and Sydenham chorea. During an elicited knee jerk, the extended lower leg may not relax immediately but may remain elevated for several seconds due to sustained contraction of the quadriceps femoris.
Very brisk reflexes—even with a few beats of clonus can be seen in anxious individuals, as well as in hyperthyroidism and in tetany.
Electrolyte disturbances • Absent reflexes is seen with hypermagnesemia. • In the **Holmes Adie syndrome,** absent deep tendon reflexes are seen.

SENSORY SYSTEM EXAMINATION

Sensations can be grossly divided into primary and secondary modalities

Primary modalities	Secondary modalities (cortical sensation)
Touch	Tactile localization
Pressure	2 point discrimination
Pain	Sensory inattention
Temperature	Stereognosis
Joint position sense	Graphesthesia
Vibration	These require secondary association area in parietal lobe

Note: When primary sensation are normal but secondary modalities are lost it implies a parietal lobe lesion.

Sherrington classification of sensory system	
Exteroceptive system	Information about the external environment, including somatosensory functions and special senses
Proprioceptive system	Senses the orientation of the limbs and body in space
Interoceptive system	Information about internal functions, blood pressure, or the concentration of chemical constituents in bodily fluids

PRIMARY MODALITIES

Examination of Exteroceptive System (Spinothalamic Tract)

Pain

- Ask the patient to close his eyes.
- Sharp end of pin is applied mildly sufficient to produce pain but not to penetrate the skin **[Fig. 6D(vi).1]**.
- Compare adjacent normal area and corresponding area on the opposite side.
- Indicate whether sensation is normal, decreased (or absent) or increased.
- In peripheral nerve disease, there is anesthesia more than analgesia.
- In spinal cord disease, there is analgesia more than anesthesia.
- Commonly used objects are the safety pin or broken wooden applicator stick.
- Avoid too sharp objects and hypodermic needles.
- A useful trick is to hold the pin or shaft of the applicator stick lightly between thumb and fingertip and allow the shaft to slide between fingertip and thumb. This ensures consistent stimulus intensity.

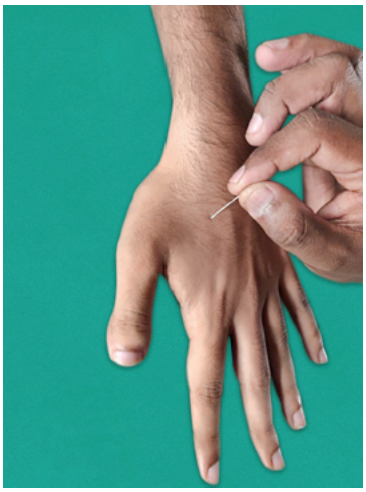


Fig. 6D(vi).1: Examination of pin prick sensation.

Temperature [Fig. 6D(vi).2]

- With the patient's eyes closed, apply the warm and cold test tubes randomly over the skin in dermatomal pattern.
- Instruct the patient to say what he feels–hot/cold/no response.
- Cold = 5°C to 10°C (41°F to 50°F) (crushed ice can be used).
- Warmth = 40°C to 45°C (104°F to 113°F) (warm water can be used).
- Temperature much lower or higher than these elicit pain rather than temperature sensations.
- In lesions of leprosy, temperature may be lost prior to pain.

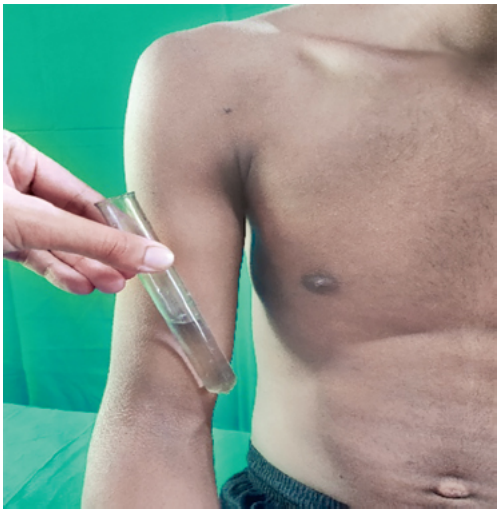


Fig. 6D(vi).2: Examination of temperature.

Tactile Sensation

- Light touch can be tested with a:
  - Wisp of cotton **[Fig. 6D(vi).3]**
  - Feather
  - Soft brush **[Fig. 6D(vi).4]**
  - Light touch of the fingertip.

- For diabetic neuropathies
  - Von Grey's hairs
  - Semmelweis monofilament
- With patient's eyes closed, gently touch the skin (preferably non-hairy region) without exerting pressure.
- Ask the patient whether he can feel the touch.
- Tactile response can be graded as per international spinal injury standards as
  - 0 = absent
  - 1 = altered response (impaired/increased)
  - 2 = normal/intact response.

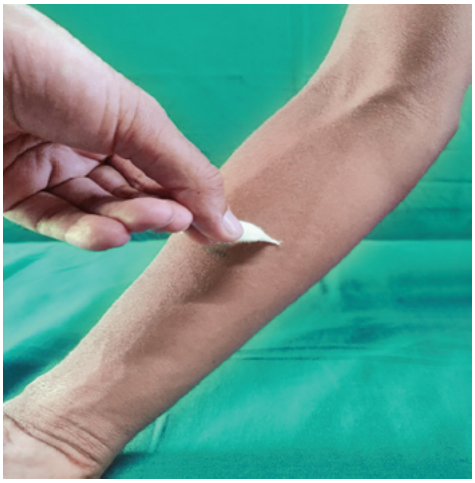


Fig. 6D(vi).3: Examination of tactile sensation with wisp of cotton.

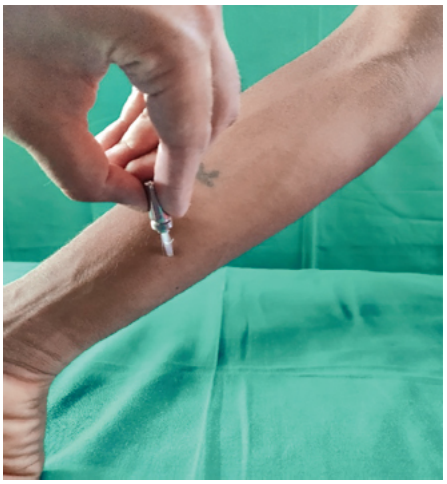


Fig 6D.vi.4: Examination of tactile sensation with soft brush.

Examination of Proprioceptive System

Proprioception (Proprioception refers to either the sense of position of a body part or motion of a body part)	
Conscious component	***Unconscious component***
Travels with the fibers subserving fine, discriminative touch.	Via spinocerebellar tract

These include: Motion Position Vibration Pressure	

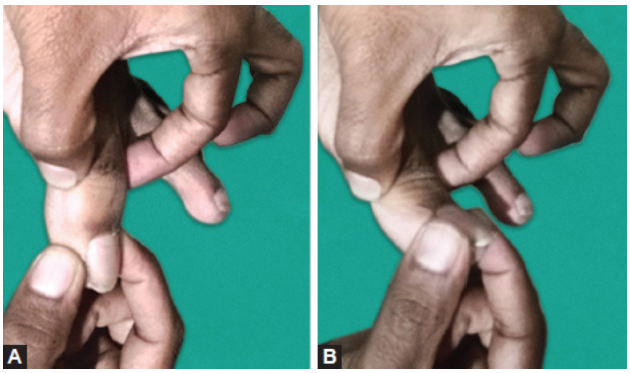
Examination of different components of proprioception: Joint motion and position:

- Usually tested together
- In the lower extremity **[Figs. 6D(vi).5A and B]**:
- Tested at the metatarsophalangeal joint of the great toe,
- In the upper extremity **[Figs. 6D(vi).6A to C]**:

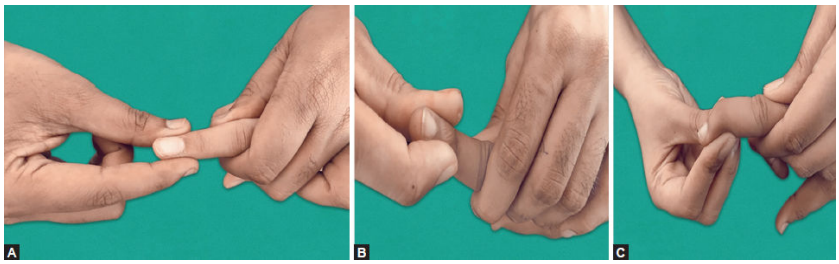
At one of the distal interphalangeal joints. If these distal joints are normal, there is no need to test more proximally.

Joint motion:

- Testing is done with the patient's eyes closed.
- It is extremely helpful to instruct the patient, eyes open, about the responses expected before beginning the test.
- Show the patient up or down movements and instruct him to reply "up" or "down".
- The examiner should hold the patient's completely relaxed digit on the sides, away from the neighboring digits, parallel to the plane of movement, exerting as little pressure as possible to eliminate clues from variations in pressure.
- The part is then passively moved up or down, and the patient is instructed to indicate the direction of movement from the last position.
- Healthy young individuals can detect great toe movements of about 1 mm, or 2° to 3°; and in the fingers virtually invisible movements, 1° or less, at the distal interphalangeal joint are accurately detected.



Figs. 6D(vi).5A and B: Examination of joint sense in the lower limb.



Figs. 6D(vi).6A to C: Examination of joint sense in upper limb.

Position sense:

- Tested by placing the fingers of one of the patient's hands in a certain position (like "OK" sign) **[Fig. 6D(vi).7]** while his eyes are closed, and then asking him to imitate it with the other hand OR do passive movement in one hand and ask the patient to do in similar way in other hand **[Fig. 6D(vi).8]**.
- This is sometimes referred to as **parietal copy**. Light touch can be tested with a wisp of cotton, tissue paper, a feather, a soft brush, light stroking of the hairs, or even using a very light touch of the fingertip. Both parietal lobes (and their connections) must be intact: one side to register the position and the other side to copy it.

Vibration (pallesthesia) [Figs. 6D(vi).9A to C]: Preferentially using a tuning fork of 128Hz due to slow decay (256 Hz is used to detect early changes in cases like subacute combined cord degeneration).

Fig. 6D(vi).7: Examination of position sense (OK sign).

- Explain procedure to patient clearly.
- Strike the tuning fork and place on the forehead and explain the difference between vibration and plain touch of tuning fork, by dampening the vibration by holding the prongs.

Fig. 6D(vi).8: Examination of position sense by asking to copy passive movement.

Fig. 6D(vi).9A: Demonstration of vibration over proximal great toe.

Fig. 6D(vi).9B: Demonstration of vibration over medial malleolus.

- Keep the vibrating tuning fork, starting from the distal most bony prominence and proceed proximally.
- Ask the patient to say when he ceases to feel the vibration.

Timed vibration test:

- It is the most sensitive and simple method to quantify defects in vibration.
- Note the time duration of perception of vibration after the tuning fork is set into vibration.
- Normally
  - ≥10 sec in lower limb.
  - ≥20 sec in upper limb.

Rhomberg's sign [Figs. 6D(vi).10A and B]:

- It is a sign of posterior column dysfunction.
- Ask the patient to stand upright with feet/heels close together, arms by the side and eyes open.
- Any significant swaying is noted.
- Now, ask the patient to close the eyes while taking adequate measures to make sure patient does not fall and hurt himself.
- Watch for swaying
  - Minimal swaying is normal.

– Immediate gross swaying is considered as positive test.

Pseudoathetosis [Fig. 6D(vi).11]:

- It is an upper limb equivalent of examination of posterior column dysfunction.
- Ask the patient to hold the upper limb in extended position and close the eyes.
- Watch for slow writhing movements of fingers (piano-playing movement) which disappear on opening the eyes.

Pressure pain:

- Tested by squeezing the Achilles tendon or calf muscle.
- Abadie's sign is loss of deep pain (seen with diseases affecting the posterior column like neurosyphilis–tabes dorsalis).

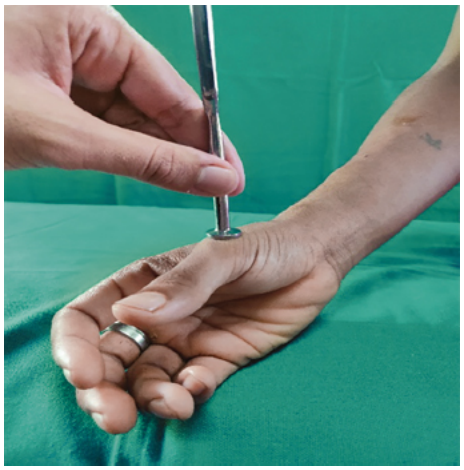


Fig. 6D(vi).9C: Demonstration of vibration over the proximal 1st metacarpopharyngeal joint.

Figs. 6D(vi).10A and B: Demonstration of Rhomberg's sign.

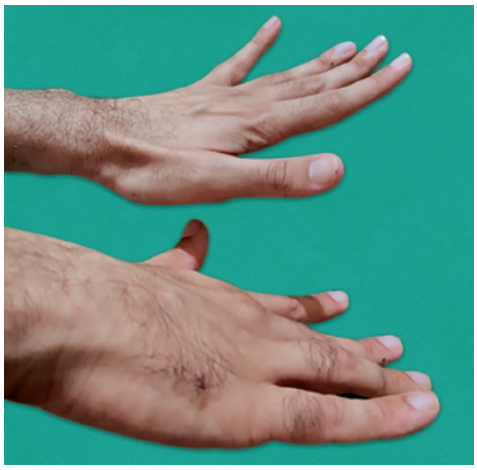


Fig. 6D(vi).11: Demonstration of pseudoathetosis in upper limb.

SECONDARY MODALITIES

Cortical Sensations

Cortical sensations cannot reliably be tested unless primary sensation is intact bilaterally.

Two-point discrimination [Fig. 6D(vi).12]: Ability to recognize simultaneous stimulation by two blunt points. Measured by the distance between the points required for recognition. The normal distances at which two points can be discriminated on various body parts:

- Tongue tip: 1 mm
- Fingertip: 2 to 4 mm
- Dorsum of fingers: 4 to 6 mm
- Palm: 8 to 12 mm
- Dorsum of hand: 20 to 30 mm
- Skin over the back : 30–40 mm.

Tactile localization (topognosis):

Ability to localize stimuli to parts of the body. Topagnosia is the absence of this ability.

Graphesthesia [Fig. 6D(vi).13]:

Ask the patient to close their eyes and identify letters or numbers that are being traced onto their palm or the tip of their finger.

Stereognosis [Figs. 6D(vi).14A and B]:

Ask the patient to close their eyes and identify various objects by touch using one hand at a time.

Tactile extinction (double simultaneous stimulation) [Figs. 6D(vi).15A and B]

- Ability to perceive a sensory stimulus when corresponding areas on the opposite side of the body are stimulated simultaneously. Loss of this ability is termed sensory extinction (perceptual rivalry/sensory suppression).
- The site of lesion is contralateral parietal lobe.

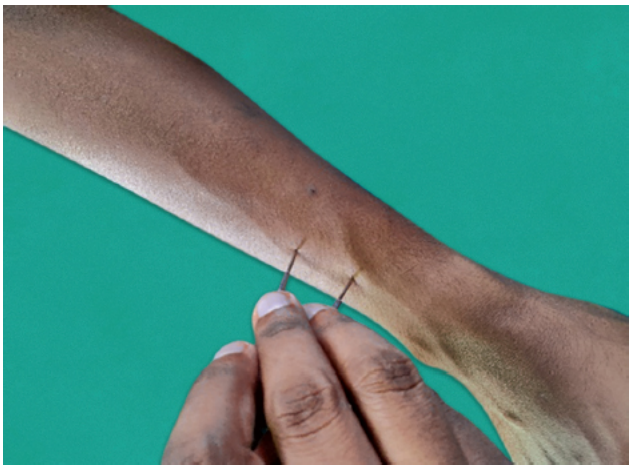


Fig. 6D(vi).12: Demonstration of 2 point discrimination.

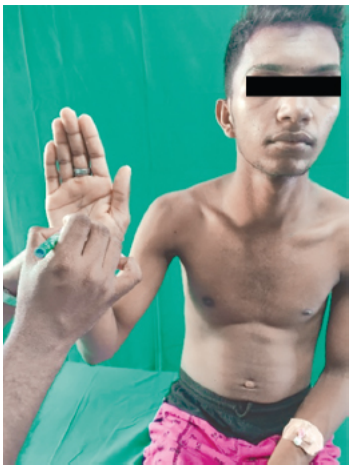


Fig. 6D(vi).13: Demonstration of graphesthesia.

Fig. 6D(vi).14A: Demonstration of stereognosis with key.

Fig. 6D(vi).14B: Demonstration of stereognosis with coin.

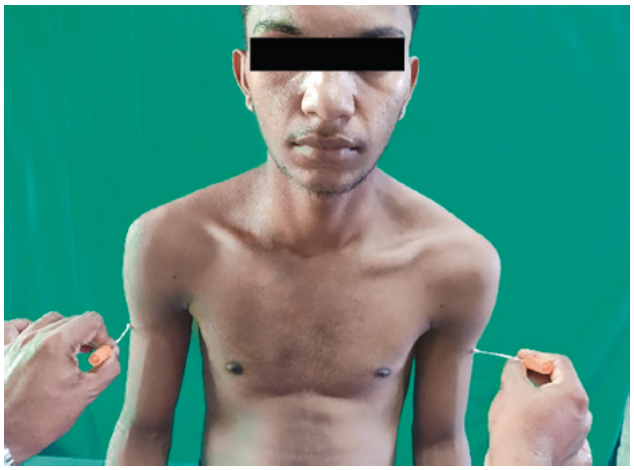


Fig. 6D(vi).15A: Demonstration of tactile extinction in upper limb.

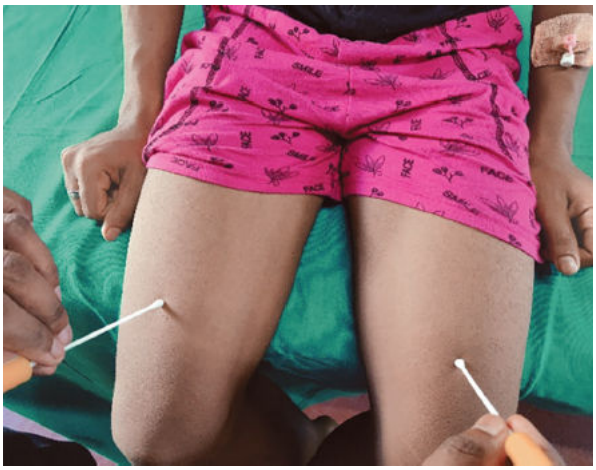


Fig. 6D(vi).15B: Demonstration of tactile extinction in lower lim

Disorders of touch	
Anesthesia	Absence of touch appreciation.
Hypoesthesia	Decrease in touch appreciation.
Hyperesthesia	Exaggeration of touch sensation, which is often unpleasant.
Paresthesia	Abnormal sensations perceived without specific stimulation. They can include wide variety of abnormal sensation except pain; episodic or constant.
Hyperpathia	Exaggerated reaction to any stimuli (touch/pressure/pain).
Disorders of pain	
Analgesia	Absence of pain appreciation.
Hypoalgesia	Decrease in pain appreciation.
Hyperalgesia	Exaggeration of pain appreciation, which is often unpleasant.
Allodynia	Perception of non-painful stimulus as painful.

Causalgia	Persistent pain, allodynia or hyperalgesia along with abnormal pseudomotor activity (edema and blood flow changes). It is also called as reflex sympathetic dystrophy.
Phantom limb pain	Individuals who have had a limb amputated may experience pain or tingling sensations that feels as if they were coming from the amputated limb, just as if that limb were still present. These individuals experience pain or tingling sensations that feel as if they were coming from the amputated limb, just as if that limb were still present.
Central or thalamic pain	Spontaneous, inexplicable, agonizing pain and other unusual sensations in the anesthetic parts.
Disorders of temperature	
Thermanalgesia	Absence of temperature appreciation
Thermhypoesthesia	Decrease of temperature appreciation
Thermhyperesthesia	Exaggeration of temperature sensation, which is often unpleasant
Disorders of posterior column sensations	
Arthranesthesia	Absence of joint position sense (**Arthresthesia**—perception of joint position sense)
Apallesthesia/Pallanesthesia	Absence of vibration sense

Barognosis (recognition of weight)

- The ability to recognize different weights.
- A set of discrimination weights consisting of small objects of the same size and shape but of graduated weights are used.

HOMUNCULUS, SENSORY PATHWAY, DERMATOMES AND CLINICAL PATTERNS OF SENSORY LOSS

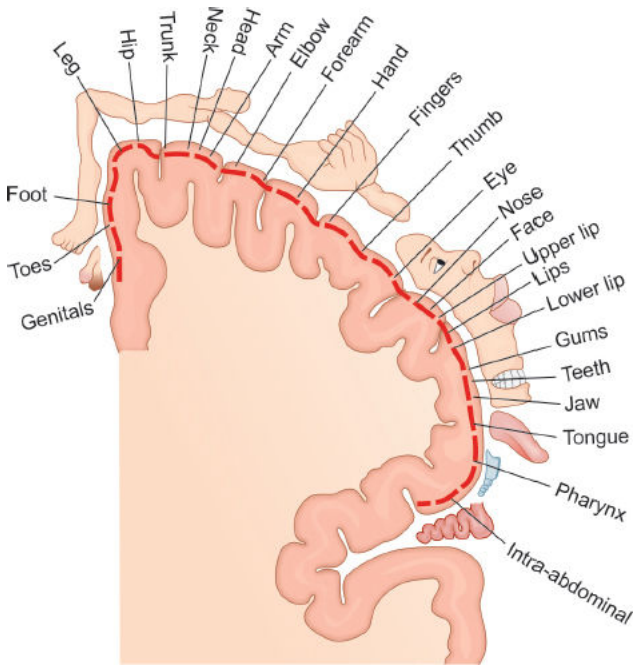


Fig. 6D(vi).16: Sensory homunculus.

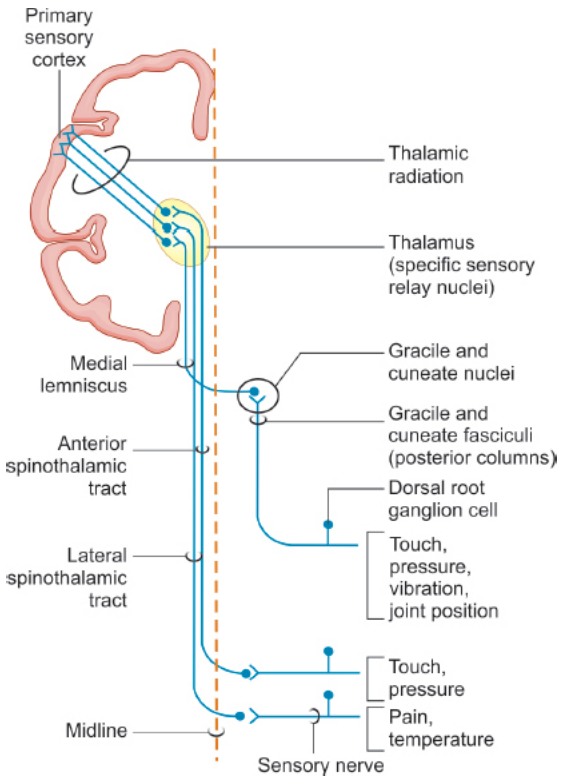


Fig. 6D(vi).17: Sensory pathway.

Sensation	Receptor	Pathway	Decussation
Pain and thermal sense from the body	Aδ and C fiber endings	Spinothalamic tract of anterolateral system (ALS)	Anterior white commissure
Nondiscriminative (crude) touch and superficial pressure from the body	Free nerve endings, Merkel's disks, peritrichial nerve endings	Spinothalamic tract of ALS	Anterior white commissure
Two-point discriminative (fine) touch, vibratory sense, proprioceptive sense from muscles and joints of body	Meissner's corpuscles, Pacinian corpuscles, muscle stretch receptors, Golgi tendon organs	First order fibers: Fasciculi gracilis and cuneatus Second order fibers: Medial lemniscus	Medial lemniscal decussation

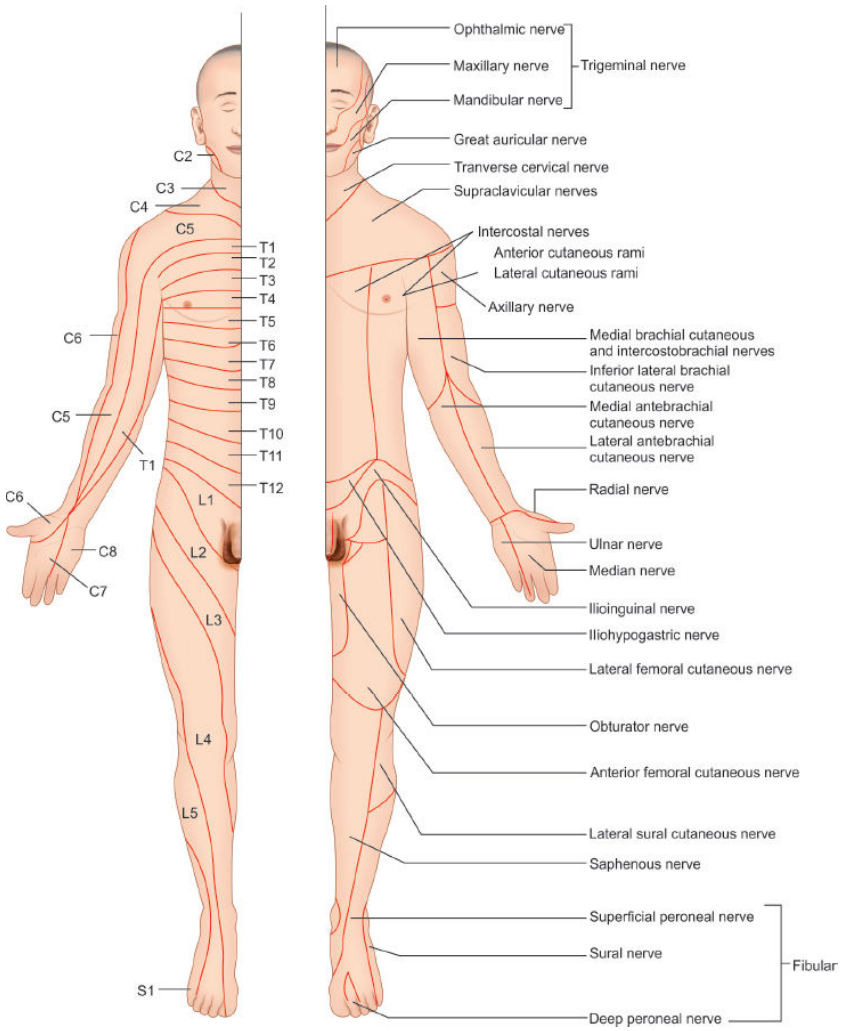


Fig. 6D(vi).18: Anterior view of skin segment innervation.

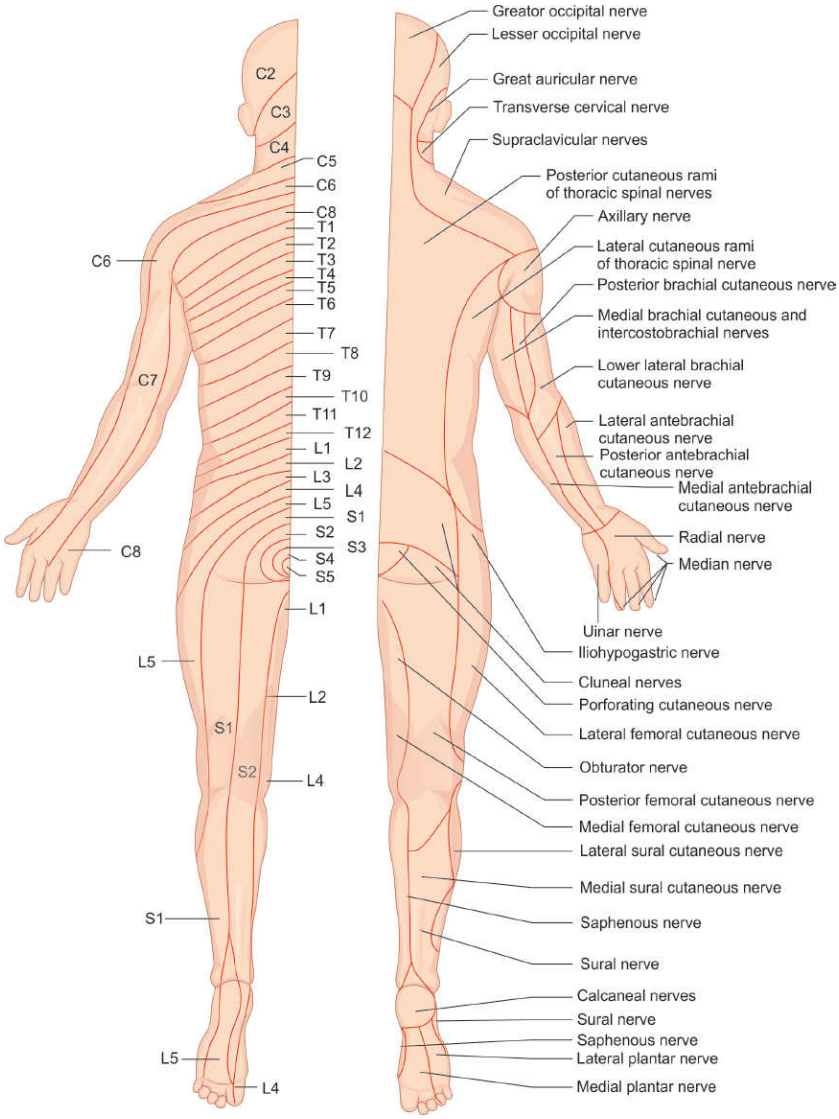
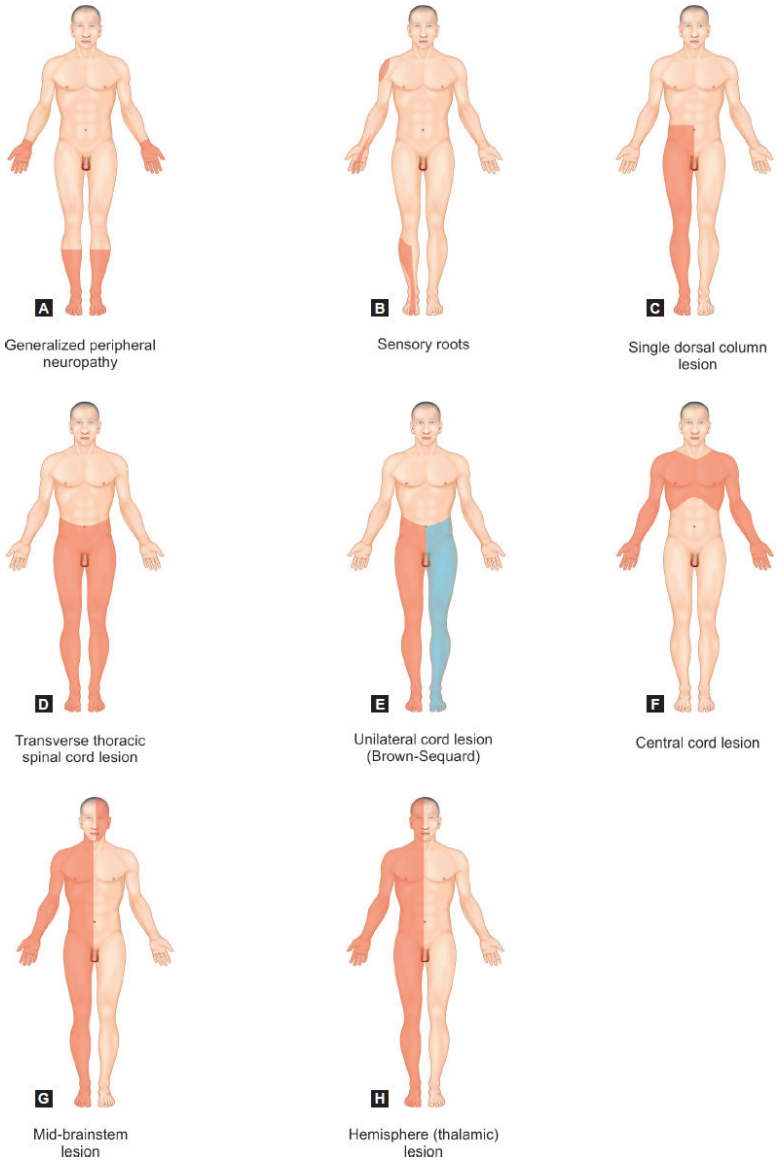


Fig. 6D(vi).19: Posterior view of skin segment innervation.

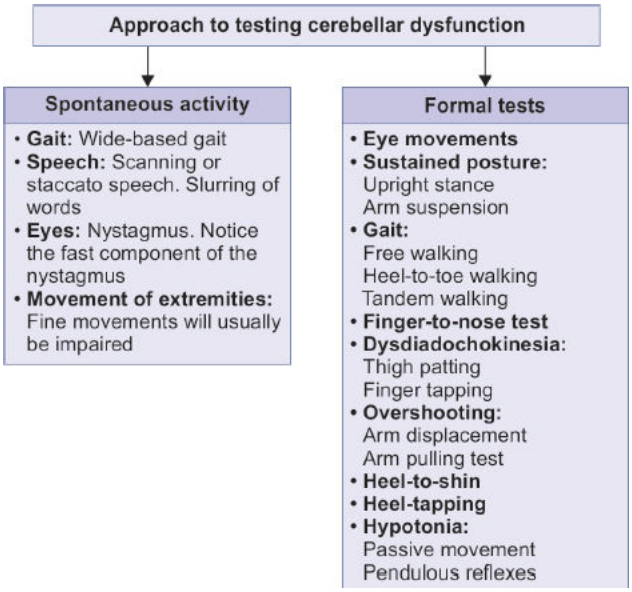


Figs. 6D(vi).20A to H: Clinical patterns of sensory dysfunction.

D(vii). CEREBELLUM AND COORDINATION

SIGNS OF CEREBELLAR DISORDERS

Deficit	Manifestation
Ataxia	Reeling, wide-based gait.
Decomposition of movement	Inability to sequence fine, coordinate acts correctly. *This is usually tested while performing the finger-nose test which requires a fine coordination between shoulder, elbow, and wrist joint. Patients with a cerebellar lesion will find it difficult to perform such movements.*
Dysarthria	Inability to articulate words correctly, usually manifesting as slurring and/or inappropriate phrasing.
Dysdiadochokinesia	Inability to perform rapid, alternating movements.
Dysmetria	Inability to control or limit the range of movement.
Hypotonia	Decrease in muscle tone.
Nystagmus	Involuntary rapid oscillation of eyeballs in a horizontal, vertical or rotationary fashion with the fast component of nystagmus maximal towards the side of the cerebellar lesion.
Scanning/Staccato speech	Slow explosive enunciation with a tendency to hesitate at the beginning of each word or each syllable. *Asking the patient to pronounce a word with multiple syllables, such as Mississippi or Venkataramana will elicit distinct pauses before each syllable.*
Tremor	Rhythmic, alternating, oscillatory movements which affects a limb as it approaches a target (Intention tremor) or of proximal musculature when attempting to bear weight (postural tremor).



Hypotonia
- Usually accompanies acute hemispheric lesions.
- Interestingly, it is seen less often in chronic lesions.
- Ipsilateral to the side of a cerebellar lesion.
- More noticeable in upper limbs and proximal muscles.
- Pendular knee jerk: Leg keeps swinging after knee jerk more than 4 times (4 or less is considered normal).

Ataxia
- Defective timing of sequential contraction of agonist/antagonist muscles.
- Results in a disturbance in smooth performance of voluntary acts (errors in rate, range, force, duration).
- May affect limbs, trunk, gait (depends on the part of cerebellum involved).

Asynergia
Lack of synergy of various muscles while performing complex movements (movements are broken up into isolated, successive parts. This is known as decomposition of movement).

Dysmetria OR abnormal excursions in movement
- **Finger-to-nose test**
  - With eyes open, the patient is asked to partially extend elbow and rapidly bring tip of index finger in a wide arc to tip of his nose.
  - In cerebellar disease, the action may manifest an intention tremor.
  - With eyes closed, sense of position in the shoulder and elbow is tested.
- **Heel-to-shin test**
  - Patient is asked to place one heel on opposite knee and slide the heel down the tibia with foot dorsiflexed.
  - Movement should be performed accurately.
  - In cerebellar disease, the arc of the movement is jerky/wavering.
  - The slide down the shin may manifest an action tremor.

Dysdiadochokinesia OR impaired performance of rapidly alternating movement
Normal coordination includes ability to arrest one motor impulse and substitute the opposite.
There are several simple clinical methods to test this:
- Alternating movements (pronate and supinate forearm and hand quickly): In cerebellar disease, the movements tend to overshoot or are inadequate resulting in irregular or inaccurate movements.
- Rapidly tap fingers on the table.
- Open and close fists.
- Stewart-Holmes rebound sign.

Have the patient pull on your hand and when they do, slip your hand out of their grasp. Normally the antagonists muscles will contract and stop their arm from moving in the desired direction. A positive sign is seen in a spastic limb where the exaggerated "rebound" occurs with movement in the opposite direction. However, in cerebellar disease, this response is completely absent causing the limb to continue moving in the desired direction. (Be careful that you protect the patient from the unrestricted movement causing them to strike themselves).

Past pointing
Overshoot is also commonly seen as part of ataxic movements and is sometimes referred to as past pointing, when the patient overshoots while reaching target (finger-to-nose test)

Cerebellar dysarthria
- Abnormalities in articulation and prosody (together or independent).
- Scanning, slurring, staccato, explosive, hesitant, garbled speech.
- Hemisphere lesions are associated with speech disorders more often than vermal lesions.
- Causes enunciation of individual syllables: *"the British Parliament" becomes "the Brit-tish Par-la-ment."*

Intention tremor—occurs during goal-directed movements. Intention tremor results when the antagonist activation that normally stops a goal-directed movement as the goal is approached is inappropriately sized or timed.

Oculomotor dysfunction
- Nystagmus frequently seen in cerebellar disorders.
- Gaze-evoked nystagmus, upbeat nystagmus, rebound nystagmus, optokinetic nystagmus may all be seen in midline cerebellar lesions.

Gait
- In cerebellar disease, the gait is staggering/lurching/wavering.
- Lesion in mid-cerebellum: Movements are in all directions.
- Lesion in lateral cerebellum: Staggering/falling is toward the side of the lesion.
- Somewhat steadied by standing or walking on a wide base.

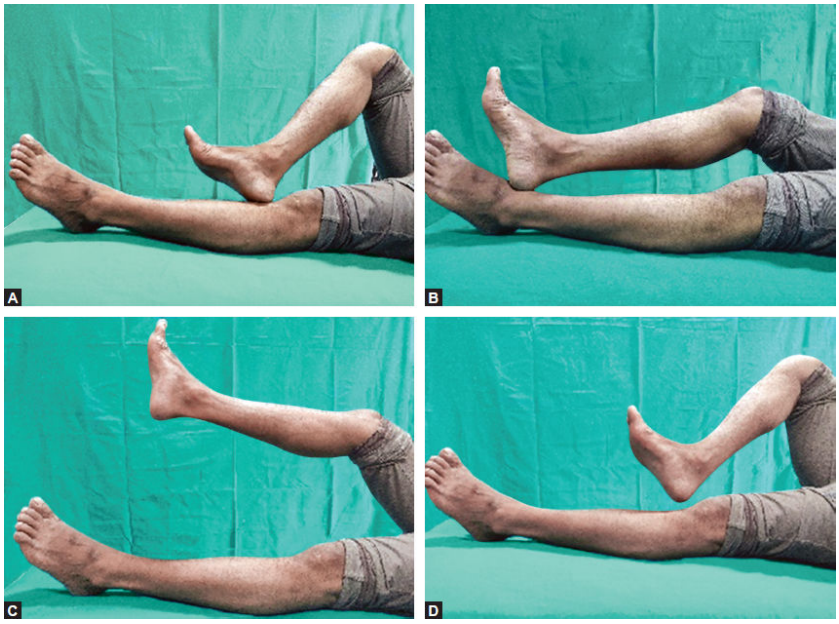
Position of feet
Ataxia from cerebellar disease is less when the patient stands on a broad base (feet widely apart).
Eyes open or closed
Cerebellar ataxia is not improved by visual orientation; ataxia from posterior column disease (disordered proprioception) is worsened with the eyes closed.
Direction of Falling

Disease of lateral lobe of cerebellum causes falling to ipsilateral side.
Lesions of midline/vermis cause indiscriminate falling depending on initial stance of the patient.

Titubation
Consists of a rhythmic body or head tremor. There is a rotatory, rocking or bobbing movement. *Clinically, this does not have significant value in localizing the lesion with respect to the part of the cerebellum involved.*

HEEL KNEE TEST [FIGS. 6D(VII).1A TO D]

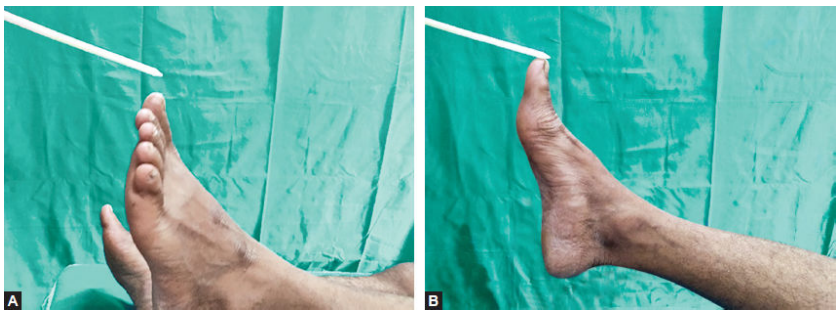
The patient is asked to touch the heel of one foot to the opposite knee and then to drag their heel in a straight line all the way down the front of their shin and back up again. In order to eliminate the effect of gravity in moving the heel down the shin, this test should always be done in the supine position.



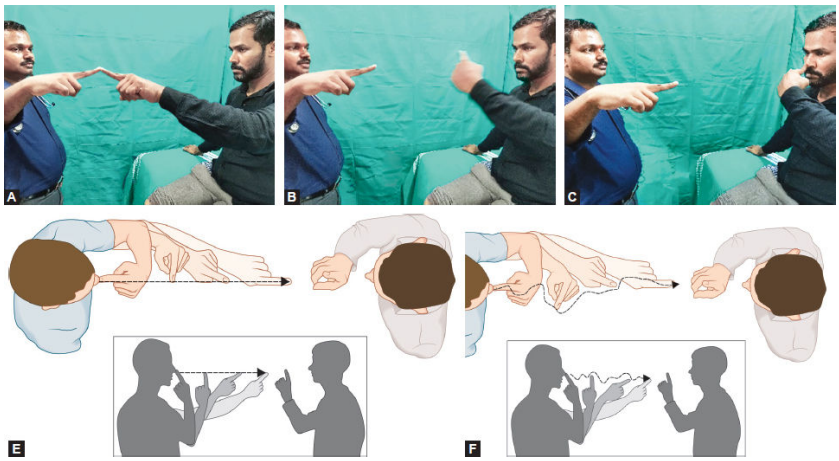
Figs. 6D(vii).1A to D: Demonstration of heel knee test.

TOE FINGER TEST [FIGS. 6D(VII).2A AND B]

Patient lies in bed and is asked to touch his great toe to the examiners fingers or any object held above the bed within his reach.



Figs. 6D(vii).2A and B: Demonstration of toe finger test.



Figs. 6D(vii).3A to E: Showing demonstration of nose finger nose test.

Nose-finger-nose test [Figs. 6D(vii).3A to E] in which the patient is asked to alternately touch their nose and the examiner's finger as quickly as possible. Abnormality of this is called as dysmetria.

FINGER NOSE TEST [FIGS. 6D(VII).4A AND B]

Figs. 6D(vii).4A and B: Demonstration of finger nose test.

Rebound Phenomenon [Fig. 6D(vii).5]

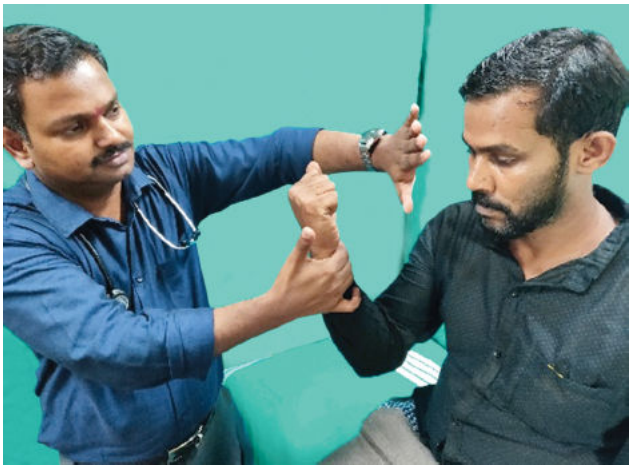
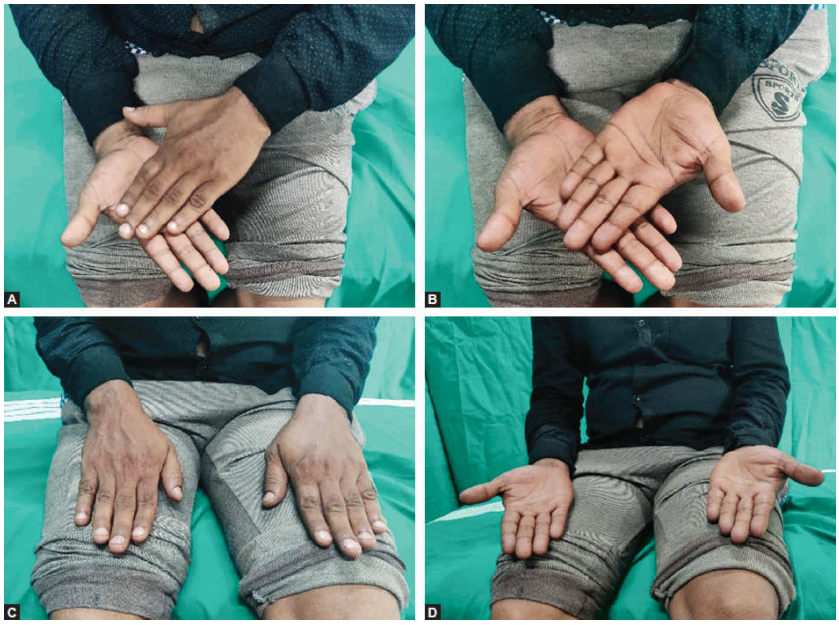


Fig. 6D(vii).5: Demonstration of rebound phenomenon.

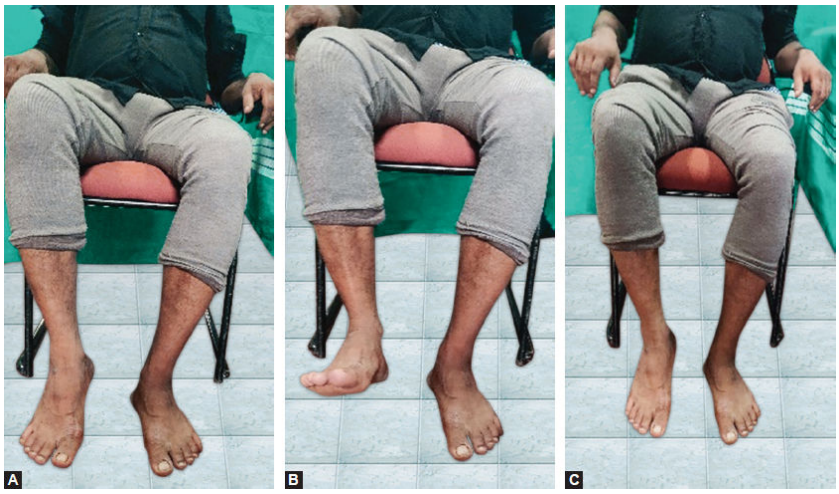
DYSDIADOKOKINESIA [FIGS. 6D(VII).6A TO D]



Figs. 6D(vii).6A to D: Demonstration of dysdiadochokinesia.

FOOT TAPPING/FOOT PAT TEST [FIGS. 6D(VII).7A TO C)]

Patient is made to sit on chair with feet touching the floor flat. He is asked to pat the floor with his forefoot. The rate, rhythm and speed of patting is compared on both sides. Even minimum cerebellar disease can be picked up by this test.

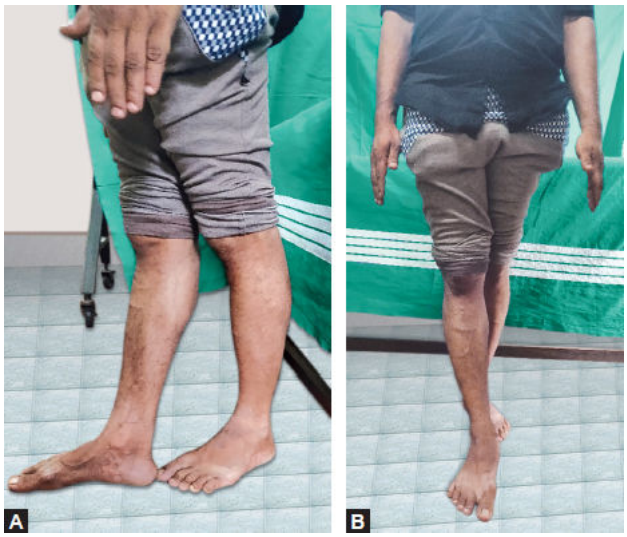


Figs. 6D(vii).7A to C: Demonstration of foot tapping.

STRAIGHT LINE WALKING [FIGS. 6D(VII).8A AND B]

Figs. 6D(vii).8A and B: Straight line walking.

TANDEM WALKING [FIGS.6D(VII).9A AND B]



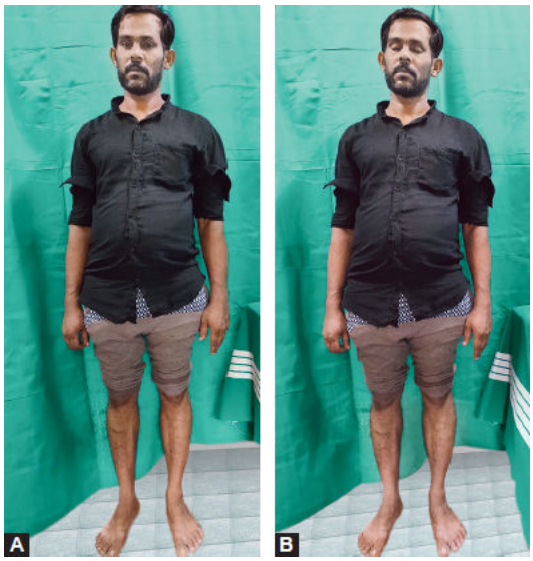
Figs. 6D(vii).9A and B: Demonstration of tandem walking.

ROMBERG TEST [FIGS. 6D(VII).10A AND B]

Patient stands still with their heels together. Ask the patient to remain still and close their eyes. If the patient loses their balance immediately, the test is positive.

To achieve balance, a person requires 2 out of the following 3 inputs to the cortex: 1. Visual confirmation of position, 2. Nonvisual confirmation of position (including proprioceptive and vestibular input), and 3. A normally functioning cerebellum.

Therefore, if a patient loses their balance after standing still with their eyes closed, and is able to maintain balance with their eyes open, then there is likely to be lesion in sensory input.



Figs. 6D(vii).10A and B: Demonstration of Romberg's sign.

APPROACH TO ATAXIA

- Ataxia, defined as impaired coordination of voluntary muscle movement affecting the rate, range, direction and force of movements.
- It is a physical finding, not a disease.
- Types of ataxia:
  1. Cerebellar
  2. Sensory
  3. Vestibular
  4. Optic
  5. Frontal

Type of ataxia	Cerebellar	Sensory	Frontal
Stance and support	Wide based	Narrow based; looking down	Wide based
Velocity	Variable	Slow	Very slow
Stride	Irregular, lurching	Regular with path deviation	Short, shuffling
Romberg	+/-	Unsteady; patient falls	+/-
Heel-shin	Abnormal	+/-	Normal
Initiation	Normal	Normal	Hesitant
Postural instability	+	+++	+++++
Falls	Late event	Frequent	Frequent
Turns	Unsteady	+/-	Multistepped; hesitant

Sensory ataxia is due to a severe sensory neuropathy, ganglionopathy or lesions of the posterior column of the spinal cord, e.g. Sjogren's syndrome, cisplatin, chronic inflammatory demyelinating polyradiculoneuropathy (CIDP), paraneoplastic disorders, subacute combined degeneration (SACD), tabes dorsalis, Miller Fischer syndrome, celiac disease.

- Ataxia more at night or while walking through narrow passages (coffee plantations).

- A history of falling into the sink or imbalance when splashing water on the face (wash-basin sign), passing a towel over the face or pulling a shirt over the head should also be sought.
- Pseudoathetosis–"piano-playing" movements—when the patient has his arms outstretched and eyes closed, the affected arm will wander from its original position.
- Vibration and position sense are usually lost together.
- Positive Romberg's test is a hallmark of sensory ataxia.

Vestibular ataxia is due to lesion of vestibular pathways resulting in impairment and imbalance of vestibular inputs, e.g. vestibular, neuronitis, and streptomycin toxicity.

- Vertigo and associated tinnitus and hearing loss.
- Direction of the nystagmus is away from the lesion.

Optic ataxia was first described in a man with lesions of the posterior parietal lobe on both sides of the brain, later known as **Balint syndrome**.

- Among the symptoms that characterize the syndrome are a restriction of visual attention to single objects and a paucity of spontaneous eye movements.
- Patients have difficulty in completing visually guided reaching tasks in the absence of other sensory cues.

Frontal lobe ataxia (Brun's ataxia) is due to involvement of subcortical small vessels, Binswanger's disease, multi infarct state or normal pressure hydrocephalus (NPH).

- The gait may appear to be a combination of awkward, magnetic (stuck to the floor), cautious, slow, and shuffling. This is also known as a frontal gait disorder, referring to the frontal lobe conditions which often cause **gait apraxia.**

CEREBELLAR ATAXIA

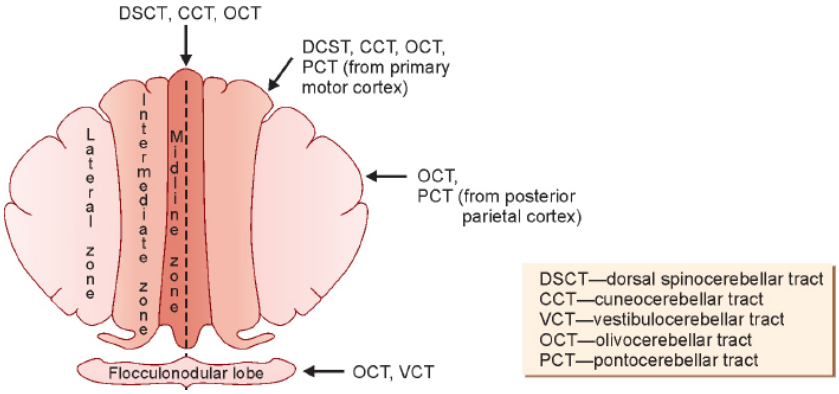


Fig. 6D(vii).11: Anatomical and functional areas of cerebellum.

Zone [Fig. 6D(vii).11]	Corresponding anatomical site	Function	Loss of function
Midline zone	Anterior and posterior parts of the vermis, fastigial nucleus	Posture, locomotion, position of head relative to trunk, control of extraocular movements	Disorders of stance/gait, truncal postural disturbances, rotated postures of the head, disturbances of eye movements
Intermediate zone	Paravermal region of cerebellum and interposed nuclei *(emboliform, globose)*	Control of velocity, force and pattern of muscle activity	—
Lateral zone	Cerebellar hemisphere and dentate nucleus	Planning of fined and skilled movement *(in connection with neurons in the Rolandic region of the cerebral cortex).*	Hypotonia, dysarthria, dysmetria, dysdiadochokinesia, excessive rebound, impaired check, kinetic and static tremors, past pointing

CAUSES OF CEREBELLAR ATAXIA

Symmetrical Cerebellar Ataxias

Acute	Subacute	Chronic
• Drugs: Phenytoin, phenobarbitone, lithium, Chemotherapeutic agents • Alcohol • Infectious: Acute viral cerebellitis, post-infectious • Toxins: Toluene, glue, gasoline, methyl mercury	• Alcohol, or Nutritional (B_1, B_{12}) • Paraneoplastic • Antigliadin or anti-GAD antibody • Prion diseases	• MSA-C • Hypothyroidism • Phenytoin toxicity

(GAD: glutamic acid decarboxylase; MSA-C: multiple system atrophy with cerebellar ataxia)

Asymmetrical Cerebellar Ataxias

Acute	Subacute	Chronic
Vascular: Cerebellar infarction or hemorrhage, subdural hematoma • Infectious: Abscess	• Neoplastic: Glioma, metastases, lymphoma • Demyelination: MS • HIV related: Progressive multifocal leukoencephalopathy	• Congenital lesions: Arnold Chiari malformation, Dandy Walker syndrome

Treatable Causes of Ataxia

- Hypothyroidism
- Ataxia with vitamin E deficiency (AVED)
- Vitamin B_{12} deficiency
- Wilson's disease
- Ataxia with antigliadin antibodies and gluten sensitive enteropathy
- Ataxia due to malabsorption syndromes
- Lyme's disease
- Mitochondrial encephalomyopathies, aminoacidopathies, leukodystrophies and urea cycle abnormalities
- Wernicke's encephalopathy

Cerebellar Syndromes	
Rostral vermis syndrome (anterior lobe) *For example, alcoholics*	Wide-based stance and gait. Ataxia of gait; proportionally less ataxia is seen on performing Heel-shin test while the patient is lying down. Normal or slightly impaired arm coordination. Infrequent hypotonia, nystagmus and/or dysarthria.
Caudal vermis syndrome (flocculonodular, posterior lobe) *For example, tumors (medulloblastoma)*	Axial disequilibrium; staggering gait. Little or no limb ataxia. Spontaneous nystagmus might be seen. Rotated postures of head.
Hemispheric syndrome (Posterior lobe, anterior variants also possible) *For example, infarcts, neoplasms, abscesses.*	Incoordination of ipsilateral limb movements. More noticeable with fine motor skills. Incoordination affects most noticeably muscles involved in speech and finger movements.
Pancerebellar syndrome *For example, infectious/parainfectious processes, hypoglycemia, paraneoplastic disorders, toxic-metabolic disorders*	Combination of all the other syndromes. Bilateral signs of cerebellar dysfunction involving trunk, limbs, cranial musculature.

LOCALIZATION OF CEREBELLAR LESIONS

Signs and symptoms	Most probable region of involvement

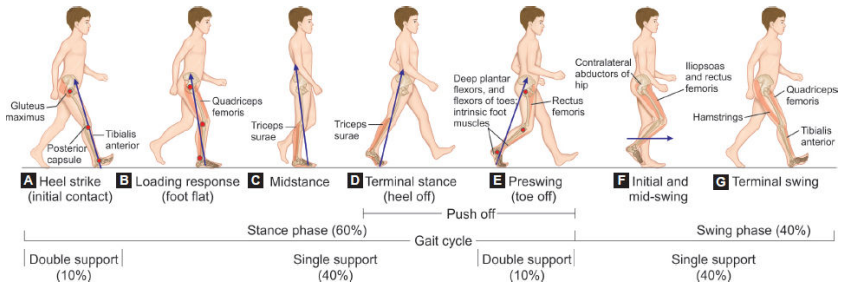
Higher cognitive changes	Lateral hemispheres
Action tremor	Dentate and interposed nuclei OR cerebellar outflow to ventral thalamus
Palatal tremor	Dentate nucleus, Guillain Mollaret triangle
Titubation	Any zone; especially anterior vermis and associated deep nuclei
Dysarthria	Posterior left hemisphere and vermis
Gait ataxia	Anterior vermis
Limb ataxia	Lateral hemispheres
Saccadic dysmetria	Dorsal vermis
Square wave jerks	Cerebellar outflow
Gaze evoked nystagmus	Flocculus and paraflocculus

Mnemonics for cerebellar signs

Danish pen	Vanishd
Dysdiadochokinesia **A**taxic gait **N**ystagmus **I**ntention tremor **S**caning/**S**taccato speech **H**ypotonia/**H**eel-shin test **P**endular knee jerk	**V**ertigo **A**taxia **N**ystagmus **I**ntentional tremor **S**canning speech **H**ypotonia **D**ysdiadochokinesia

NORMAL GAIT CYCLE [FIGS. 6D(VIII).1A TO G]

The gait cycle is the time interval or sequence of motions occurring between two consecutive initial contacts of the same foot, i.e. cycle of stance and swing by one foot.



Figs. 6D(viii).1A to G: Normal gait cycle.

Observation to be noted while the patient walks:

1. Posture of the body while walking
2. The regularity of the movement
3. The position and movement of the arms
4. The relative ease and smoothness of the movement of the legs
5. The distance between the feet both in forward and lateral directions
6. The ability to maintain a straight course
7. The ease of turning
8. Stopping
9. Position of feet and posture just before initiation of gait.

ABNORMALITIES OF GAIT

Neurogenic gait disorders should be differentiated from those due to skeletal abnormalities (characterized by pain producing an antalgic gait, or limp).

Gait abnormalities incompatible with any anatomical or physiological deficit may be due to functional disorders.

Pyramidal (Circumduction/Hemiplegic) Gait [Fig. 6D(viii).2]

- Lesions of the upper motor neuron lesions produce characteristic extension of the affected leg. There is tendency for the toes to strike the ground on walking and outward throwing/swing of lower limbs. This movement occurring at the hip joint is called circumduction. There is leaning towards the opposite normal side. The arm of the affected side is adducted at the shoulder and flexed at the elbow, wrist, and fingers.
- In hemiplegia/hemiparesis, there is a clear asymmetry between affected and normal sides on walking, but in paraparesis both lower legs swing slowly from the hips in extension and are stiffly dragged over the ground (walking in mud).

Fig. 6D(viii).2: Circumduction gait.

Foot Drop (High Stepping/Slapping Gait) [Fig. 6D(viii).3]

In normal walking, the heel is the first part of the foot to hit the ground. A lower motor neuron lesion affecting the leg will cause weakness of ankle dorsiflexion, resulting in a less controlled descent of the foot, which makes slapping noise as it hits the ground. In severe cases, the foot will have to be lifted higher at the knee to allow room for the inadequately dorsiflexed foot to swing through, resulting in a high-stepping gait. Cause, e.g. common peroneal nerve palsy.

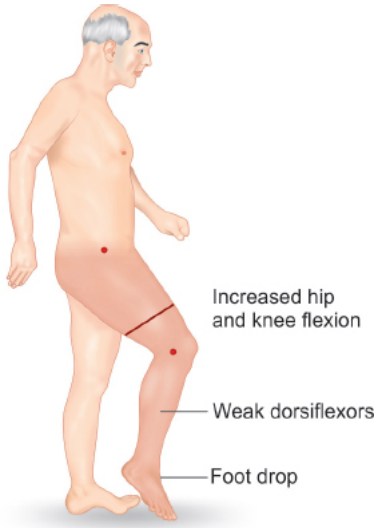


Fig. 6D(viii).3: High stepping gait.

Myopathic Gait/Waddling Gait [Fig. 6D(viii).4]

- During walking, alternating transfer of the body's weight through each leg, needs adequate hip abduction.
- **Causes:** Weakness of proximal lower limb muscles (e.g. polymyositis and muscular dystrophy) causes difficulty rising from sitting. The hips are not properly fixed by these muscles and trunk movements are exaggerated, and walking becomes a waddle or rolling. The pelvis is poorly supported

by each leg. This may be seen with bilateral congenital dislocation of hip (**Trendelenburg gait**). The patient walks on a broad base with exaggerated lumbar lordosis.

Gluteus Medius Gait or Abductor Lurch

Lurch of body towards affected side in every stance phase (abductor lurch). Seen with congenital coxa vara, gluteus medius paralysis, polio, and Perthes disease.

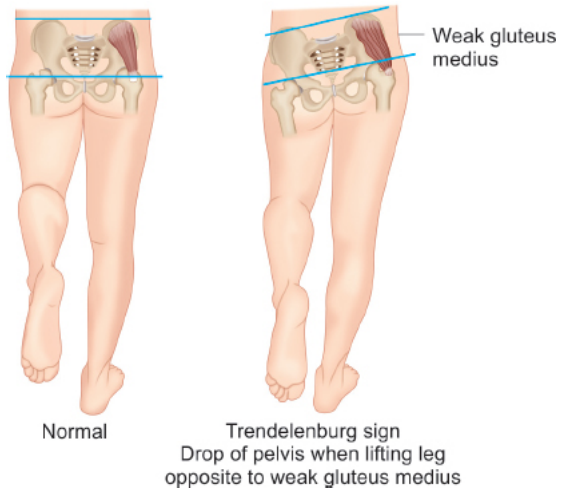


Fig. 6D(viii).4: Waddling gait.

Ataxic Gait (Cerebellar Ataxia: Broad-based Gait) [Fig. 6D(viii).5]

- In this type of gait, the patient, unstable, tremulous and reels in any direction (including backwards) and walks on a broad base. Ataxia describes this incoordination. The patient finds difficulty in executing tandem walking.
- **Causes:** Lesions of the cerebellum, vestibular apparatus or peripheral nerves. When walking, the patient tends to veer to the side of the affected cerebellar lobe. When the disease involves cerebellar vermis, the trunk becomes unsteady without limb ataxia, with a tendency to fall backwards or sideways and is termed truncal ataxia.

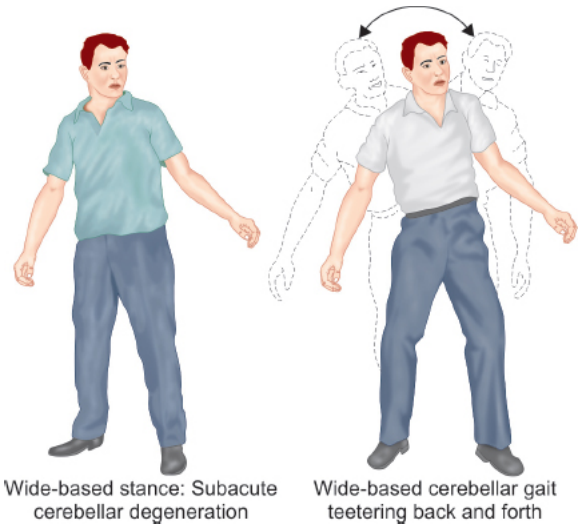


Fig. 6D(viii).5: Cerebellar/ataxic gait.

Apraxic Gait

- In an apraxic gait, the acquired walking skills become disorganized. On examination of the legs, the power, cerebellar function, and proprioception are normal. Leg movement is normal when sitting or lying and the patient can carry out complex motor tasks (e.g. bicycling motion). But patient cannot initiate and organize the motor act of walking. The feet appear stuck to the floor and the patient cannot walk.
- **Causes:** Diffuse bilateral hemisphere disease or diffuse frontal lobe disease (e.g. tumor, hydrocephalus, and infarction).

Marche à petits pas

- It is characterized by small, slow steps, and marked instability. In contrast to the festination found in Parkinson's disease, it lacks increasing pace and freezing.
- **Cause:** Small-vessel cerebrovascular disease and accompanying bilateral upper motor neuron signs.

Extrapyramydal/Shuffling/Festinant Gait [Fig. 6D(viii).6]

- It is characterized by stooped posture and gait difficulties with problems initiating walking and controlling the pace of the gait. Patients make a series of small, flat footed shuffles, and become stuck while trying to start walking or when walking through doorways (freezing). The center of gravity will be moved forwards to aid propulsion and difficulty in stopping. It is characterized by muscular rigidity throughout extensors and flexors. Power is preserved, pace is shortened and slows to a shuffle, and its base remains narrow. There is a stoop and diminished arm swinging and gait becomes festinant (hurried) with short rapid steps. Patient will be having difficulty in turning quickly and initiating movement. Retropulsion, i.e. small backward steps are taken involuntarily when a patient halts.
- **Cause:** Parkinsonism.

[**Kinesia paradoxa**—presented in Parkinson's disease patients, who generally cannot move but under certain circumstances of need exhibit a sudden, brief period of mobility (walking or even running)]

Scissoring Gait [Figs. 6D(viii).7A and B]

Seen classically with cerebral palsy due to bilateral spasticity.

Sensory Ataxia: Stamping Gait [Fig. 6D(viii).8]

- It is characterized by broad based, high stepping, stamping gait, and ataxia due to loss of proprioception (position sense). This type of ataxia becomes more prominent by removal of sensory input (e.g. walks with eyes closed) and becomes worse in the dark. Romberg's test is positive.
- **Cause:** Peripheral sensory (large fiber) lesions (e.g. polyneuropathy), posterior column lesion (vitamin B_{12} deficiency or tabes dorsalis).

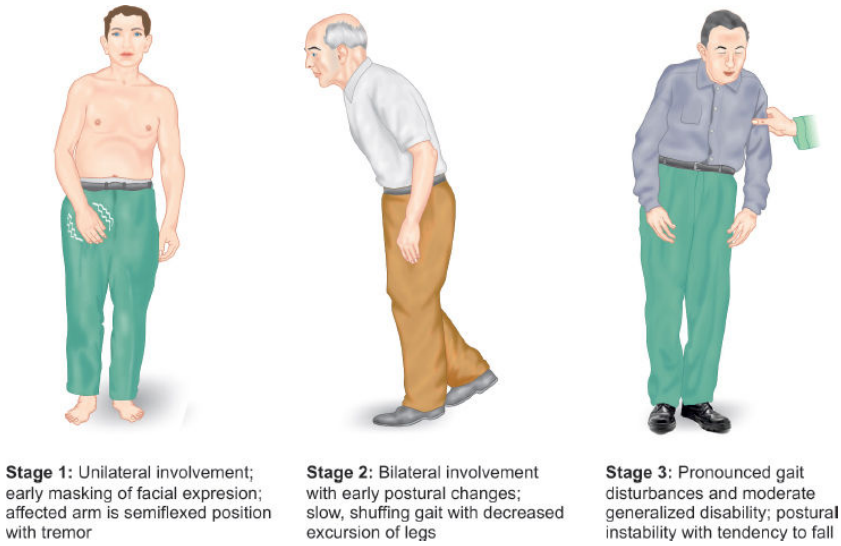
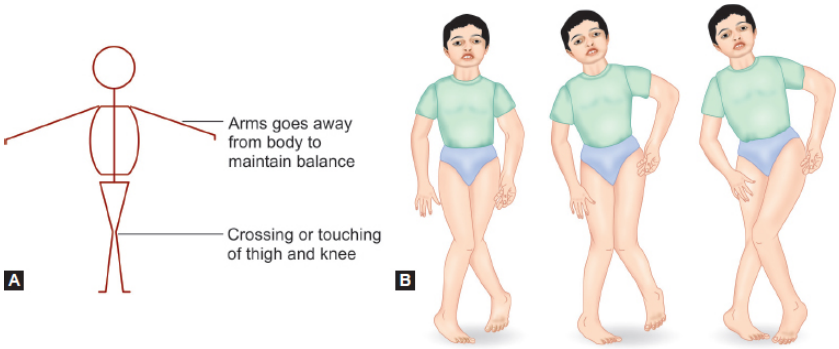


Fig. 6D(viii).6: Stages of Parkinson's gait.



Figs. 6D(viii).7A and B: Scissoring gait.

Fig. 6D(viii).8: Sensory ataxia.

Choreiform Gait (Hyperkinetic Gait)

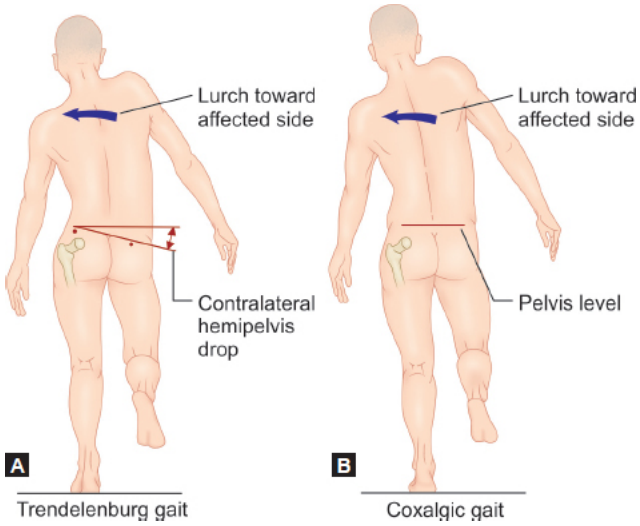
- The patient will display irregular, jerky, and involuntary movements in all extremities. Walking may accentuate their baseline movement disorder.
- **Cause:** Sydenham's chorea, Huntington's disease, and other forms of chorea, athetosis or dystonia.

Antalgic or Painful Gait

Decreased duration of stance phase as the painful limb is unable to bear full weight. It is seen in any painful lesion of the lower extremity, i.e. foot, knee, and hip.

Coxalgic Gait [Figs. 6D(viii).9A and B]

In patients with hip pain, the upper trunk is typically shifted towards the affected side during the stance phase on the affected leg. This is an unconscious adaptive maneuver which reduces the force exerted on the affected hip during the stance phase.



Figs. 6D(viii).9A and B: Trendelenburg gait versus coxalgic gait.

Toe-walking or Equinus Gait

Heel strike is avoided. It is seen in patients with heel pain, clubfoot, congenital short Achilles tendon, and cerebral palsy.

Quadriceps Weakness Gait

Inability to maintain knee extension at heel-strike and patient may push on thigh to extend the knee and lock. It is seen in quadriceps paralysis.

Astasia-Abasia

It is a psychogenic pattern of walking in which the patient seems to alternate between a broad base for stability and a narrow, tightrope-like stance, with contortions of the trunk, and limbs that give the appearance of an imminent fall.

Alderman's Gait

Patient walks with chest and head thrown backwards with protuberant abdomen and legs thrown wide apart. It is seen in tuberculosis of lower thoracic and upper lumbar vertebra.

GAIT ABNORMALITIES ANALYSIS

Gait initiation, maintenance, and termination	Difficulty starting	PD, atypical parkinsonism
	Freezing of gait	PD, atypical parkinsonism
	Inability to stop (festination)	PD, atypical parkinsonism
Stance width	Narrowed base of support	PD, spastic paraparesis
	Widened base of support	Cerebellar ataxia, sensory ataxia, vestibular ataxia
	Scissoring of the legs	Spastic paraparesis
	Unable to walk in a straight line, sideways deviation (veering) of gait	Unilateral vestibular ataxia, unilateral cerebellar ataxia
Step length, height, and cadence	Reduced step height	PD, parkinsonism; foot drop
	Small steps	PD, atypical parkinsonism, normal pressure hydrocephalus
	Irregular step size	Cerebellar ataxia, vestibular ataxia, chorea
	Reduced stance phase on the affected side (limping)	Pain (antalgic gait)
Arm swing	Unilaterally reduced	Hemiparesis, dystonia, PD
	Bilaterally reduced	PD, parkinsonism, dystonia
	Excessive	Chorea, levodopa-induced dyskinesias, NPH
	Tremor appearing in hand during walking	PD, parkinsonism
Movement fluidity	Dropped foot, lifting the leg higher than normal (steppage gait)	Neuropathy of common fibular nerve or sciatic nerve, L5 radiculopathy, Charcot–Marie–Tooth disease
	Knees giving way (buckling of the knees)	Quadriceps weakness (for example, limb-girdle myopathy, IBM)
	Locking of the knees	Cerebellar ataxia
	Pelvis drop at side of the swing leg, resulting in alternating lateral trunk movements (waddling gait and bilateral Trendelenburg gait)	Bilateral proximal muscle weakness in the leg and hip girdle
	Bizarre gait pattern	Chorea
Gait speed	Slow	PD
	Fast	Vestibular disease, Alzheimer's disease

(PD: Parkinson's disease; NPH: normal pressure hydrocephalus; IBM: inclusion body myositis)

BEDSIDE TESTS TO DIAGNOSE PES CAVUS AND PES PLANUS

Wet Test [Fig. 6D(viii).10]

There are three basic foot types, each based on the height of the arches. The quickest and easiest way to determine your foot type is by taking the "wet test," below. (1) Pour a thin layer of water into a shallow pan. (2) Wet the sole of your foot. (3) Step onto a shopping bag or a blank piece of heavy paper. (4) Step off and look down. Observe the shape of your foot

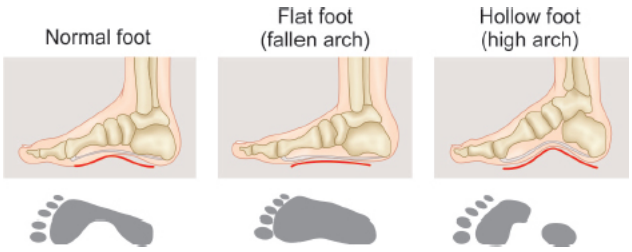


Fig. 6D(viii).10: Wet test and appearance.

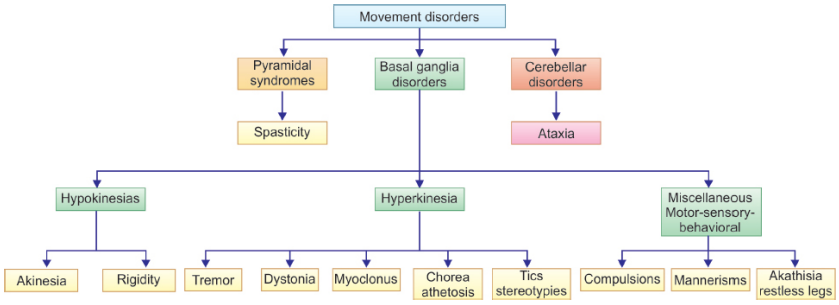
MOVEMENT DISORDERS

Dyskinesia is abnormal uncontrolled movement and is a common symptom of many movement disorders **[Flowcharts 6D(ix).1 and 6D(ix).2]**.

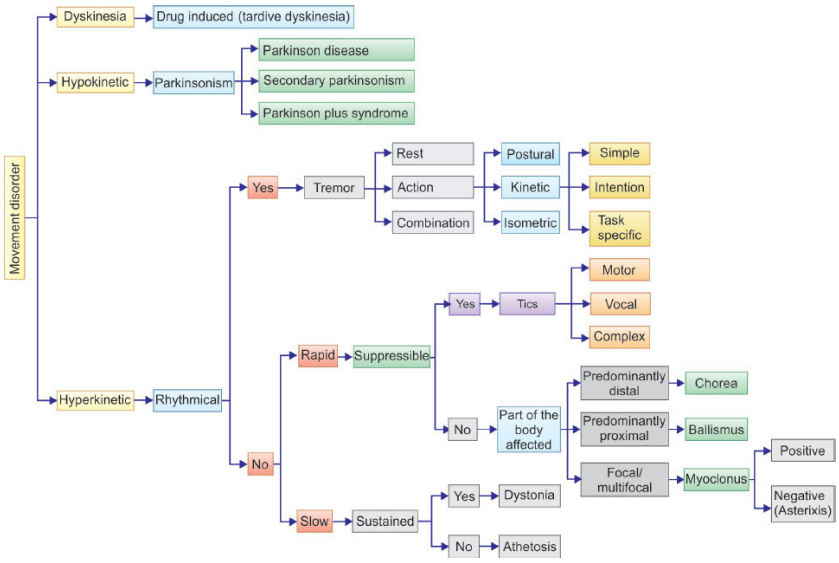
Movement disorders disrupt motor function by:

1. Abnormal, involuntary, unwanted movements (hyperkinetic movement disorders).
2. Curtailing (restricting) the amount of normal free flowing, fluid movement (hypokinetic movement disorders).

Flowchart 6D(ix).1: Categorization of movement disorders.



Flowchart 6D(ix).2: Systematic approach to movement disorders.



Site of Lesion

1. Parkinsonism → Contralateral substantia nigra
2. Unilateral hemiballismus → contralateral subthalamic nucleus
3. Chronic chorea → Caudate nucleus/putamen
4. Athetosis, dystonia → Contralateral putamen or thalamus
5. Myoclonus → Cerebellar cortex/thalamus

6. Rhythmic palatal/facial myoclonus → Central tegmental tract, inferior olivary nucleus, olivodentate fibers.

TREMOR

Tremor: Series of involuntary, relatively rhythmic, purposeless, oscillatory movements due to intermittent muscle contractions:

- Simple tremor involves only a single muscle group
- Compound tremor involves several muscle groups
  - Several elements in combination
  - Resulting in a series of complex movements
- May be unilateral or bilateral
- Most commonly involves distal parts of the extremities—fingers or hands
- May also affect the arms, feet, legs, tongue, eyelids, jaw, and head
- May occasionally involve the entire body
  - Rate may be slow, medium, or fast
    - » Slow: Oscillations of 3 to 5 Hz
    - » Rapid: Oscillations of 10 to 20 Hz
  - Amplitude may be fine, coarse, or medium
  - The relationship to rest or activity is the basis for classification into two primary tremor types:
    - » Resting
    - » Action

<table>
<tr><th colspan="3">Resting (Static)</th></tr>
<tr><td colspan="3">• Tremors are present mainly during relaxation (e.g. with the hands in the lap)
• Attenuate when the part is used
• Rest tremor is seen primarily in PD and other Parkinsonian syndromes</td></tr>
<tr><th colspan="3">Action Tremors</th></tr>
<tr><td>Postural tremors become evident when the limbs are:
Maintained in an antigravity position (e.g. arms outstretched)
Types of postural tremor:
• Enhanced physiological tremor (EPT)
• Essential tremor (ET)</td><td>Kinetic tremor: Appears when making a voluntary movement
May occur at the beginning, during or at the end of the movement. For example, intention (terminal) tremor seen primarily in cerebellar disease</td><td>Task specific tremor: Occurs when performing highly skilled, goal-oriented tasks. For example, while writing or speaking</td></tr>
</table>

CHOREA

- Characterized by involuntary, irregular, purposeless, random, nonrhythmic hyperkinesias.
- Movements are spontaneous, abrupt, brief, rapid, jerky, and unsustained.
- Movements are actually random and aimless:
  - Rather than disrupting a voluntary task, it appears as if fragments of movements intrude; in some cases, there is loss of motor tone, known as “**motor impersistence”,** which appears due to lapses in the ability to perform desired action.

- When asked to hold the hands outstretched, there may be constant random movements of individual fingers (**piano-playing** movements).
- If the patient holds the examiner's finger in her fist, there are constant twitches of individual fingers (**milkmaid grip**):
  - **"Jack in the box" tongue/** harlequin's tongue: Patient is unable to maintain tongue in protruded state and the tongue moves in and out.
- Blink rate is increased.

Causes

- Hereditary: Huntington's disease, benign chorea
- Drugs: Antiparkinsonian drugs, oral contraceptives
- Toxin: Alcohol, carbon monoxide poisoning
- Infections: Sydenham's chorea, encephalitis
- Metabolic: Hyperthyroidism, hypocalcemia
- Immunological: SLE, polyarteritis nodosa
- Vascular
- Pregnancy (Chorea gravidarum)

ATHETOSIS

- Involuntary, irregular, coarse, somewhat rhythmic, and writhing or squirming in character (twisting).
- Hyperkinesias are slower, more sustained, and larger in amplitude than those in chorea.
- May involve the extremities, face, neck, and trunk.
- In the extremities, they affect mainly the distal portions, the fingers, hands, and toes:
  - Affected limbs are in constant motion (athetosis means "without fixed position")
  - Choreoathetosis refers to movements that lie between chorea and athetosis in rate and rhythmicity, and may represent a transitional form.

Causes

- Cerebral palsy
- Congenital due to perinatal injury to the basal ganglia.

HEMIBALLISMUS

Dramatic neurologic syndrome of wild, flinging (forceful), incessant (uninterrupted or continuous) movements that occur on one side of the body.

Due to infarction or hemorrhage in the region of the contralateral subthalamic nucleus.

- More rapid and forceful
- Involve the proximal portions of the extremities.
- When fully developed, there are continuous, violent, swinging, flinging, rolling, throwing, flailing (thrashing) movements of the involved extremities.
- They are usually unilateral, and involve one entire half of the body.
- Rarely, they are bilateral (biballismus or paraballismus) or involve a single extremity (monoballismus).

MYOCLONUS

Single or repetitive, abrupt, brief, rapid, lightning-like, jerky, arrhythmic, asynergic, involuntary contractions, involving portions of muscles, entire muscles, or groups of muscles.

- Seen principally in the muscles of the extremities and trunk, but the involvement is often multifocal, diffuse, or widespread.

- May involve the facial muscles, jaws, tongue, pharynx, and larynx.
- Myoclonus may appear symmetrically on both sides. Such synchrony may be an attribute unique to myoclonus.

Myoclonus has been classified in numerous ways including the following:

i. Positive versus negative;
ii. Epileptic versus nonepileptic;
iii. Stimulus sensitive (reflex) versus spontaneous;
iv. Rhythmic versus arrhythmic;
v. Anatomically (peripheral, spinal, segmental, brainstem, or cortical)
vi. By etiology (physiologic, essential, epileptic, and symptomatic)

- Encephalitis
- Juvenile myoclonic epilepsy (JME, Janz syndrome)
- Drug overdose
- Hypnic jerks (appear during the process of falling asleep)
- Hiccup
- Creutzfeldt–Jakob disease
- Subacute sclerosing panencephalitis (SSPE)
- Anoxic encephalopathy (Lance-Adams syndrome)

TIC

A “tic” is an involuntary movement or vocalization that is usually sudden onset, brief, repetitive, stereotyped but nonrhythmical in character, can be suppressed.

Types

Motor tics are associated with movements. Categorized as simple or complex.

Simple motor tics involve only a few muscles usually restricted to a specific body part.

- Examples of simple motor tics include: Eye blinking, shoulder shrugging, facial grimacing, neck stretching, mouth movements, jaw clenching, and spitting.

Vocal/phonic tics are associated with sound

Simple vocal tics consist of sounds that do not form words, such as, throat clearing, grunting, coughing, and sniffing.

Common complex vocal tics include: Repeating words or phrases out of context.

- Coprolalia: Use of socially unacceptable words, frequently obscene.
- Palilalia: Repeating one’s own sounds or words.
- Echolalia: Repeating the last-heard sound, word, or phrase.

Gilles de la Tourette syndrome—associated with chronic motor and phonic tics.

DYSTONIA

- Refers to a syndrome of involuntary sustained or spasmodic muscle contractions involving cocontraction of the agonist and the antagonist.
- The movements are usually slow and sustained, and they often occur in a repetitive and patterned manner.
- They can be unpredictable and fluctuate.

Partial or focal	Generalized
• Spasmodic torticollis • Blepharospasm • Oromandibular dystonia • Writers cramp • Hemiplegic dystonia after stroke	• Dystonia musculorum deformans (idiopathic torsion dystonia) • Dopamine responsive dystonia: In childhood and generally involves the legs only. • Drug-induced dystonia (metoclopramide, phenothiazine, haloperidol, chlorpromazine) • Symptomatic dystonia (after encephalitis, Wilsons disease)

Blepharospasm and Oromandibular Dystonia

Involuntary prolonged tight eye closure (blepharospasm) is associated with dystonia of mouth, tongue or jaw muscles (jaw clenching and tongue protrusion).

Writer's Cramp = Mogigraphia = Scrivener's Palsy

Symptoms usually appear when a person is trying to do a task that requires fine motor movements such as writing or playing a musical instrument.

MYOKYMIA

Myokymia, a form of involuntary muscular movement, usually can be visualized on the skin as vermicular or continuous rippling movements.

AKATHISIA

Akathisia is a movement disorder characterized by a feeling of inner restlessness and a compelling need to be in constant motion, as well as by actions such as:

- Rocking while standing or sitting.
- Lifting the feet as if marching on the spot.
- Crossing and uncrossing the legs while sitting.

RESTLESS LEGS SYNDROME/"EKBOM'S SYNDROME"

- Spontaneous, continuous leg movements associated with paresthesia.
- These sensations occur only at the rest and relieved by movement.
- Causes: Familial, lumbar root disease, polyneuropathy, renal failure, and iron deficiency.

SYNKINESIS/MIRROR MOVEMENTS

Mirror movements are characterized by involuntary movements on one side of the body mirroring voluntary movements of the other side.

FASCICULATIONS

Fasciculations are visible, fine and fast, sometimes vermicular contractions of fine muscle fibers that occur spontaneously and intermittently but usually do not generate sufficient force to move a limb. Described as verminosis, because they look like *worms* moving below the dermis.

Involuntary contraction of the muscle fibers innervated by a motor unit.

Causes of Fasciculations

Fasciculations in healthy subjects	Coffee; exhaustive physical activity/fatigue; stress; **benign fasciculations**
Fasciculations associated with movement disorders	Spinocerebellar degeneration-type 3; spinocerebellar degeneration-type 36; Parkinsonism (multiple system atrophy, ALS-plus syndromes)
Motor neuron diseases	**Amyotrophic lateral sclerosis;** progressive spinal muscular atrophies; benign monomelic amyotrophy; postpolio syndrome; Kennedy disease
Systemic diseases	**Hyperthyroidism**; hypophosphatemia, calcium disorders secondary to hyperparathyroidism, paraneoplastic myopathy

Drugs and/or intoxications by heavy metals pollutants	**Organophosphorus poisoning;** neostigmine; corticosteroids; succinylcholine; elemental mercury intoxication; atropine, lithium, nortriptyline; flunarizine; isoniazid

SIGNS OF MENINGEAL IRRITATION

Nuchal Rigidity/Meningeal Stiffness

Meningeal tightness is a contracture of the paravertebral muscles, a defense against the secondary pain stemming from inflammation of the meninges.

Painful and permanent, it sometimes presents with the subject lying down, curled up with his or her back to the light, head back, and extremities half-bent. All attempts to flex the head provoke insurmountable and painful resistance. There is extreme neck stiffness; rotational and side-to-side movements are possible but aggravate the headache **[Fig. 6D(x).1]**.

Fig. 6D(x).1: Examination of neck stiffness.

In **Kernig's sign**, patient is kept in supine position, hip and knee are flexed to a right angle, and then knee is slowly extended by the examiner. The appearance of resistance or pain during extension of the patient's knees beyond 135° constitutes a positive Kernig's sign **[Figs. 6D(x).2 and 6D(x).3]**.

Brudzinski's Sign

Josef Brudzinski described 4 maneuvers for the clinical diagnosis of meningitis: The cheek sign, symphyseal sign, Brudzinski's leg sign/reflex, and Brudzinski's neck sign.

1	**The cheek sign**	A positive cheek sign is elicited by applying pressure on both cheeks inferior to the zygomatic arch that leads to spontaneous flexion of the forearm and arm
2	**Symphyseal sign [Fig. 6D(x).4]**	A positive symphyseal sign occurs when pressure applied to the pubic symphysis elicits a reflex hip and knee flexion and abduction of the leg
3	**Brudzinski's leg sign/reflex [Fig. 6D(x).5]**	Brudzinski's contralateral reflex sign consists of reflex flexion of a lower extremity after passive flexion of the opposite extremity
4	**Brudzinski's neck sign [Figs. 6D(x).6 and 6D(x).7]**	Brudzinski's neck sign is performed with the patient in the supine position. The examiner keeps one hand behind the patient's head and the other on chest in order to prevent the patient from rising. Reflex flexion of the patient's hips and knees after passive flexion of the neck constitutes a positive Brudzinski's sign

Fig. 6D(x).2: Demonstration of Kernig's sign.

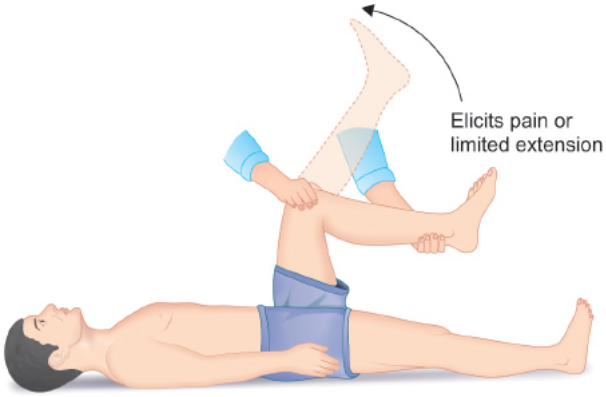


Fig. 6D(x).3: Illustration of Kernig's sign.

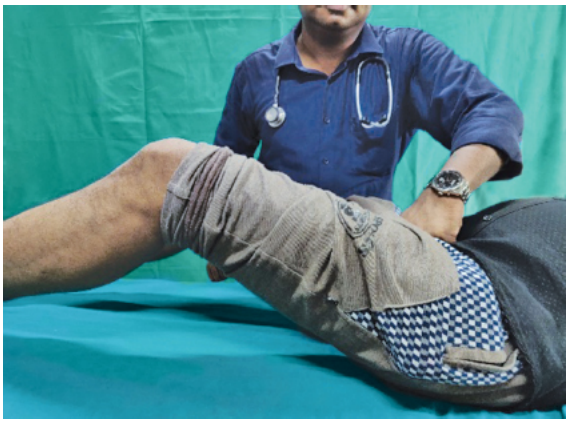


Fig. 6D(x).4: Symphyseal sign.

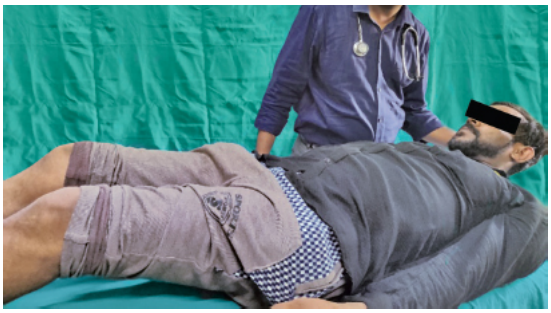


Fig. 6D(x).7: Brudzinski's neck sign.

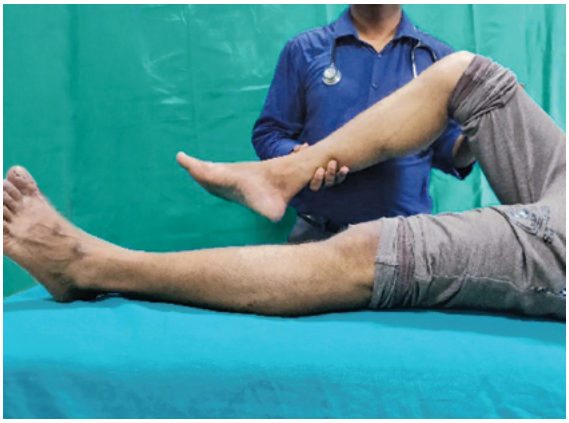


Fig. 6D(x).5: Brudzinski's leg sign/reflex.

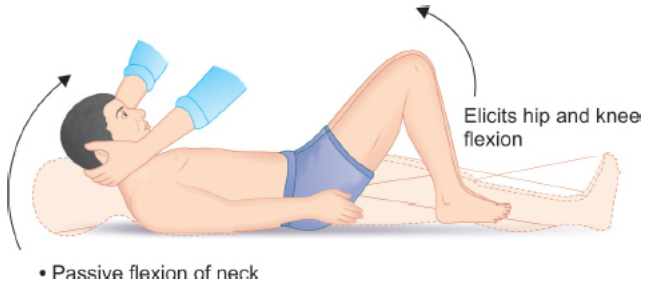


Fig. 6D(x).6: Illustration of Brudzinski's sign.

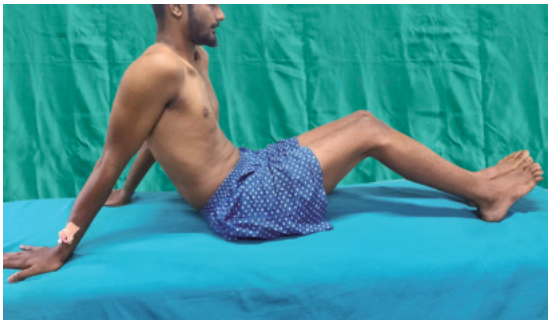


Fig. 6D(x).8: Tripod sign (Amoss's sign).

Tripod sign, also known as the "Amoss's sign", is a useful sign of meningeal irritation.

The patient is asked to sit up in bed. This action requires active movement involving flexion of the neck. Although a normal patient sits up without supporting himself, a patient with meningeal irritation tries to sit up by supporting himself with his hands placed far behind him in the bed (like a tripod), in order to take the weight off the spine and prevent its flexion **[Fig. 6D(x).8]**. Severe meningeal irritation may result in the patient assuming the tripod position with the knees and hips flexed, the back arched lordotically, the neck extended, and the arms brought back in a plane posterior to the pelvis to support the thorax.

MENINGISM

Meningism, also called meningismus or pseudomeningitis, is a set of symptoms similar to those of meningitis but not caused by meningitis. Whereas meningitis is inflammation of the meninges (membranes that cover the central nervous system), meningism is caused by nonmeningitic irritation of the meninges usually associated with acute febrile illness, especially in children and adolescents.

Causes

Meningism:
- Meningitis
- Subarachnoid hemorrhage.

Other conditions that mimic meningism (also resist cervical rotation):
- Cervical spondylosis
- After cervical fusion
- Parkinson's disease
- Raised intracranial pressure especially if there is impending tonsillar herniation
- Acute dystonic reaction

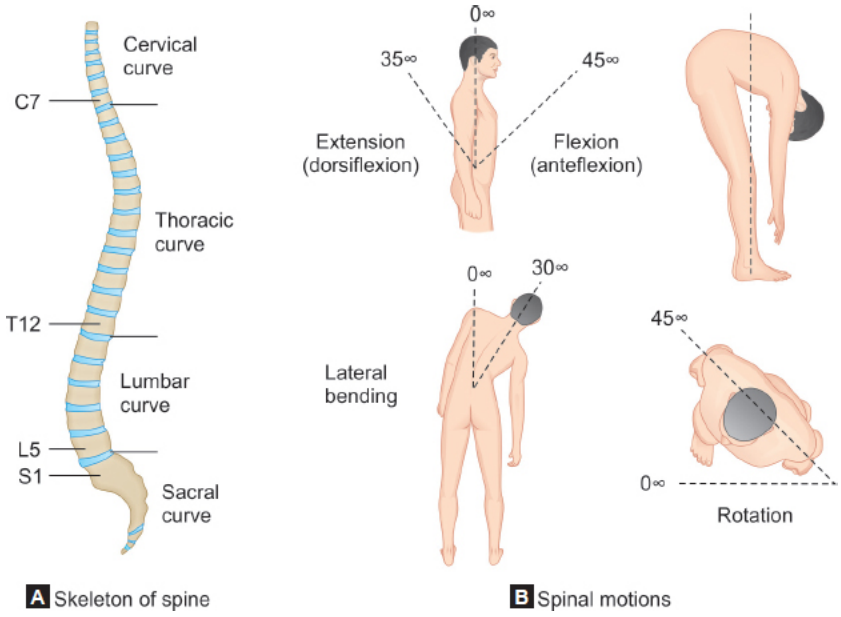
• Tetanus • Strychnine poisoning.
Intermittent neck stiffness is characteristic of Arnold-Chiari malformation.

EXAMINATION OF SKULL

- Size of skull—microcephaly, macrocephaly
- Shape/deformities
- Tenderness—fracture/metastasis
- Crackpot sound on percussion—hydrocephalus
- Bruits on auscultation—arteriovenous malformation (AVM), hemangioma.

EXAMINATION OF SPINE

- Inspection—deformities, curvature—kyphosis, scoliosis, lordosis, dimple, tuft of hair, Pott's spine, and meningioma
- Palpation—tenderness, paraspinal spasm, and deformities
- Movements **[Figs. 6D(x).9A and B]**.



Figs. 6D(x).9A and B: Movements of spine. (Details discussed under rheumatology section)

AUTONOMIC NERVOUS SYSTEM TESTING

Common autonomic symptoms	Signs
• Orthostatic intolerance • Dizziness • Lightheadedness • Fatigue	• Pupils—mid-dilated sluggish reacting pupil • Pedal edema • Resting tachycardia • Postural hypotension • Palpable urinary bladder • Sweating abnormalities

• "Coat hanger" headache • Nausea • Palpitations • Near syncope and syncope **Genitourinary** • Bladder urgency or frequency • Incontinence • Nocturia • Erectile dysfunction • Ejaculatory disturbances	
Common autonomic symptoms	**Signs**
Gastrointestinal • Diarrhea • Constipation • Fecal incontinence • Postprandial fullness, cramping, or bloating **Sudomotor** • Hyperhidrosis • Hypohidrosis and anhidrosis	
Tests	
Cardiovagal innervation (parasympathetic innervation) • Heart rate (HR) response to deep breathing • Valsalva ratio, and • HR response to standing (30:15 ratio)	**"Spoon test":** A kitchen soup spoon, with its curved surface resting on the skin, was held between the thumb and forefinger, and was drawn slowly on the skin, using sufficient energy to overcome its weight without lifting it from the skin. When "sympathectomized" skin was crossed, the pull was smooth and unopposed; but where sweat gland innervation and sympathetic function was intact, the skin was moist, and the flow of the spoon was interrupted, and became sticky requiring readjustment of the strength of pull
Adrenergic • Beat-to-beat blood pressure (BP) responses to the Valsalva maneuver, sustained handgrip/diastolic hand grip test ** and • BP and HR responses to tilt-up or active standing	"Sustained handgrip test (SHT): This parameter indicates cardiac sympathetic response and DBP response to the sustained handgrip test—taken as the difference between the DBP just before release of handgrip and the mean of three resting DBP readings. The change in mean DBP in response to sustained handgrip test was interpreted as: • ≥16 mm Hg was taken as normal • 11–15 mm Hg as borderline • ≤10 mm Hg as abnormal
Sudomotor: • Quantitative sudomotor axon reflex test (QSART) • Thermoregulatory sweat test (TST) • Sympathetic skin response (SSR), and • Silastic sweat imprint	

Head-Up Tilt-Table Testing

The patient lies supine on the tilt table. Beat-to-beat and oscillometric BP instruments are attached to each arm. ECG monitoring should take place throughout the test. Once the patient is comfortable, with feet resting on the footboard, a baseline BP is recorded for at least 3 minutes. The patient is then slowly tilted upright to an angle of 60–80°.

During testing, the patient is asked to report any symptoms. Both BP and HR are recorded throughout tilt-table testing, after which the patient is returned to a horizontal supine position.

Three well-described patterns of neurally-mediated syncope can occur during head-up tilt-table testing:

1. Vasodepression resulting in hypotension without bradycardia.
2. Cardioinhibition with a marked bradycardia (fewer than 40 beats/min) with or without significant hypotension.
3. Mixed, with both bradycardia and hypotension.

Valsalva Ratio

The Valsalva maneuver consists of respiratory strain which increases intrathoracic and intra-abdominal pressures and alters hemodynamic and cardiac functions.

- The patient is supine or with head slightly elevated to about 30°.
- Have the patient strain against 40 mm Hg applied for 15 seconds by blowing into a mouthpiece attached to a sphygmomanometer.
- Following cessation of the Valsalva strain, the patient relaxes and breathes at a normal comfortable rate.
- The ECG is monitored during the strain and 30–45 seconds following its release.
- The maximal heart rate of phase II actually occurs about 1 seconds following cessation of the strain.
- The minimal heart rate occurs about 15–20 seconds after releasing the strain.

DISEASES ASSOCIATED WITH AUTONOMIC DYSFUNCTION [TABLE 6D(X).1]

Table 6D(x).1: Diseases commonly associated with autonomic dysfunction.

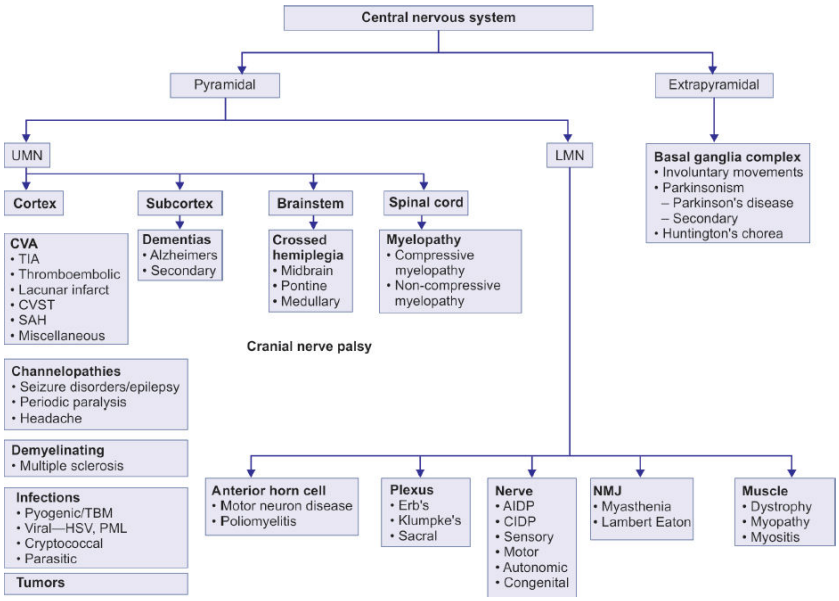
- **Preganglionic autonomic failure:**
  - Multiple system atrophy
  - Parkinson's disease with autonomic failure
- **Ganglionic and postganglionic disorders**
  - Pure autonomic failure
- **Peripheral neuropathies and neuronopathies with autonomic dysfunction**
  - *Acute and subacute (preganglionic and postganglionic):*
    - Acute pandysautonomia
    - Guillain-Barré syndrome
    - Paraneoplastic pandysautonomia
    - Others (porphyria, toxins, durgs)
  - *Chronic small-fiber (postganglionic) neruopathies:*
    - Diabetes
    - Amyloidosis
    - Hereditary (familial dysautonomia, Fabry's disease)
  - *Subacute or chronic sensory and autonomic ganglionopathies:*
    - Paraneoplastic
    - Sjogren's syndrome
  - *Other peripheral neuropathies:*
    - Infections (human immunodeficiency virus)
    - Connective tissue disease (systemic lupus erythematosus)
    - Metabolic-nutritional (alcohol, uremia, vitamin B_{12} deficiency)

E. A PPROACH TO COMMON NEUROLOGICAL CASES

Approach to following cases have been discussed in this section:

1. Approach to cerebrovascular accident
2. Approach to spinal cord diseases
3. Approach to neuropathy
4. Approach to movement disorders

Flowchart 6E.1: Diseases stratification of nervous system.



(UMN: upper motor neuron; LMN: lower motor neuron; CVA: cerebrovascular accident; TIA: transient ischemic attack; CVST: cerebral venous sinus thrombosis; SAH: subarachnoid hemorrhage; TBM: tuberculous meningitis; HSV: herpes simplex virus; PML: promyelocytic leukemia; SACD: subacute combined degeneration; AIDP: acute inflammatory demyelinating polyneuropathy; CIDP: chronic inflammatory demyelinating polyneuropathy; NMJ: neuromuscular junction)

1. APPROACH TO CEREBROVASCULAR ACCIDENT

Table 6E.1: Signs of upper and lower motor neuron disease.

Sign	*Upper motor neuron*	*Lower motor neuron*
Atrophy	None (rarely disuse atrophy)	Severe wasting
Fasciculations	None	Common
Tone	Hypertonia—rigidity/spasticity	Decreased (hypotonia)
Distribution of weakness	Distal predominant/regional	Predominantly proximal (except neuropathy)/segmental
Tendon reflexes	Exaggerated/hyperactive	Hypoactive/lost
Babinski sign	Present	Absent

Flexor spasms, clonus	Present	Absent

- A stroke (cerebrovascular accident is a vague term which should be avoided) is defined as a syndrome of rapid (abrupt) onset of a neurologic deficit that is attributable to a focal vascular cause **(Flowchart 6E.2)**.
- World Health Organization (WHO) definition: Stroke is a "rapidly developing clinical signs of focal (or global) disturbance of cerebral function, with symptoms lasting for 24 hours or longer or leading to death, with no apparent cause other than of vascular origin".
- **Progressing stroke (or stroke in evolution):** It is a stroke in which the focal neurological deficit worsens after the patient first presents. It may be due to increasing volume of infarction, secondary hemorrhage in the infarcted area, or increasing cerebral edema.
- **Complete stroke:** Rapid onset with persistent focal neurological deficit which does not progress beyond 96 hours.
- **Evolving stroke:** Gradual stepwise development of neurological deficits. Focal cerebral deficits that develop slowly (over weeks to months) are unlikely to be due to stroke and are more suggestive of tumor or inflammatory or degenerative disease.

Terminologies

Several terms are used to classify strokes mainly based on the duration and evolution of symptoms.

- **Transient ischemic attack (TIA):** Described later
- **Reversible ischemic neurological deficit (RIND):** In some cases, deficits last for longer than 24 hours but resolve completely or almost completely within a few days.
- **Stuttering hemiplegia:** Internal carotid lesions are characterized by repeated episodes of TIA followed by fully evolved stroke.

Flowchart 6E.2: Types of stroke.

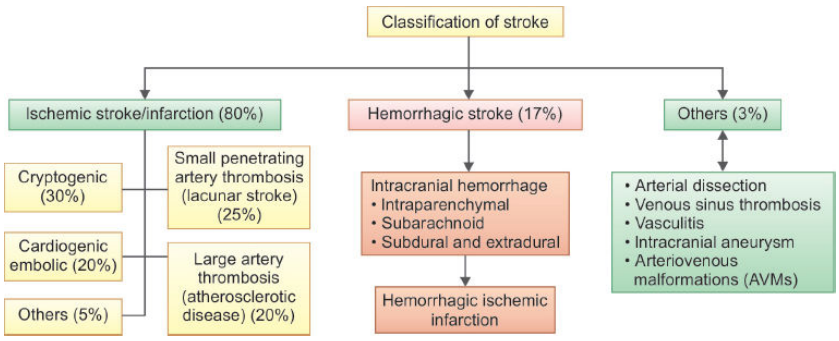


Table 6E.2: Risk factor for stroke.	
Risk factors in patients of all age groups	
High-risk	
• Hypertension (including isolated systolic) • Smoking • Diabetes mellitus • Atrial fibrillation • Drugs: Cocaine, amphetamine • Dilated cardiomyopathy • Endocarditis	• High cholesterol • Obesity • Vasculitis: Systemic vasculities [e.g. polyarteritis nodosa—PAN), granulomatosis with polyangiitis (Wegener's) etc.], primary CNS vasculitis • Meningitis (syphilis, tuberculosis, fungal, bacterial, zoster)
Low-risk	
Migraine	• Recent myocardial infarction

• Oral contraceptives or alcohol • Patent foramen ovale	• Prosthetic valve • Sleep apnea
Additional risk factors that are more common in young patients	
Hypercoagulable disorders	
• Protein C and S deficiencies • Antithrombin III deficiency • Antiphospholipid antibody syndrome • Factor V Leiden mutation • Prothrombin G20210A heterozygous mutation	• Sickle-cell anemia • Hyperhomocysteinemia • Thrombotic thrombocytopenic purpura • Arterial dissection • Infections (e.g. syphilis, HIV) • Systemic malignancy

(CNS: central nervous system; HIV: human immunodeficiency virus)

Table 6E.3: Causes for young stroke.

- **Cardiac**
  - Congenital heart disease, patent foramen ovale
  - Atrial myxoma
  - Atrial fibrillation and other arrhythmia
  - Cardiomyopathy, myocarditis, myocardial infarction
  - Cardiac surgery, cardiac catheterization
  - Endocarditis, rheumatic heart disease
  - Prosthetic valve
- **Hematologic**
  - Sickle cell disease, iron deficiency anemias, polycythemia vera
- **Hypercoagulable states**
  - Inherited prothrombotic states, protein C and S deficiency, antithrombin III deficiency, factor V Leiden gene mutation, prothrombin gene mutation
  - Antiphospholipid antibody syndrome
  - Hyperhomocysteinemia
  - Myeloproliferative disorders (e.g. leukemia, lymphoma)
  - Pregnancy exposure to hormonal treatments, such as anabolic steroids and erythropoietin, nephrotic syndrome
- **Vascular**
  - **Noninflammatory**
    - Arterial dissection
    - Secondary to connective tissue disease (Ehlers-Danlos, Marfan)
    - Moyamoya disease
    - Hypertension
    - Radiation vasculopathy
    - Vasculitis and postinfectious vasculopathy
    - Migraine
    - Cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy (CADASIL) Fibromuscular dysplasia, Susac's syndrome, Sneddon's syndrome, Fabry's disease
  - **Inflammatory**
    - Takayasu arteritis
    - Giant cell arteritis
    - Kawasaki disease
    - Polyarteritis nodosa
    - Human immunodeficiency virus (HIV)
    - Bacterial meningitis

  Illicit drug use: Cocaine, amphetamine

Table 6E.4: Differences between hemorrhagic, thrombotic, and embolic strokes.

Feature	***Hemorrhagic stroke (Intracerebral or subarachnoid hemorrhage)***	***Ischemic stroke***	
		Thrombotic	***Embolic***
Time of onset of stroke	During activity	Suddenly and often during sleep or in the early morning (4 AM)	Any time (usually during activity)
Rapidity of onset and progression	Over minutes and hours	On waking up or over hours	Rapid within seconds deficit maximum at onset
Transient ischemic attacks (TIAs)	Absent	Precedes stroke	Precedes stroke
Vomiting	Recurrent	Absent or occasional	Absent or occasional
Headache	Severe and prominent	Mild or absent	Mild or absent
Early resolution (within minutes or days)	Unusual	Variable	Possible
Meningeal irritation	May be present	Absent	Absent

Carotid bruit and absence of pulse	Not observed	Highly supports the diagnosis	Possible
Valvular heart disease and atrial fibrillation	Not found	Unusual	Highly supports the diagnosis
CT scan findings	Hemorrhage	• Early stage: Normal • Later: Pale infarct	• Early stage: Normal • Later: Pale infarct

Localization of Stroke

Site of lesion	Predominant clinical features
Cortex	• Monoplegia common (brachial-MCA territory; crural-ACA territory) • Hemiplegia (may be present but never dense) • Contralateral 7th cranial nerve palsy (UMN variant) • Seizures • Aphasias (in dominant hemisphere) • Apraxias (in nondominant hemisphere)
Subcortical (usually secondary to hypoperfusion)	• Monoplegias common • Transcortical aphasias common
Internal capsule lesion	• Contralateral hemiplegia (dense) • Contralateral hemisensory loss • 7th cranial nerve palsy (UMN variant) • Homonymous hemianopia • Broca's like aphasia (only site to have subcortical aphasia). **Note:** *Most common etiology being ischemic and hence is territory specific. Since different parts of internal capsule has blood supply from different blood vessels, all the above-mentioned features may not be present at same time. However, if present, it suggests hemorrhage or tumor compressing internal capsule*
Brainstem lesion	• Discussed in separate table
High cervical cord lesion (Brown-Sequard syndrome)	• Ipsilateral hemiplegia • Ipsilateral loss of posterior column sensation • Contralateral loss of pain and temperature sensation • Usually no cranial nerve involvement

(ACA: anterior cerebral artery; MCA: middle cerebral artery; UMN: upper motor neuron)

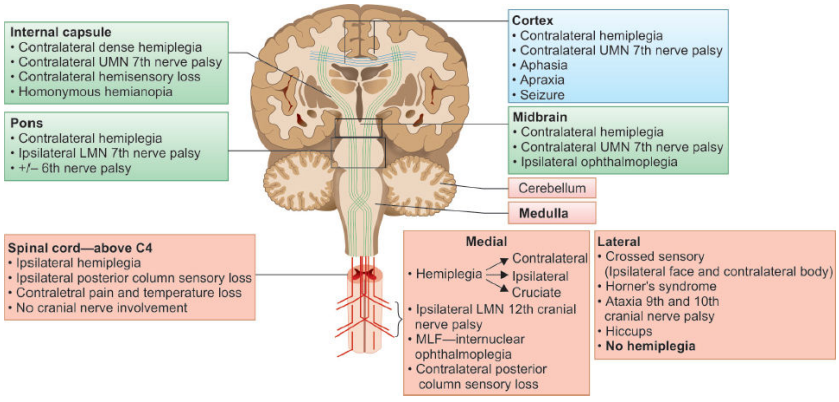


Fig. 6E.1: Localization of hemiplegia.

(UMN: upper motor neuron; LMN: lower motor neuron; MLF: medial longitudinal fasciculus)

Middle cerebral artery lesions and clinical features			
Internal carotid artery	***M1 branch of MCA***	***Stem of MCA***	***M2 branches of MCA***
Both anterior cerebral artery (ACA) and middle cerebral artery (MCA) territory involved along with ophthalmic artery causing amaurosis fugax	• Global aphasia • Dense hemiplegia (as internal capsule is also involved due to involvement of lenticulostriate branches of MCA)	• Global aphasia • Internal capsule spared	• Superior division • Inferior division (differences described below)

M2 stroke		
Division of M2	***Superior division***	***Inferior division***
Motor involvement	Face, arm > leg	Nil
Sensory	Face, arm	Nil
Vision	Nil	Quadrantanopia
Language	Broca's aphasia	Wernicke's aphasia
Nondominant	Hemineglect	Constructional apraxia

Brainstem syndromes			
Site of lesion/syndrome	***Blood supply and tracts involved***	***Ipsilateral features***	***Contralateral features***
Midbrain			
Benedict's syndrome (Claude's + Weber)	Interpeduncular branches of basilar artery, PCA—posterior cerebral artery (midbrain tegmentum—CN III fibers; red nucleus; CST; SCP)	Ipsilateral CN III palsy	Ataxia + Hyperkinesia and tremor ("rubral tremor") + Hemiparesis
Claude's syndrome	PCA (midbrain tegmentum—CN III fibers; red nucleus; SCP)	Ipsilateral CN III palsy	Ataxia + Tremor ("rubral tremor")
Weber's syndrome	Paramedian branches of the basilar artery, PCA	Ipsilateral CN III palsy	Hemiparesis
Nothnagel syndrome	Basilar penetrating artery, mesencephalic artery (midbrain tectum Ipsilateral or bilateral CN III)	Oculomotor palsies; ataxia	
Parinaud syndrome	Midbrain dorsum (quadrigeminal plate region; pretectum; periaqueductal gray matter)	Impaired upgaze; convergence retraction nystagmus; dilated pupils with light near dissociation	
Top of basilar artery syndrome	• Midbrain • Thalamus • Portion of temporal and occipital lobe involved	• Behavioral abnormalities • Ocular finding • Visual defects • Pupillary abnormalities • Motor deficits	
Artery of Percheron stroke	Single thalamic perforating artery from the proximal PCA	• Altered sensorium • Vertical gaze palsy • Memory impairment	
Pons			
Raymond Ceston	Long circumferential branch of basilar artery (CN VI; CST)	6th nerve palsy	Hemiparesis

syndrome			
Millard-Gubler syndrome	Basilar artery (CN VII; CST)	7th nerve palsy (± Lateral rectus palsy)	Hemiparesis
Foville's syndrome	Basilar artery (CN VII; lateral gaze center, CST)	7th nerve palsy + Horizontal gaze palsy	Hemiparesis
Pierre-Marie-Foix syndrome	AICA	• 6th + 7th nerve palsy • Horner's syndrome	Hemiparesis
Medulla			
Wallenberg syndrome (lateral medullary syndrome)	Vertebral artery > PICA (Lateral medullary Tegmentum—spinal tract of CN V and its nucleus; nucleus ambiguus; emerging fibers of CNs IX and X; LST; descending sympathetic fibers; vestibular nuclei; inferior cerebellar peduncle; afferent spinocerebellar tracts; lateral cuneate nucleus)	• Loss of pain and temperature of face • Ipsilateral decreased corneal reflex • Ipsilateral weakness of soft palate • Ipsilateral loss of gag reflex • Ipsilateral paralysis of vocal cord • Ipsilateral central Horner's syndrome • Nystagmus • Cerebellar ataxia of Ipsilateral limbs • Lateropulsion • **Hiccups**	Loss of pain and temperature of body
Dejerine syndrome (medial medullary syndrome)	Vertebral > anterior spinal artery	Ipsilateral tongue weakness	Hemiparesis
Avellis' syndrome	Medullary tegmentum	Ipsilateral palatal and vocal cord weakness;	Loss of pain and temperature
Jackson's syndrome	Medullary tegmentum	Ipsilateral flaccid paralysis of soft palate, pharynx, and larynx; flaccid weakness and atrophy of SCM and trapezius (partial), and of the tongue	
Schmidt's	Lower medullary tegmentum	Ipsilateral paralysis of soft palate, pharynx, and larynx; flaccid weakness and atrophy of SCM and trapezius (partial)	
Céstan-Chenais	Due to vertebral artery occlusion below origin of the PICA; (nucleus ambiguus; ICP; sympathetics; CST; ML)	Ipsilateral weakness of soft palate, pharynx, and larynx; cerebellar ataxia; Horner's syndrome	Contralateral hemiparesis with loss of posterior column function
Internuclear ophthalmoplegia (INO)	MLF lesion in the midbrain	Ipsilateral adduction palsy	Contralateral gaze evoked nystagmus
Wall eyed bilateral internuclear ophthalmoplegia (WEBINO)	Bilateral MLF lesion in the brain	Bilateral adduction deficit and primary gaze position exotropia	
PCA syndromes			
Gerstmann syndrome	Parietal lobe	Inability to write (dysgraphia or agraphia), the loss of the ability to do mathematics (acalculia), the inability to identify one's own or another's fingers (finger	

		agnosia), and inability to make the distinction between the right and left side of the body.	
Anton syndrome	Bilateral occipital cortex involvement due to bilateral PCA infarct	Anton's syndrome describes the condition in which patients deny their blindness despite objective evidence of visual loss, and moreover confabulate to support their stance	Anosognosia (or lack of awareness of defect) and confabulation
Balint syndrome	Parieto-occipital lobes on both sides of the brain	Inability to perceive the visual field as a whole (simultanagnosia), difficulty in fixating the eyes (oculomotor apraxia), and inability to move the hand to a specific object by using vision (optic ataxia)	

(CN: cranial nerve; CST: corticospinal tract; SCP: superior cerebellar peduncle; AICA: anterior inferior cerebellar artery; PICA: posterior inferior cerebellar artery; LST: lateral spinothalamic tract ; SCM: sternocleidomastoid muscle; ICP: intracranial pressure; CST: corticospinal tract ; ML: medial leminiscus ; MLF: medial longitudinal fasciculus; PCA: posterior cerebral artery)

Transient Ischemic Attacks

Transient ischemic attack (TIA) is characterized by a brief episode of neurological dysfunction (sudden loss of function) in which symptoms and signs resolve completely after a brief period within 24 hours (usually within 30 minutes).

- Transient ischemic attack is defined as a transient episode of neurologic dysfunction caused by focal brain, spinal cord, or retinal ischemia, without acute infarction. However, TIAs may herald a stroke.
- Newly proposed definition classifies those with new brain infarction as ischemic strokes regardless of whether symptoms persist.

Clinical features: Hemiparesis and aphasia are most common. Other features include amaurosis fugax (sudden transient loss of vision in one eye), hemisensory loss, hemianopic visual loss, diplopia, vertigo, vomiting, choking and dysarthria, ataxia, etc.

Types of Transient Ischemic Attack

- Large artery low-flow TIA—recurrent, short lasting episodes of stereotyped symptoms (shotgun TIA/thrombotic TIA)
- Embolic TIA—longer lasting less frequent episodes with varied symptoms, changing territories
- Lacunar TIA.

Small Vessel (Lacunar) Stroke

- Small penetrating arterial branches of 200–800 μm in diameter, supply the deep brain parenchyma. Each of these small branches can be occluded either by atherothrombotic disease at its origin or by the development of occlusive vasculopathy—lipohyalinotic thickening (consequence of hypertension) **(Table 6E.5)**.
- Thrombosis of these vessels causes small infarcts that are referred to as lacunae. These infarcts range in size from 0.2 mm to 15 mm in diameter.

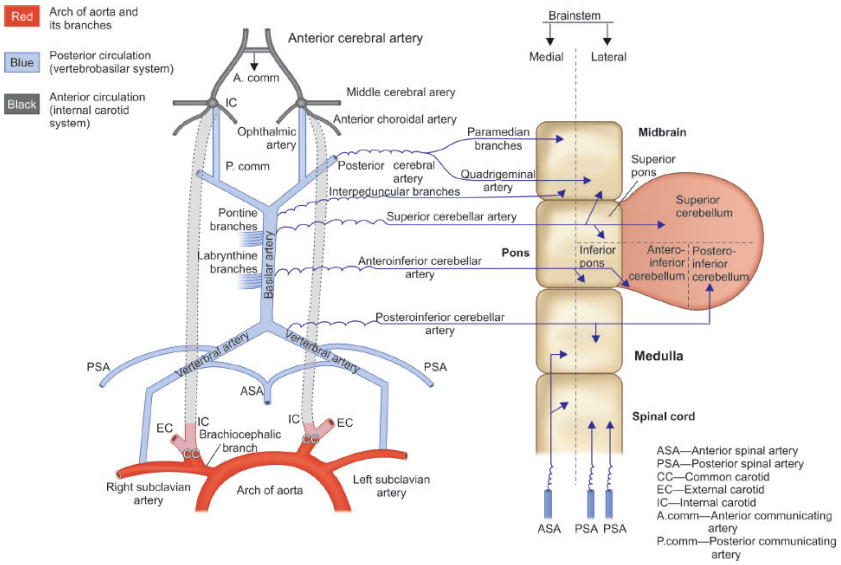


Fig. 6E.2: Cerebrovascular system (a comprehensive diagram of arterial system).

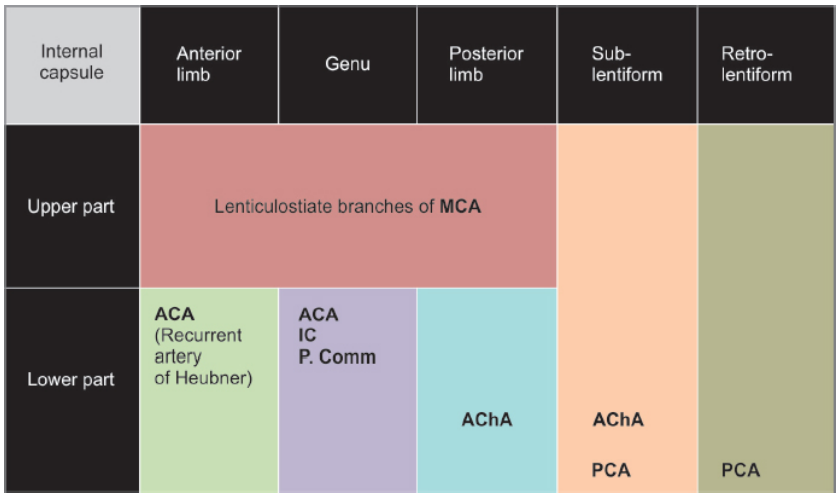


Fig. 6E.3: Blood supply of internal capsule.
(ACA: anterior cerebral artery; MCA: middle cerebral artery; PCA: posterior cerebral artery; AChA: anterior choroidal artery; IC: internal carotid artery (direct branches); P. Comm: posterior communicating artery)

Table 6E.5: Signs and symptoms of lacunar stroke depending on location of lesion.			
Syndrome	***Signs/symptoms***	***Localization***	***Vascular supply***
Pure motor	Contralateral hemiparesis or hemiplegia. Affects face, arm and leg equally	• Posterior limb of internal capsule • Corona radiata-Basis pontis	Lenticulostriate branches of the middle cerebral artery (MCA) or perforating arteries from basilar artery
Pure sensory	Contralateral hemisensory loss. Persistent or transient numbness and/or tingling on one side of the body	• Ventral posterolateral (VPL) nucleus of thalamus	Lenticulostriate branches of MCA. Small thalamoperforators of posterior cerebral artery (PCA)
Mixed sensorimotor	Contralateral weakness and numbness.	Thalamus and	Lenticulostriate branches of MCA

	Hemiparesis or hemiplegia with ipsilateral sensory impairment	adjacent posterior limb of internal capsule	
Dysarthria-clumsy hand	Slurred speech and weakness of contralateral hand (fine motor)	Basis pontis	Basilar artery perforators
Ataxic-hemiparesis	Combination of cerebellar and motor symptoms. Contralateral hemiparesis and ataxia out of proportion to weakness	• Internal capsule-posterior limb • Basis pontis • Corona radiata	• Lenticulostriate branches of MCA • Perforating arteries of basilar artery
Hemiballismus/hemichorea	Contralesional limb flailing/dyskinesis	Subthalamic nucleus	Perforating arteries of anterior choroidal or posterior communicating artery (PCOM)

2. APPROACH TO SPINAL CORD DISEASES

Spinal Cord Anatomy

The spinal cord originates at the medulla and continues caudally to terminate at the filum terminale, a fibrous extension of the conus medullaris is that terminates at the coccyx.

The adult spinal cord is approximately 45 cm long, oval or round in shape, and enlarged in the cervical and lumbar regions, where neurons that innervative the upper and lower extremities, respectively are located. The meninges that cover the spinal cord are continuous with those of the brainstem and cerebral hemispheres.

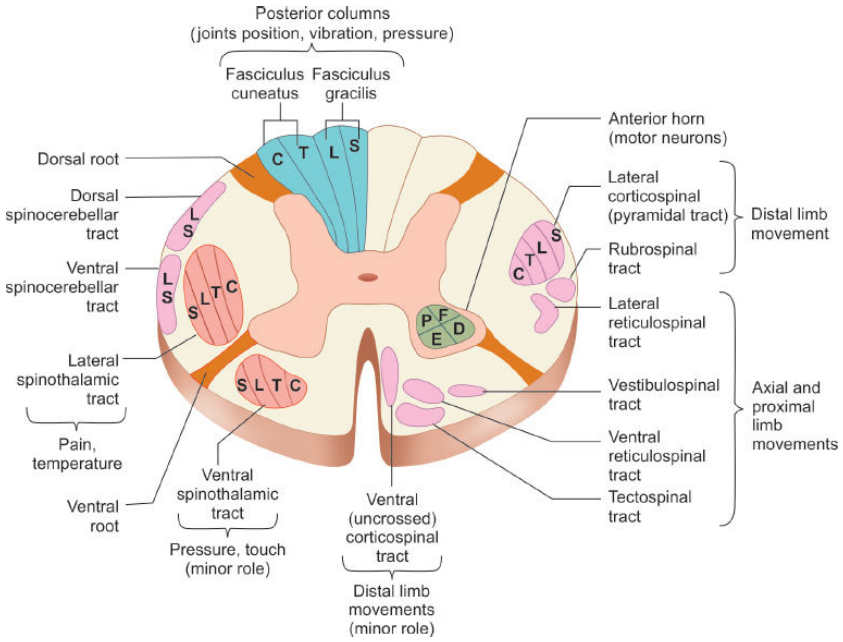


Fig. 6E.4: Tracts of spinal cord.

- The adult cord consists of 31 segments, each containing an exiting ventral motor root and entering dorsal sensory root.
- During embryologic development, growth of the cord lags behind that of the vertebral column, and in the adult spinal cord ends at approximately the first lumbar vertebral body. The lower spinal nerves take an increasingly downward course to exit via the appropriate intervertebral foramina.

- The first seven pairs of cervical spinal nerves exit above the same-numbered vertebral bodies, whereas all the subsequent nerves exit below the same-numbered vertebral bodies; this situation is due to the presence of eight cervical spinal cord segments but only seven cervical vertebrae.
- The approximate relationship between spinal cord segments and the corresponding vertebral bodies is shown in the following table:

Spinal cord level	Corresponding vertebral body
• Upper cervical • Lower cervical • Upper thoracic • Lower thoracic • Lumbar • Sacral • Coccygeal	• Same as cord level • 1 level higher • 2 levels higher • 2 to 3 levels higher • T 10 to T11 • T12 to L1 • L1

Features Suggestive of Involvement of Spinal Cord

- Presence of sensory deficit and/or motor weakness in both lower limbs and/or upper limbs.
- Bladder and bowel involvement
- Brown-Sequard type of clinical picture
- Presence of definite sensory level
- Vertebral pain.

VASCULAR SUPPLY OF SPINAL CORD (FIG. 6E.5)

- **The anterior spinal artery:** Union of the anterior spinal branches of the vertebral artery and descends within the anterior median fissure.
- **The two posterior spinal arteries:** Originate from the vertebral arteries and descend in the posterolateral sulcus.
- By themselves not sufficient and depend on feeder arteries that join them along their course (6–10 join the ASA and 10–20 join the PSA).
- **Thirty-one pairs of small radicular arteries:** Supply corresponding nerve roots.
- **Some of them give a branch to spinal arteries:** The radiculospinal branches.
- **C1-4:** Vertebral artery.
- **C5-T2:** Ascending and deep cervical artery.
- **T3 to T8:** Intercostal artery.
- **T9 and below:** Artery of Adamkiewicz—supplies most of the lower one-third of spinal cord; arises from a left-sided intercostal or lumbar artery (T8-L3).

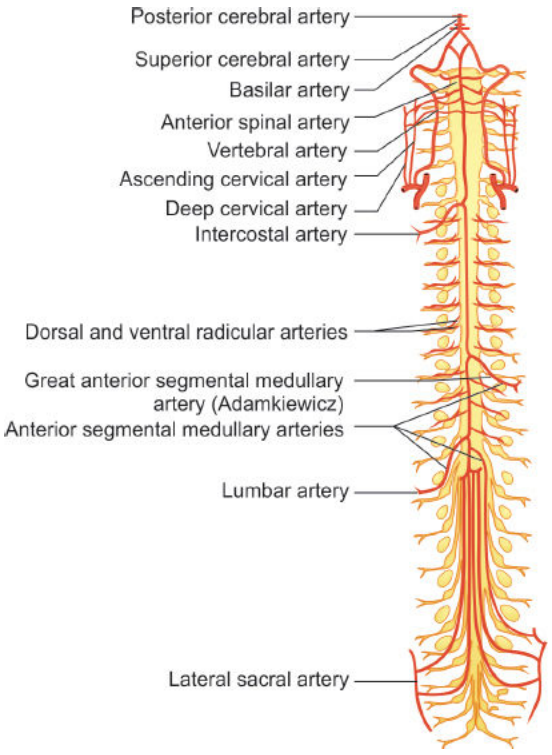


Fig. 6E.5: Vascular supply of spinal cord.

DIFFERENTIATION BETWEEN COMPRESSIVE AND NONCOMPRESSIVE MYELOPATHY

Features	Compressive	Noncompressive
Bony deformity	+	–
Bony tenderness	+	–
Girdle like sensation	+	–
Upper level of sensory loss	+	–
Zone of hyperesthesia	+	–
Root pain	+	–
Onset and progress	Gradual	May be acute
Symmetry	Asymmetrical	Majority are symmetrical
Flexor spasm	Common	Usually absent
Pattern of neurodeficit	U-shaped (Ellsberg phenomenon)	Bilaterally symmetrical
Bladder and bowel movement	Late	Early (acute transverse myelitis)
Selective tract involvement	Rare	Usually seen

Flowchart 6E.3 depicts the types of spinal cord diseases.

Compressive myelopathies examples

• Trauma • Tumor • Tuberculosis • Myeloma • Metastasis		
Extramedullary extradural • Caries spine • Metastasis • Intervertebral disc prolapse • Spondylosis • Fluorosis • Trauma to vertebra • Epidural abscess • Epidural hematoma • Hematomyelia	**Extramedullary intradural** • Meningioma • Neurofibroma • Schwannoma • Patchy arachnoiditis • Arteriovenous malformations • Lipoma • Sarcoma • Dermoid	**Intramedullary** • Ependymoma • Chordoma • Glioma

Flowchart 6E.3: Types of spinal cord diseases.

Noncompressive myelopathies examples
Inflammatory
• Infectious—viral, bacterial, fungal, and parasitic • Autoimmune—SLE, Sjogren's, sarcoidosis, Bechet syndrome, MCTD, polyarteritis nodosa, pANCA positive vasculitis • Demyelinating—MS, NMO, ADEM, and postviral postvaccinial • Paraneoplastic—lung carcinoma, breast, and ovary • Encephalomyelitis
Noninflammatory
• Inherited—HSP, inherited metabolic disorders • Metabolic—vitamin B_{12}, copper, folate and vitamin E deficiency—AIDS associated • Toxic—cassava, lathyrism, fluorosis, SMON, nitrous oxide, TOCP, and Konzo • Vascular—anterior spinal artery thrombosis, AVM, and dural arteriovenous fistula • Degenerative—familial spastic paraplegia • Physical agents—electrical injury, Caisson's disease, and radiation myelopathy

(SLE: systemic lupus erythematosus; MCTD: mixed connective tissue disease; pANCA: perinuclear antineutrophil cytoplasmic antibodies; MS: multiple sclerosis; NMO: neuromyelitis optica; ADEM: acute disseminated encephalomyelitis; HSP: hereditary spastic paraplegia; AIDS: acquired immunodeficiency syndrome; SMON: subacute myelo-optic neuropathy; TOCP: triorthocresyl phosphate; AVM: arteriovenous malformation)

Discriminate Between Extramedullary and Intramedullary Lesions

Features	Extramedullary	Intramedullary
Radicular pain	Common **Intradural:** Unilateral **Extradural:** Bilateral	Unusual
Vertebral pain	Common (extradural)	Unusual
Funicular pain	Rare	Common

Motor deficit	Ascending motor weakness, i.e. sacral → lumbar → thoracic → cervical	Descending pattern of loss, i.e. cervical → thoracic → lumbar → sacral
Upper motor neuron involvement	Early and prominent	Less pronounced; late feature
Lower motor neuron involvement	Segmental	Marked with widespread atrophy, fasiculations seen
Reflexes	Brisk early feature	Less brisk, later feature
Sensory deficit	Ascending sensory loss, i.e. sacral → lumbar → thoracic → cervical Saddle anesthesia Hemisection—contralateral loss of pain and temperature, ipsilateral loss of joint position	• Descending pattern of loss, i.e. cervical → thoracic → lumbar → sacral • Dissociative sensory loss • Suspended sensory loss (Jacket pattern)
Sacral sensation	Lost (early)	Sacral sparing
Autonomic involvement (bladder and bowel)	Late	Early
Trophic changes	Usually not marked	Common
Vertebral tenderness	May be present (extradural)	No bony tenderness in vertebrae
Changes in CSF	Frequent (increased protein, cells)	Rare

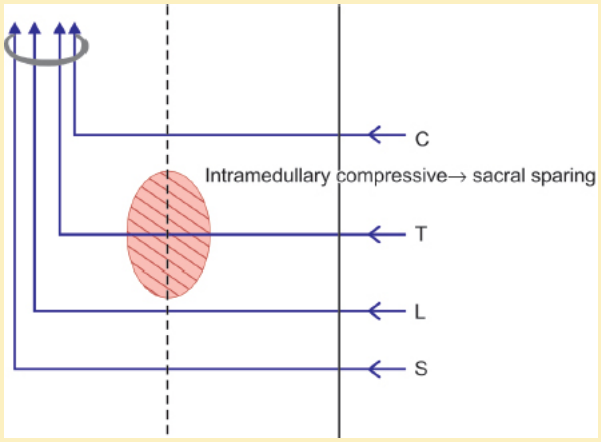


Fig. 6E.6: Arrangement of motor fibers.

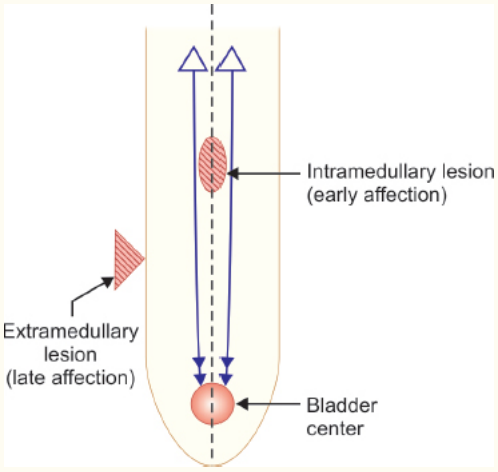


Fig. 6E.7: Bladder involvement in spinal cord disease.

Differences Between Presentation of Intradural and Extradural Lesion

Features	Extradural	Intradural
Mode of onset	Usually symmetrical	Asymmetrical
Root pain	Less common	More common
Spinal tenderness	Common	Uncommon
Spinal deformity	Present	Absent

Patterns of Spinal Cord Disease

1. Complete cord transection syndrome

2. Brown-Sequard syndrome/hemisection of the cord
3. Central cord syndrome (syringomyelia)
4. Posterior column syndrome (tabes dorsalis)
5. Posterolateral cord syndrome (SACDC)
6. Combined AHC—pyramidal tract syndrome (ALS)
7. AHC syndrome
8. Anterior spinal artery occlusion.

Complete Cord Transection

Causes	Features
• Trauma • Metastatic carcinoma • Multiple sclerosis • Spinal epidural hematoma • Autoimmune disorders • Postvaccinial syndromes	• **Sensory:** – All sensations are affected – Sensory level is usually 2 segments below the level of lesion – Segmental paresthesia occurs at the level of lesion • **Motor:** – Paraplegia due to corticospinal tract involvement – First spinal shock followed by hypertonic hyperreflexia paraplegia – Loss of abdominal and cremasteric reflexes – At the level of lesion LMN signs occur • **Autonomic:** – Urinary retention and constipation – Anhidrosis, trophic skin changes, vasomotor instability below the level of lesion – Sexual dysfunction can occur

Brown-Sequard Syndrome

Due to damage to one lateral half of spinal cord.

Causes	Features
• Caused by extramedullary lesions • Usually caused by penetrating injuries (gunshot) or tumor	• **Sensory:** – Ipsilateral loss of proprioception due to posterior column involvement – Contralateral loss of pain and temperature due to involvement of lateral spinothalamic tract 1 or 2 segments below • **Motor:** – Ipsilateral spastic weakness due to descending corticospinal tract involvement – Lower motor neuron signs at the level of lesion

Central Cord Syndrome

Causes	Features
• Most common cause is syringomyelia • Other causes are hyperextension, injuries of neck, intramedullary tumors and trauma • Associated with Arnold Chiari type 1 and 2 and Dandy Walker malformation	• **Sensory:** – Pain and temperature are affected – Touch and proprioception are preserved – Dissociative anesthesia – Shawl like distribution of sensory loss • **Motor:** – Upper limb weakness > Lower limb weakness • **Other features include:** – Horner's syndrome – Kyphoscoliosis – Sacral sparing – Neuropathic arthropathy of shoulder and elbow joint Early bladder involvement (exception—syringomyelia)

Posterior Column Syndrome

Cause	Features
Occurs due to neurosyphilis, diabetes mellitus	• **Sensory:** – Impaired position and vibration sense in lower limb – Sensory ataxia – Positive Romberg's sign, sink sign and Lhermitte's sign • **Abadie's sign** positive • Urinary incontinence • Absent knee and ankle jerk (areflexia and hypotonia) • Charcot's joint • Miotic and irregular pupil not reacting to light—Argyll Robertson pupil

Posterolateral Column Disease

Causes	Features
• Vitamin B_{12} deficiency • AIDS • HTLV associated myelopathy • Cervical spondylosis	• **Sensory:** – Paresthesia in feet – Loss of proprioception and vibration in legs – Sensory ataxia – Positive Romberg's sign • **Bladder** atonia • **Motor:** – Corticospinal tract involvement—spasticity, hyperreflexia, bilateral Babinski sign • AIDS-associated dementia and spastic bladder is present • HTLV associated myelopathy—slowly progressive paraparesis and an increase in CSF IgG antibodies to HTLV1

(AIDS: acquired immunodeficiency syndrome; HTLV: human T-cell lymphotropic virus; CSF: cerebrospinal fluid; IgG: immunoglobulin G)

Anterior Horn Cell Syndromes

Cause	Features
Spinal muscular atrophy (SMA)	• **Motor:** – Weakness, atrophy, and fasciculations – Hypotonia with depressed reflexes – Muscles of trunk and extremities are affected • **Sensory system** is not affected

Anterior Spinal Artery Syndrome

Cause	Features
Occurs due to syphilitic arteritis, aortic dissection, atherosclerosis of aorta, SLE, AIDS, and AV malformation	• **Motor:** – Flaccid and areflexic paraplegia • **Sensory:** – Loss of pain and temperature – Preservation of position and vibration • **Autonomic:** – Urinary incontinence – Spinal cord infarction usually occurs in T1 to T4 and L1 segment • Abrupt onset, radicular, or girdle pain

Postspinal Artery Syndrome

Cause	Features

Rare	• Loss of proprioception and vibratory sense • Pain and temperature is preserved • Absence of motor deficit

Anterior Horn Cell and Pyramidal Tract

Cause	Features
ALS—amyotrophic lateral sclerosis	• LMN signs • UMN signs • Sensations preserved • Onuf's nucleus spared—hence no bladder and bowel involvement

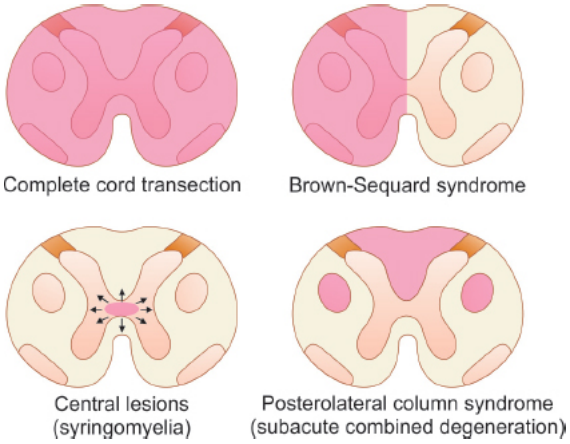


Fig. 6E.8: Spinal cord syndromes 1.

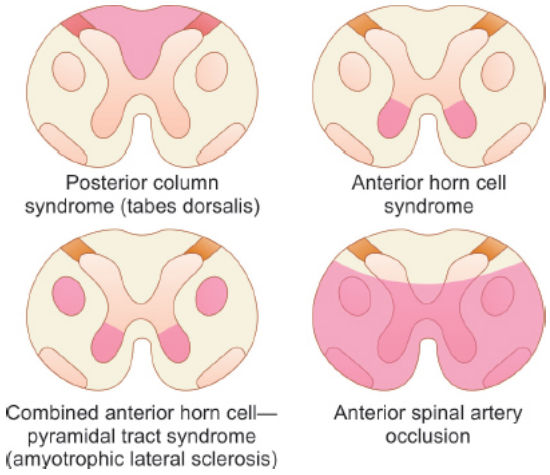


Fig. 6E.9: Spinal cord syndromes 2.

Difference Between Paraplegia in Flexion and Paraplegia in Extension

Features	Paraplegia in extension	Paraplegia in flexion
Definition	Lower limb takes an extension attitude and extensor muscles are spastic	Lower limb muscles take an attitude of flexion
Pathology	Only pyramidal tract involved	Both pyramidal and extrapyramidal tract involved (reticulospinal tracts). Occurs in late stage of paraplegia

Evolution	Early	Late
Tone	Clasp knife spasticity in extensor group	Tone is increased in flexor groups
Deep tendon reflex (DTR)	• Deep tendon reflexes are exaggerated • Clonus may be present	• DTR's are present but diminished • No clonus
Plantar reflex	Extensor plantar response	Extensor plantar associated with flexor spasm
Mass reflex**	Absent	Present

Note: ****Mass reflex:** Any stimulation (scratching of skin) below the level of lesion produces an interoceptive response resulting in flexor spasms, spontaneous emptying of bowel and bladder, profuse sweating and piloerection and seminal emission.

Cord Involvement at Multiple Sites

- Arachnoiditis (in tubercular, there is patchy involvement)
- Neurofibromatosis
- Multiple sclerosis
- Secondary deposits
- Cervical spondylitis.

Causes of Spastic Paraplegia (UMN Type Lesion)

A. **Gradual onset**

- Cerebral causes—parasagittal meningioma, hydrocephalus, etc.
- Spinal causes:
  - Compressive or transverse lesion in the spinal cord
  - Noncompressive or longitudinal lesion or systemic disease of the spinal cord.
- Motor neuron disease (MND), e.g. amyotrophic lateral sclerosis
- Multiple sclerosis, Devic's disease
- Friedreich's ataxia
- Subacute combined degeneration (i.e. from vitamin B_{12} deficiency)
- Lathyrism
- Syringomyelia
- Hereditary spastic paraplegia
- Erb's spastic paraplegia
- Tropical spastic paraplegia
- Radiation myelopathy.

B. **Sudden onset**

- Cerebral causes—thrombosis of unpaired anterior cerebral artery, superior sagittal sinus thrombosis
- Spinal causes:

  Compressive causes:
  - Injury to the spinal cord (fracture-dislocation or collapse of the vertebra)
  - Prolapsed intervertebral disc
  - Spinal epidural abscess or hematoma.

  Noncompressive causes:
  - Acute transverse myelitis
  - Thrombosis of anterior spinal artery
  - Hematomyelia (from arteriovenous malformation, angiomas, or endarteritis)
  - Radiation myelopathy electrical injury.

Causes of Flaccid Paraplegia (LMN Type)

- UMN lesion in shock stage, transverse myelitis, spinal injury
- Lesion involving anterior horn cells:
  - Acute anterior poliomyelitis
  - Progressive muscular atrophy (variety of MND).
- Diseases affecting nerve root—tabes dorsalis, radiculitis, Guillain-Barré (GB) syndrome
- Diseases affecting peripheral nerves:
  - Acute infective polyneuropathy (GB syndrome)
  - High cauda equina syndrome
  - Disease of peripheral nerves involving both the lower limbs
  - Lumbar plexus injury (psoas abscess or hematoma).
- Diseases affecting myoneural junction:
  - Myasthenia gravis, Lambert-Eaton syndrome
  - Periodic paralysis due to hypo- or hyperkalemia.
- Diseases affecting muscles—myopathy.

Causes of Quadriplegia

Weakness of all the 4 limbs can occur in the lesions from cortex to C5 level of spinal cord and various LMN lesion affecting anterior horn cells, roots, peripheral nerve, NM junction, and muscles.

Upper motor neuron causes	Lower motor neuron causes
• Cerebral palsy • Bilateral brainstem lesion (glioma) • Craniovertebral anomaly • High cervical cord compression • Multiple sclerosis • Motor neuron disease	• Acute anterior poliomyelitis • Guillain-Barré syndrome • Peripheral neuropathy • Myopathy or polymyositis • Myasthenia gravis and crisis • Periodic paralysis • Snake bite, organophosphate poisoning, etc.

SPECIFIC LOCALIZING SIGNS AT VARIOUS LEVELS

Features of Cervical Signs at Cord Lesion

In general, cervical cord disorders are best localized by the pattern of weakness that ensues, whereas sensory deficits have less localizing value.

- High cervical cord lesions (lesions above C5) are frequently life threatening, produce quadriplegia and weakness of diaphragm, the main respiratory muscle innervated by the phrenic nerve (C3-C5).
- Extensive lesions near the junction of the cervical cord and medulla are usually fatal owing to involvement of adjacent medullary centers, which results in vasomotor and respiratory collapse.
- Compressive lesions near the foramen magnum may produce weakness of the ipsilateral shoulder and arm followed by weakness of the ipsilateral leg, then the contralateral leg, and finally the contralateral arm (cartwheel pattern or Ellsberg phenomenon).
- Lesions at C4-C5 produce quadriplegia with preserved respiratory function.
- At the midcervical (C5-C6) level, there is relative sparing of shoulder muscles and loss of biceps and brachioradialis reflexes.
- Lesions at C7 spare the biceps but produce weakness of finger and wrist extensors and loss of the triceps reflex.
- Lesions at C8 paralyze finger and wrist flexion, and the finger flexor reflex is lost.
- Horner's syndrome (miosis, ptosis, and facial hypohidrosis) may also occur ipsilateral to cervical lesions at any level.

Features of Thoracic Cord Lesion

Lesions of the thoracic cord are best localized by identification of a sensory level on the trunk.

- Useful markers in terms of sensory dermatomes are at the nipples (T4), xiphisternum (T6), subcostal margins (T8), umbilicus (T10), and pubic symphysis (T12)
- The abdominal wall musculature, supplied by the lower thoracic nerves is observed during movements of respiration or coughing or by asking the patient to interlock the fingers behind the head in the supine position and attempt to sit up.
- Lesions at T9-T10 paralyze the lower, but spare the upper, abdominal muscles, resulting in upward movement of the umbilicus when the abdominal wall contracts (Beevor's sign) and in loss of lower, but not upper, superficial abdominal reflexes.
- With unilateral lesions, attempts to contract the abdominal wall produce movement of the umbilicus to the normal side; superficial abdominal reflexes are absent on the involved side.
- Midline back pain is a useful localizing sign in the thoracic region.

Feature of Lumbar Cord

Effect of various root lesions in lumbar region:

Roots	Motor deficit (most rapidly demonstrated)
L2	Hip flexion and thigh adduction
L3	Knee extension and thigh adduction
L4	Inversion of foot
L5	Dorsiflexion to toes and foot
S1	Plantar flexion and eversion of foot

- Lesions at L2-L4 paralyze flexion and abduction of the thigh, weaken leg extension at the knee, and abolish the patellar reflex.
- Lesions at L5-S1 paralyze movements of the foot and ankle, flexion at the knee, and extension of the thigh, and abolish the ankle jerk (S1).
- A cutaneous reflex useful in localization of lumbar cord disease is the cremasteric reflex, which is segmentally innervated at L1-L2.

Features of Sacral Cord/Conus Medullaris

The conus medullaris is the tapered caudal termination of the spinal cord, comprising the lower sacral and single coccygeal segments. Isolated lesions of the conus medullaris spare motor and reflex functions in the legs.

The Conus Syndrome (Fig. 6E.10)

- Bilateral saddle anesthesia (S3-S5), prominent bladder and bowel dysfunction (urinary retention and incontinence with lax anal tone), and impotence
- The bulbocavernous (S2-S4) and anal (S4-S5) reflexes are absent
- Muscle strength is largely preserved.

Cauda Equina Syndrome—Asymmetric, Atrophic, and Areflexic Paralysis of Lower Limbs (Fig. 6E.10)

- The cluster of nerves derived from the lower cord as they descend to their exits in the intervertebral foramina (L2-3 to coccygeal nerve roots).
- Cauda equina lesions are characterized by severe low back or radicular pain, asymmetric leg weakness or sensory loss, variable areflexia in the lower extremities, and relative sparing of bowel and bladder function.
- Mass lesions in the lower spinal canal may produce mixed clinical picture in which elements of both cauda equina and conus medullaris syndromes coexist.

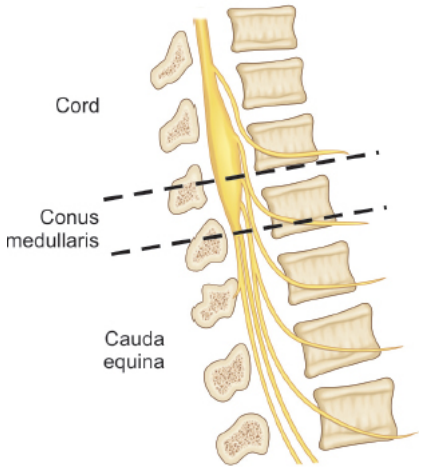


Fig. 6E.10: Conus-cauda equina syndrome.

	Conus medullaris syndrome (S2-4)	Cauda equina syndrome (L3 root and below)
Presentation	Sudden and bilateral	Gradual and unilateral
Reflexes	Knee jerk is preserved but ankle jerk is affected	Both knee and ankle jerks are affected
Radicular pain	Less severe	More severe
Low back pain	More	Less
Sensory symptoms and sings	Numbness is symmetrical and bilateral, sensory dissociation occurs, saddle anesthesia present	Numbness is asymmetrical, may be unilateral, no necessary dissociation
Motor strength	Typically symmetric hyperreflexia, distal paresis of lower limbs	Asymmetric areflexic paraplegia
Impotence	Frequent	Less frequent
Sphincter dysfunction	Overflow urinary incontinence and fecal incontinence, tend to present early in course of disease	Urinary retention tends to present late in course of disease
Trophic changes	Common	Less marked

Epiconus: Lesion of lumbar cord at the level of L4-S2 characterized by a flaccid paralysis of legs (only the roots are affected causing peripheral paralysis, i.e. distal paraplegia). Reflex but not conscious evacuation of the bladder is present, and rectum is preserved. Sexual potency is lost.

What are the Different Types of Spinal Pain?

- Radicular pain is characterized as a unilateral, lancinating, dermatomal pain often exacerbated by cough, sneeze, or Valsalva's maneuver. Radicular pain is common with extradural growths and rare with intramedullary lesions. An example of an extramedullary tumor causing radicular pain is the neurilemmoma (usually an intradural extramedullary lesion).
- Vertebral pain is characterized by an aching pain localized to the point of the spine involved in the compressive process and often accompanied by point tenderness. Spinal pain is common with neoplastic or inflammatory extradural lesions and infrequent with intramedullary or intradural extramedullary lesions.
- Funicular (central) pain is common with intramedullary lesions and very unusual with extradural lesions. It is described as deep, ill-defined painful dysesthesias, usually distant from the affected spinal cord level (and therefore of poor localizing value), probably related to dysfunction of the spinothalamic tract or posterior columns.

- With dysfunction of the posterior columns in the cervical region, neck flexion may elicit a sudden "electric-like" sensation down the back or into the arms (Lhermitte's sign or "barber's chair syndrome).

APPROACH TO PERIPHERAL NEUROPATHY

Various nerve fibers and their functions are depicted in **Figure 6E.11**.

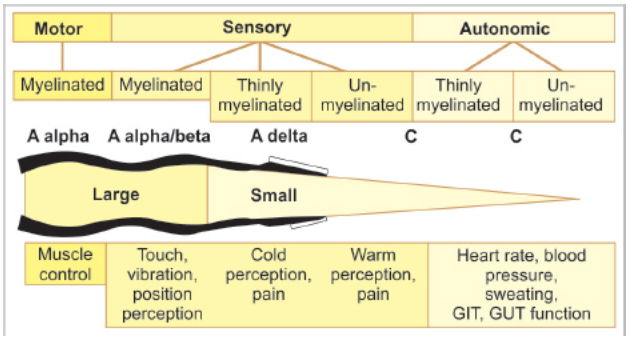


Fig. 6E.11: Various nerve fibers and their function.

Clinical Types of Neuropathy

1. **Polyneuropathy:** It is the most common variety of neuropathy. The nerve fibers are affected in a length-dependent pattern; toes and soles are affected first and hands later. A majority of these cases occur due to metabolic, toxic, or systemic disorders.

Causes of polyneuropathy
• **D**iabetes mellitus • **A**lcohol • **N**utritional (B_{12} deficiency) • **G**uillain-Barré syndrome • **T**oxins (Pb, As, Zn, and Hg) • **H**ematologic (paraproteins) • **E**ndocrine (hypothyroid) • **R**heumatologic (systemic lupus erythematosus, rheumatoid arthritis, and vasculitis) • **A**myloid • **P**orphyria • **I**nfectious (syphilis, human immunodeficiency syndrome) • **S**arcoid • **T**umor (paraneoplastic) **"DANG THERAPIST"**

2. **Mononeuropathy:** Mononeuropathy refers to single peripheral nerve involvement and usually occurs due to trauma, compression, or entrapment.

Causes of mononeuropathy	
• **Acute:** Sustained pressure, e.g. tourniquet • **Chronic:** Entrapment.	
Causes (according to site of compression)	
• Carpal tunnel	Median nerve
• Cubital tunnel	Ulnar nerve
• Spiral groove of humerus	Radial nerve
• Inguinal ligament	Lateral cutaneous of thigh (meralgia paresthetica)
• Neck of fibula	Common peroneal nerve
• Flexor retinaculum (Tarsal tunnel)	Posterior tibial nerve

Entrapment neuropathies are commonly seen in

- Endocrinal (diabetes mellitus, myxedema, acromegaly)
- Amyloidosis
- Hereditary neuropathy susceptible to pressure palsy
- Pregnancy
- Arthritis (rheumatoid)

3. **Multiple mononeuropathies/mononeuritis multiplex** refers to the involvement of multiple, separate noncontiguous peripheral nerves either simultaneously or sequentially.

Causes of mononeuritis multiplex
• Leprosy (most common) • Diabetes mellitus • Vasculitis • Sarcoidosis • Amyloidosis • Malignancy • Neurofibromatosis • HIV infection • Idiopathic multifocal motor neuropathy

PATHOLOGIC CLASSIFICATION OF NEUROPATHIC DISORDERS (FIGS. 6E.12A AND B)

1. Neuronopathies (pure sensory or pure motor):

- Sensory neuronopathies (ganglionopathies)
- Motor neuronopathies (motor neuron disease)

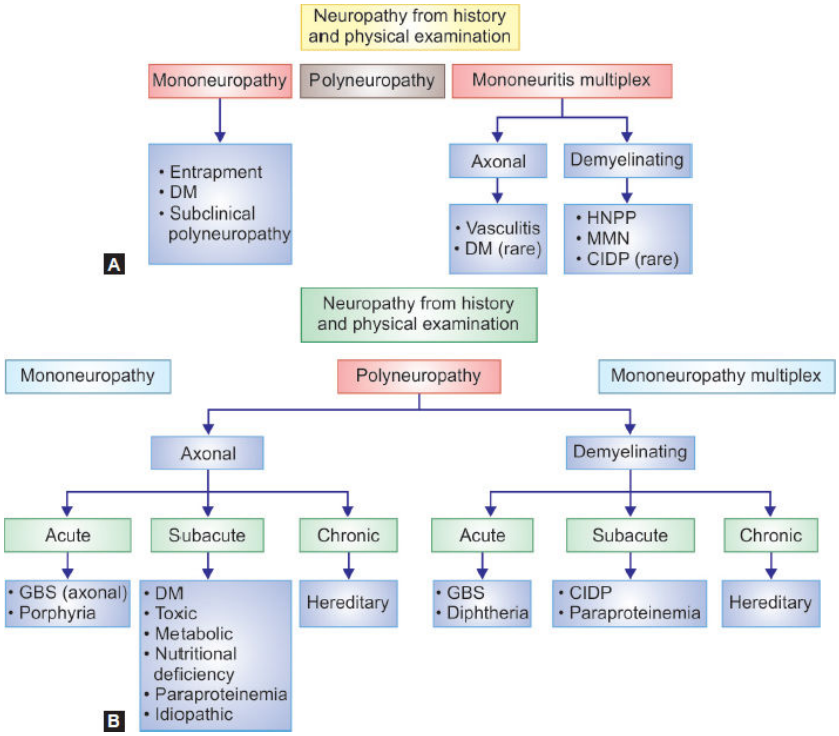
Sensory neuronopathy	Motor neuronopathy
• Ganglion cells predominantly affected • Both proximal and distal involvement • Sensory ataxia is common • No weakness • But awkward movement due to sensory disturbances **Example:** • Cancer (paraneoplastic) • Sjogren's syndrome • Cisplatin and other analogs • Vitamin B_6 toxicity • HIV–related sensory neuronopathy	Disorder of anterior horn cells. Weakness, fasciculation, atrophy not truly a process of peripheral nerves

2. Peripheral neuropathies (usually sensorimotor):

- Myelinopathies
- Axonopathies

Axonal neuropathy	Demyelinating neuropathy
Usually gradual and insidious onset	Usually acute or subacute
Large and long axons are affected early, hence initially lower extremeties are affected	Diffuse process, starts in lower limbs. But not always distal
Stocking-glove sensory motor loss results in symmetrical distal clinical signs in legs and arms	Generalized weakness and mild sensory loss
Distal involvement	Proximal and distal involvement

Ankle jerk lost early and proximal tendon reflexes preserved	All reflexes are lost early
Muscle wasting common	Relatively absent
Cerebrospinal fluid (CSF) proteins normal	CSF proteins elevated (since nerve roots are involved)
Slow recovery	Rapid recovery
Residual deformity common	Residual deformity less common
Nerve conduction normal or slightly lowered	Nerve conduction is slowed



Figs. 6E.12A and B: Classification of neuropathy based on history and examination.

(DM: diabetes mellitus; HNPP: hereditary neuropathy with liability to pressure palsies; CIDP: chronic inflammatory demyelinating polyneuropathy; MMN: multifocal motor neuropathy; GBS: Guillain–Barré syndrome)

APPROACH TO POLYNEUROPATHY

What is the onset and temporal evolution? • Acute (days to 4 weeks) • Subacute (4–8 weeks) • Chronic (>8 weeks)	
Acute onset	• Guillain-Barré syndrome • Acute intermittent porphyria • Critical illness polyneuropathy • Thallium toxicity
Subacute onset	• Toxins or medications • Nutritional deficiency • Metabolic abnormality

	• Paraneoplastic syndrome
Chronic	• Hereditary motor and sensory neuropathy (HMSN) • CIDP • CKD
Relapsing/remitting course	• Guillain-Barré syndrome • CIDP • HIV/AIDS • Porphyria

(CIDP: chronic inflammatory demyelinating polyneuropathy; CKD: chronic kidney disease; HIV: human immunodeficiency virus; AIDS: acquired immunodeficiency syndrome)

What systems are involved? Motor (or) sensory (or) autonomic (or) mixed	
Motor symptoms	
Negative symptoms	***Positive symptoms***
• Weakness • Wasting • Loss of dexterity In the early stage, weakness in peripheral neuropathy is distal; however, early proximal weakness is a feature of demyelinating neuropathy and porphyric neuropathy	• Cramps • Tremors • Fasciculations • Spasms
Neuropathic disorders that may have only motor symptoms at presentation • Motor neuron disease • Lead intoxication • Acute porphyria • Guillain-Barre Syndrome • Hereditary motor neuropathy • CIDP • Diphtheria • Brachial neuritis • Diabetic lumbosacral plexus neuropathy	
Sensory symptoms	
Negative symptoms	***Positive symptoms***
Numbness, loss of sensation in hands and feet	Burning, pain, walking on cotton wool, band-like sensation on feet or trunk, stumbling, tingling, pins, and needles
Large fiber neuropathy—neuropathy of signs/ataxic neuropathy There are few symptoms (numbness, ataxia) but lots of signs (loss of vibration, joint position sense, diminished reflexes, Romberg's sign positive)	**Small fiber neuropathy—neuropathy of symptoms** Lots of symptoms (PAIN—burning, shock like, stabbing, prickling, shooting, lancinating, allodynia, tight band like pressure. Insensitive to heat and cold) but very few signs (loss of pain, temperature)
Examples: • Sjogren's syndrome • Vitamin B_{12} neuropathy • Cisplatin • Pyridoxine neurotoxicity • Friedreich's ataxia	**Examples:** • Diabetes • Amyloidosis • Fabry's disease • HIV • Tangier's disease • Hereditary sensory and autonomic neuropathy • Sjogren's syndrome • Chronic idiopathic small fiber sensory neuropathy
Small and large fiber neuropathy—pan sensory: Global sensory loss **Example:** • Carcinomatous sensory neuropathy	

- Hereditary sensory neuropathy
- Diabetic sensory neuropathy
- Vacor intoxication
- Xanthomatous neuropathy of primary biliary cirrhosis

Peripheral neuropathies that are often associated with pain
- Cryptogenic sensory or sensorimotor neuropathy
- Diabetes mellitus
- Vasculitis
- Guillain–Barré syndrome
- Amyloidosis
- Toxic (arsenic and thallium)
- HIV related distal symmetrical polyneuropathy
- Fabry's disease

Autonomic symptoms

Enquire if the patient has fainting spells or orthostatic lightheadedness, sweating abnormalities or any bowel, bladder, or sexual dysfunction.

Examples:

Acute:
- Pandysautonomia
- Botulism
- Porphyria
- Guillain-Barré syndrome
- Amiodarone
- Vincristine

Chronic:
- Amyloid
- Diabetes
- Sjogren's
- HSN 1 and 3
- Chagas disease
- Paraneoplastic

PATTERNS OF NEUROPATHY

Pattern 1

Symmetric Proximal and Distal Weakness with Sensory Loss

Inflammatory demyelinating polyneuropathy (GBS and CIDP).

Pattern 2

Symmetric Distal Weakness with Sensory Loss

Metabolic disorders, hereditary toxins drugs.

Pattern 3

Asymmetric Distal Weakness with Sensory Loss
- Multiple nerves—vasculitis
- Single nerves/regions—compressive mononeuropathy and radiculopathy.

Pattern 4

Asymmetric Distal Weakness without Sensory Loss
- Motor neuron disease—with upper motor neuron findings
- Multifocal motor neuropathy—without upper motor neuron findings.

Pattern 5

Asymmetric Proximal and Distal Weakness with Sensory Loss

- Polyradiculopathy or plexopathy due to diabetes mellitus
- Meningeal carcinomatosis.

Pattern 6

Symmetric Sensory Loss without Weakness

Cryptogenic sensory polyneuropathy (CSPN), metabolic (diabetes and others) drugs, and toxins.

Pattern 7

Symmetric Sensory Loss and Distal Areflexia with Upper Motor Neuron Findings

B_{12} deficiency, HIV, and hepatic disease.

Pattern 8

A Symmetric Proprioceptive Sensory Loss without Weakness

Sensory neuronopathy (ganglionopathy).

Pattern 9

Autonomic Symptoms and Signs

Neuropathies associated with autonomic dysfunction.

Pattern 10

Syndrome of Acute Ascending Motor Paralysis

- Guillain-Barré syndrome/acute idiopathic polyneuritis
- Diphtheria
- Porphyria
- Triorthocresyl phosphate (TOCP) poisoning
- Paraneoplastic
- Postvaccinial.

Pattern 11

Syndrome of Subacute Sensory Motor Neuropathy

- Deficiency—alcoholic beriberi, pellagra, and vitamin B_{12}
- Toxins = arsenic, lead, Hg, and Pb
- Drugs = nitrofurantoin, INH, dapsone, disulfuram, and clioquinol
- Uremic
- DM, PAN and sarcoidosis.

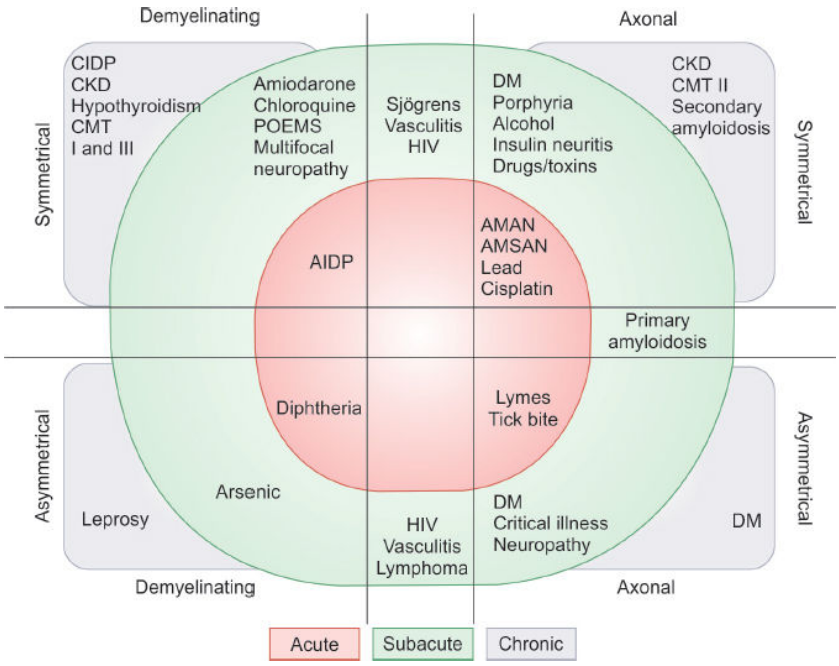


Fig. 6E.13: Simplified diagram showing types of polyneuropathy.
(CIDP: chronic inflammatory demyelinating polyneuropathy; CKD: chronic kidney disease; CMT: Charcot-Marie-Tooth; POEMS: polyneuropathy, organomegaly, endocrinopathy, monoclonal gammopathy and skin changes; DM: diabetes mellitus; HIV: human immunodeficiency virus; AIDP: acute inflammatory demyelinating polyneuropathy; AMAN: acute motor axonal neuropathy; AMSAN: acute motor and sensory axonal neuropathy)

General examination in neurpoathy	
Purpura, livedo reticularis	Vasculitis
Skin hypopigmentation	Leprosy
Hyperpigmentation	Osteosclerotic myeloma—POEMS
Bullous lesions	Variegate porphyria
Purpura	Vasculitis, cryoglobulinemia
Ichthyosis	Refsum's disease
Mee's lines	Arsenic/thallium intoxication
Alopecia	Thallium poisoning
Curled hair	Giant axonal neuropathy
Nerve thickening	• Leprosy • CMT • CIDP • Amyloidosis • Neurofibromatosis • Refsum's disease • Dejerine-Sottas disease • Roussy Levy syndrome • Acromegaly • Idiopathic

(POEMS: polyneuropathy, organomegaly, endocrinopathy, monoclonal gammopathy and skin changes; CMT: Charcot-Marie-Tooth; CIDP: chronic inflammatory demyelinating polyneuropathy)

CRANIAL NERVE EXAMINATION IN NEUROPATHY

- Anosmia—Refsum's disease and B_{12} deficiency
- Optic atrophy—demyelinating disease may suggest an inherited syndrome, B_{12} deficiency
- Anisocoria and impaired pupillary light reflexes—parasympathetic damage and may be isolated, as in Adie's syndrome, diabetic neuropathy or acute dysautonomia as in GBS
- Impaired ocular mobility suggests botulism or Miller Fisher syndrome
- Facial weakness—GBS, CIDP, Lymes disease, and leprosy
- Trigeminal sensory loss—Sjogren neuropathy
- Lower cranial nerve palsies—Kennedy's disease.

Medications causing neuropathies	
• **Axonal** – Vincristine – Paclitaxel – Nitrous oxide – Colchicine – Isoniazid – Hydralazine – Metronidazole – Pyridoxine – Didanosine – Lithium – Dapsone – Phenytoin – Cimetidine – Disulfiram – Chloroquine – Ethambutol – Amitriptyline	• **Demyelinating** – Amiodarone – Chloroquine – Suramin – Gold • **Neuronopathy** – Thalidomide – Cisplatin – Pyridoxine

COMMON NEUROPATHIES

Guillain-Barré Syndrome (Tables 6E.6 and 6E.7)

Table 6E.6: Diagnostic criteria of GBS.
Required features • Progressive weakness in both arms and legs • Areflexia (or hyporeflexia)
Features supportive of diagnosis • Progression of symptoms over days to 4 weeks • Relative symmetry • Mild sensory signs or symptoms • Cranial nerve involvement, especially bilateral facial weakness • Recovery beginning 2–4 weeks after progression ceases • Autonomic dysfunction • Absence of fever at onset • Typical CSF (albuminocytologic dissociation) • EMG/nerve conduction studies (characteristic signs of a demyelinating process in the peripheral nerves)
Features casting doubt on the diagnosis

- Asymmetrical weakness
- Persistent bladder and bowel dysfunction
- Bladder or bowel dysfunction at onset
- >50 mononuclear leukocytes/mm^3 or presence of polymorphonuclear leukocytes in CSF
- Distinct sensory level

Features that rule out the diagnosis
- Hexacarbon abuse
- Abnormal porphyrin metabolism
- Recent diphtheria infection
- Lead intoxication
- Other similar conditions: Poliomyelitis, botulism, hysterical paralysis, toxic neuropathy

(CSF: cerebrospinal fluid; EMG: electromyogram)

Table 6E.7: Variants of GB syndrome.

Common variants	*Less common variants*
• Acute motor and sensory axonal neuropathy (AMSAN) • Acute motor axonal neuropathy (AMAN) • Miller-Fisher variant • Pure motor variants • Pure sensory variants • Pure dysautonomia variant • Pharyngeal-cervical-brachial variant • Paraparetic variant (Ropper variant)	• Acral paresthesias with diminished reflexes in either arms or legs • Facial diplegia or abducens palsies with distal paresthesias • Isolated postinfectious ophthalmoplegia • Bilateral foot drop with upper limb paresthesias • Acute ataxia without ophthalmoplegia • Bickerstaff's brainstem encephalitis (BBE)

Diabetes Mellitus (Box 6E.1)

Box 6E.1: Classification of diabetic neuropathy.

Polyneuropathy
- Symmetrical, mainly sensory and distal
- Asymmetrical, mainly motor and proximal (including amyotrophy)

Mononeuropathy and mononeuritis multiplex
- Cranial nerve lesions
- Isolated peripheral nerve lesions

Autonomic (visceral) neuropathy
- Cardiovascular
- Gastrointestinal
- Genitourinary
- Sudomotor
- Vasomotor
- Pupillary

Polyradiculopathies
- Diabetic amyotrophy (lumbar polyradiculopathy)
- Thoracic polyradiculopathy
- Diabetic neuropathic cachexia

Treatment-induced neuropathy of diabetes

Neuropathies with HIV Infection

- **Seroconversion**
  - Guillain-Barre syndrome
  - Chronic inflammatory demyelinating polyneuropathy (CIDP).
- **Symptomatic stage:** Mononeuritis multiplex axonal type subacute or chronic
- **Late symptomatic stage:** Distal symmetrical sensory polyneuropathy, most common neuropathy frequently coexists with symptomatic encephalopathy and myelopathy
  - Toxic polyneuropathy (drugs)
  - Subacute asymmetrical polyneuropathy of cauda equina, caused by cytomegalovirus.

HEREDITARY NEUROPATHIES

Neuropathy is the sole or primary part of the disease	Neuropathy is part of a more generalized neurological or multisystem disorder
• *Charcot-Marie-tooth disease*—CMT1 (demyelinating) and CMT2 (axonal) • HMSN-III (or *Dejerine–Sottas neuropathy)* • Hereditary sensory and autonomic neuropathy (HSAN)	• Spinocerebellar atrophy (SCA)—Friedreich ataxia (FA) • Hereditary spastic paraplegia neuropathy (i.e. complicated HSP, HMSN 5) • Familial amyloid (transthyretin, gelsolin, ApoA1)
• Distal hereditary motor neuropathy (dHMN) • Hereditary brachial plexus neuropathy (HBPN) • Hereditary neuropathy with liability to pressure palsies (HNPP)	• Leukodystrophy • Lipoprotein deficiency • Porphyrias

APPROACH TO A PATIENT WITH PARKINSON'S DISEASE

Idiopathic Parkinson's Disease (Paralysis Agitans)

It is a chronic, progressive disorder in which idiopathic parkinsonism occurs without evidence of more widespread neurologic involvement.

Clinical Manifestations

Motor symptoms: Always asymmetrical in onset and become bilateral within a year (Table 6E.8).

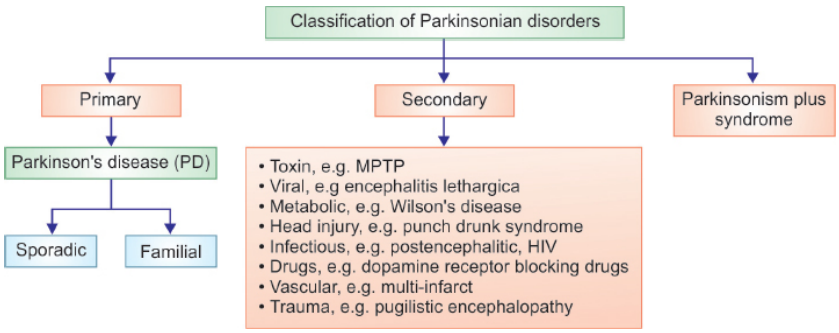
- **Tremor** is an early and presenting symptom in 70% of patients.
  - Frequency is 4–6 Hz tremor and is typically most prominent at rest and worsens with emotional stress.
  - Typically tremor starts with the fingers and hands at rest.
  - Often described as pill rolling of finger and wrist, because the patient appears to be rolling something between thumb and forefinger.
  - Disappears on voluntary movement and sleep.
- **Rigidity:**
  - Stiffness on passive limb movement is described as "lead pipe" rigidity because the increase in muscle tone is present throughout the range of movement. Unlike spasticity, it is not dependent on speed of movement.
  - When tremor is superimposed on the rigidity, a ratchet like jerkiness is felt, described as "cogwheel" rigidity.
- **Akinesia or bradykinesia**
  - Poverty/slowing of movement is the hallmark of parkinson's disease (PD). Slowness/difficulty of initiating voluntary movement and an associated reduction in automatic movements, such as swinging of the arms when walking.

  - There is fixity of facial expression (facial immobility—mask like face) with widened palpebral fissures and infrequent blinking.
  - Repetitive tapping (at about 2 Hz) over the glabella (glabellar tap) produces a sustained blink response (Myerson's sign), in contrast to the response of normal subject.
- **Postural changes:** A stooped posture is a characteristic feature.
- **Gait changes:** Slow shuffling, freezing and reduced arm swing, small stride length, slow turns, festinating gait (tendency to advance rapid short steps) and catching center of gravity. Feet may be glued to floor. Postural instability and freezing may result in fall forward.
- **Reduced eye blink.**

Table 6E.8: Nonmotor symptoms of Parkinson's disease.

Autonomic dysfunction • Orthostatic hypotension • Urinary incontinence • Constipation • Sexual problems	**Neuropsychiatric** • Anxiety • Depression • Apathy • Psychosis • Dementia	**Sensory problems** • Reduced sense of smell (hyposmia) • Pain
Sleep disorders • Restless legs • Insomnia • Daytime somnolence	**Rheumatological** • Frozen shoulder • Periarthritis • Swan neck deformity	**Other** • Seborrhea

Flowchart 6E.4: Classification of Parkinsonsian disorder.



(MPTP: manganese, 1-methyl 4-phenyl tetrahydropyridine; HIV: human immunodeficiency virus)

Table 6E.9: Hoehn and Yahr stage of Parkinson's disease.

Stage	*Disease state*
I	Unilateral involvement only, minimal or no functional impairment
II	Bilateral or midline involvement, without impairment of balance
III	First sign of impaired righting reflex, mild to moderate disability
IV	Fully developed, severely disabling disease; patient still able to walk and stand unassisted
V	Confinement to bed or wheelchair unless aided

Table 6E.10: Causes of secondary Parkinsonism.

Toxin: Manganese, 1-methyl 4-phenyl -1,2,3,6-tetrahydropyridine (MPTP), carbon monoxide, manganese, mercury, carbon disulfide, cyanide, methanol **Viral:** Encephalitis lethargica, Creutzfeldt-Jakob disease **Metabolic:** Wilson's disease	**Drugs:** Dopamine receptor blocking drugs, reserpine, tetrabenazine, alpha methyl dopa, lithium, flunarizine, cinnarizine **Vascular:** Multi-infarct, Binswangers disease **Trauma:** Pugilistic encephalopathy

Head injury: Punch drunk syndrome **Infectious:** Postencephalitic, human immunodeficiency virus (HIV), subacute sclerosing panencephalitis (SSPE), Prion diseases	**Others:** Parathyroid abnormalities, hypothyroidism, brain tumors, paraneoplastic, normal pressure hydrocephalus (NPH), psychogenic

Table 6E.11: Parkinson plus syndromes and its features.

Syndrome	*Features*
Progressive supranuclear palsy (PSP, Steele-Richardson-Olszewski syndrome)	Slow ocular saccades, eyelid apraxia, and restricted eye movements with particular impairment of downward gaze and reptilian stare **[Fig. 6D(iii).37]**. Frequently experience hyperextension of the neck with early gait disturbance and falls. MRI may reveal a characteristic atrophy of the midbrain with relative preservation of the pons (the 'hummingbird sign' on midsagittal images)
Multiple-system atrophy (MSA) • Parkinsonian (MSA-P) or striatonigral degeneration • Cerebellar (MSA-C) or olivopontocerebellar atrophy Autonomic (MSA-A) • form or Shy-Drager syndrome	Parkinsonism in conjunction with cerebellar signs and/or early and prominent autonomic dysfunction, usually orthostatic hypotension. Cerebellar and brainstem atrophy (the pontine 'hot cross buns' sign in MSA-c)
Corticobasal ganglionic degeneration (Rebeitz-Kolodny-Richardson syndrome)	Asymmetric dystonic contractions and clumsiness of one hand coupled with cortical sensory disturbances manifest as apraxia, agnosia, focal myoclonus, or alien limb phenomenon
Dementia with lewy bodies	Early onset dementia, visual hallucinations
Parkinsonism-dementia complex of Guam	Motor neuron disease plus Parkinson's
Guadeloupean parkinsonism	Levodopa-unresponsive parkinsonism, postural instability with early falls, and pseudobulbar palsy

Rheumatology

CHAPTER 7

A. CASE SHEET FORMAT

HISTORY TAKING

Name:

Age:

Sex:

Residence:

Occupation:

Chief Complaints

1. _____ × days
2. _____ × days
3. _____ × days

History of Presenting Illness

Joint pain:
- Duration:
- Onset:
- No. of joints involved:
- Symmetry:
- Progression:
- Variation:
- Aggravating factors:
- Relieving factors:

Morning stiffness:
- Duration of stiffness:
- Onset:
- Progression:
- Variation:
- Aggravating:
- Relieving factors:

Deformities:
- Duration:
- Onset:

Ulcers:
- Duration:
- Onset:
- Progression:

Fever:
- Episodic or continuous
- Grade

- Chill and rigors
- Aggravating factors
- Relieving factors
- Variation
  - Diurnal variation

History of:
- Petechiae
- Purpura
- Other bleeding manifestations
- Breathing difficulty
- Dyspnea on exertion
- Numbness and tingling of legs
- Skin lesions
- Endocrine abnormalities

Past history:
- Asthma
- Chronic obstructive airway disease
- Tuberculosis
- History of contact with tuberculosis
- Diabetes mellitus (DM)
- Hypertension (HTN)
- Ischemic heart disease (IHD)
- Seizure disorder

Family history:
(Draw pedigree chart representing three generations)

Personal history:
- Bowel habits
- Bladder habits
- Appetite
- Loss of weight
- Occupational exposure
- Sleep
- Dietary habits and taboo
- Food allergies
- Smoking (in smoking Index or Pack years)
- Alcohol history (__ grams of alcohol/day or ___ units of alcohol/week)

Menstrual and obstetric history:
- G__P__L__A__
- Age of menarche __
- Menopause at __
- Flow – Amenorrhea/Oligomenorrhea/Menorrhagia

Summarize:

Differential diagnosis:

1.
2.
3.

EXAMINATION

Rheumatological examination includes a thorough general examination and systemic examination along with examination of locomotor system.

General Examination

Patient

- Conscious
- Oriented
- Cooperative
- Obeying commands

Body Mass Index (BMI)

- Weight (in kg)/Height^2 (in meters)
- Grading according to WHO for Southeast Asian countries

Vitals

- **Pulse**
  - Rate
  - Rhythm
  - Volume
  - Character
  - Vessel wall thickening
  - Radio-radial delay and radio-femoral delay
  - Peripheral pulses
- **Blood pressure**
  - Right arm
  - Left arm
  - Both lower limbs
- **Respiration**
  - Rate
  - Abdominothoracic (male) or thoracoabdominal (female)
  - Usage of accessory muscles
- **Jugular venous pulse**
  - Waveform
- **Jugular venous pressure**
  - __ cm of blood above sternal angle (+ 5 cm water)
- **Temperature** ____ degree of Celsius or Fahrenheit measured at _____ site.

Physical Examination

- Pallor
- Icterus

- Cyanosis
- Clubbing
- Lymphadenopathy [systemic lupus erythematosus (SLE) and Still's disease)
- Edema

Other head to toe

- Skin
- Nails
- Oral cavity
- Mucous membrane
- Eyes

Locomotor System Examination

Rapid screening of the locomotor system can be done by **GALS screen** (**G**ait-**A**rms-**L**egs-**S**pine). With the patient undressed, observe the patient from front, back, and sides. Observe his gait, check his arms (inspect and palpate), check his legs (inspect and palpate), and check his spine (inspect and palpate).

Examination of the Individual Joints

[Regional Examination of Musculoskeletal System (REMS)]

We have 14 joint areas in the body on either side namely:

1. Proximal and distal interphalangeal joints
2. Metacarpophalangeal joints
3. Carpometacarpal joints of thumb
4. Wrist joint
5. Elbow joint
6. Shoulder joint
7. Acromioclavicular joint
8. Sternoclavicular joint
9. Temporomandibular joint
10. Hip joint
11. Knee joint
12. Ankle joint
13. Subtalar joint
14. Small joints of foot including midtarsal, metatarsophalangeal, and interphalangeal joints.

Each of the joints is examined under the following headings:

Inspection: Look for swelling, skin, and deformity

Palpation

- Look for tenderness and warmth
- Palpate for synovial thickening
- Look for **crepitus** (crepitus can also be auscultated) (Fine crepitus—synovitis or bursitis; Coarse crepitus—cartilage or bone damage)
- Look for range of movement of joint (both active and passive movements)

Example: *At knee joint there is swelling on inspection and on palpation synovial thickening present, warmth and tenderness present, crepitus felt. The range of movement is painful and restricted in both active and passive movement at the joint.* Also examine the **tendons, bursae, ligaments, synovium, and muscles** around the joint.

Examination of Spine

Look for the curvature of the spine. Normally there is cervical lordosis, thoracic kyphosis, lumbar lordosis, and sacral kyphosis. List if any deformities present.

Movements of the spine	
Cervical spine	Rotation Flexion Extension Lateral bending
Thoracolumbar spine	Flexion Extension Lateral bending Rotation Schober's test Straight leg raising test
Sacroiliac joint	Direct pressure Patrick's test Gaenslen's test

B. DIAGNOSIS FORMAT

Based on chronicity
Acute/chronic

Based on symmetry
Symmetrical/nonsymmetrical

Based on inflammation
Inflammatory/noninflammatory

Based on number of joints involved
Mono/oligo/polyarthritis

Associated features
- With/without deformities
- With/without axial spine involvement
- With systemic manifestations in the form (Pleural effusion, anemia, uveitis, etc.)

Disease severity
- DAS28
- Simplified and clinical disease activity indices (SDAI and CDAI)
- Rheumatoid arthritis severity scale (RASS)

EXAMPLES

Example 1

Chronic symmetrical inflammatory polyarthritis with swan neck deformity of fingers, with no axial spine involvement, with systemic features in the form of anemia and interstitial lung disease—I would like to consider diagnosis of **rheumatoid arthritis.**

CDAI score 7

Example 2

Chronic recurrent inflammatory monoarthritis involving right first MTP joint with deformities, without axial spine involvement or systemic manifestations—I would like to consider diagnosis of **gout.**

NOTES

C. DISCUSSION ON SYMPTOMMATOLOGY AND EXAMINATION

DISCUSSED IN THE FOLLOWING HEADINGS

1. Sympatomatology
2. Examination of skin, hands and eyes
3. Examination pattern of musckuloskeletal system
4. Examination of upper limbs
5. Examination of lower limbs
6. Examination of spine
7. Examination of other joints
8. Examination of other systems in rheumatological disorders
9. Discussion on common rheumatological diseases
10. Scoring systems

1. SYMPTOMATOLOGY

Arthralgia (subjective): Only pain around the joint

Arthritis (objective): Pain + other signs of inflammation (redness/swelling/increased temperature/loss of function)

Synovitis: Inflammation of synovial membrane

Tenosynovitis: Inflammation of the tendon sheath

Enthesitis: Inflammation of site of attachment of ligament, tendon or capsule to the periosteum or bone

Myositis: Inflammation of muscle

Arthritis–presentation	
Duration	Acute (presenting within hours to days) Chronic (persisting for weeks or longer)
Number of joints involved	Monoarticular (only 1 joint) Oligoarticular/pauciarticular (2–4 joints) Polyarticular (5 joints or more)
If more than one joint is involved	Symmetric (or) asymmetric Additive (or) migratory
Type	Inflammatory or noninflammatory (see below)
Deformities	Present (or) absent Deformities are usually seen in: • Rheumatoid arthritis • Psoriatic arthritis • Osteoarthritis • Reiter's disease • Chronic gout
Precipitating factors like	Sexually transmitted disease (STD) Infection Trauma Alcohol Diarrhea

Associated features	Constitutional symptoms: Fever, fatigue, and weight loss Extra-articular manifestations and systemic manifestations Comorbid conditions

Note: Treatment history should be taken in detail.

Inflammatory Versus Noninflammatory Disease

Features	Inflammatory (rheumatoid arthritis)	Noninflammatory (osteoarthritis)
Age of onset	Usually 20–40 years but may begin at any age	Most commonly over 50 years of age
Speed of onset	Rapid over weeks to months	Slow; over years
Systemic symptoms	Fatigue, low-grade fever, anorexia. Extra-articular manifestations: Rheumatoid nodules, Sjogren's syndrome, Felty syndrome	No systemic symptoms
Joint affection	Symmetrical	Asymmetrical
Joint symptoms	Painful, swollen, stiff joints, and muscle aches	Joints painful without swelling
Joints involved	Primarily affects small joints [metacarpophalangeal (MCP) and proximal interphalangeal (PIP)] with sparing of dip	Affects large weight bearing joints (hip, knee or the spine). Affects proximal interphalangeal (PIP) and distal interphalangeal (DIP) joints
Stiffness	Morning stiffness for >1 hour. Stiffness occurs after periods of rest/inactivity (the so-called "gel phenomenon")	Morning stiffness for <30 minutes. Stiffness is generally mild and occurs after periods of activity
Relation of movement with pain	Movement or mild to moderate activity decreases pain	Movement increases the pain (worsens with activity) and improves with rest
Examination of joint	Swollen, red, warm, tender, and painful	Swollen, cool, and hard on palpation. When severely inflamed (as in acute gout or septic arthritis), can have erythema of the overlying skin
Radiological findings	Bony erosions, soft-tissue swelling, angular deformities, periarticular osteopenia	Loss of joint space and damage to articular cartilage, osteophytes
Rheumatoid factor (RF) factor and antinuclear antibody (ANA)	Positive	Negative
Erythrocyte sedimentation rate (ESR) and C-reactive protein	Both are often raised	Usually normal but transient elevation of ESR may occur due to synovitis
White blood cell (WBC) count in the synovial fluid	WBC count is >2,000/mm^3 in septic arthritis and not in rheumatoid arthritis	WBC count is <2,000/mm^3

Causes of Arthritis

Acute monoarthritis	
Inflammatory	Crystal disease (e.g. gout), infectious disease, spondyloarthropathy, rheumatoid arthritis
Mechanical	Trauma, avascular necrosis
Acute polyarthritis	
Infectious	Bacterial, human immunodeficiency virus (HIV)
Noninfectious	Rheumatoid arthritis, spondyloarthropathy, other connective tissue diseases, crystal (gout), sarcoidosis, malignancy, leukemia, sickle cell anemia

Chronic monoarthritis	
Inflammatory	Crystal disease, infectious disease (e.g. tuberculosis, fungal), spondyloarthropathy, rheumatoid arthritis
Noninflammatory	Osteoarthritis, avascular necrosis, neuropathic arthropathy, villonodular synovitis
Chronic polyarthritis	
Inflammatory	Rheumatoid arthritis, spondyloarthropathy, other connective tissue diseases
Mechanical	Osteoarthritis
Crystal	Gout
Metabolic	Infiltrative, metabolic, hypothyroidism

2. EXAMINATION OF SKIN, HANDS, AND EYES

Skin changes in rheumatology	
Erythema	Septic arthritis Crystal arthropathy
Palpable purpura (Fig. 7C.1)	Vasculitis
Ulcers over skin (Fig. 7C.2)	Vasculitis
Rash	Systemic lupus erythematosus (SLE) [malar or discoid rash **(Fig. 7C.3)**] Vasculitis Drugs Stills disease
Violaceous scaly lesions	Psoriasis
Keratoderma blennorrhagica **Circinate balanitis**	Reiter's disease
Mucosal ulcers (Fig. 7C.4)	Behcet's disease SLE
Dryness of skin	Sjogren's disease
Thickened hard skin (Fig. 7C.5)	Systemic sclerosis Scleroderma
Pyoderma gangrenosum	Inflammatory bowel disease
Palmar erythema	Rheumatoid arthritis
Photosensitivity	Development of rash on exposure to sunlight of less than 30 minutes (SLE)
Digital gangrene	Raynaud's and medium vessel vasculitis
Alopecia	SLE Scleroderma
Heliotrope rash and Gottron's papules	Dermatomyositis
Salt and pepper appearance	Scleroderma (most prominently on the upper back and chest)
Livedo reticularis (Fig. 7C.6)	SLE Antiphospholipid antibody (APLA) syndrome Sneddon's syndrome, Polyarteritis nodosa
Raynaud's	Systemic sclerosis Vasculitis Mixed connective tissue disorder

Sclerodactyly	Progressive systemic sclerosis

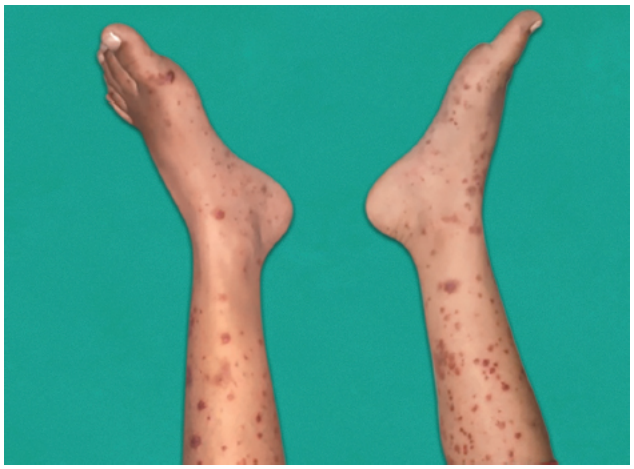


Fig. 7C.1: Palpable purpura over lower legs in Henoch–Schönlein purpura.

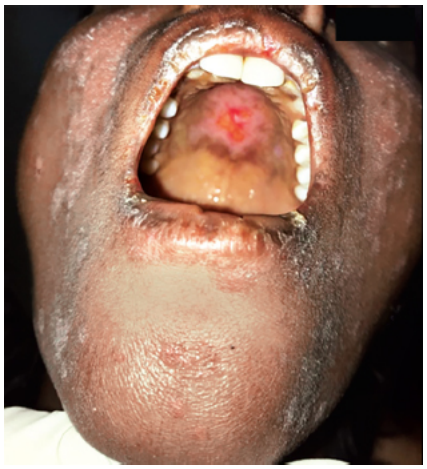


Fig. 7C.4: Mucosal ulcers in SLE.

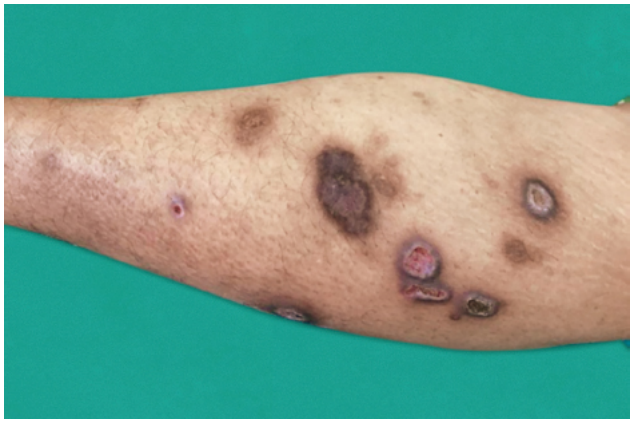
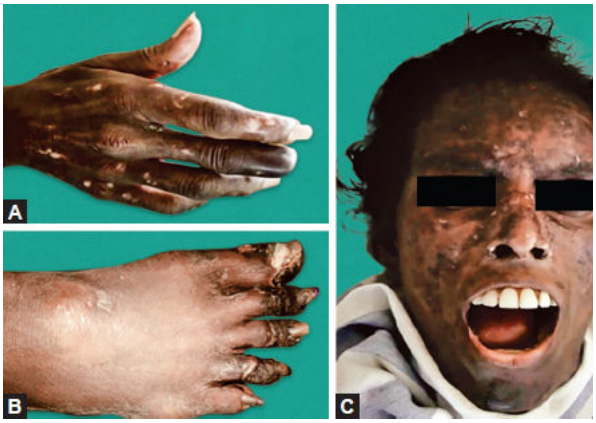


Fig. 7C.2: Ulcers on the leg in medium vessel vasculitis.



Figs. 7C.5A to C: Systemic sclerosis. (A and B) Shiny and thickened skin of hands and feet; (C) Mask-like face with decrea oral aperture.

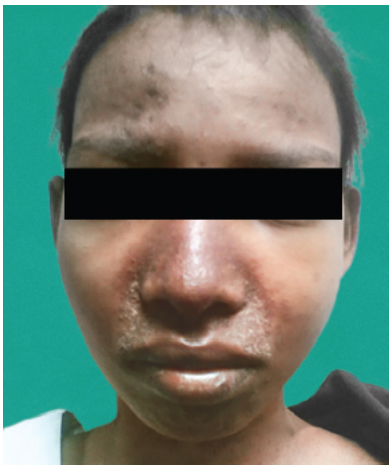


Fig. 7C.3: Systemic lupus erythematosus with malar rash and alopecia.

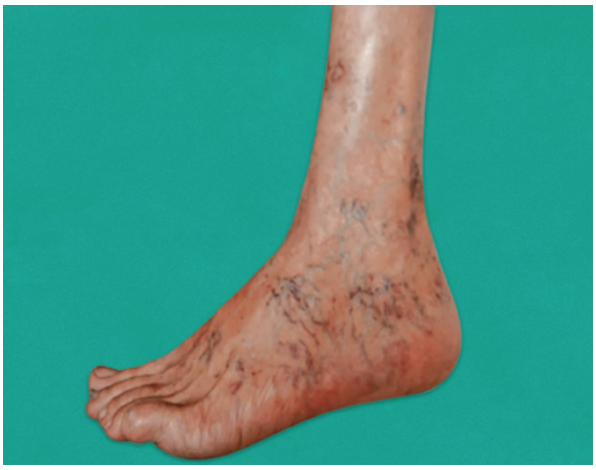


Fig. 7C.6: Livedo reticularis-mottled reticulated vascular patte that appears as a lace-like purplish discoloration of the skin. It is to swelling of the venules caused by obstruction of capillaries

Subcutaneous Nodules—differential Diagnosis

- Rheumatoid arthritis
- Rheumatic fever
- Gout
- Erythema nodosum*
- Sarcoidosis
- SLE
- Hyperlipidemia.

Erythema nodosum (Fig. 7C.7)

It is a type of panniculitis characterized by painful reddish nodules in the subcutaneous tissue most commonly seen on the shin.

Common causes include:

- Tuberculosis
- Leprosy
- Sulfonamides and other drugs
- Streptococcal infection
- Sarcoidosis
- Inflammatory bowel disease.

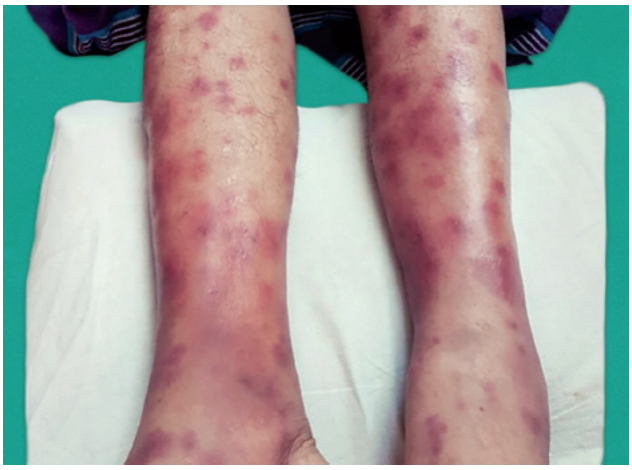


Fig. 7C.7: Erythema nodosum.

Nail Changes

Clubbing	Fibrosing alveolitis Hypertrophic Osteoarthropathy
Pitting and onycholysis (Fig. 7C.8)	Psoriasis*
Splinter hemorrhages	Vasculitis

Nail Changes in Psoriasis

Involvement is common and may be observed up to 50% of patients with psoriasis. These include:

a. "Thimble pitting" of the nail plate;
b. Distal separation of the nail plate from the nail bed (onycholysis);
c. Yellow-brown discoloration underneath the nail plate ("oil drop" sign);
d. Subungual hyperkeratosis; and
e. Thickening of the nail (onychodystrophy).

For diagnosis of nail involvement: >6 nails should be involved with each nail should have >20 pits.

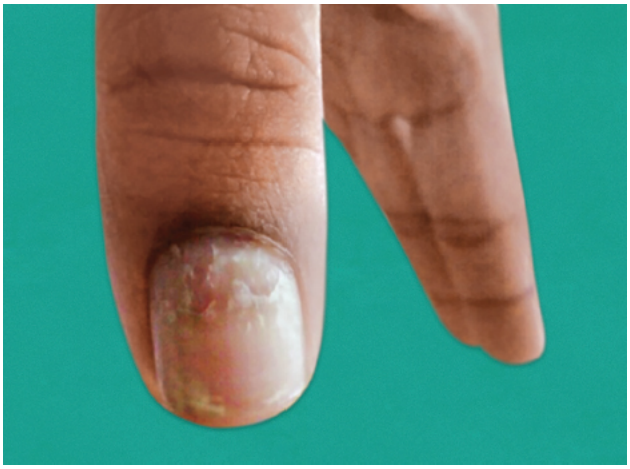


Fig. 7C.8: Nail changes in psoriasis.

Eye Changes

Dryness of eyes	Sjogren's syndrome
Episcleritis/scleritis (Fig. 7C.9)	Rheumatoid arthritis
Iritis/iridocyclitis	Ankylosing spondylitis
Conjunctivitis	Reiter's disease
Tenosynovitis of superior oblique	Rheumatoid arthritis (Brown's syndrome)
Scleromalacia perforans	Rheumatoid arthritis

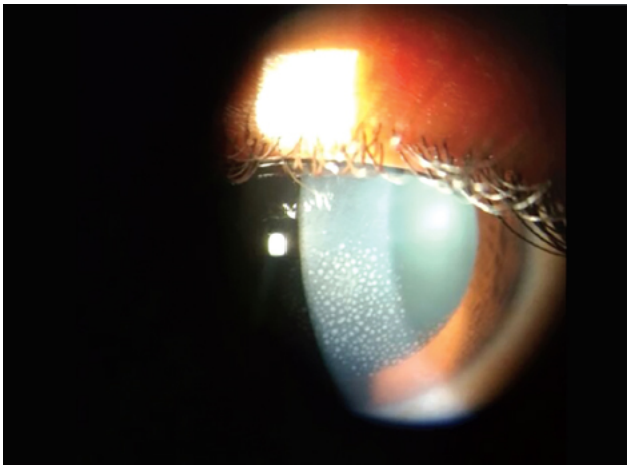


Fig. 7C.9: Slit-lamp examination showing keratitis.

3. EXAMINATION PATTERN OF MUSCULOSKELETAL SYSTEM

Gait, arms, legs, spine (gals) screening	
Gait	Observe the gait
Arms	Examine the range of movement of joints. Joint deformities. Synovial thickening.
Legs	Examine the range of movement of joints. Joint deformities. Synovial thickening. Special tests.
Spine	Look for spine deformity. Special test.

Regional examination of musculoskeletal system (REMS) examination (Look, feel, move)	
Look for	• Swellings • Redness • Rashes • Scars • Muscle wasting
Feel for	• Temperature • Swelling • Tenderness

Move	• Full range of movement—active and passive (refer the table and figure) **(Figs. 7C.10A to H)** • Restriction—mild/moderate/severe
Function	• Functional assessment of joint
All the joints have to be examined in the above headings.	

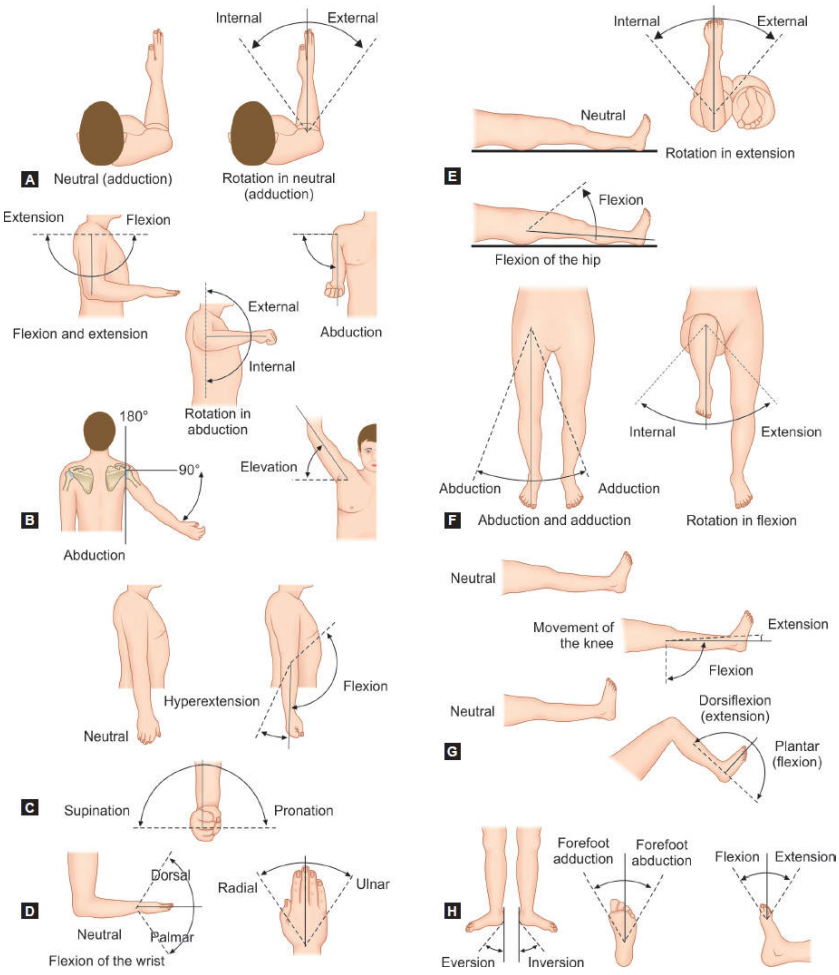
Range of movement of joints (Figs. 7C.10A to H):

	Flexion	Extension	Abduction	Adduction	Rotation
Wrist	70°	70°	30°	30°	
MCP	45°	90°			
PIP	120°				
DIP	90°	10°			
Elbow	160°	5°			
Shoulder	160°	60°	175°	50°	70°
Hip	110°	30°	30°	30°	45°
Knee	130°				
Ankle	40° (dorsiflexion)	50° (plantar flexion)			

Others:

Subtalar joint—has 5° of inversion and eversion.

Midtarsal joint—has 30° of inversion and eversion.



Figs. 7C.10A to H: Demonstration of range of movement of joints.

4. EXAMINATION OF UPPER LIMBS

Examination of shoulder

Examination of glenohumeral joint (Fig. 7C.11):

- Examine for tenderness and swelling along the joint line as shown in the **Figure 7C.11**.

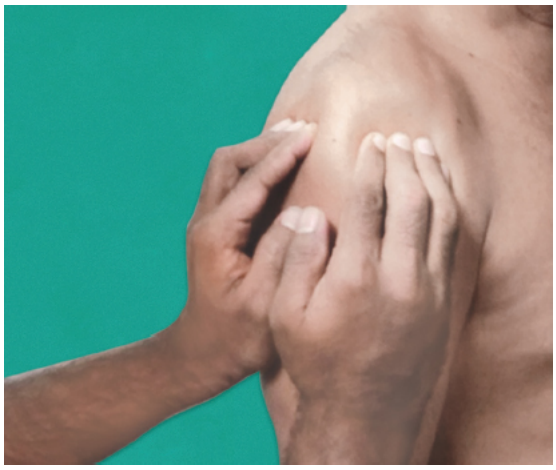


Fig. 7C.11: Image showing examination of tenderness and swelling along the joint line of shoulder joint.

Impingement test (Fig. 7C.12):

Fig. 7C.12: Demonstration of impingement test.

Apprehension test (Fig. 7C.13):

- Flex the patients elbow to 90°
- Abduct the patients shoulder to 90°
- Now attempt external rotation of the shoulder
- Apprehension to the test is considered positive suggesting glenohumeral instability with possibility of labral tear.

Fig. 7C.13: Demonstration of apprehension test.

Examination of Elbow (Fig. 7C.14)

- Palpate the joint for tenderness and synovial thickening along the joint line as shown in the **Figure 7C.14**.

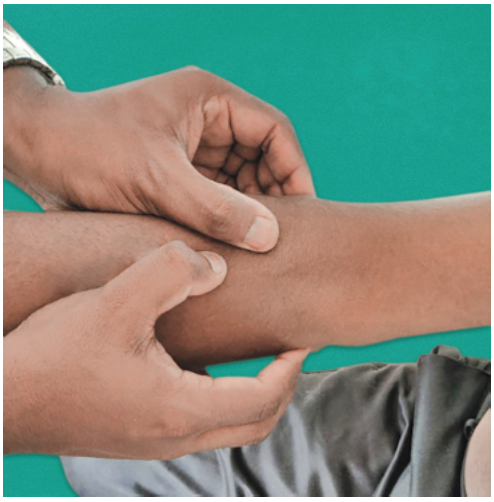


Fig. 7C.14: Palpation of elbow.

Examination of wrist joint

(Two-thumb technique) (Fig. 7C.15)

- The examiner's thumb should follow the third metacarpal bone on the dorsal aspect of the hand until a dimple is reached at the capitate level.
- Continuous pressure is exerted by the thumb.
- The other thumb is used to intermittently apply pressure approximately half an inch away on the wrist joint in order to identify swelling and/or tenderness.

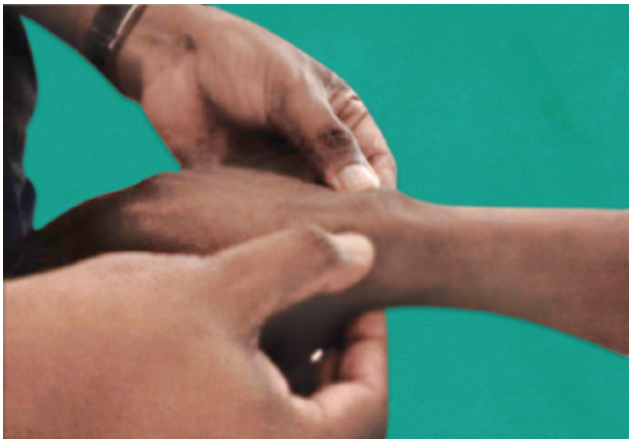


Fig. 7C.15: Examination of wrist joint.

Prayer sign (Fig. 7C.16):

- The patient is asked to dorsiflex both the wrist and hold the palms together actively as in praying
- Pain or inability to perform this activity would suggest joint involvement or carpal tunnel syndrome
- Also seen with diabetic cheiroarthropathy.

Fig. 7C.16: Demonstration of prayer sign.

Metacarpophalangeal Joint Assessment (Figs. 7C.17A to C)

a. **Scissor technique:** A scissor-like shape is made with the fingers. The patient's hand is held from the sides at the MCP level.
b. The MCPs are flexed to 90°. The thumbs are used to palpate the joint—one to apply pressure to the joint, the other to assess for effusion, swelling, and/or tenderness.
c. **Squeeze test:** Squeeze the metacarpophalangeal joints as shown in the **Figure 7C.17C** and watch for tenderness.

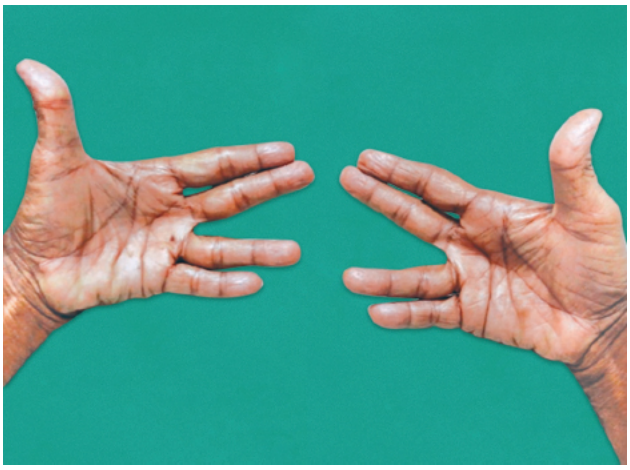


Fig. 7C.17A: Examination of metacarpophalangeal joint.

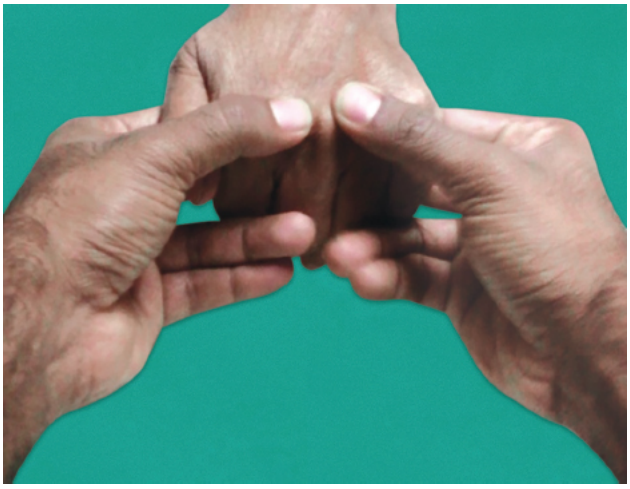


Fig. 7C.17B: Examination of metacarpophalangeal joint.

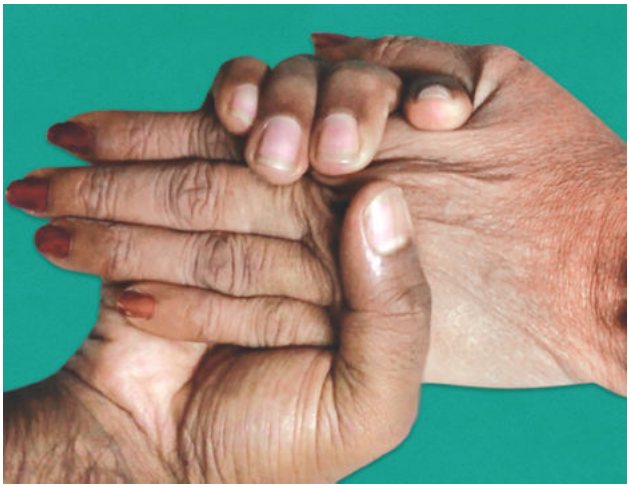


Fig. 7C.17C: Squeeze test of hand for assessment of metacarpophalangeal joint.

Interphalangeal Joint Assessment (Fig. 7C.18)

(Four-finger technique)

Each interphalangeal joint is held by the thumb and index finger of one hand of the examiner. Pressure is applied until the distal finger becomes whitened due to low blood supply. The thumb and index finger of the examiner's other hand are used palpate the joint to identify effusion, swelling, and/or tenderness.

Fig. 7C.18: Examination of interphalangeal joints (four finger technique).

Deformities of hand	
Spindling of the fingers	It is the earliest finding characterized by swelling of the proximal, but not the distal interphalangeal joints.
Swan-neck deformity (Figs. 7C.19 and 7C.20)	It is due to hyperextension of the proximal interphalangeal joints (PIP) with flexion of the distal interphalangeal joints (DIP). At DIP joint, there is elongation or rupture of attachment of the extensor tendon to the base of the distal phalanx; this results in mallet deformity of distal joint and in addition, an extensor tendon imbalance, leading to hyperextension deformity at PIP joint.
Boutonniere' or "button-hole" deformity (Figs. 7C.19 and 7C.21)	This deformity is due to flexion of the PIP joints and extension of the DIP joints. Disruption of the central slip of the extensor tendon and the triangular ligament allows each of the conjoint lateral bands of the digit to slide volarly resulting in a pathologic flexion force and an extension lag; all tendons traversing the PIP joint in this setting elicit flexion of the joint.
Ulnar deviation (Fig. 7C.22)	It results from subluxation of the metacarpophalangeal (MCP) joints, with subluxation of the proximal phalanx to the volar side of the hand.
Hitchhiker's thumb (Fig. 7C.23)	A condition where the thumb can bend backwards to an angle of almost 45°. Thumb flexes at the metacarpophalangeal joint and hyperextends at the interphalangeal joint.
"Z" deformity (Fig. 7C.24)	It is due to radial deviation of the wrist, ulnar deviation of the digits with palmar subluxation of the first MCP joint with hyperextension of the first interphalangeal (IP) joint.
Carpal tunnel syndrome	Due to synovial proliferation in and around the wrists producing compression of the median nerve.
Bow string sign	Prominence of the tendons in the extensor compartment of the hand.
Heberden's nodes (Fig. 7C.25)	DIP swelling in osteoarthritis.
Bouchard's node (Fig. 7C.25)	PIP swelling in osteoarthritis.
Sausage digits (Fig. 7C.26)	Dactylitis involving both PIP and DIP as seen in psoriatic arthritis.

Pencil in cup deformity	Psoriatic arthritis.
Arthritis mutilans	Psoriatic arthritis.

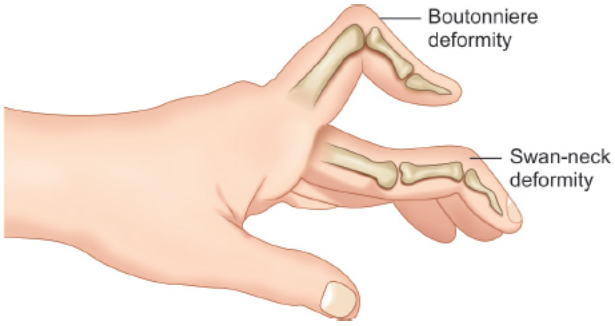


Fig. 7C.19: Boutonniere and swan—neck deformity.

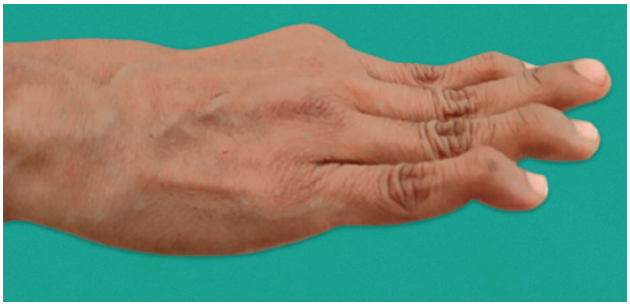


Fig. 7C.20: Swan-neck deformity.

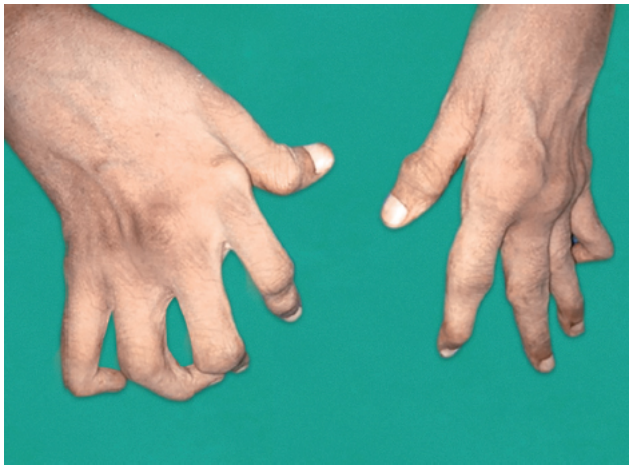


Fig. 7C.21: Boutonniere deformity.

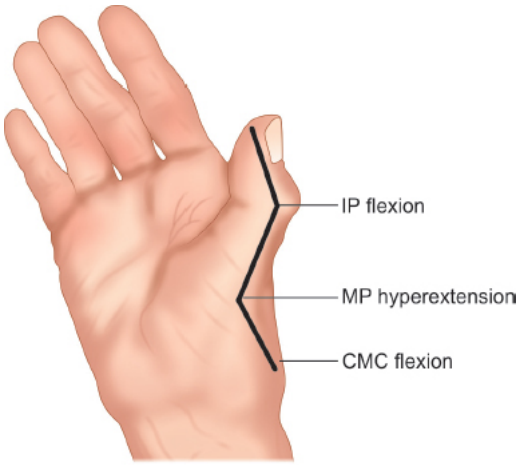


Fig. 7C.24: Z-shaped deformity of thumb in RA.

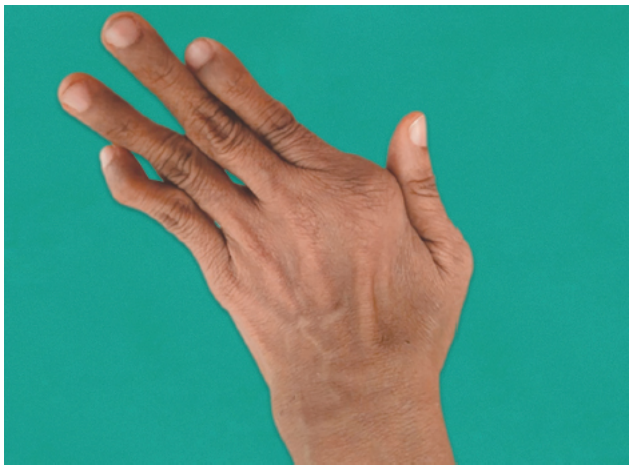


Fig. 7C.22: Ulnar deviation of hand.

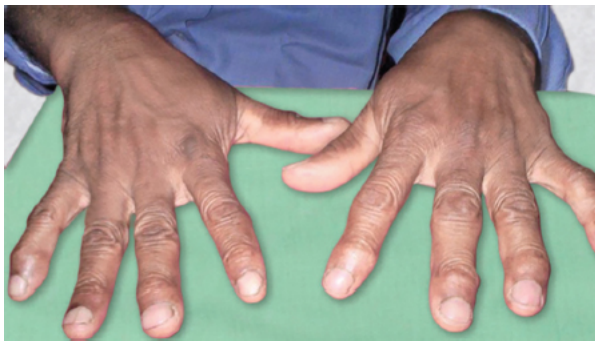


Fig. 7C.25: Osteoarthritis showing Heberden's nodes (on DIP) a Bouchard's nodes (on PIP).

Fig. 7C.23: Hitchhiker's thumb.

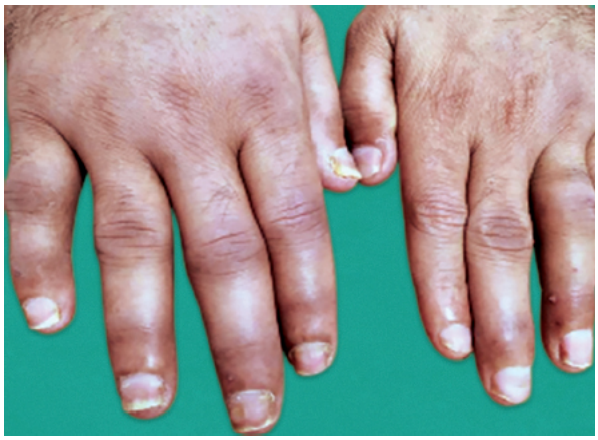


Fig. 7C.26: Sausage digits in psoriatic arthritis and psoriatic na

5. EXAMINATION OF LOWER LIMB

Examination Hip Joint

Trendelenburg test (Fig. 7C.27)

- Assesses the proximal hip muscles strength.
- This involves patient alternately standing on each leg alone.
- In a negative test, the pelvis remains level.
- In an abnormal test, the pelvis will dip to the contralateral side suggesting gluteus medius weakness.
- This test is abnormal, if the hip is involved either due to arthritis or avascular necrosis. Also proximal muscle weakness can be secondary to drugs used like steroids.

Thomas test (Fig. 7C.28)

- To look for fixed flexion deformity of hip.
- Keep one hand under the patient's back to ensure that there is no lumbar lordosis.
- Fully flex one hip.

- If the opposite leg lifts off the couch, there is a fixed flexion deformity (normally as the pelvis tilts, the hip would extend allowing the leg to remain on the couch).

Examination of Knee Joint

- Palpation of knee joint to look for tenderness and synovial thickening **(Fig. 7C.29)**.

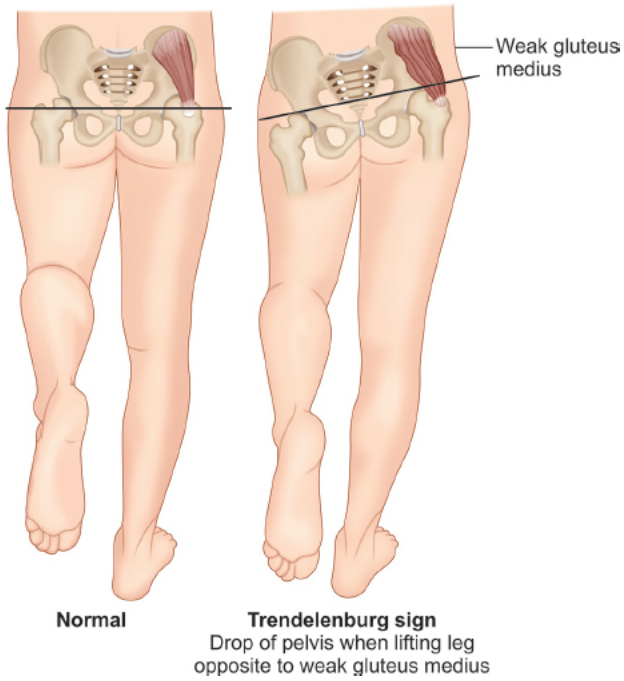


Fig. 7C.27: Trendelenburg sign.

Fig. 7C.28: Demonstration of Thomas test.

Fig. 7C.29: Demonstration of palpation of knee joint.

Patellar Tap Test

- Used to detect effusion in the knee joint.
- Slide your hand down the patient's thigh compressing the suprapatellar pouch **(Fig. 7C.30)**.
- This forces all the fluid to collect behind the patella.
- With two fingers of the other hand push the patella down gently **(Fig. 7C.31)**.
- In a positive test, the patella will bounce back with the tap.

Bulge Sign/Cross Fluctuation Sign (Figs. 7C.32A and B)

- Stroke the medial side of the knee upwards towards the suprapatellar pouch.
- This empties the medial compartment of the fluid.
- Now stroke the lateral side downwards.
- The medial side will now refill and bulge indicating joint effusion.

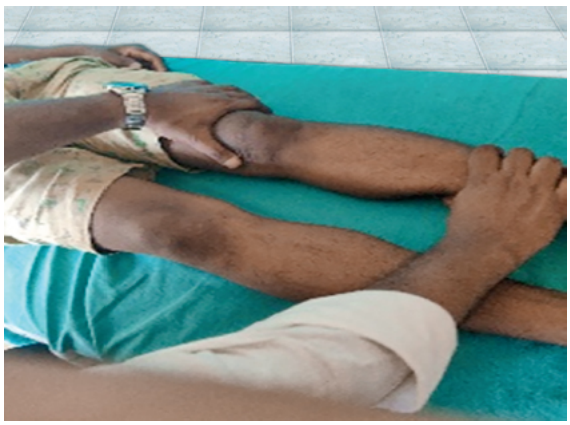


Fig. 7C.30: Slide your hand down the patient's thigh compressing the suprapatellar pouch.

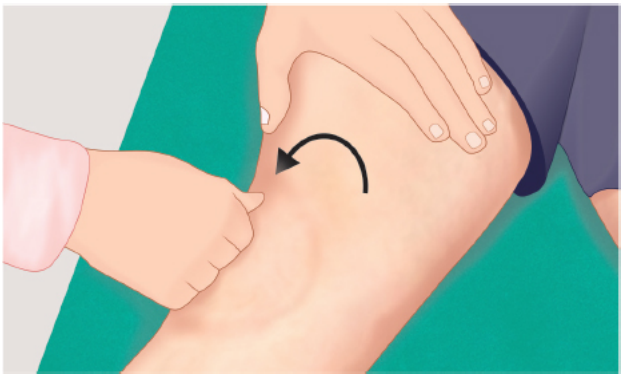


Fig. 7C.32B: The cross fluctuation sign (bulge sign): Stroke the lateral side downwards.

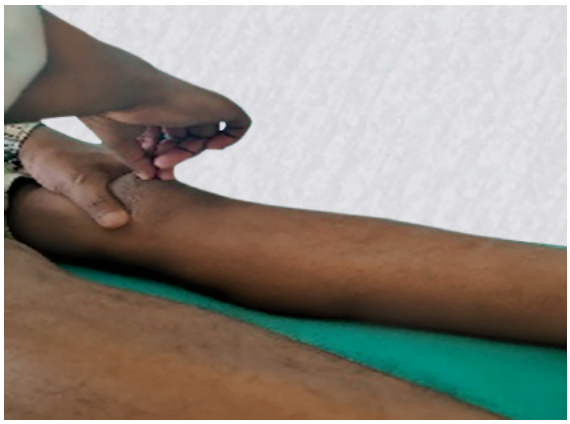


Fig. 7C.31: With two fingers of the other hand push the patella down gently.

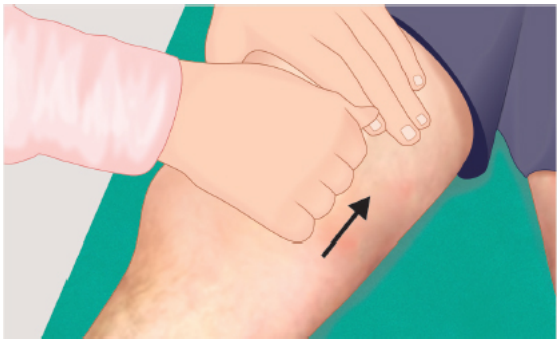


Fig. 7C.32A: The cross fluctuation sign (bulge sign): Stroke the medial side of the knee upwards towards the suprapatellar pouch.

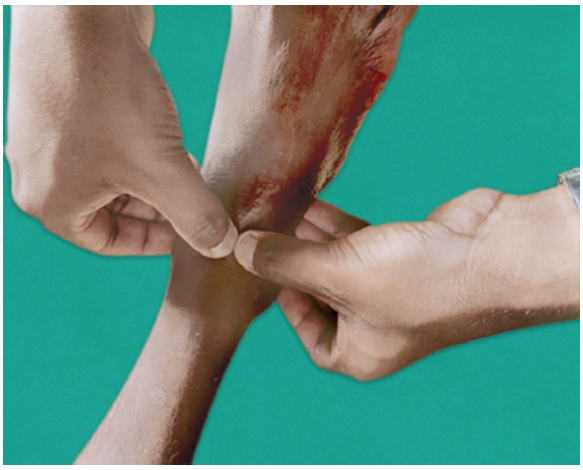


Fig. 7C.33: Examination of ankle joint.

Examination of Ankle Joint

- Palpate the bare area of the ankle [bare area is the triangular area in front of the ankle, between the two tendons of extensor hallucis longus (EHL) and extensor digitorum longus (EDL)] for tenderness and synovial thickening **(Fig. 7C.33)**.

Examination of Achilles Tendon for Swelling

- Palpate the Achilles tendon for swelling and tenderness **(Fig. 7C.34)**. Enthesitis is classically seen in case of seronegative spondyloarthropathies.

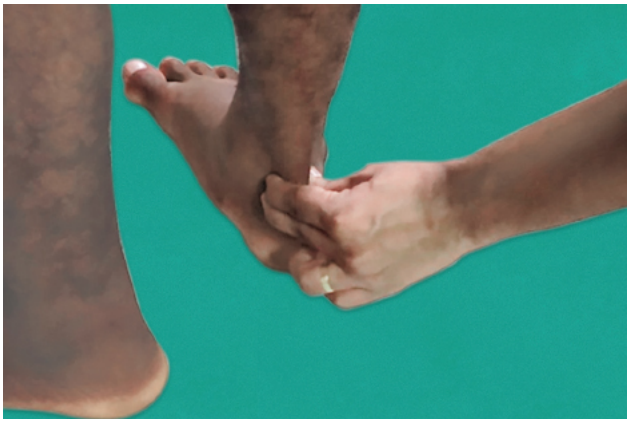


Fig. 7C.34: Examination of swelling over Achilles tendon.

Examination of Metatarsophalangeal Joints

- Squeezing the metatarsophalangeal joints to look for pain **(Fig. 7C.35)**

Fig. 7C.35: Examination of metatarsophalangeal joints.

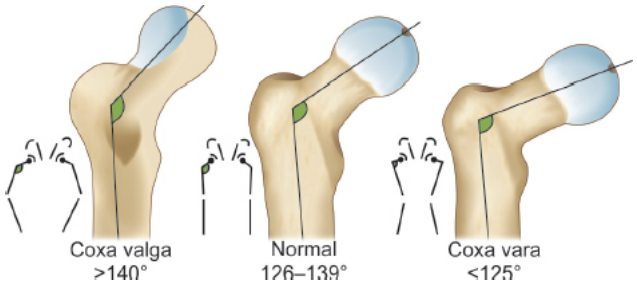
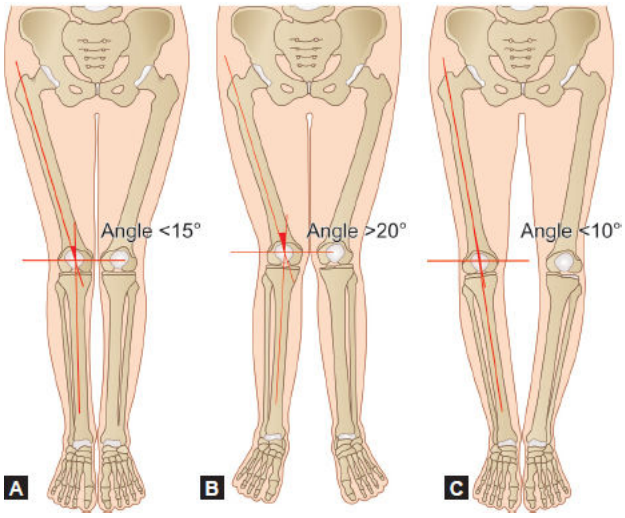


Fig. 7C.36: Hip joint deformities.



Figs. 7C.37A to C: Knee joint deformities. (A) Normal; (B) Genu vaigus (kknock knees); (C) Genu varus (bow legs).

Deformities of leg:

Hip joint (Fig. 7C.36)	Coxa vara/valgum
Knee joint (Figs. 7C.37A to C)	Genu varum (bow legs)/genu valgum (knock knee)
Foot (Fig. 7C.38)	Hallux varus/hallux valgus/hammer toes
Metatarsophalangeal (Fig. 7C.39)	Gout/podagra

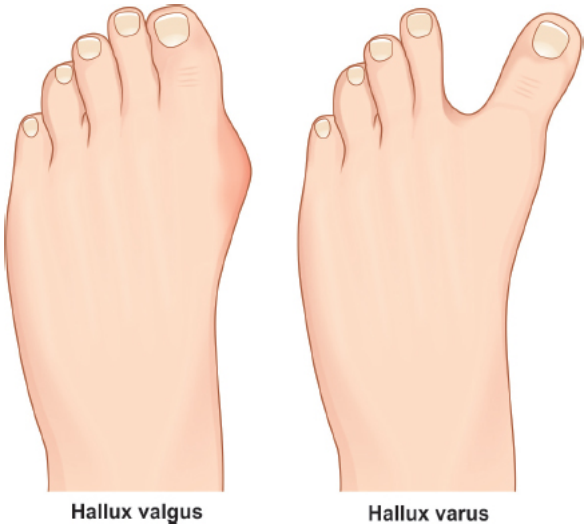


Fig. 7C.38: Hallux valgus and hallux varus deformity.

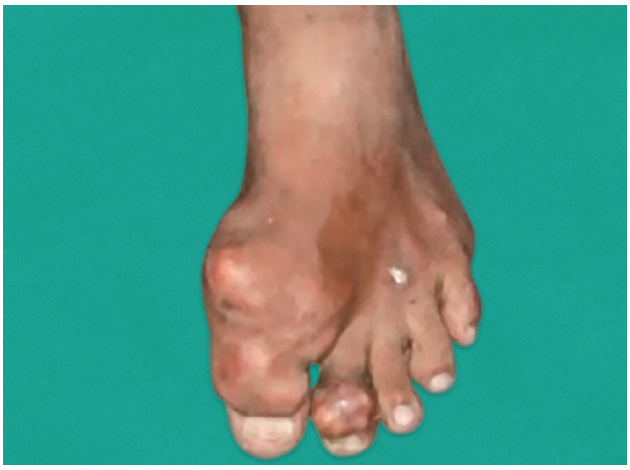


Fig. 7C.39: Acute gouty arthritis involving the first metatarsophalangeal (MTP) joint (termed podagra).

6. EXAMINATION OF SPINE

Occiput to wall Distance/Flesche test (Fig. 7C.40)

- Ask the patient to stand erect against a wall, with heels and buttocks placed against a wall.
- Now, ask the patient to extend the neck maximally.
- The distance between the occiput and the wall is measured in degree of flexion deformity of cervical spine.
- Normally the occiput to wall distance is zero.
- It is increased in cervical flexion deformity as in ankylosing spondylitis.

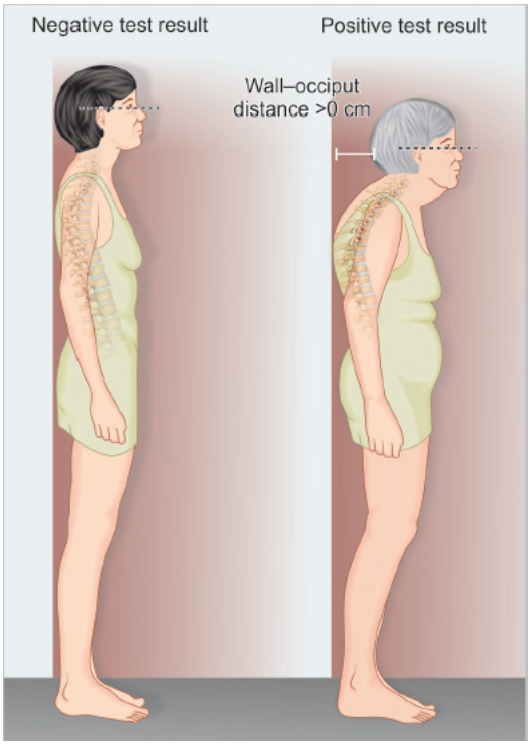


Fig. 7C.40: Demonstration of Flesche test.

Schober's Test (Fig. 7C.41)

- Mark a point approximately at L5 (A)
- Now mark two horizontal lines, one 10 cm above (B) and one 5 cm below L5 (C)
- Ask the patient to touch his/her toes
- Normally the distance between two lines increases by 5 cm (total >20 cm)
- If the increase is less than 5 cm, it suggests restriction.

Modified Schober's Test (Fig. 7C.42)

- Mark a line connecting two posterior superior iliac spine.
- Draw a parallel line 10 cm above this line.
- Now ask the patient to bend and touch his toes as much as possible.
- The distance between the two lines must be >15 cm. If it is less than 15 cm, it indicates restricted movement of the lumbar spine as seen **in ankylosing spondylosis.**

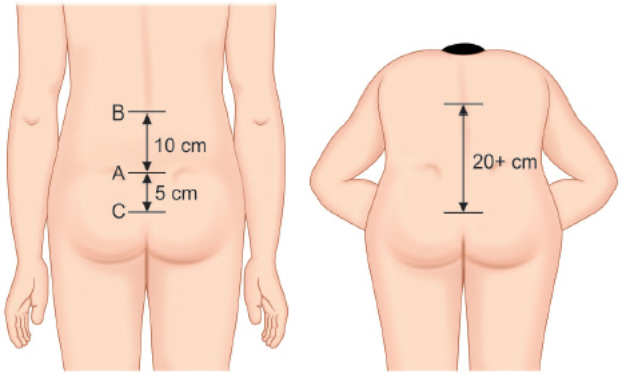


Fig. 7C.41: Demonstration of Schober's test.

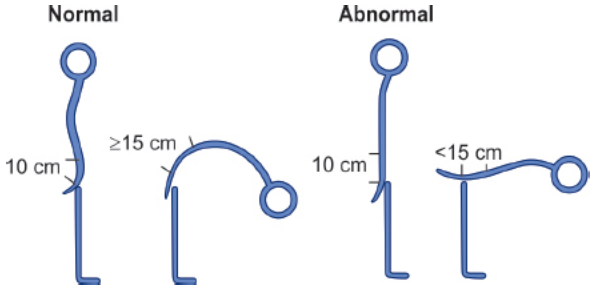


Fig. 7C.42: Demonstration of modified Schober's test.

Straight Leg Raising Test (Fig. 7C.43)

- Patient lying in supine position, the heel of the leg (with knee extended) is cupped by examiner and elevated slowly.
- The test is considered positive if sciatic pain is reproduced between 35° and 70° of elevation.
- The straight leg raise (SLR) test is best for eliciting L4, L5, or S1 radiculopathy.

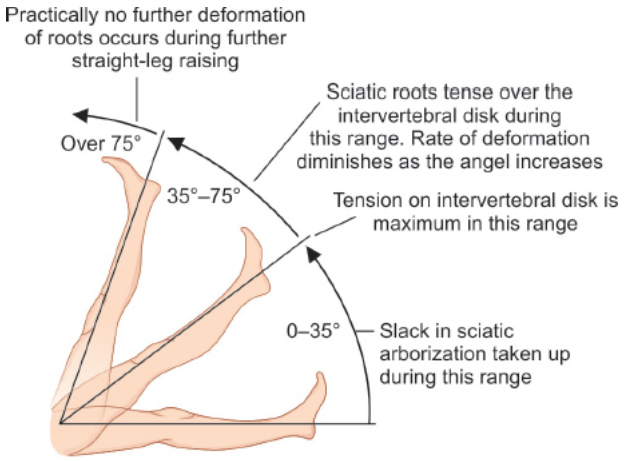


Fig. 7C.43: Straight leg raising test.

Patrick's Test (Figure-of-4 test) (Fig. 7C.44)

- One leg is guided into "figure-of-4" position with the ipsilateral ankle resting across the contralateral thigh.
- The ipsilateral knee is then pressed downwards with one hand while providing counter pressure with the other hand on the contralateral anterior superior iliac spine.
- Pain indicates **sacroiliac joint involvement.**

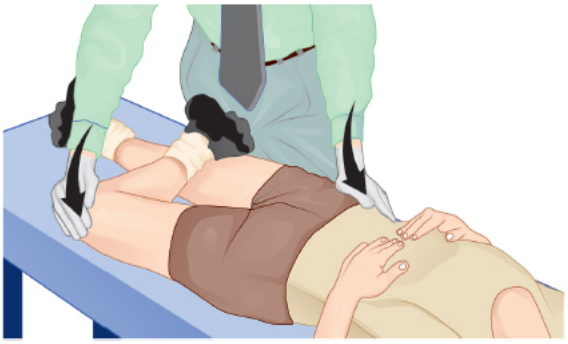


Fig. 7C.44: Demonstration of Patrick's test (figure-of-4).

Gaenslen Maneuver (Fig. 7C.45)

- Ask the patient to lie down on supine.
- One hip if flexed maximally and the other hip is extended by allowing the leg to dangle off the side of the examining table as shown in the **Figure 7C.45**.
- Pain **indicates sacroiliac joint involvement.**

Fig. 7C.45: Demonstration of Gaenslen test.

Deformities of spine (Fig. 7C.46)	
Lordosis	Anterior curvature
Kyphosis	Posterior curvature
Scoliosis	Lateral curvature
Knuckle deformity or step deformity	Prominence of one spinous process
Gibbus deformity (e.g. Pott's spine/metastasis)	Prominence of two spinous processes

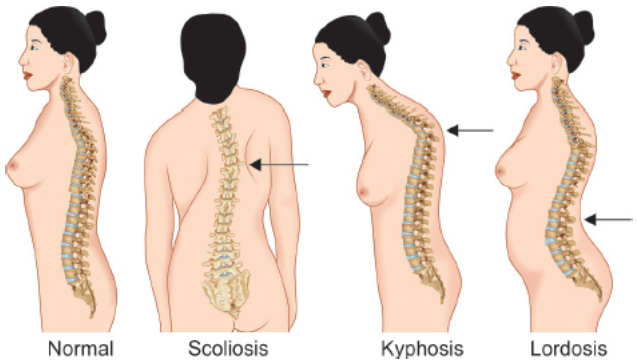


Fig. 7C.46: Various spine deformities.

7. EXAMINATION OF OTHER JOINTS

Temporomandibular Joints (Fig. 7C.47)

- Palpate the temporomandibular joint by asking the patient to open the mouth.
- Observe for tenderness, synovial thickening, and crepitus.

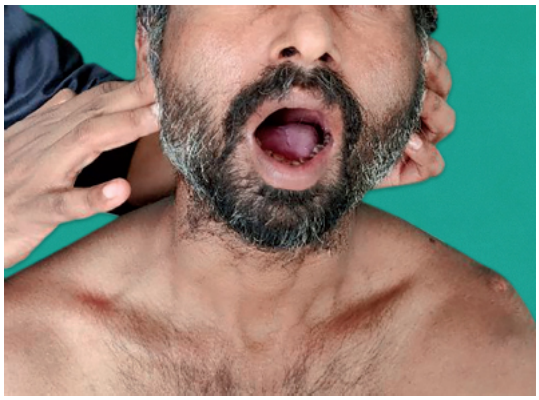


Fig. 7C.47: Examination of temporomandibular joint (TMJ).

Examination of Sternoclavicular Joint (Fig. 7C.48)

- Palpate the sternoclavicular joint.
- Look for tenderness and synovial thickening.

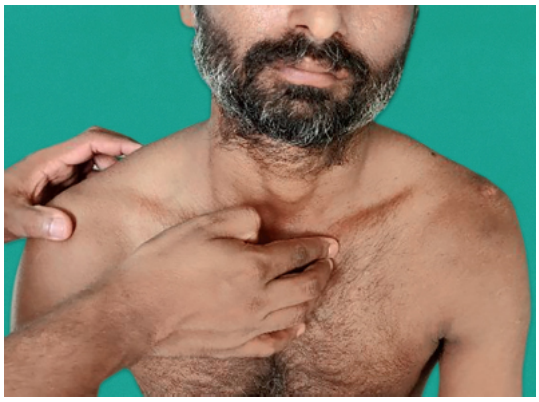


Fig. 7C.48: Examination of sternoclavicular joint.

8. EXAMINATION OF OTHER SYSTEMS IN RHEUMATOLOGICAL DISORDERS

Cardiovascular system	
Pericarditis	RA SLE
Endocarditis	SLE
Aortitis and aortic regurgitation	RA Psoriasis Ankylosing spondylitis Reiter's
Conduction defects	SLE
Nervous system	
Myelopathy	RA—atlantoaxial dislocation Vasculitis

Neuropathy (entrapment/mononeuritis multiplex)	RA SLE Vasculitis (especially PAN)
Stroke	RA SLE APLA Vasculitis
Myopathy	Polymyositis Dermatomyositis
Respiratory system	
Upper respiratory tract	Wegener's granulomatosis
Pleural effusion	RA SLE
Fibrosis	RA SLE Systemic sclerosis
Lung nodules	RA (Caplan's syndrome)
Alveolar hemorrhage	Microscopic polyangiitis Goodpasture's syndrome Wegener's granulomatosis
Asthma	Churg–Strauss syndrome
Decreased chest expansion	Ankylosing spondylosis
Gastrointestinal system	
Oral ulcers	SLE Behcet's disease
IBD	Seronegative spondyloarthropathies
Hepatosplenomegaly	SLE RA Stills disease
GI bleeding	Henoch–Schönlein purpura Other vasculitis Analgesic use
Genitourinary system	
Urethritis	Reactive arthritis
Glomerulonephritis	SLE Microscopic polyangiitis Goodpasture's syndrome Wegener's granulomatosis
Renal failure	Analgesics use Vasculitis
Endocrinology	
Diabetes	Steroid induced
Thyroid disease	Associated autoimmune conditions
Blood	
Anemia	SLE

Thrombocytopenia Pancytopenia	RA (Felty's syndrome)

9. DISCUSSION ON COMMON RHEUMATOLOGICAL DISEASES

Rheumatoid Arthritis

American College of Rheumatology (ACR) criteria for rheumatoid arthritis.	
Morning stiffness Arthritis of 3 joint areas Arthritis of the hands Symmetric arthritis Rheumatoid nodules Serum rheumatoid factor positive Radiographic changes These criteria ***must be*** present for more than 6 weeks. Presence of ***four or more criteria*** favors definite diagnosis of RA.	
European League against Rheumatism (EULAR) classification criteria for rheumatoid arthritis: 2010.	
A. Joint involvement (Fig. 7C.49)	
1 large joint (shoulder, elbow, hip, knee, ankle)	0
2–10 large joints	1
1–3 small joints (MCP, PIP, thumb IP, MTP, wrists) + involvement of large joints	2
4–10 small joints + involvement of large joints	3
>10 joints (at least 1 small joint)	5
B. Serology (at least one test result is needed for classification)	
Negative RF and negative ACPA	0
Low-positive RF or low-positive ACPA (≤3 times ULN)	2
High-positive RF or high-positive ACPA (≥3 times ULN)	3
C. Acute-phase reactants (at least one test result is needed for classification)	
Normal CRP and normal ESR	0
Abnormal CRP or abnormal ESR	1
D. Duration of symptoms	
<6 weeks	0
≥6 weeks	1
Above criteria yields a score of 0–10. A score of **≥6 required for definitive diagnosis of RA.** *A score of <6/10 are not classifiable as RA, but their status to be reassessed over time.*	

(ACPA: Anticitrullinated protein antibodies; CRP: C-reactive protein; ESR: erythrocyte sedimentation rate; IP: interphalangeal joint; MCP: Metacarpophalangeal joint; MTP: metatarsophalangeal joint; PIP: proximal interphalangeal joint; RF: rheumatoid factor; ULN: upper limit of normal)

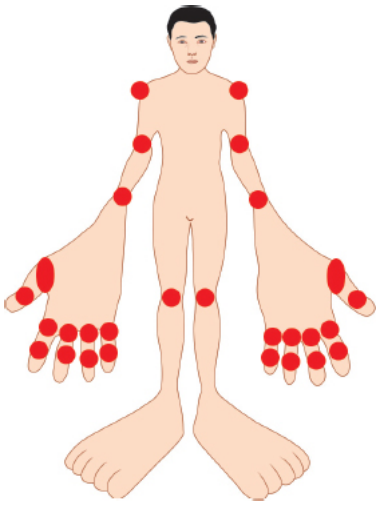


Fig. 7C.49: The 28 joints to be examined in rheumatoid arthritis include the 5 proximal interphalangeal joints of the 2 hands, the 5 metacarpophalangeal joints of the 2 hands, the 2 wrists, the 2 elbows, the 2 shoulders, and the 2 knees.

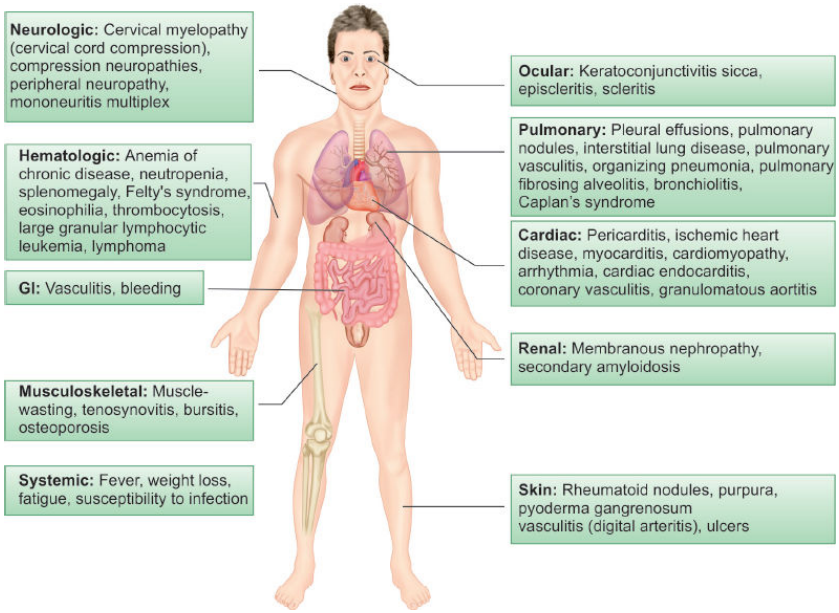


Fig. 7C.50: Extra-articular manifestations of rheumatoid arthritis.

Systemic Lupus Erythomatosis (Fig. 7C.51)

Systemic Lupus International Collaborating Clinics (SLICC) Classification 2012 criteria	
Biopsy proven LUPUS NEPHRITIS and ANA/anti-DNA (or) atleast four criteria (one needs to be immunological)	
Clinical	***Immunological***
Acute cutaneous LE	ANA
Chronic cutaneous LE	Anti-dsDNA
Oral ulcer	Anti-Sm
Alopecia	aPL antibodies
Synovitis	Low complement
Serositis	Direct Coombs' test positive

Renal Neurologic Hemolytic anemia Leukopenia/lymphopenia Thrombocytopenia	

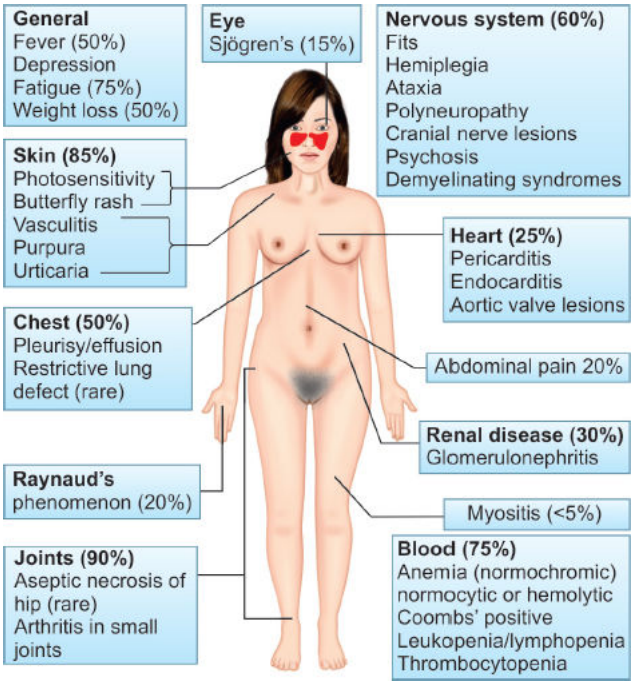


Fig. 7C.51: Clinical features of systemic lupus erythematosus (SLE).

Differences between rheumatoid arthritis and SLE		
Features	***Rheumatoid arthritis***	***Systemic lupus erythematosus***
Smoking	Predisposing factor	No relation
Female:Male	3:1	9:1
Type of arthritis	Erosive	Nonerosive
Deformities	Common	Rare, Jaccoud's arthropathy (10%)
Systemic involvement	Relatively less	Marked
Nodules	Rheumatoid nodules	Absent
Malar (skin) rash	Nil	Striking feature: Malar rash, discoid rash
Photosensitivity	Absent	Photosensitivity present
Oral ulcer and alopecia	Absent	Present
Spine involvement	Involves cervical spine	Rare
Pyoderma gangrenosum	May develop	Rare
Renal involvement	Uncommon	Common and severe
Platelet abnormality	Thrombocythemia	Thrombocytopenia
Serology	RA factor and ACPA	ANA and anti-dsDNA

Criteria for diagnosis	ACR/EULAR	SLICC/ACR
Response to DMARDs	Present	Less response

(ACPA: anticyclic citrullinated peptide antibodies; ACR: American College of Rheumatology; ANA: antinuclear antibodies; DMARD: disease-modifying antirheumatic drugs; dsDNA: double-stranded deoxyribonucleic acid; EULAR: European league against rheumatism; RA: rheumatoid arthritis; SLICC: systemic lupus international collaborating clinics)

Osteoarthritis (Fig. 7C.52)

Osteoarthritis (OA) is a **noninflammatory**, **slowly progressive joint disease**, mainly **involving the cartilage**. It shows **progressive destruction of articular cartilage** of weight-bearing joints of **genetically susceptible older persons**. It leads to narrowing of joint space, subchondral bone thickening, and finally **painful and nonfunctioning joints**.

Fibromyalgia

Fibromyalgia syndrome (FMS) is characterized by chronic widespread pain, and is defined as pain for more than three months both above and below the waist.

Diagnostic Criteria for FMS

- At least 3 months of widespread pain that is bilateral, above and below the waist.
- It includes axial skeletal pain and pain to palpation at a minimum of 11 of 18 predefined tender points **(Fig. 7C.53)**.
- The diagnosis of other diseases does not exclude the diagnosis of FMS.

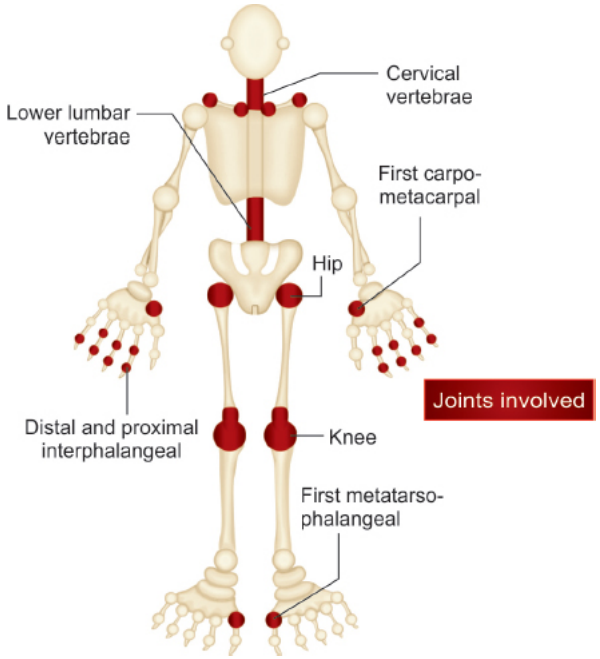


Fig. 7C.52: Pattern of joint involvement in osteoarthritis.

10. SCORING SYSTEMS FOR SEVERITY OF DISEASE

Disease activity score 28 (DAS28)

DAS28 is a common measurement of disease activity in RA and provides score that tells you how well controlled your RA is and whether treatment is working.

Twenty-eight joints (20 hand joints, 2 shoulder joints, 2 elbow joints, 2 wrist joints, and 2 knee joints) are examined throughout your body. Each joint is squeezed and the number of tender and swollen joints is calculated.

DAS28	Implication
Less than 2.6	Disease remission Usually no action necessary except Continue current medication
2.6–3.2	Low disease activity May merit change in therapy for some patients
3.2–5.1	Moderate disease activity May merit change in therapy
More than 5.1	Severe disease activity require change in therapy Consider biologic treatment

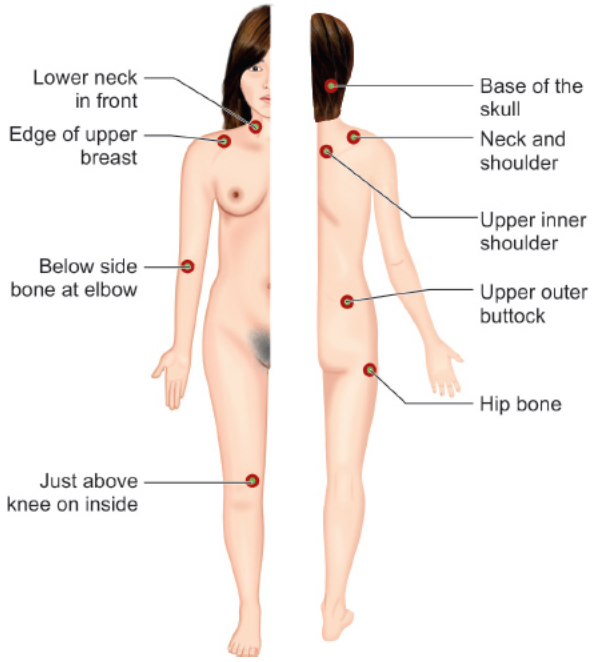
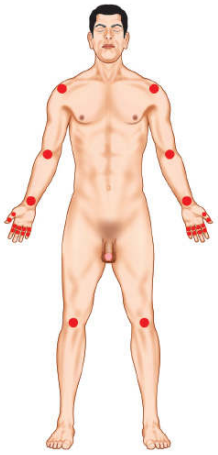


Fig. 7C.53: Trigger points in fibromyalgia.

Clinical Disease Activity Index (CDAI) (Fig. 7C.54)

Clinical Disease Activity Index (CDAI)

Joint	Left		Right	
	Tender	Swollen	Tender	Swollen
Shoulder				
Elbow				
Wrist				
MCP 1				
MCP 2				
MCP 3				
MCP 4				
MCP 5				
PIP 1				
PIP 2				
PIP 3				
PIP 4				
PIP 5				
Knee				
Total	**Tender:**		**Swollen:**	



Patient global assessment of disease activity

Considering all the ways your arthritis affects you, rate how well you are doing on the following scale:

Very	o	o	o	o	o	o	o	o	o	o	o	o	o	o	o	o	o	o	o	o	Very
Well	0	0.5	1.0	1.5	2.0	2.5	3.0	3.5	4.0	4.5	5.0	5.5	6.0	6.5	7.0	7.5	8.0	8.5	9.0	9.5	10	Poor

Your name_____**Date of birth**_____ **Today's date**_____

Provider global assessment of disease activity

Very	o	o	o	o	o	o	o	o	o	o	o	o	o	o	o	o	o	o	o	o	Very
Well	0	0.5	1.0	1.5	2.0	2.5	3.0	3.5	4.0	4.5	5.0	5.5	6.0	6.5	7.0	7.5	8.0	8.5	9.0	9.5	10	Poor

How to score the CDAI

Variable	Range	Value
Tender joint score	(0–2.8)	
Swollen joint score	(0–2.8)	
Patient global score	(0–10)	
Provider global score	(0–10)	
Add the above values to calculate the CDAI score	(0–76)	

CDAI Score Interpretation	
0.0–2.8	Remission
2.9–10.0	Low activity
10.1–22.0	Moderate activity
22.1–76.0	High activity

Fig. 7C.54: Clinical disease activity index.

NOTES

Comprehensive Geriatric Assessment

CHAPTER 8

CASE SHEET FORMAT

HISTORY TAKING

Name:

Hospital number:

Age:

Sex:

Date of examination:

Address/contact:

Name/relationship of contact person:

Contact address/number:

Problem list	Duration

Past medical history:

Medical condition	Duration
Vision impaired	
Hearing impaired	

Cancer	
OA	
Thyroid	

Family History:

Hypertension	
Diabetes	
Heart disease	
Dementia	
Cancer	

Social Assessment:

Married:	Yes	No
Spouse living:	Yes	No
Living with:		
No. of children		
How often do you see them?		
Who assists you?		
Is it sufficient?	Yes	No
Native language		
Type of house	Independent	Apartment
Stairs	Present	Absent

Personal History:

Do you exercise daily?	Yes	No

If yes, minutes/day?		
What type?		
Weight loss/gain (3 kg)	Yes	No
Smoker	Yes	No
	Duration	
Alcohol	Yes	No
	Duration	

Level of Independence (tick one of them)	Independent	
	Dependent	
	Needs assistance	

Caregiver fatigue	Yes	No

10-minute comprehensive screening				
Memory	3 objects named		Yes	No
Depression	Are you often sad/depressed?		Yes	No
Falls	Fallen more than twice in last 1 year		Yes	No
	Able to walk around chair?		Yes	No
Urinary incontinence	Lost urine/got wet in past 1 year?		Yes	No
Memory recall	One object	Two objects	Three objects	None
Draw the face of clock				

Vision	Difficulty in	Right	Left

	reading	eye	eye
Hearing		Right ear	Left ear
6, 1, 9 test—Stand behind the patient and say 6, 1 and 9 in normal tone and in whisper	Normally		
	Softly		
Constipation		Yes	No
Insomnia		Yes	No

Physical Functional Capacity:

Are you able to ……?

Run/walk fast to catch a bus	Yes	No
Do heavy work at home	Yes	No
Go shopping for groceries/clothes	Yes	No
Get to places out of walking distance?(drive/take a bus)	Yes	No
Bath using shower/bucket	Yes	No
Put on clothes/footwear	Yes	No

Basic Activities of Daily Living:

Bath	Yes	No	**Transfer**	Yes	No
Dress	Yes	No	**Toilet**	Yes	No
Toilet	Yes	No	**Feeding**	Yes	No

Montreal cognitive assessment score	
Geriatric depression score	

Physical Examination:

Height (m)	

Weight (kg)	
Body mass index (BMI) (W/H^2)	
Pulse	
Blood pressure (BP) (sitting/supine)	
BP (standing 1 minute/3 minutes)	
Anemia	Yes/No
Skin	Normal/abnormal
Teeth	Normal/abnormal
Any other GPE abnormality	

Systemic Examination:

	Normal/abnormal	Describe
Joints		
Cervical spine		
Thoracic spine		
Lumbar spine		
RS		
CVS		
P/A		

Neurological examination			R	L
Muscle strength	**Upper limb**			
		Shoulder		
		Elbow		
		Wrist		

		Small muscles of hand		
	Lower limb			
		Hip		
		Knee		
		Ankle		
Tone (describe)	Rigidity/hypotonia/spasticity			
Balance	Normal/abnormal	Sensory Cerebellar Vestibular		
Gait				
Timed up and go test (seconds)				

Current Treatment Details:

.....

Polypharmacy: Yes/No

Investigations:

Investigations	Date	Values
Complete blood picture		
Creatinine		
Electrolytes, blood sugar		
PSA (for males)		
Urine routine		
Ultrasonography (USG) abdomen and pelvis		

DIAGNOSIS FORMAT

Comprehensive Geriatric Assessment Report

Acute Illness	
Comorbidity	
Geriatric giants	
Other age-related problems	
Social problems	
Economic problems	
Prescription modification	

Examples:

Acute illness	Delirium secondary to hyponatremia Postoperative fracture neck of femur
Comorbidity	Diabetes, hypertension, dyslipidemia.
Geriatric giants	Delirium Incontinence
Other age-related problems	Cataract Stress incontinence
Social problems	Stress incontinence Living alone Feels lonely Has no body for emergency help
Economic problems	Present, not earning
Prescription modification	Avoid diuretics and beta-blockers

DISCUSSION

Comprehensive geriatric assessment (CGA) **(Fig. 8.1)** is a multidimensional, multidisciplinary diagnostic, and therapeutic p rocess conducted to determine the medical, mental, and functional problems of older people with frailty so that a coordinated and integrated plan for treatment and follow-up can be developed.

Factors which make assessment/treatment of elderly different are as follows:

- Individuals become more dissimilar as they grow
- Abrupt decline in any system is always due to disease and not due to normal aging
- Multiple pathology
- Missing symptoms (e.g. angina in an elderly patient with osteoarthritis—may not manifest)
- Masking symptoms (e.g. history of fall and fracture neck of femur in an elderly female-masked a coexistent hemiparesis due to an internal capsule infarct).

When an older person is identified as being at risk of frailty, whether in an acute hospital, day hospital, community or residential care, they should be considered for a CGA. CGA should be initiated as soon as possible after admission to hospital by a skilled, senior member of the multidisciplinary team, and used to identify reversible medical problems, target rehabilitation goals, and plan all the components of discharge and postdischarge support needs.

The CGA multidisciplinary team may include:

- Medical, e.g. geriatrician, psychiatry of old age, palliative care specialist, and general practitioner (GP)
- Nursing
- Medical social worker
- Physiotherapy

- Occupational therapy
- Speech and language therapy
- Dietetics
- Pharmacists
- Podiatry.

Benefits of Comprehensive Geriatiric Assessment

- Improves diagnostic accuracy
- Optimizes medical and rehabilitation treatment
- Enhances health and functional outcomes
- Informs the development of individualized care plans
- Assists in avoiding the potential complications of hospitalization
- Facilitates effective discharge planning.

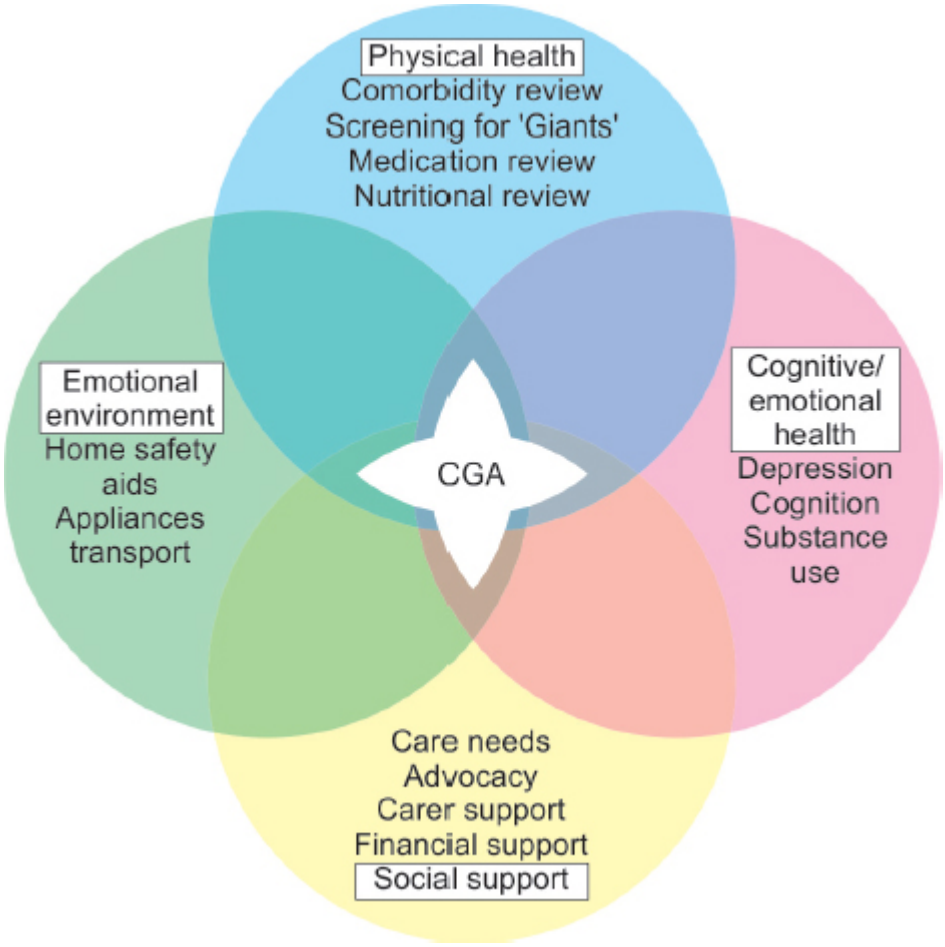


Fig. 8.1: Components of comprehensive geriatric assessment (CGA).

The four main dimensions covered in a CGA should include physical, functional, psychological, and social assessment as follows:

Four main dimensions	
Physical assessment	***Functional assessment***
• Presenting complaint • Past medical history • Medication reconciliation and review • Nutritional status • Alcohol • Immunization status	• Activities of daily living • Balance • Mobility

• Advanced directives	
Psychological assessment	***Social assessment***
• Cognition and mood	• Living arrangements • Social support • Career stress • Financial circumstances • Living environment

Identifying Elderly Patients who Would Benefit from Such an Assessment

Strongly consider if they have three or more of the "Red Flags" namely
• >75 years • Needs help with activities of daily living/instrumental activities for daily living (ADLs/IADLs) by caregiver • Lives alone • Falls • Delirium/confusion • Incontinence • >2 admissions to acute care hospital/year • "Failure to thrive"

Basic activities of daily living

Basic activities of daily livings (BADLs) are fundamental activities such as personal cares which are basic to independent living. Loss of basic ADLs places a heavy burden on the caregivers and is a marker of complete dependence.

For assessing autonomy in daily activities:

- Toileting, self-hygiene, bathing, grooming, dressing, feeding, and ambulation (stairs too).
- For each of the questions, enquire whether the person can perform it independently, whether he/she needs assistance or he/she is completely caregiver-dependent.

Instrumental activities of daily living

Instrumental activities of daily living (IADLs) are complex tasks which enable an older adult to live independently and safely. They are not necessary for fundamental existence in the way that basic ADLs are necessary, but are an indicator of functional independence. Assessment of IADLs is useful during baseline and follow-up assessments among older adults. Loss of IADLs may be the first indication of deterioration in an older adult.

- Complex tasks and roles you do at home
- Shopping, meal planning and preparation, housekeeping, laundry, transit, financial management, using a telephone, medication management, and driving.

Geriatric Giants (Fig. 8.2)

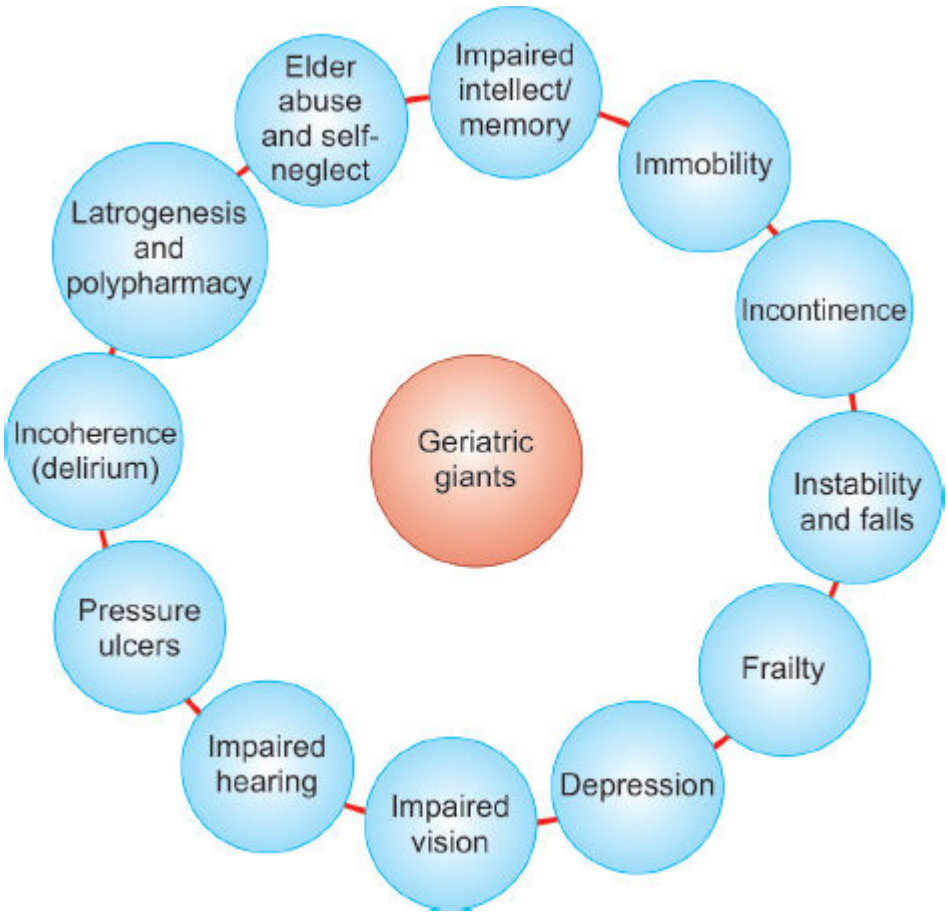


Fig. 8.2: Modern geriatric giants.

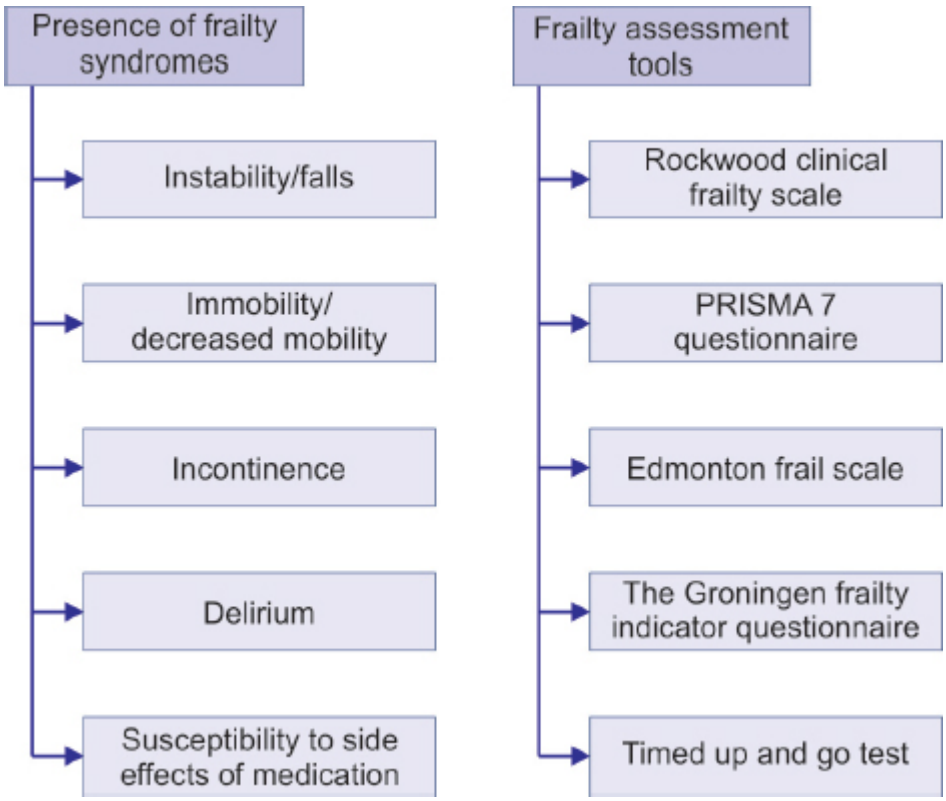
FRAILTY SYNDROME

Frailty is defined as the loss of an individual's ability to withstand minor stresses because of decreased functional reserve of several organ systems.

Two main criteria used in diagnosing frailty are Linda Fried/Johns Hopkins Frailty Criteria and the Rockwood Frailty Index.

Five key elements form the core of the frailty cycle
Frailty is defined as the presence of three or more of following conditions
1. Unexplained weight loss (>5% over a year) 2. Poor endurance and energy (self-reported) 3. Poor strength (in lowest 20th percentile) 4. Slow walking speed (Poor "Get up and Go" test) 5. Low physical activity (lowest 20th percentile)

Identifying Frailty



Objective measures of physical function	
Timed up and go (TUG) test (Fig. 8.3)	>30 seconds: Fall risk
6-meter walk	<5.8 seconds
Gait speed	>6.0 seconds
6-minute walk	<300 m: Mortality <400 m: Functional Impairment

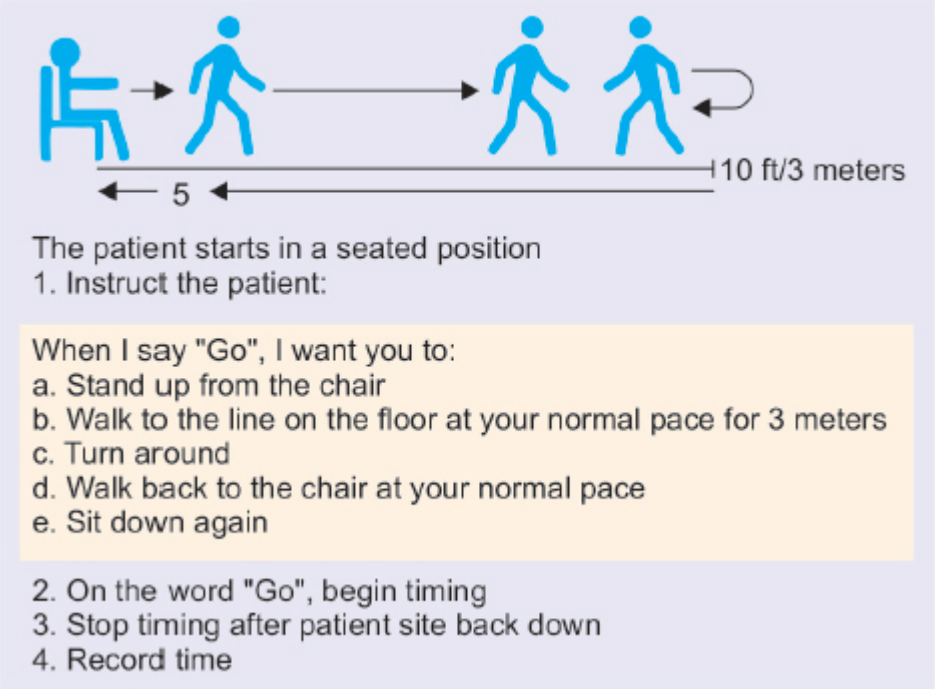


Fig. 8.3: Timed up and go (TUG) test.

DEMENTIA

Causes of dementia are given in Box 8.1.

Mini-Mental State Examination

- For screening of cognitive impairments
- Time required: 15 minutes

- Mini-mental state examination test a broad range of cognitive functions including orientation, recall, attention, calculation, language manipulation, and constructional praxis.

Box 8.1: Causes of dementia.

Degenerative/inherited:

- Alzheimer's disease—60–70%
- Neurodegenerative disorders: Frontotemporal dementia (including Pick's disease)—Lewy body disease, Parkinson's disease, Huntington's disease

Vascular dementia (10–20%): Diffuse small vessel disease

Neoplastic: Primary/secondary deposits

Traumatic: Chronic subdural hematoma, post-head injury

Infections: Creutzfeldt–Jakob disease, human immunodeficiency virus (HIV), syphilis

Toxic/nutritional: Alcohol, thiamine deficiency, vitamin B_{12} deficiency

Prion disease

Reversible dementia

For assessing cognitive impairment we use Mini-Mental State Examination (MMSE), Montreal Cognitive Assessment (MoCA), and Mini-Cog^{TM}.

Score	Interpretation
27–30	Normal
20–26	Mild impairment
10–19	Moderate impairment
Below 10	Severe impairment

Montreal Cognitive Assessment

Montreal Cognitive Assessment (MoCA) is a 30-point test that is more sensitive for the detection of mild cognitive impairment, and it includes items that sample a wider range of cognitive domains including memory, language, attention, visuospatial, and executive functions.

Mini-Cog™

The Mini-Cog™ serves as an effective triage tool to identify individuals in need of more thorough evaluation. **The Clock drawing test (CDT)** component of the Mini-Cog™ allows clinicians to quickly assess numerous cognitive domains including cognitive function, memory, language comprehension, visual-motor skills, and executive function and provides a visible record of both normal and impaired performance that can be tracked over time.

The Clock Drawing Test

Ask patient to draw the face of a clock. After numbers are on the face, ask patient to draw hands to read 10 minutes after 11:00 (or 20 minutes after 8:00).

INCONTINENCE

Involuntary loss of urine or stool in sufficient amount or frequency to constitute a social and/or health problem.

Types of urinary incontinence and causes

- **Urge incontinence:** Other names—detrusor hyperactivity, detrusor instability, irritable bladder, and spastic bladder. Infection, tumor, stones, atrophic vaginitis or urethritis, stroke, Parkinson's disease, and dementia

- **Stress incontinence:**
  - Hypermotility of bladder neck and urethra; associated with aging, hormonal changes, trauma of childbirth or pelvic surgery

– Intrinsic sphincter problems; due to pelvic/incontinence surgery, pelvic radiation, trauma, and neurogenic causes
• **Overflow incontinence:** – Bladder outlet obstruction; stricture, benign prostatic hyperplasia (BPH), cystocele, fecal impaction. – Noncontractile bladder (hypoactive detrusor or atonic bladder); diabetes, multiple sclerosis (MS), spinal injury, and medications
• **Functional incontinence**

FALLS IN THE ELDERLY (TABLE 8.1)

Table 8.1: Falls in elderly.

Intrinsic factors	*Extrinsic factors*
• Medical conditions	• Medications
• Impaired vision and hearing	• Improper usage of assistive devices
• Age-related changes	• Environment

Common pathologies associated with fall are given in Box 8.2.

Box 8.2: Common pathologies associated with fall.

- Ophthalmologic diseases
- Arthritis
- Foot problems
- Neurologic illness
- Parkinson's and related disorders
- Strokes
- Peripheral neuropathy
- Dizziness and disequilibrium

Balance test

- Done to asses the risk of falls
- **Side-by-side:** Feet side-by-side, touching;
- **Semi-tandem:** Side of the heel of one foot touching the big toe of the other;
- **Tandem:** Heel of one foot directly in front of and touching the toes of the other foot.

Note: People unable to hold a position for 10 seconds are not asked to attempt further stands.

Approach to Psychiatric Illness

CHAPTER 9

Dr Vaddi Rohit, Dr Sriraksha Nayak

CASE SHEET FORMAT

HISTORY TAKING

Name:

Sex:

Age:

Address:

Telephone No.:

Sociodemographic Data

Marital status:

1. Single
2. Married
3. Divorced.
4. Others___.

Religion:

1. Hindu
2. Muslim
3. Christian
4. Others.

Education:

1. Nil
2. Primary
3. Graduate
4. Postgraduate
5. Other
   Specify qualification _____.

Occupation:

1. Nonprofessional service

2. Professional
3. Homemaker
4. Student
5. Retired
6. Other
   Specify vocation _____.

Distance:

1. Local
2. Up to 100 km
3. Over 100 km.

Family:

1. Nuclear
2. Extended
3. Living alone.

Patients and informants report:

- Reliability: Satisfactory/unsatisfactory
- Adequacy of information: Adequate/inadequate.

History of Illness:

Presenting complaints and duration:

(Mention in chronological order).

History of presenting illness:

(Describe nature of onset as acute/subacute/insidious; precipitating events; physical illness, pharmacological treatment, and psychosocial events; evolution and course of each symptom, epiphenomena; relevant negative history; and nature of treatment received during the course).

Past psychiatric illness:

(Describe past episodes symptoms and signs; deficits; treatment received; response to treatment; compliance to treatment or reasons for poor compliance if applicable; and probable diagnosis).

Total duration of illness:

Course of the illness:

(Continuous/episodic/remittent/episodic with progressive deficits/episodic with stable deficits/incomplete remission/complete remission).

No. of episodes/exacerbations:

Past physical illness:

(Describe as in past psychiatric illness).

Family history:

(Enquire for consanguinity between parents).

Family tree:

(Family of origin up to three generations if possible).

Family history of mental illness:

(Specify mental illness/mental retardation/suicide/epilepsy/substance abuse/abnormal or odd personalities. Also elicit history of dementia, movement disorders, other neurological disorders, hypertension (HTN), type 2 diabetes mellitus (T2DM), etc. where relevant. Attempt to obtain at least two generations family history).

Premorbid personality:

How does he describe himself? What are his strengths and abilities? Is he shy or makes friends easily? Are the relations close or lasting? Does he always want to be the center of attraction? What is his mood like? Can he express feelings of love, anger, frustration or sadness? Does he ever lose control over his feelings? Has he been violent?

MENTAL STATUS EXAMINATION

I. General details of examination
II. Consciousness, rapport, and general behaviors
III. Cognitive status
IV. Examination of thought
V. Mood and effect
VI. Perception
VII. Other psychiatric phenomenon
VIII. Other phenomena
IX. Insight.

(Discussed below under the section Discussion on examination)

GENERAL AND SYSTEMIC EXAMINATION

Vitals Examination

- Pulse:
- Respiratory rate:
- Blood pressure:

Physical Examination

- Pallor
- Icterus
- Cyanosis
- Clubbing
- Lymphadenopathy
- Pedal edema.

Respiratory

- Inspection:
- Palpation:
- Percussion:
- Auscultation:

Cardiovascular System

- Inspection:
- Palpation:
- Percussion:
- Auscultation:

Gastrointestinal System

- Inspection:
- Palpation:
- Percussion:
- Auscultation:

Nervous System

- Higher mental functions:
- Cranial nerves:
- Sensory system:
- Motor system:
- Reflexes:
- Cerebellar system:
- Meningeal signs:

DIAGNOSIS FORMAT

Axis I:

Clinical syndromes and physical diagnosis.

Axis II:

Disabilities (disabilities range from normal to complete loss of function; where normal is given grade of 1 and loss of function grade 4):

- **A.** Personal:
- **B.** Occupational:
- **C.** Family:
- **D.** Social:

Total duration:

Current duration:

Axis III:

Environmental/circumstantial and personal lifestyle/life management factors.

Examples

Example 1:

Axis I:

Bipolar affective disorder, current episode mania without psychotic symptoms, and hypertension.

Axis II:

Total duration of illness 5 years current episode 1 week

- **A**3**B**2**C**2**D**2.

AXIS III:

Family history of other mental and behavioral disorders.

Example 2:

Axis I:

Paranoid schizophrenia episodic with stable deficit and diabetes mellitus.

Axis II:

Total duration of illness 8 years current duration 1 month.

- A3B2C2D4.

Axis III:

Problems in relationship with spouse or partner.

MENTAL STATUS EXAMINATION

I. General details of examination:

- Language of interview:
- Use of interpreter: Y/N
- Time taken for interview:
- Date and time:

Describe explicitly the observations (e.g. behavior, mental phenomena on tests, etc.) that are interpreted as clinical signs and symptoms.

II. Consciousness, rapport, and general behaviors:

- **Consciousness** Level and stability. If the subject is not fully alert, mention amount of stimulation needed for arousal and duration of time patient can maintain attention.
- **Rapport:**
- **General appearance and behavior** (described as follows):
    i. Appearance and appropriateness of the situation, personal cleanliness
    ii. Body build [record body mass index (BMI)]
    iii. Handedness
    iv. Grooming: Note whether patient is well-groomed or has poor hygiene
    v. Facial expression and posture
    vi. Otto Veraguth sign: Increased forehead marking seen in depression
    vii. Signs of anxiety: Excess perspiration and tensed voice
    viii. Attitude towards examiner (cooperative/guarded/playful/hostile/agitated, etc.)
    ix. Motor behavior (describe under following qualities—rate or speed, purposiveness, goal-directedness, response to command, environmental stimuli, and repetitiveness)

x. Arousal—determine the level of consciousness (especially in case of delirium).

III. Cognitive status:

i. **Attention and concentration:**

(Assessed by clinical behavior of patient and by asking the patient to count backwards, e.g. counting 100 minus 7 consecutively (or) calling out months of year or days of the week in backwards fashion.)

ii. **Language functions:**

(Neurological aspects—comment on phonation, articulation, comprehension (give a three stage command, e.g. “place index finger of right hand on your nose and then on your left ear”), naming (name a pencil and watch), repetition (repeat “No ifs, ands, or buts”), reading (ask patient to read and obey a written command on a piece of paper stating “Close your eyes”), writing (ask the patient to write a sentence. Assess if it is sensible and has a subject and a verb.)

iii. **Orientation:**

(Assess by clinical behavior. Time—day, date, week, month, year; place—room, hospital, city, etc.; person—self and others)

iv. **Memory:**

(Examiner names three objects (e.g. apple, table, and penny). Patient asked to repeat them, later asked for names of three objects learned earlier.

IV. Thought:

Speech:

Assess fluency and speed (e.g. slow/retarded speech in depression and word-finding difficulty).

Thought abnormalities:

a. Thought formation:

Is the speech coherent? Or is there loosening of association? (Suggests schizophrenia).

b. Thought possession:

Ask the patient if the thoughts are his own or if it is controlled by external source. It can be:

Thought insertion	Someone else's thoughts are being put in one's mind
Thought withdrawal	One's thoughts are being removed from one's mind
Thought broadcast	Many people are getting to know one's thoughts

c. Thought content:

Determined by asking "What are your main concerns?" Look for presence of delusions and obsessions.

Delusions
Definition: *It is a false, unshakeable belief that is out of keeping with the patient's social and cultural background and is due to internal morbid process.*
Types of delusion: • Persecutory (e.g. belief that others are out to harm me) • Grandiose [e.g. belief that one has special powers or status (suggests mania)] • Nihilistic (e.g. conviction that "My head is missing", "I have no body", and "I am dead") • Erotomanic delusions (e.g. believing a movie star loves them) • Somatic delusions (e.g. believing head is filled with air/worms—parasitosis) • Delusions of reference (e.g. belief that the story in a book is referring to them) • Delusions of **control/passivity** (e.g. believing one's thoughts and movements are controlled by aliens) • Other delusions are delusion of misinterpretation, hypochondriacal delusion, delusion of jealousy, and delusion of infidelity.

Obsessions
Definition: *An obsession is a thought that persists and dominates an individual thinking despite individual awareness beyond the point of relevance.*
Types: Theme of obsessions can be of following types: • Cleanliness: Fears of contamination • Forbidden or taboo thoughts: Aggressive, sexual, and religious obsessions • Harm (e.g. thoughts about harm on oneself or others).

V. Mood and affect:

Mood: Ask the patient how he felt in the last 1 week. Example: Sad/happy/anxious/tensed/worried.

Affect: Assessed by observing facial expression, posture, and movements. Example: Depressed/elated-elevated mood with excess energy and reduced need for sleep (suggests mania), feeling guilty or hopeless, thoughts of self-harm (suggests depression).

VI. Perception:

Observe for presence of hallucination or illusion.

Hallucination:

- It is perception without external stimuli, i.e. wakeful sensory experiences of stimuli that are not actually present. It is a disorder of thought perception.
- It can occur in any sensory modality, most common being auditory (thought echo, command hallucination, running commentary) or visual (e.g. seeing “visions”).
- Other types include tactile (cocaine bug and phantom limb), olfactory, and gustatory.
- Pseudohallucinations are a type of mental image that, although clear and vivid, lack the substantiality of perception and are located in subjective space (e.g. inside the head). They are involuntary and are seen in full consciousness.

Illusions:

In contrast to hallucinations**, illusions** are misperceptions of real external stimuli (e.g. mistaking a shrub for a person in poor light).

VII. Other psychotic phenomena:

(Somatic passivity, made action/affect/impulse.)

VIII. Other phenomena:

(Depersonalization/derealization, body image disturbances, specify negative symptoms, and other phenomena not listed above.)

IX. Insight:

(Verbatim report on the presence, nature, remedy of the problem, and possible outcomes, i.e. awareness/attribution/acceptance of intervention and comment on Grade I–VI of levels.)

Grading of insight	
Grade I	Denies illness
Grade II	Ambivalent about the illness

Grade III	Aware of illness but attribution is to **external causes** like black magic and medical illness
Grade IV	Admission of illness and recognition that symptoms or failures in social judgment are due to irrational feelings or disturbances; without applying that knowledge to future experience
Grade V	**Intellectual insight,** but able to apply knowledge appropriately
Grade VI	**Emotional insight**—fully aware of illness, consequences, and need for appropriate treatment and can make decision to choose treatment options

DISCUSSION ON DISEASES AND DIAGNOSTIC CRITERIA

Relationship of various psychiatric illnesses has been shown in **Figure 9.1**.

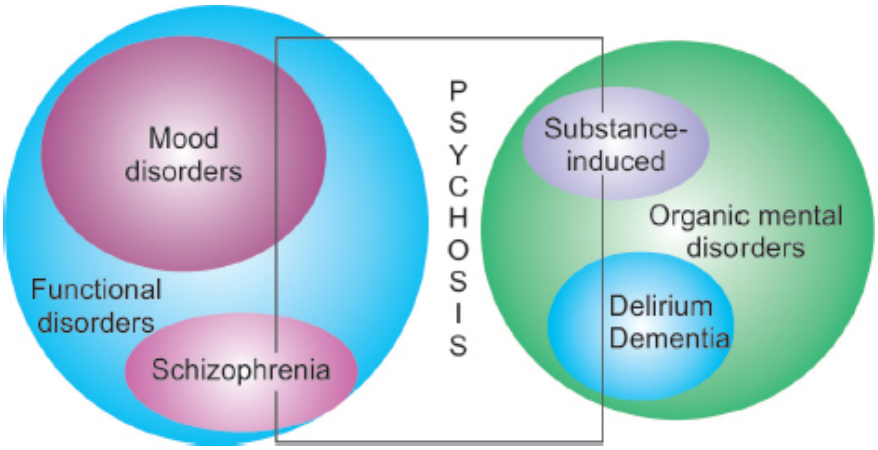


Fig. 9.1: Relationship of various psychiatric illnesses.

Table 9.1 presents differences between psychosis and neurosis.

Table 9.1: Differences between psychosis and neurosis.

Feature	*Psychosis*	*Neurosis*
Contact with reality	Lost	Preserved
Interpersonal behavior	Marked disturbance in reality and behavior	Preserved
Empathy	Absent	Present
Insight	Absence of understanding current symptoms	Present symptoms are recognized as undesirable
Symptoms	Delusions, illusions and hallucinations	Usually physical or psychic symptoms
Dealing with reality	Capacity is grossly reduced	Preserved

Examples	Schizophrenia	Anxiety, phobia, depression

Mood Disorders (Fig. 9.2 and Table 9.2)

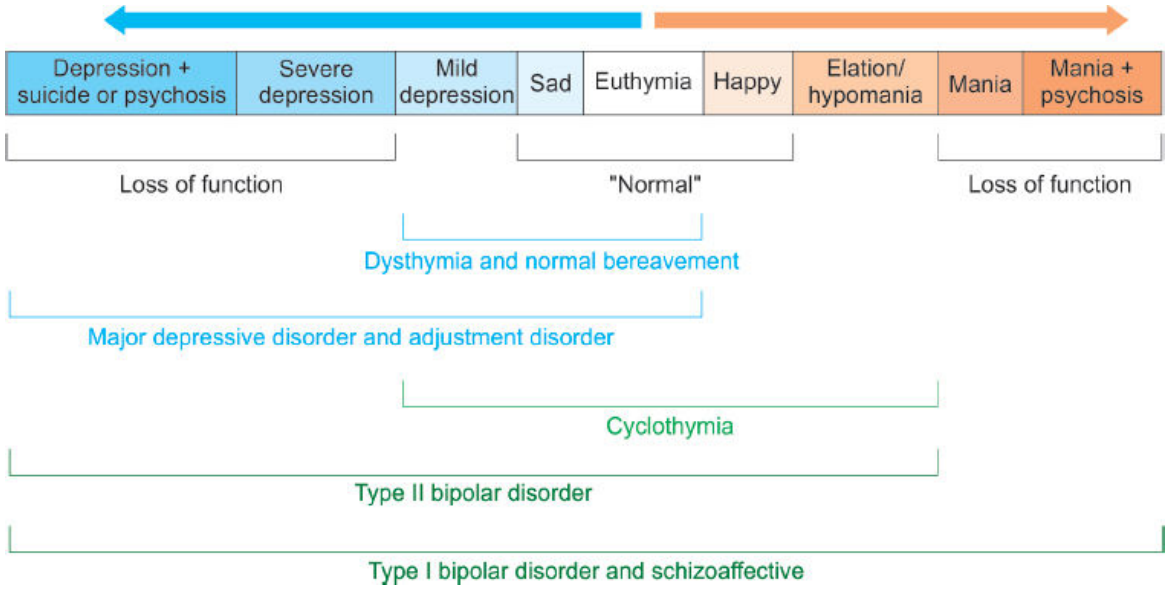


Fig. 9.2: Spectrum of mood disorders.

Table 9.2: Classification of mood disorder.		
Unipolar	***Bipolar***	***Mood disorders with known etiology***
Major depressive disorder	Bipolar I disorder	Substance-induced mood disorder
Dysthymic disorder	Bipolar II disorder	Mood disorder due to general medical condition
	Cyclothymic disorder	Depression, mania, bipolar disorders, depression-affect, mood, syndrome

Diagnostic criteria for manic episode	
Three to four of the following criteria are required during the elevated, expansive or irritable mood, lasting at least for 1 week.	
Self-esteem	Highly inflated and grandiosity
Sleep	Decreased need for sleep and rested after only a few hours
Speech	Pressured

Thoughts	Racing thoughts and flight of ideas
Attention	Easy distractibility
Activity	Increased goal-directed activity
Hedonism	High excess involvement in pleasurable activities (sex, spending, and travel)

Diagnostic criteria for major depressive episode	
General criteria for a major depressive episode require five or more of the below symptoms to be present for at least 2 weeks; one symptom must be depressed mood or loss of interest or pleasure. The symptoms must also cause distress or impairment.	
Mood	Depressed mood most of the day, nearly everyday (dysphoria)
Sleep	Insomnia or hypersomnia
Interest	Marked decrease in interest and pleasure in most activities (anhedonia)
Guilt	Feelings of worthlessness or inappropriate guilt
Energy	Fatigue or low energy nearly everyday
Concentration	Decreased concentration or increased Indecisiveness
Appetite	Increased or decreased appetite or weight gain or loss
Psychomotor	Psychomotor agitation or retardation
Suicidality	Recurrent thoughts of death, suicidal ideation, suicidal plan, and suicide attempt

Psychotic Disorders

Schneider's 11 first rank symptoms of schizophrenia
3 Thought phenomenon
• Thought insertion • Thought withdrawal • Thought broadcasting
3 Made phenomenon
• Made volition acts • Made feelings • Made impulse

3 Disorders of thought perception
Auditory hallucinations: • Audible thoughts • Voices arguing • Voices commenting on ones' action
2 Special phenomenon
• Somatic passivity phenomenon • Delusional perception

Negative and positive symptoms of schizophrenia	
Negative symptoms	***Positive symptoms***
Alogia: "Lack of words", including poverty of speech and of speech content in response to a question **Affective flattening:** Decreased expression of emotion, such as lack of expressive gestures **Avolition-apathy:** • Loss of function • Impaired concentration • Diminished social engagement **Anhedonia-asociality:** Few friends, activities, interests, impaired intimacy, little sexual interest **Attention impairment**	**Hallucinations** **Delusions** **Bizarre behavior** **Conceptual disorganization** **Aggressive/agitated**

Delirium
(For a definitive diagnosis, symptoms, mild or severe, should be present in each one of the following areas)
1. Impairment of consciousness and attention.
2. Global disturbance of cognition (illusions and hallucinations—most often visual; impairment of immediate recall and of recent memory but with relatively intact remote memory; disorientation for time as well as, in more severe cases, for place and person).
3. Psychomotor disturbances (hypo- or hyperactivity and unpredictable shifts from one to the other.
4. Disturbance of the sleep-wake cycle (insomnia or, in severe cases, total sleep loss or reversal of the sleep-wake cycle; daytime drowsiness; nocturnal worsening of symptoms).

5. Emotional disturbances, e.g. depression, anxiety or fear, irritability, euphoria or wondering perplexity.

Table 9.3 presents differences between delirium, dementia, and psychosis.

Table 9.3: Differences between delirium, dementia and psychosis.

Condition	*Onset*	*Pattern*	*Orientation*	*Attention*	*Memory*	*Duration*
Delirium	Acute	Fluctuating	Usually impaired	Impaired/fluctuating	Impaired	Hours or days
Dementia	Insidious	Progressive	Normal or impaired	~Normal	Impaired	Months or years
Psychosis	Variable	Variable	~Normal	Normal or impaired	Normal or impaired	Variable

Table 9.4 presents differences between delirium and dementia.

Table 9.4: Differences between delirium and dementia.

Features	*Delirium*	*Dementia*
Onset	Rapid (hours to days)	Gradual (years)
Course	Wide fluctuations; may continue for weeks if cause is not found	Slow but continuous decline
loss of consciousness (LOC)	Hyperalert to difficult to arouse	Normal
Orientation	Disoriented, confused	Disoriented, confused
Attention	Always impaired	May be intact; may focus on one thing for long periods
Sleep	Always disturbed	Usually normal
Behavior	Agitated, restless	May be agitated or apathetic; may wonder
Memory	Especially recent memory impairment	Especially recent memory impairment
Cognition	Disordered reasoning	Disordered reasoning and calculation

	Thought content: Incoherent, confused, delusional	
Perception	Illusions, hallucinations	No change
Judgment	Poor	Poor, socially inappropriate

Substance Abuse Disorder (Alcohol)

Alcohol dependence syndrome
(A definite diagnosis of dependence should usually be made only if three or more of the following have been present together at some time during the previous year)
1. A strong desire or sense of compulsion to take the substance.
2. Difficulties in controlling substance-taking behavior in terms of its onset, termination, or levels of use.
3. A physiological withdrawal state when substance use has ceased or been reduced.
4. Evidence of tolerance, such that increased doses of the psychoactive substance are required in order to achieve effects originally produced by lower doses.
5. Progressive neglect of alternative pleasures or interests because of psychoactive substance use.
6. Persisting with substance use despite clear evidence of overtly harmful consequences, such as harm to the liver through excessive drinking.

CAGE questionnaire
Affirmative answers to any two of the following questions (or to the last question alone) are suggestive of alcohol abuse.
1. Have you ever felt that you should cut down your drinking? 2. Have you ever felt annoyed by others criticizing your drinking? 3. Have you ever felt guilty about your drinking? 4. Have you ever had a morning drink (Eye-opener) after hangover?

Table 9.5 presents consequences of alcohol misuse and dependence.

Table 9.5: Consequences of alcohol misuse.	
Acute alcohol intoxication	***Features of alcohol withdrawal syndrome***
• Disturbances in emotional and behavioral state	• Psychological: Restlessness, anxiety, panic attacks

• Medical symptoms: Due to hypoglycemia, aspiration of vomit, respiratory depression • Complication of other medical problems • Accidents, injuries developed in fights	• Autonomic: Tachycardia, sweating, pupil dilatation, nausea, vomiting • Delirium tremens: Agitation, hallucinations, illusions, delusions • Seizures

Table 9.6 presents consequences of harmful alcohol use. **consequences of harmful alcohol use**.

Table 9.6: Consequences of harmful alcohol use.

1. Medical
• Neurological: Peripheral neuropathy, dementia, cerebral hemorrhage, cerebellar degeneration, Marchiafava-Bignami syndrome, subacute combined degeneration of the cord myopathy, ventricular enlargement and cognitive impairment. • Hepatic: Fatty change and cirrhosis, hepatocellular carcinoma • Gastrointestinal: Esophagitis, esophageal varices, Mallory-Weiss syndrome, esophageal carcinoma, gastritis, malabsorption, pancreatitis, parotid enlargement • Skin: Palmar erythema, spider naevi, Dupuytren's contractures, telangiectasias • Cardiac: Cardiomyopathy, hypertension • Respiratory: Pneumonia, tuberculosis • Musculoskeletal: Myopathy, fractures • Endocrine and metabolic: Pseudo-Cushing's syndrome, hypoglycemia, gout • Reproductive: Hypogonadism, infertility, fetal alcohol syndrome
2. Psychiatric and cerebral
• Depression • Alcoholic hallucinosis • Alcoholic 'blackouts' • **Wernicke's encephalopathy:** – Nystagmus – Ophthalmoplegia – Ataxia – Confusion • **Korsakoff's syndrome** – Short-term memory deficits – Confabulation

Semilong Cases

CHAPTER 10

SEMILONG/THERAPEUTIC CASES

Therapeutic cases are common cases that will be encountered in outpatient settings. In examination of such cases, candidate is expected to take a brief focused history, do general examination and relevant systemic examination pertaining to the case. Also, the candidate is expected to formulate a management plan for the patient which would include relevant investigations, treatment strategy, and appropriate referral.

Common therapeutic cases kept are diabetes mellitus (DM), chronic kidney disease, thyroid disorders (hypothyroid/hyperthyroid), obesity, hypertension (HTN), fever, chronic obstructive pulmonary disease (COPD), bronchial asthma, anemia, pedal edema, and anasarca.

The format of case taking would include following:

1. History:
    a. Demographic details and presenting complaints
    b. Duration of disease and presence of complications
    c. Treatment details, any surgeries/interventions, and history of hospitalizations
    d. Personal history
    e. Diet history
2. General physical examination:
    a. Vitals
    b. Anthropometry

3. Systemic examination:
    a. Skin
    b. Cardiovascular
    c. Respiratory
    d. Neurological
    e. Gastrointestinal
    f. Musculoskeletal
4. Complete diagnosis
5. Investigations
6. Treatment plan.

A: Diabetes Mellitus	
History	• Type of diabetes • Duration • Any complications—microvascular/macrovascular • Other coexistent diseases—hypertension, etc. • Treatment history • Diet history • Family history • History of hypoglycemia
Vitals	Pulse—peripheral pulses, resting tachycardia, and vessel wall thickening Hypertension and postural hypotension Raised jugular venous pressure (JVP) Pedal edema (renal, cardiac, insulin induced, and autonomic neuropathy)
Anthropometry	Body mass index (BMI), waist circumference, and waist-hip ratio
Skin	Ulcers Signs of insulin resistance (acanthosis nigricans, skin tags, and visceral obesity) Diabetic dermopathy (shin spots) and blisters *Taenia*, intertrigo, balanoposthitis **(Figs. 10A.1 and 2)**, vulvovaginitis, oral thrush, folliculitis, and carbuncle
Cardiovascular	Orthostatic hypotension, resting tachycardia, evidence of hypertension, and heart failure

Respiratory	Pneumonia and tuberculosis
Neurological	Polyneuropathy and autonomic dysfunction Retinopathy **(Figs. 10A.3 and 4)**
Gastrointestinal	Gastroparesis, constipation, and nocturnal diarrhea
Musculoskeletal	Carpal tunnel syndrome, diabetic Cheiroarthropathy, Charcot's joint, frozen shoulder, and Dupuytren's contracture
Others	Genitourinary—urinary incontinence, recurrent infection, impotence, erectile dysfunction, and retrograde ejaculation Examination of foot—ulcers, callosities, and vascular and neurological examination
Complete diagnosis	For example, type 2 diabetes mellitus with hypertension and obesity with nonproliferative retinopathy, chronic symmetrical sensorimotor polyneuropathy with autonomic dysfunction
Investigations	Hemoglobin A1c (HbA1c), fasting blood sugar (FBS), postprandial blood sugar (PPBS), serum creatinine, fasting lipid profile, urine routine and microalbuminuria, electrocardiogram (ECG), and thyroid stimulating hormone (TSH)
Treatment plan	Nutritional and lifestyle modification Drugs including insulin Management of complication
Referral	Ophthalmology, nephrology, and neurology

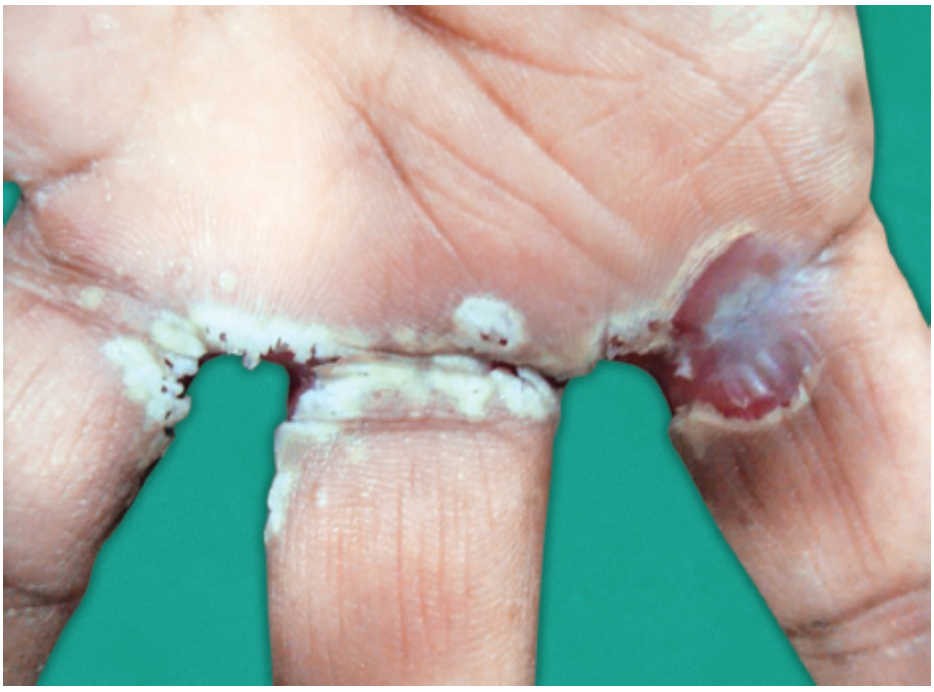


Fig. 10A.1: Intertrigo.

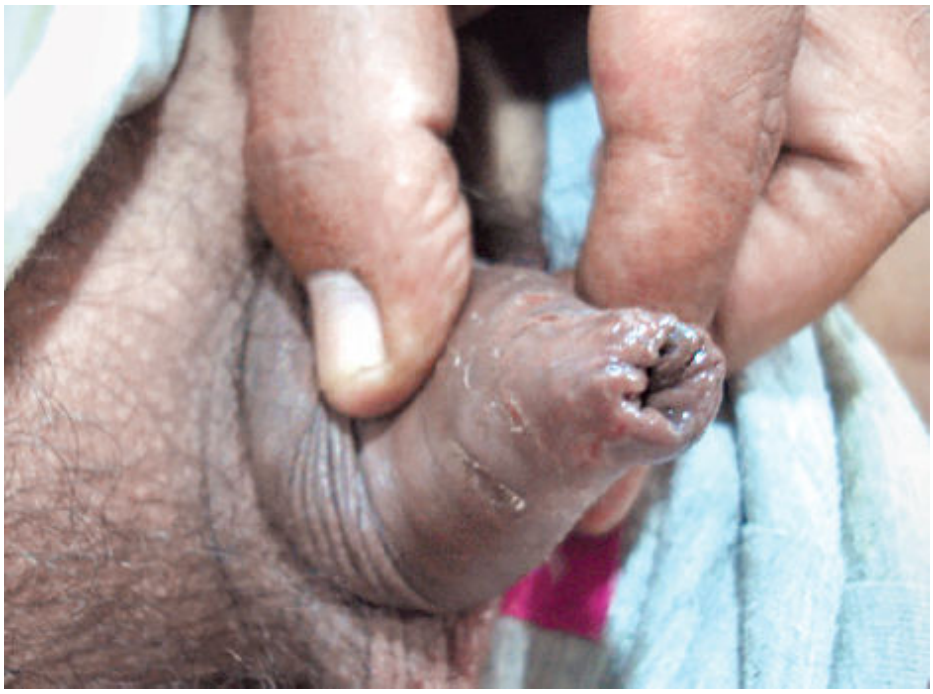


Fig. 10A.2: Balanoposthitis.

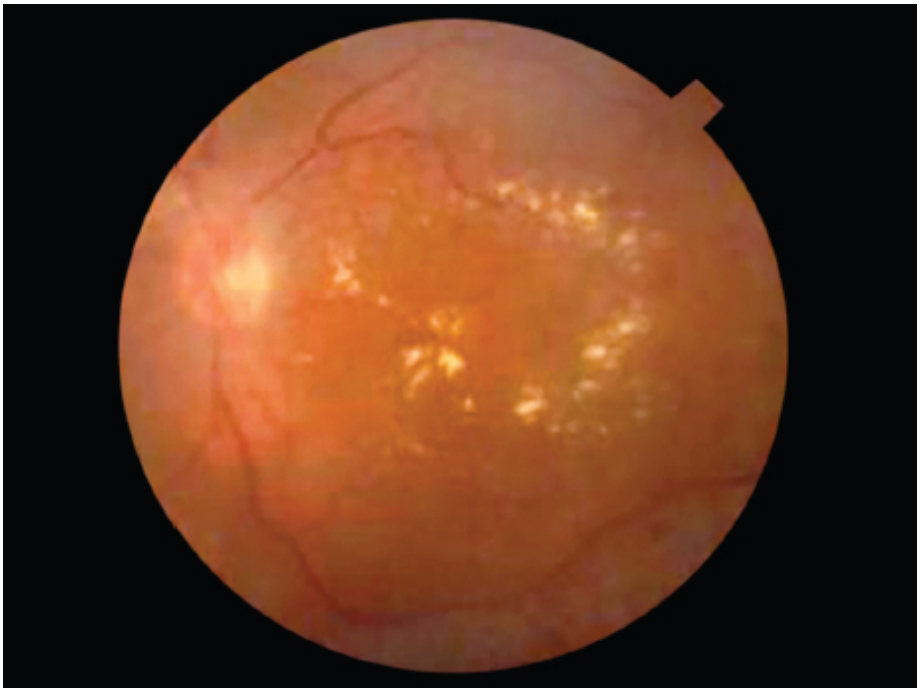


Fig. 10A.3: Nonproliferative diabetic retinopathy.

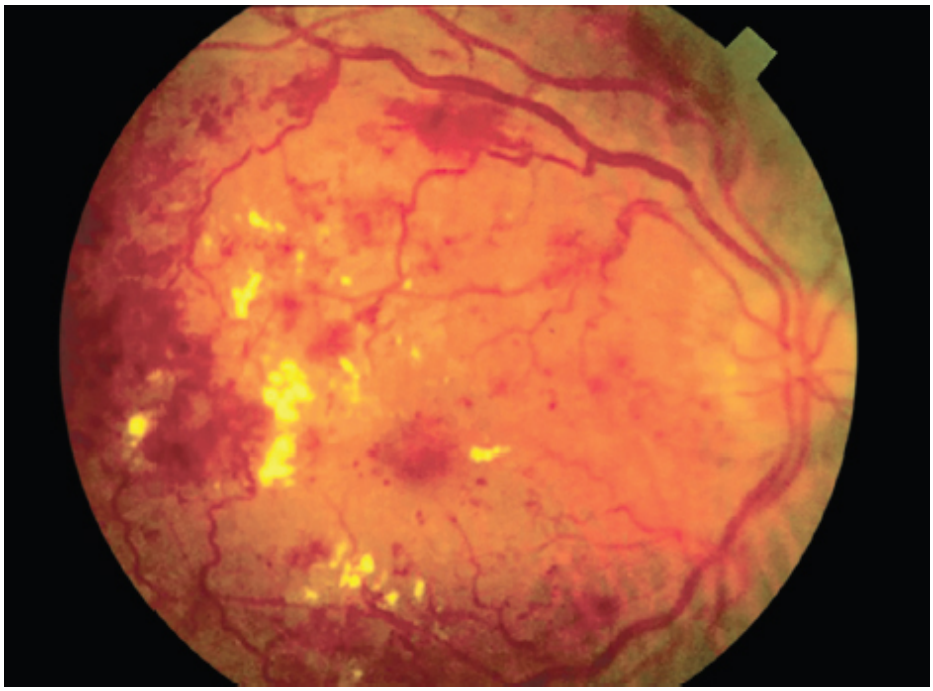


Fig. 10A.4: Proliferative diabetic retinopathy.

B: Hypertension

History	Duration Complications Treatment details
Vitals	Signs of atherosclerosis (vessel thickening, bruits, and xanthelasma) Peripheral pulses and radio-femoral delay—coarctation Pulse rate and rhythm Blood pressure (BP) to be checked in all four limbs and postural BP Edema (cardiac, renal, and drug induced) Pallor [chronic kidney disease (CKD)]
Anthropometry	BMI and waist-hip ratio
Skin	Hyperpigmentation, striae, signs of CKD, and thyroid disease
Cardiovascular	Signs of left ventricular hypertrophy (LVH) (heaving apex, S4) and heart failure
Respiratory	Obstructive sleep apnea (OSA)
Neurological	Fundus—hypertensive retinopathy Evidence of stroke
Renal	Palpable kidney (polycystic kidney) and renal bruit (renal artery stenosis).
Complete diagnosis	Hypertension (primary/secondary) with LVH and retinopathy **(Fig. 10B.1)**
Investigations	ECG, creatinine, urine routine and protein, echocardiography, FBS, lipid profile, serum uric acid, and evaluation of secondary causes—thyroid, ultrasonography (USG) abdomen
Treatment plan	Nutritional and lifestyle modification Drugs Management of complication
Referral	Ophthalmology and nephrology

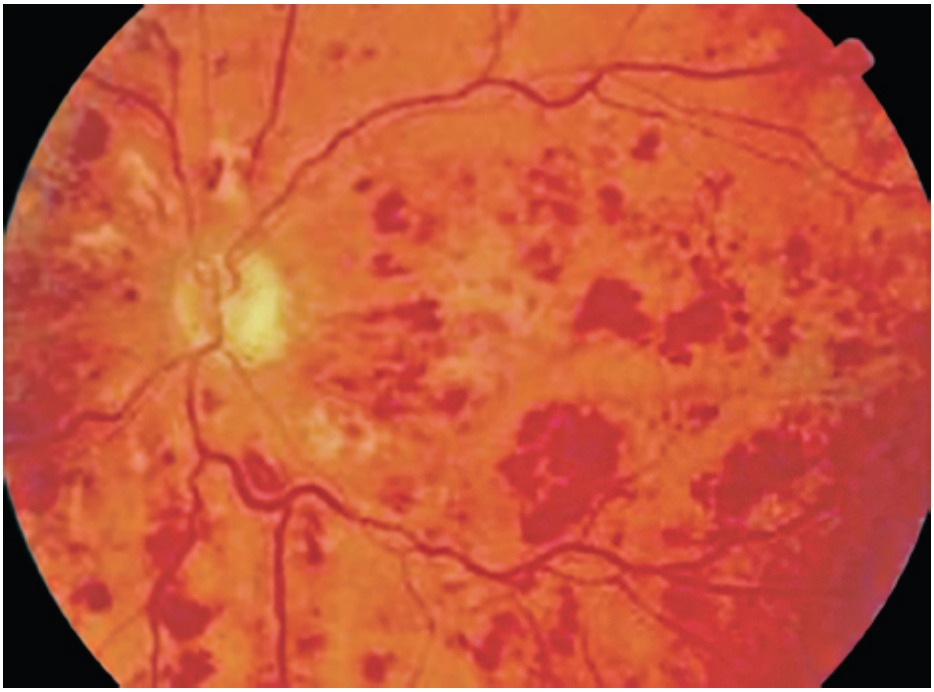


Fig. 10B.1: Fundus image of hypertensive retinopathy.

C: Chronic Kidney Disease (Fig. 10C.1)	
History	Duration Treatment details and dialysis History for etiology—DM, HTN, drugs, chronic glomerulonephritis, etc. Symptoms of uremia
Vitals	Hypertension, pallor, edema, and raised JVP
Anthropometry	BMI
Skin	Pruritus/itching, rash, uremic frost, metastatic calcification, arteriovenous (AV) fistula **(Fig. 10C.2)** and dialysis catheter
Cardiovascular	Atherosclerosis, heart failure, hypertension, and pericarditis
Respiratory	Pulmonary edema, pleural effusion, and interstitial lung disease
Neurological	Peripheral neuropathy, encephalopathy, proximal myopathy, seizures, myoclonic twitching, coma, and restless leg syndrome
Gastrointestinal	Loss of appetite (anorexia), nausea, vomiting, diarrhea, GI bleed

Musculoskeletal	Bone pains
Others	Women: Amenorrhea and menorrhagia Males: Erectile dysfunction and oligospermia
Complete diagnosis	For example, chronic kidney disease (stage—) secondary to diabetes, and patient has peripheral neuropathy
Investigations	Serum creatinine, urea, electrolytes, arterial blood gas (ABG), ECG, ECHO, ultrasound abdomen, urine analysis, and complete blood count (CBC) with peripheral smear
Treatment plan	Nutritional and lifestyle modification Drugs Medical management Hemodialysis
Referral	Nephrology

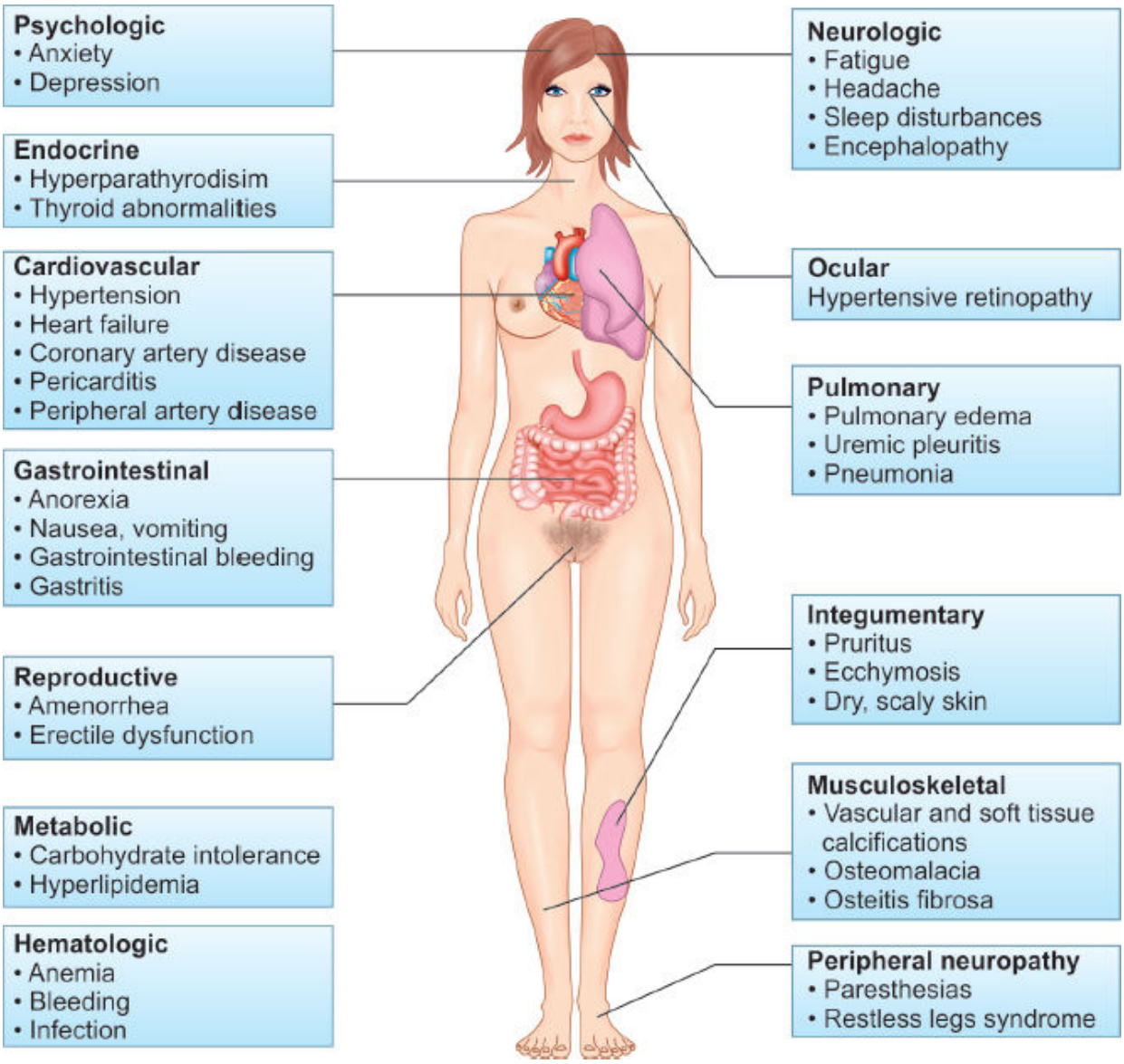


Fig. 10C.1: Various clinical manifestations of chronic kidney disease (CKD).

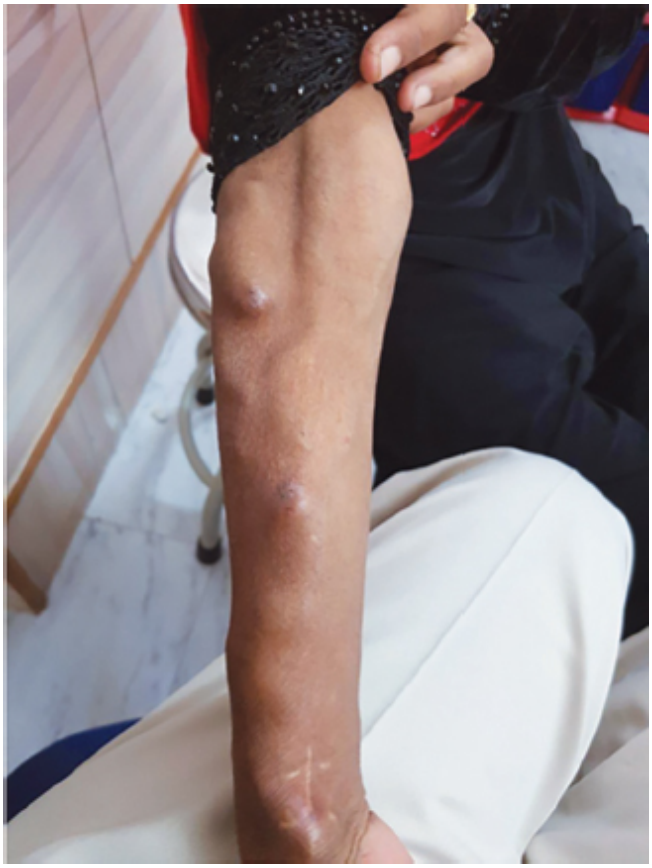


Fig. 10C.2: Arteriovenous fistula (AV) created for dialysis.

D: Hypothyroidism	
History	Lethargy, somnolence, weight gain, goiter, cold intolerance, and hoarse voice Family history Drug history
Vitals	Bradycardia, nonpitting edema, diastolic hypertension, and thyromegaly Pallor Anemia
Anthropometry	Obesity
Skin	Myxedema **(Fig. 10D.1)** (non pitting edema of the skin of hands, feet, and eyelids), dry flaky skin and hair, alopecia, vitiligo, purplish lips and malar flush, carotenemia, erythema ab igne, xanthelasmas, and madarosis (thinning of lateral one-third of eyebrows)

Cardiovascular	Angina, bradycardia, hypertension (diastolic), cardiac failure, pericardial effusion, dyslipidemia and hyperhomocysteinemia
Respiratory	Pleural effusion and OSA
Neurological	Aches and pains, muscle stiffness, delayed relaxation of tendon reflexes (Woltman's sign), carpal tunnel syndrome, depression, psychosis, cerebellar ataxia, deafness, myotonia, proximal myopathy, pseudohypertrophy of muscles, and Hashimoto encephalopathy
Gastrointestinal	Reduced appetite, constipation, ileus, ascites, and macroglossia
Musculoskeletal	Carpal tunnel syndrome
Others	Menorrhagia, infertility, galactorrhea (hyperprolactinemia), impotence and hyponatremia
Complete diagnosis	Primary hypothyroidism possibly secondary to Hashimoto's disease with bilateral carpal tunnel syndrome and infertility
Investigations	TSH, free thyroxine (FT4), thyroid peroxidase (TPO) antibodies, FBS, lipid profile, CBC with smear, and ECG
Treatment plan	Thyroxine supplementation Monitoring with TSH
Referral	Endocrinology

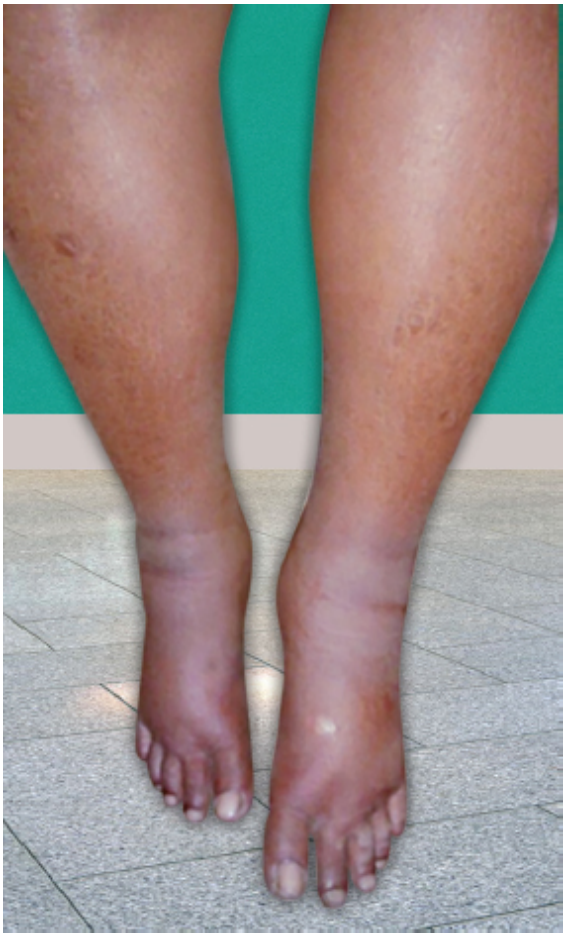
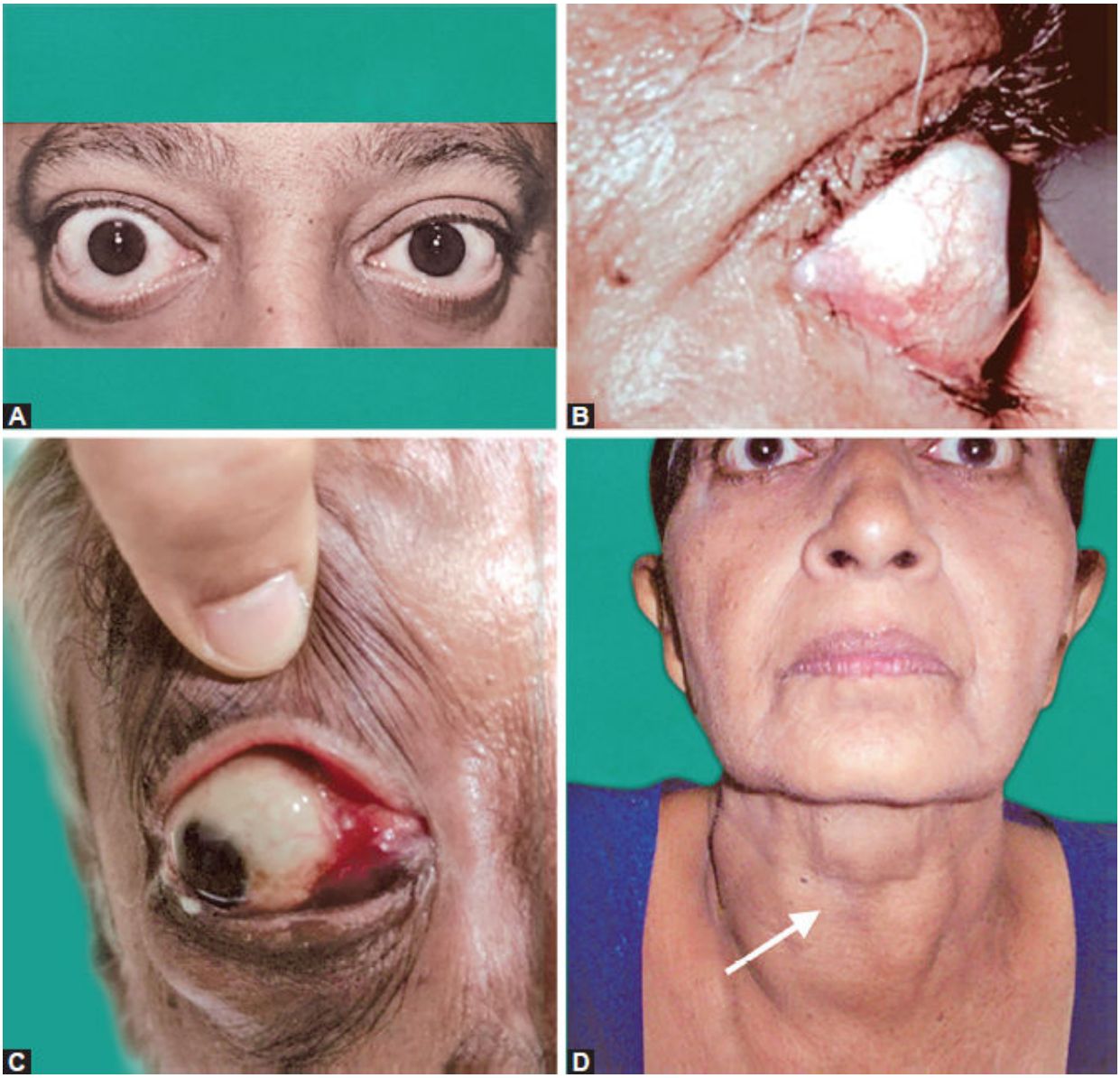


Fig. 10D.1: Non pitting pedal edema-myxedema.

E: Hyperthyroidism	
History	Weight loss, heat intolerance, fatigue, gynecomastia, apathy, and thirst
Vitals	Tachycardia, irregularly irregular pulse [atrial fibrillation (AF)], and hypertension Anemia Thyroid: Diffuse or nodular enlargement, warmth and bruit (due to increased vascularity)
Anthropometry	Low BMI
Skin	Soft, warm, and moist. Increased sweating, pruritus, palmar erythema, spider nevi, onycholysis, pretibial myxedema (Graves'), pigmentation, alopecia, and clubbing (thyroid acropachy)

Cardiovascular	Exertional dyspnea, palpitations, angina, sinus tachycardia, atrial fibrillation, wide pulse pressure, cardiac failure, cardiomyopathy, and “scratchy” midsystolic murmur (Means–Lerman scratch)
Neurological	Nervousness, irritability, psychosis, emotional lability, and fine tremors Inability to concentrate, hyperreflexia, proximal myopathy, bulbar myopathy, ill-sustained clonus
Gastrointestinal	Increased appetite, vomiting, diarrhea, and steatorrhea
Others	Menstrual disturbances (amenorrhea or oligomenorrhea), repeated abortions, infertility, loss of libido, and impotence. Eye signs **(Figs. 10E.1A to D):** Lid lag, exophthalmos, proptosis, extraocular diplopia, exposure keratitis, and lagophthalmos (classically seen in Graves’ disease)
Complete diagnosis	Primary hyperthyroidism due to Graves’ disease with thyroid ophthalmopathy and atrial fibrillation
Investigations	TSH, FT4, FT3, TSH receptor antibody, radioactive iodine (RAI) scan, USG neck, ECG, and CBC
Treatment plan	Antithyroid drugs Surgery/radioactive iodine ablation ablation Follow-up
Referral	Endocrinology, nuclear medicine, ophthalmology, and surgery



Figs. 10E.1A to D: (A and B) Exophthalmos (front and side view); (C) Infiltration of extraocular muscles in hyperthyroidism; (D) Eye signs and enlarged nodular goiter (arrow).

F: Cushing's Syndrome (Fig. 10F.1)	
History	• Onset • Duration • Any complications—cardiovascular system (CVS) and respiratory system (RS) • Other coexistent diseases

	• Treatment history—chronic steroid use with indication
Vitals	Hypertension Pedal edema
Anthropometry	BMI—truncal obesity
Skin (Figs. 10F.2A to D)	Moon face, buffalo hump, plethora, and purple striae. Easy bruisability, and ecchymosis. Thinning of hair, skin infections, and acne
Cardiovascular	Hypertension, coronary artery disease, and heart failure
Respiratory	Infections—pneumonia and tuberculosis
Neurological	Proximal myopathy, emotional lability, nervousness, irritability, and psychosis
Gastrointestinal	Pain abdomen and peptic ulcer disease
Musculoskeletal	Backache, osteoporosis, and fractures
Others	Females: Hirsutism, acne, and menstrual disturbances Male: Gynecomastia, impotence, and loss of libido
Complete diagnosis	For example, Cushing's syndrome probably due to glucocorticoid therapy
Investigations	Serum electrolytes (hypokalemia and hypochloremia), glucose tolerance test (GTT), CT/MRI abdomen (adrenal lesion) and brain (pituitary tumor), serum cortisol and adrenocorticotropic hormone (ACTH), low dose/high dose dexamethasone suppression test, and 24-hour urinary free cortisol excretion
Treatment plan	Adrenal adenoma/carcinoma—surgical resection Ectopic ACTH—treatment of primary and medical/chemical adrenalectomy Management of complications
Referral	Endocrinology and surgery

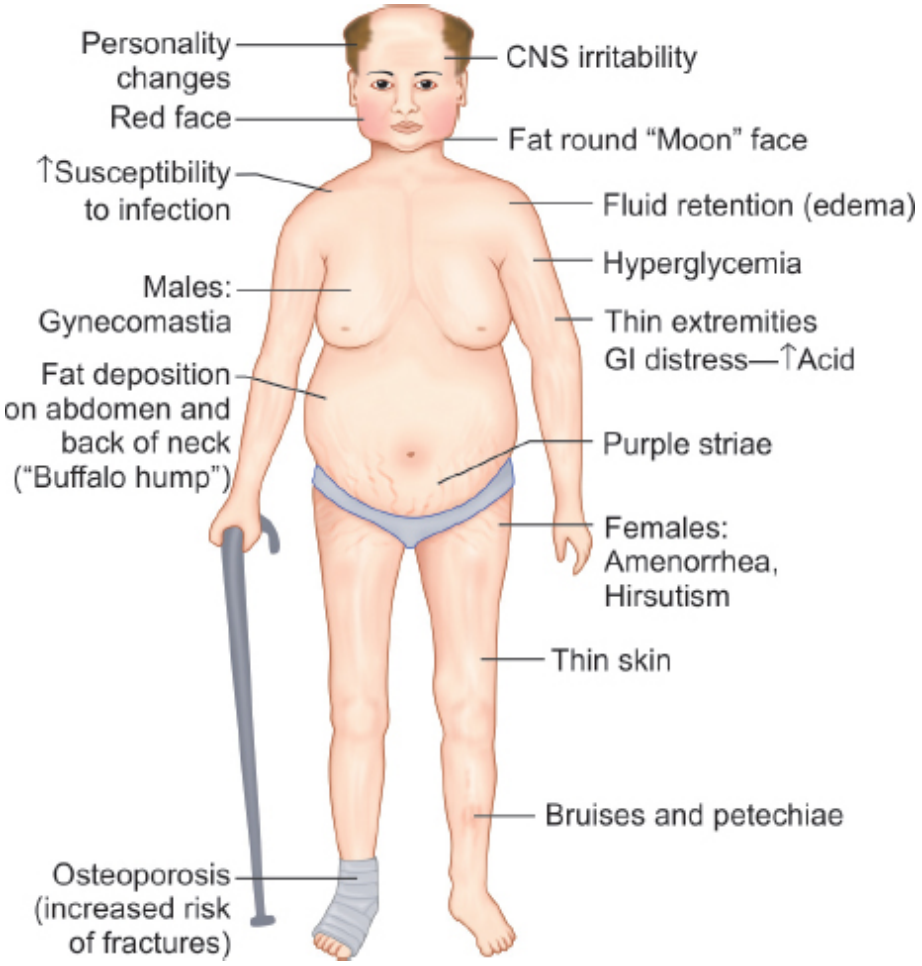
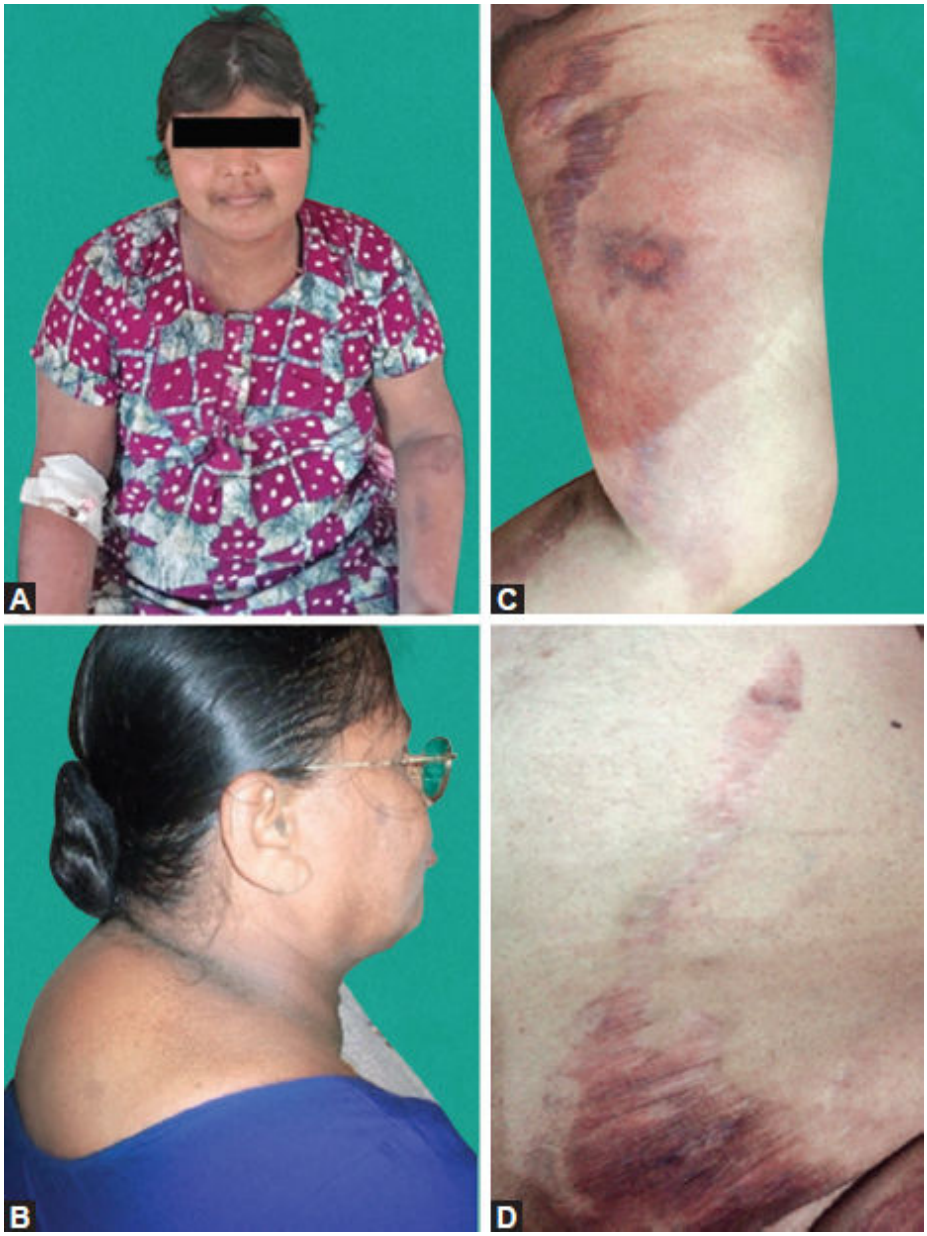


Fig. 10F.1: Clinical features of Cushing's syndrome.



Figs. 10F.2A to D: Features of Cushing's syndrome. (A) Cushing's habitus, obesity and moon facies; (B) Buffalo hump; (C and D) Pigmented striae.

G: Acromegaly (Figs. 10G.1 to 3)	
History	• Onset • Duration • Any complications—CVS and RS • Other coexistent diseases

	• Husky voice to be noted
Vitals	Hypertension
Anthropometry	BMI Gigantism
Skin	Thick skin with hypertrichosis and exaggerated nasolabial fold Hyperhidrosis, skin tags, and acanthosis nigricans
Cardiovascular	Hypertension, cardiomegaly, cardiomyopathy, and congestive cardiac failure (CCF)
Respiratory	OSA
Neurological	Proximal myopathy, bitemporal hemianopia, blindness (optic atrophy), headache, and cranial nerve palsy
Gastrointestinal	Organomegaly
Musculoskeletal	Prognathism, carpal tunnel syndrome, osteoporosis, kyphoscoliosis, dental malocclusion, and frontal bossing
Others	Macroglossia, spade-shaped hand, and increased heel pad thickness Females: Mild hirsutism, menstrual disturbances, and galactorrhea Male: Impotence and loss of libido
Complete diagnosis	Acromegaly due to pituitary tumor with impaired glucose tolerance (IGT)
Investigations (Figs. 10G.3A to C)	Basal fasting growth hormone (GH) levels, insulin-like growth factor-1 (IGF-1) level, X-ray (skull, hand, and feet), GTT, MRI brain (pituitary tumor), and visual field examination
Treatment plan	Medical: Octreotide, pegvisomant, and bromocriptine Transsphenoidal surgical removal of pituitary adenoma Management of complications
Referral	Endocrinology, neurosurgery, and ophthalmology

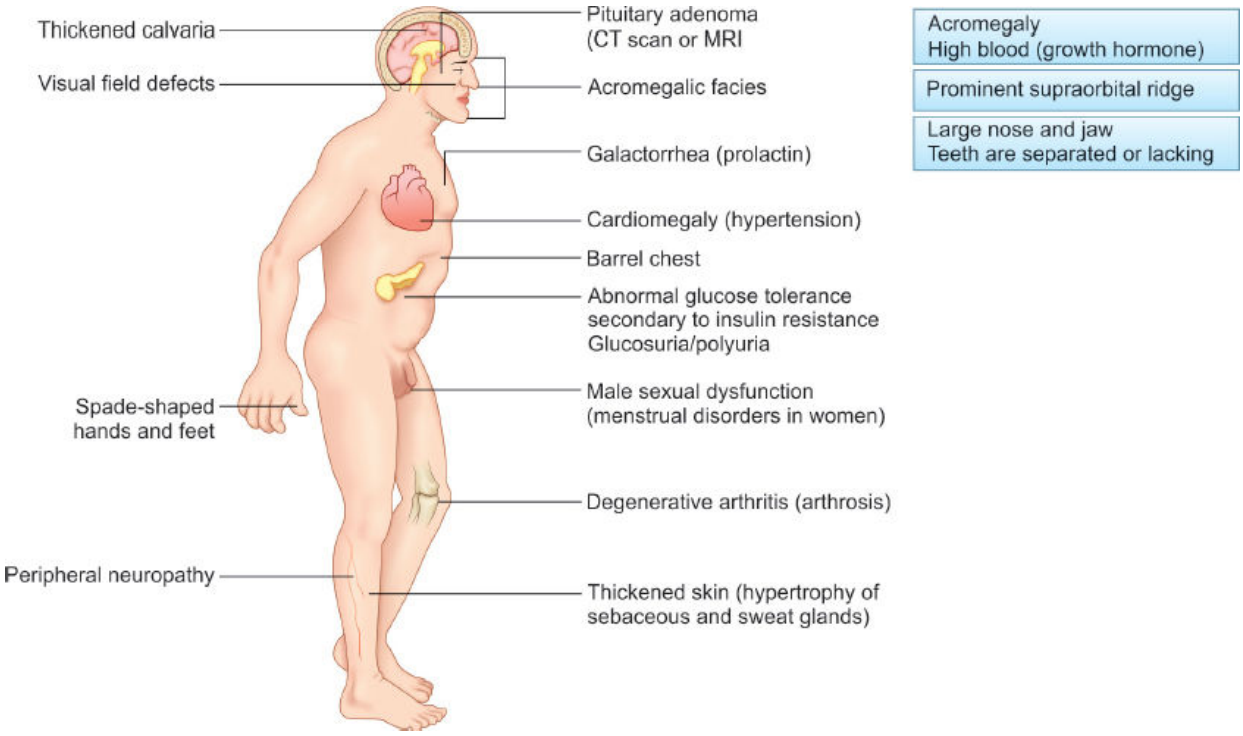
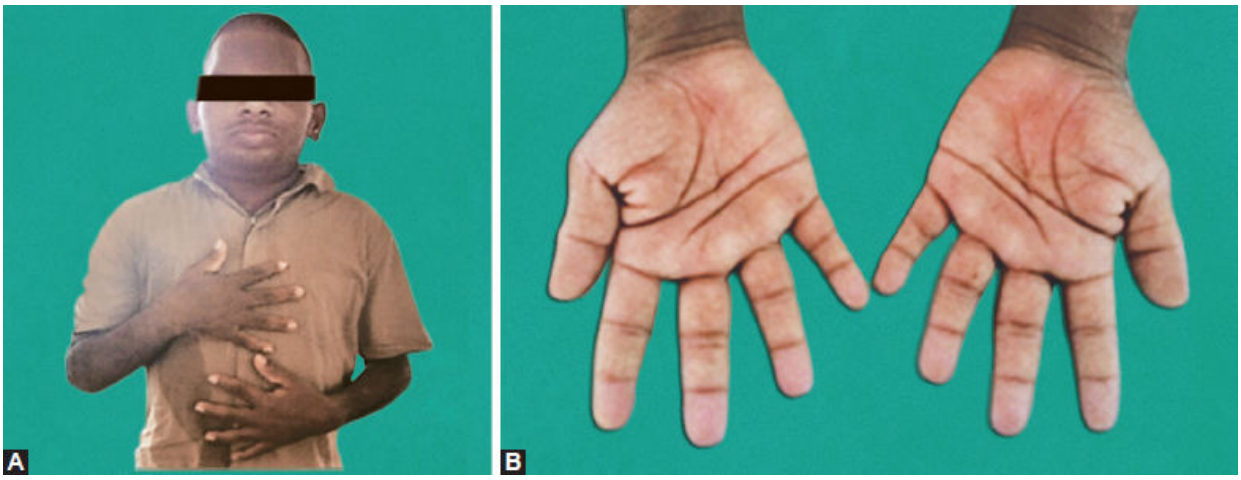
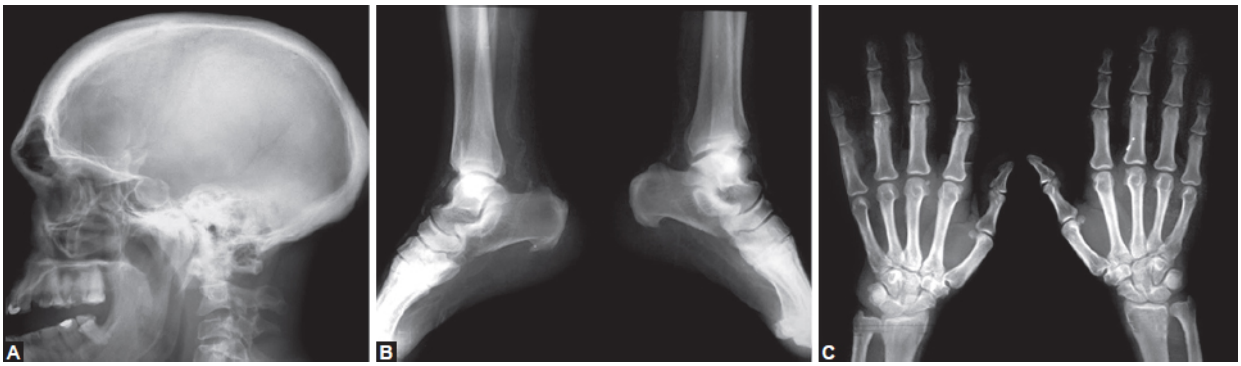


Fig. 10G.1: Summary of various clinical features of acromegaly (diagrammatic).



Figs. 10G.2A and B: Acromegalic facies and thick and spade-shaped hands.



Figs. 10G.3A to C: X-ray findings in acromegaly. (A) Lateral X-ray skull showing sellar enlargement, thickening of the calvarium, enlargement of the frontal and maxillary sinuses, and enlargement of the jaw; (B) X-ray ankle shows increased thickness of the heel pad in acromegaly; (C) X-ray of hand showing increased soft tissue bulk and "arrowhead" tufting of the distal phalanges.

Simplified Approach to ECG (Reading and Diagnosis)

CHAPTER 11

CONDUCTION SYSTEM OF THE HEART (FIG. 11.1)

The rate and rhythm of the heart are controlled by the sinoatrial node (SA node) situated at the junction of superior vena cava and right atrium.

- The impulse from the SA node spreads through the atrial musculature and down to the atrioventricular (AV) node that is situated above the tricuspid valve.
- Passage through the AV node is relatively slow, accounting for the normal physiological delay in ventricular depolarization.
- The impulse then travels downward to the bundle of His and through its branches (right bundle branch and left bundle branch) to the Purkinje network of fibers that convey the impulse to the ventricular endocardium and then epicardium.
- The SA node is the normal pacemaker of the heart as it has the fastest inherent discharge rate. However, potential pacemaking properties also exist in the cells of the AV node, bundle of His, and Purkinje fibers.
- Sinoatrial node—dominant pacemaker with an intrinsic rate of 60–100 beats/minute.
- Atrioventricular node—back-up pacemaker with an intrinsic rate of 40–60 beats/minute.
- Ventricular cells—back-up pacemaker with an intrinsic rate of 20–45 bpm.

ECG WAVEFORMS AND INTERVALS

The electrocardiogram (ECG) ordinarily is recorded on special graph paper that is divided into 1-mm^2 grid-like boxes. Since the ECG paper speed is generally 2 mm/s, the smallest (1 mm) horizontal divisons correspond to 0.04 (40 ms), with heavier lines at intervals of 0.20 s (200 ms). Vertically, the ECG graph measures the amplitude of a specific wave or deflection (1 mV = 10 mm with standard calibration; the voltage criteria for hypertrophy are given in millimeters) **(Fig. 11.2)**.

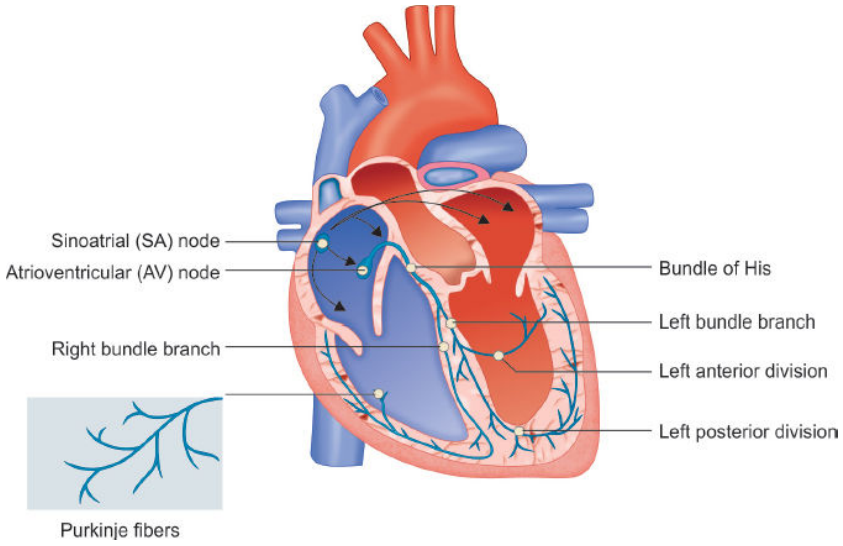


Fig. 11.1: Conduction system of the heart.

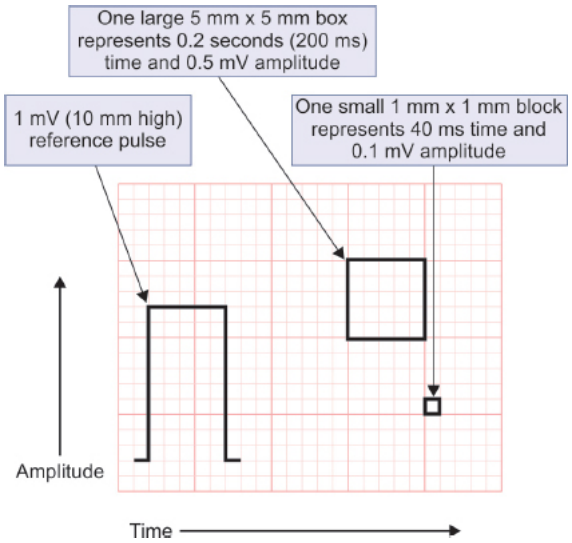


Fig. 11.2: ECG grid and standardization.

The ECG waveforms are labeled alphabetically **(Fig. 11.3)**, beginning with the P wave, which represents atrial depolarization. The QRS complex represents ventricular depolarization, and the ST-T-U complex (ST segment, T wave, and U wave) represents ventricular repolarization. The J point is the junction between the end of the QRS complex and the beginning of the ST segment. *Atrial repolarization is usually too low in amplitude to be detected, but it may become apparent in conditions such as acute pericarditis and atrial infarction.*

There are four major ECG intervals; R-R, PR, QRS, and QT. The heart rate (beats per minute) can be computed readily from the inter beat [R-number of small (0.04 s) units into 1,500]. The PR interval measures the time (normally 120–200 ms) between atrial and ventricular depolarization, which includes the physiologic delay imposed by stimulation of cells in the AV junction area. The QRS interval (normally 100–110 ms or less) reflects the duration of ventricular depolarization. The QT interval incudes both ventricular depolarization and repolarization times and varies inversely with the heart rate. A rate-related ("corrected" Bazett's correction) QT interval, QTc, can be calculated as QT/Type equation here. R-R and normally is 0.44 s (some references give QTc upper normal limits as 0.43 s in men and 0.45 s in women.

Also, a number of different formulas have been proposed, without consensus, for calculating the QTc). The QRS complex is subdivided into specific deflections or waves. If the initial QRS depletion in a particular lead is negative, it is termed a Q wave; the first positive deflection is termed an R wave. A negative deflection after an R wave is an S wave. Subsequent positive or negative waves are labeled "R" and "S", respectively. Lowercase letters (qrs) are used for waves of relatively small amplitude. An entirely negative QRS complex is termed a QS wave.

- *U Wave:Small, rounded, and upright wave following T wave. Most easily seen with a slow heart rate. Indicates repolarization of Purkinje fibers.*

ECG Leads (Figs. 11.4A and B)

The 12 conventional ECG leads record the difference in potential between electrodes placed on the surface of the body. These leads are divided into two groups: Six limb (extremity) leads and six chest (precordial) leads. The limb leads record potentials transmitted onto the frontal plane, and the chest leads record potentials transmitted onto the horizontal plane.

The spatial orientation and polarity of the six frontal plane leads are represented on the hexaxial diagram. The six chest leads are unipolar recordings obtained by electrodes in the following positions; lead V1, fourth intercostal space, just to the right of the sternum; lead V2, fourth intercostal space, just to the left of the sternum; lead V3, midway between V2 and V4: Lead V4, midclavicular line, fifth intercostal space; and lead V5, anterior axillary line, same level as V4; and lead V6, midaxillary line, same level as V4 and V5.

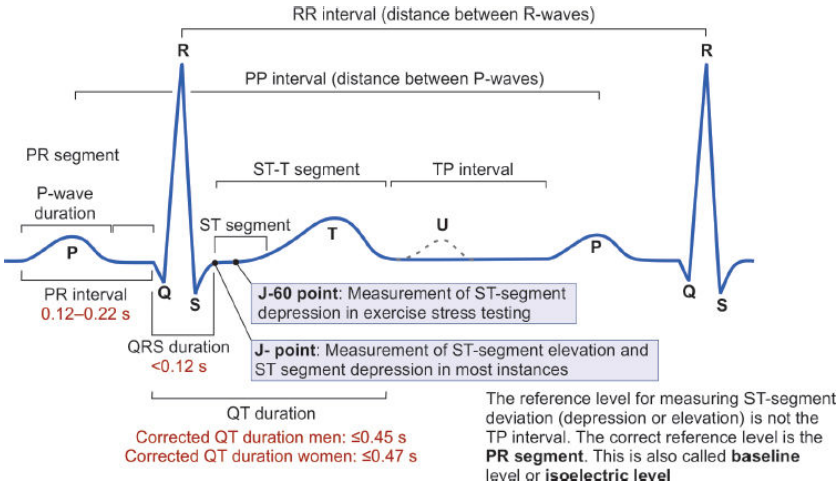
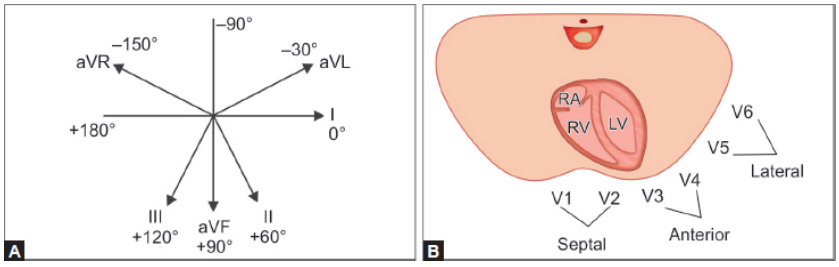


Fig. 11.3: Normal waves, segments and Intervals.



Figs. 11.4A and B: Anatomical relation of leads.

Anatomic Groups of ECG Leads

I Lateral	aVR None	V1 Septal	V4 Anterior
II Inferior	aVL Lateral	V2 Septal	V5 Lateral
III Inferior	aVF Inferior	V3 Anterior	V6 Lateral

Together, the frontal and horizontal plane electrodes provide a three-dimensional representation of cardiac electrical activity. Each lead can be likened to a different video camera angle "looking" at the same events—atrial and ventricular depolarization and repolarization—from different spatial circumstances. For example, right precordial leads V3R, V4R, etc. are useful in detecting evidence of acute right ventricular ischemia. Bedside monitors and ambulatory ECG (Holter) recordings, usually employ only one or two modified leads. The ECG leads are configured so that a positive (upright) deflection is recorded in a lead, if a wave of depolarization spreads toward the positive pole of the lead, and a negative deflection is recorded, if the wave spreads toward the negative pole. If the mean orientation of the depolarization vector is at right angles to a particular lead axis, a biphasic (equally positive and negative) deflection will be recorded.

READING 12-LEAD ECGS

The best way to read 12-lead ECGs is to develop a step-by-step approach (just as we did for analyzing a rhythm strip). In these modules, we present a seven-step approach:

1. Calculate RATE
2. Determine RHYTHM
3. Determine QRS AXIS
4. Check individual WAVES
5. Calculate INTERVALS
6. Assess for HYPERTROPHY
7. Look for evidence of infarction/dyselectrolytemia.

Step 1: Determining the Heart Rate (Fig. 11.5A)

Rule of 300/1500

Count the number of "big boxes" between two QRS complexes, and divide this into 300 (smaller boxes with 1,500) for regular rhythms.

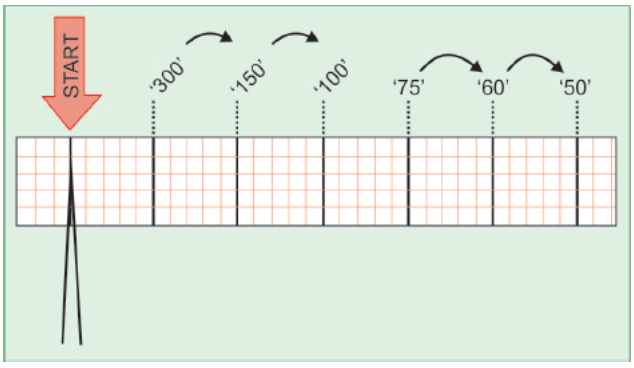


Fig. 11.5A: Calculation of heart rate.

6 Second Rule

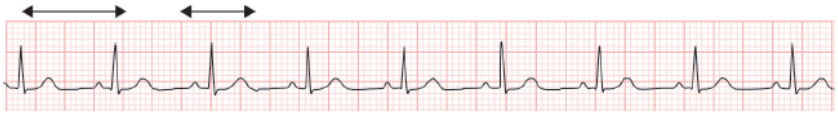
- ECGs record 6 seconds of rhythm per page

- Count the number of beats present on the ECG in 6 seconds
- Multiply by 10
- This is useful for irregular rhythms.

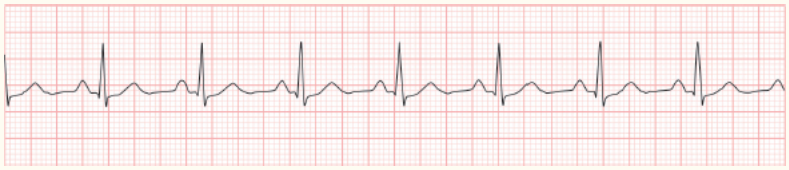
Interpretation	bpm	Causes
Normal	60–99	—
Bradycardia	<60	Hypothermia, increased vagal tone (due to vagal stimulation or drugs), athletes (fit people) hypothyroidism, beta blockade, marked intracranial hypertension, obstructive jaundice, uremia, structural SA node disease, or ischemia
Tachycardia	>100	Any cause of adrenergic stimulation (including pain); thyrotoxicosis; hypovolemia; vagolytic drugs (e.g. atropine) anemia, pregnancy; vasodilator drugs, including many hypotensive agents; fever, myocarditis

Step 2: Determine Regularity

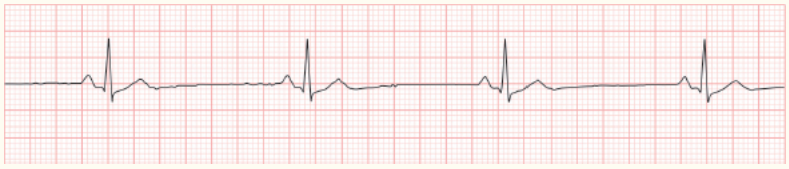
- Look at the R-R distances (using a caliper or markings on a pen or paper).
- Regular (are they equidistant apart)? Occasionally irregular? Regularly irregular?
- Irregularly irregular?—atrial fibrillation (AF).



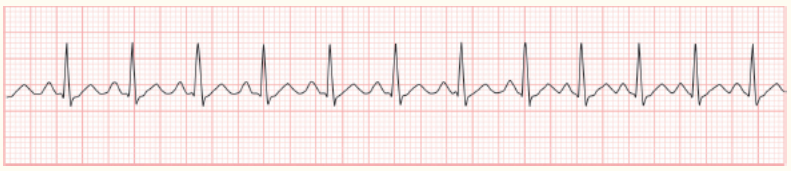
Sinus rhythm
Cardiac impulse originates from the sinus node. Every QRS must be sinus node. Every QRS must be preceded by a P wave.



Sinus bradycardia
Rhythm originates in the sinus node. Rate of less than 60 beats per minute.

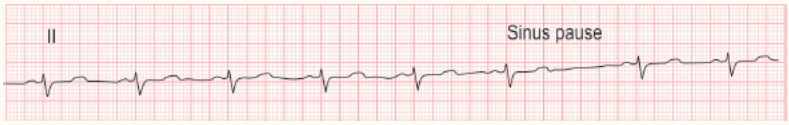


Sinus tachycardia
Rate >100 bpm, otherwise, normal



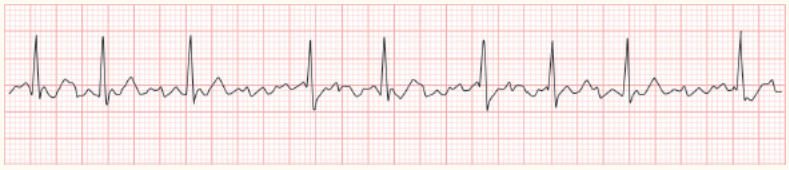
Sinus pause
In disease (e.g. sick sinus syndrome), the SA node can fail in its pacing function. If failure is brief and recovery is prompt, the result is only a missed beat (sinus pause). If recovery is delayed and no other focus assumes pacing function, cardiac arrest

follows.



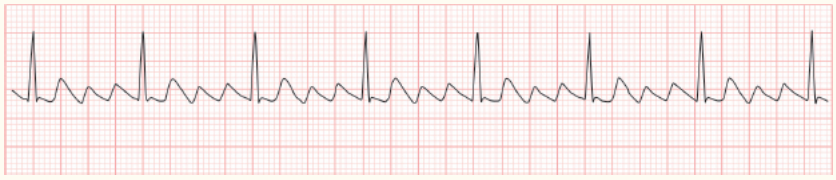
Atrial fibrillation

Atrial rate approximately 400–600; Ventricular rate approximately 150 bpm; irregularly irregular, baseline irregularity, no visible p waves, QRS occurs irregularly with its length usually <0.12 s, fibrillary waves.



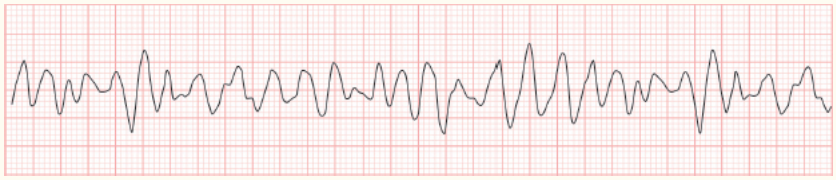
Atrial flutter

Atrial rate =~300 bpm, P waves absent but have flutter waves, ECG baseline adapts “saw-toothed” appearance.



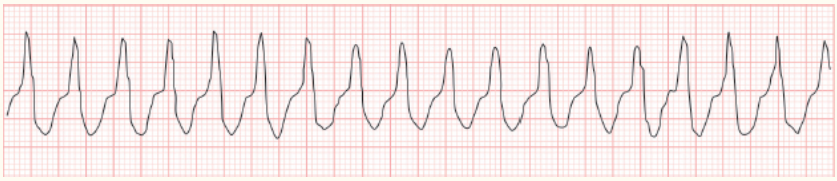
Ventricular fibrillation

Rate cannot be discerned, rhythm unorganized, QRS broad >0.12 s



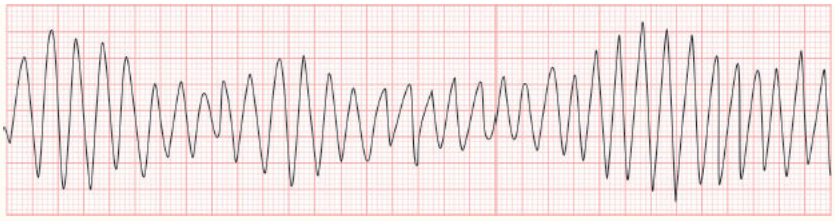
Ventricular tachycardia

Rate = 100–250 bpm, broad QRS, regular



Torsades de Pointes

Literally meaning twisting of points is a distinctive form of polymorphic ventricular tachycardia characterized by a gradual change in the amplitude and twisting of the QRS complexes around the isoelectric line.



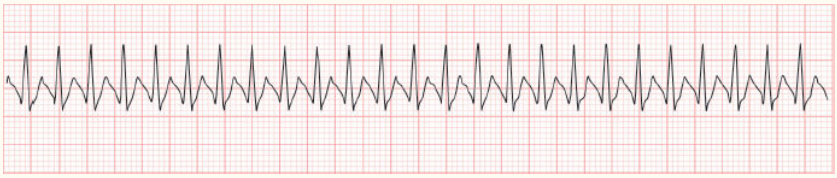
Supraventricular tachycardia

Tachycardic rhythm originating above the ventricular tissue. Atrial and ventricular rate = 150–250 bpm. Regular rhythm, p is usually not discernable.

Note:

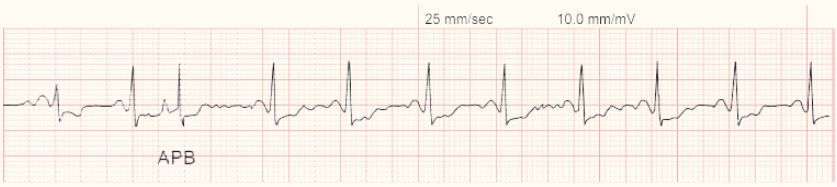
Types of SVT:

- Sinoatrial node reentrant tachycardia (SANRT)
- Ectopic (unifocal) atrial tachycardia (EAT)
- Multifocal atrial tachycardia (MAT)
- A-fib or A flutter with rapid ventricular response. Without rapid ventricular response both usually not classified as SVT
- Atrioventricular (AV)-nodal reentrant tachycardia (AVNRT— commonest)
- Permanent (or persistent) junctional reciprocating tachycardia (PJRT)
- Atrioventricular reentrant tachycardia (AVRT)



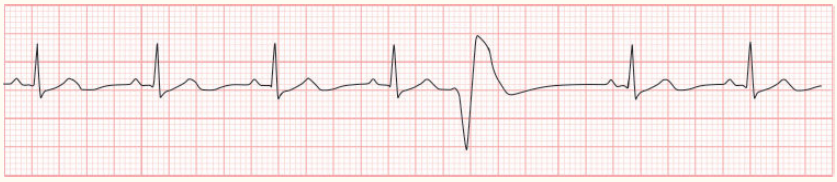
Atrial premature beat (APB)

Arises from an irritable focus in one of the atria. APB produces different looking P wave, because depolarization vector is abnormal. QRS complex has normal duration and same morphology.



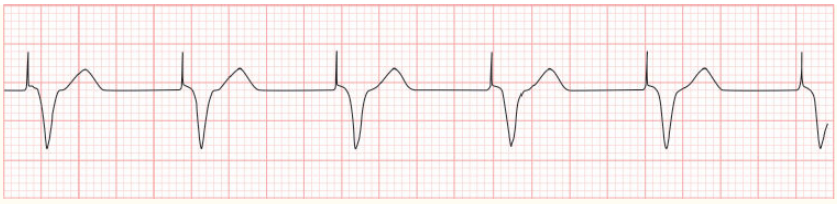
Premature ventricular complexes (PVCs)

- Occasionally irregular rhythm, broad QRS arising from ventricles
- No P-wave associated with PVCs. It can be monomorphic/polymorphic.



Artificial pacemaker

Sharp, thin spike, before each complex, ventricular paced rhythm shows wide ventricular pacemaker spikes.



Step 3: Determining the Axis

- Normal QRS axis from −30° to +110°.
- −30° to −90° is referred to as a left axis deviation (LAD).
- +110° to +180° is referred to as a right axis deviation (RAD).
- −180° to −90° is referred as north-west axis/extreme axis/axis in no man's land as depicted in **f ig. 11.5 b**.

Axis	LI	LIII or aVF	TIP (Fig. 11.5C)
Normal	Positive	Positive	Both up
Right	Negative	Positive	Meet-**R**EACHING
Left	Positive	Negative	Separate-**L**EAVING
Northwest	Negative	Negative	Both down

- QRS complex in leads I and aVF.
- Determine if they are predominantly positive or negative.
- The combination should place the axis into one of the 4 quadrants above.

Cardiac axis	Causes
Left axis deviation	• Left anterior hemiblock, left ventricular hypertrophy, Wolff-Parkinson-White syndrome, inferior myocardial infarction (MI), ostium primum atrial septal defect (ASD), and ventricular tachycardia • Normal variation in pregnancy, obesity; ascites
Right axis deviation	Normal finding in children and tall thin adults, right ventricular hypertrophy (RVH), chronic lung pulmonary disease (COPD), left posterior hemiblock, Wolff-Parkinson-White syndrome, and anterolateral MI
North West	Dextrocardia, severe emphysema, hyperkalemia, lead transposition, artificial cardiac pacing, and ventricular tachycardia

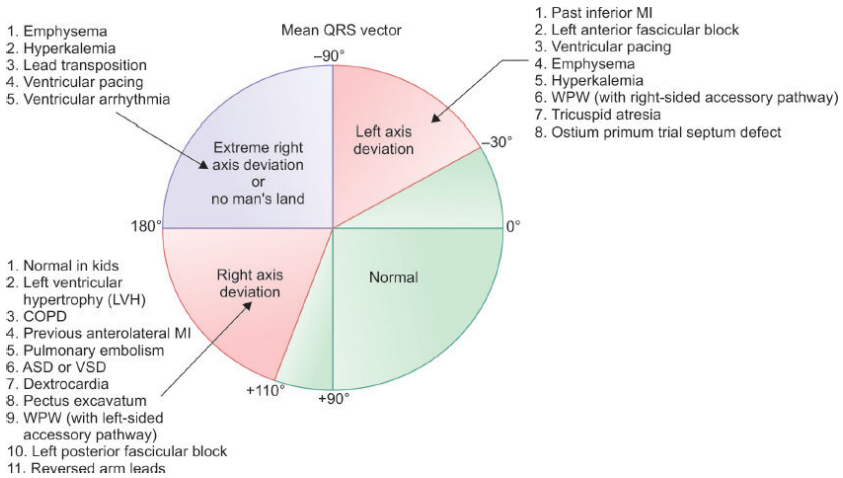


Fig. 11.5B: Pictorial representation of AXIS deviation with examples.
(COPD: chronic obstructive pulmonary disease; ASD: atrial septal defects; VSD: ventricular septal defects)

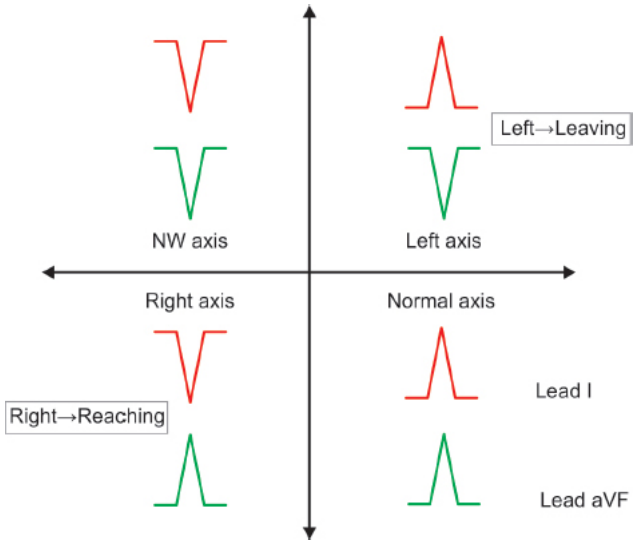


Fig. 11.5C: Axis determination based on direction of lead I and lead aVF.

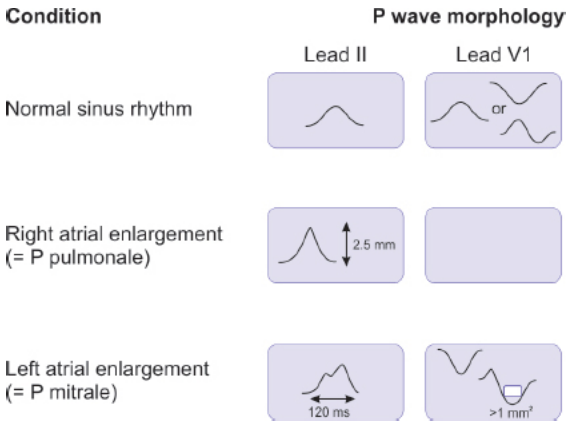
Step 4: Check Individual Waves

Assess P Waves

- Always positive in lead I and II
- Always negative in lead aVR
- <2.5 small squares in duration
- <2.5 small squares in amplitude
- Commonly biphasic in lead V1

- Best seen in leads II
- Tall (>2.5 mm), pointed P waves **(P pulmonale)—**suggests right atrial enlargement
  - Seen in chronic obstructive pulmonary disease (COPD), atrial septal defect (ASD), TS, Ebstein anomaly **(Himalayan P waves**)
- Notched/bifid (“M” shaped) P wave **(P “mitrale”)** in limb leads—suggests left atrial enlargement
  - Seen in MS, MR, and systemic hypertension
- Absent P waves—atrial fibrillation/flutter

- Inverted P waves in lead II—dextrocardia



QRS-Complex

Normal characteristics:

- *Duration:* 0.04–0.11 seconds.
  - **Broad/wide QRS** (>0.12 s)
    - » Ventricular hypertrophy
    - » Intraventricular conduction disturbance
    - » Aberrant ventricular conduction
    - » Ventricular pre-excitation
    - » Ventricular ectopic or escape pacemaker
    - » Ventricular pacing by cardiac pacemaker.
- Q <0.04 s, <25% of R wave
- Height of QRS—**Sokolow index** (SV2 + RV5) <35 mm (<45 mm for young)
  - Increased in RV/LV hypertrophy
  - Decreased—**low voltage QRS** (<5 mV in limb leads/<10 mV in chest leads)
    - » Obese patient
    - » Restrictive cardiomyopathy
    - » Pericardial effusion
    - » Hypothyroidism
    - » Hypothermia
    - » Myocarditis.
- Axis of ventricular depolarization −30 to +110° (abnormalities already discussed)
- **v entricular activation time (VAT)—**time from start of q wave till top of R wave. Normal of LV <0.04 s (V5 and V6 leads), RV <0.03 s (V1 Lead).
  - Prolonged in ischemia, bundle branch block
- **Precordial R wave progression**, i.e. R wave amplitude progressively increases from V1 to V6.
  - Absent R wave progression sign of anterior wall MI.

Q Waves

- The normal Q wave in lead I is due to septal depolarization
- It is small in amplitude—less than 25% of the succeeding R wave, or less than 3 mm
- Its duration is <0.04 sec or one small box
- It is seen in L1 and sometimes in V5 and V6
- The pathological Q wave of infarction in the respective leads is due to dead muscle

- It is deep in amplitude—more than 25% of the succeeding R wave, or more than 4 mm. Its duration is >0.04 sec or >1 small box
- Pathological Q waves may be seen in cardiomyopathies—hypertrophic obstructive cardiomyopathy (HOCM), infiltrative myocardial disease
- Absent Q waves in V5–V6 is most commonly due to left bundle branch block (LBBB).

T Wave

- Normally repolarization directs from epicardium to endocardium = T wave is concordant with QRS complex
- Ischemic area: A repolarization is delayed, an action potential is extended
- Vector of repolarization is directed from ischemic area:
  - Subendocardial ischemia—to epicardium—T wave elevation
  - Subepicardial ischemia—to endocardium—T wave inversion.
- Asymmetrical T wave inversion— the first half having more gradual slope than the second half
- Symmetrical→T wave inversiobn seen in ischemia
- Amplitude rarely exceeds 10 mm.

Causes of T wave inversions	Tall T waves (more than two-thirds of neighboring QRS)
• CAD/ischemia • Cardiomyopathies—hypertrophic • Myocarditis and pericarditis • Wellens syndrome • Pulmonary embolism • Raised ICT—CNS bleed • Ventricular hypertrophy • Bundle branch block • Pacing • Persistent juvenile T wave pattern	• Hyperkalemia—Steeple T waves • Hyperacute MI • Benign early repolarization (BER)

U Waves

- The U wave is a wave on an electrocardiogram that is not always seen. It is typically small, and, by definition, follows the T wave. U waves are thought to represent repolarization of the papillary muscles or Purkinje fibers
- Normal U waves are small, round and symmetrical and positive in lead II. It is the same direction as T wave in that lead.
- Prominent U waves are most often seen in hypokalemia, but may be present in hypercalcemia, thyrotoxicosis, or exposure to digitalis, epinephrine, and class 1A and 3 antiarrhythmics, as well as in congenital long QT syndrome, and in the setting of intracranial hemorrhage.
- An inverted U wave may represent myocardial ischemia or left ventricular volume overload.

The Osborn wave (J wave) is a positive deflection at the J point (negative in aVR and V1), characteristically seen in hypothermia (typically temperature <30°C), but also can be seen in raised ICT, hypercalcemia
Epsilon wave is a small positive deflection buried in the end of the QRS complex. It is the characteristic of arrhythmogenic right ventricular dysplasia (ARVD).

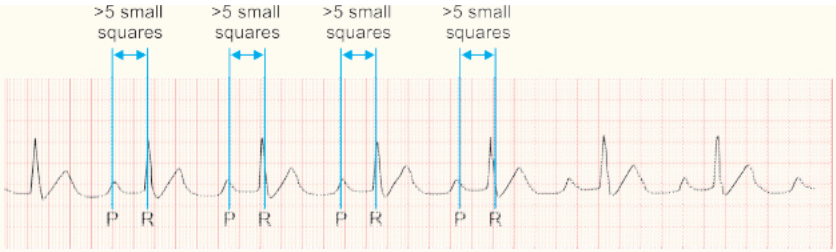
Step 5: Calculate Intervals

PR Interval (Figs. 11.6A to C)

Normal: 0.12–0.20 seconds.

Long PR interval may indicate heart block.

First degree heart block

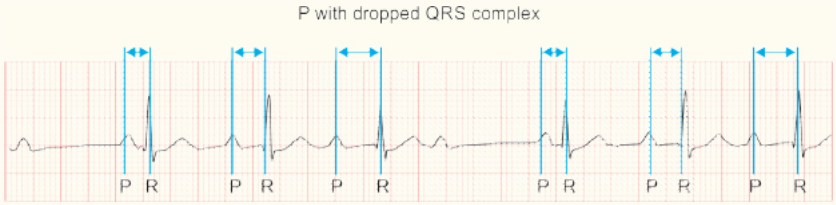


P wave precedes QRS complex but PR intervals prolong (>5 small squares) and remains constant from beat to beat

Second degree heart block

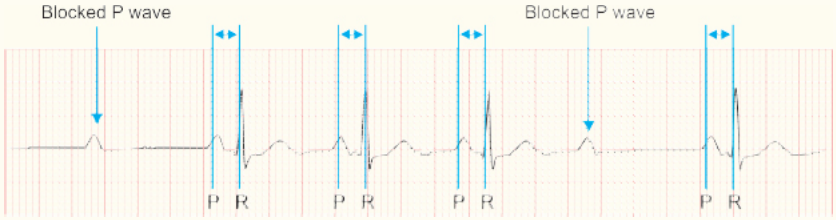
1. **Mobitz Type I or Wenckebach**

- Runs in cycle, first PR interval is often normal. With successive beat, PR interval lengthens until there will be a P wave with no following QRS complex.
- The block is at AV node, often transient, may be asymptomatic.



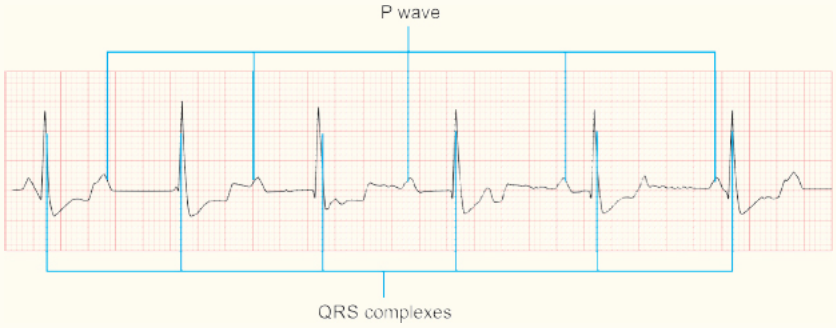
2. **Mobitz Type 2**

- PR interval is constant, duration is normal/prolonged. Periodically, no conduction between atria and ventricles—producing a p wave with no associated QRS complex (blocked P wave).
- The block is most often below AV node, at bundle of His or BB,
- May progress to third degree heart block.



Third degree heart block (complete heart block)

- No relationship between P waves and QRS complexes.
- An accessory pacemaker in the lower chambers will typically activate the ventricles—escape rhythm. Atrial rate = 60–100 bpm. Ventricular rate based on site of escape pacemaker. Atrial and ventricular rhythm, both are regular.



Causes of Conduction Block

- CAD, acute MI, remote MI, pulmonary embolism

- Drugs
- Aortic stenosis, SABE + abscesses in conduction
- Cardiac trauma and hyperkalemia
- Lenegre's disease (idiopathic fibrosis of conduction)
- Lev's disease (calcification of the cardiac skeleton)
- Cardiomyopathy—dilated and hypertrophic
- Infiltrative—Chagas disease
- Myxedema, amyloidosis, and ventricular hypertrophy
- Idiopathic.

Short PR interval

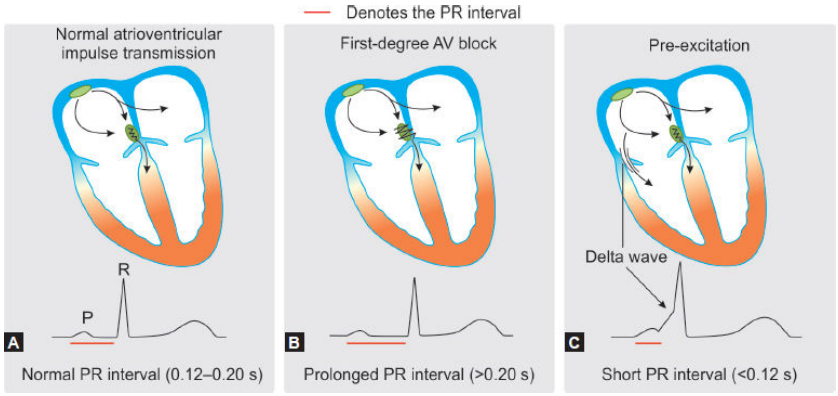
1. Tachycardia
2. Pre-excitation syndromes
   a. Lown–Ganong–Levine syndrome
   b. Wolff-Parkinson-White (WPW) syndrome
   c. Mahaim pathway.

The diagnostic triad of WPW consists of a wide QRS complex associated with a relatively short PR interval and slurring of the initial part of the QRS (delta wave), with the latter effect being due to aberrant activation of ventricular myocardium. The presence of a bypass tract predisposes to re-entrant supraventricular tachyarrhythmias.

QT Interval

It represents the time taken for ventricular depolarization and repolarization.

- The duration of the QT interval is proportionate to the heart rate. The faster the heart beats, the faster the ventricles repolarize so the shorter the QT interval. Therefore what is a "normal" QT varies with the heart rate.
- QT interval should be 0.35–0.45 s.
- For each heart rate you need to calculate an adjusted QT interval, called the "corrected QT" (QTc): QTc = QT/square root of RR interval—**Bazett's formula**.



Figs. 11.6A to C: (A) Normal atrioventricular impulse transmissions; (B) First-degree AV block; (C) Pre-excitation.

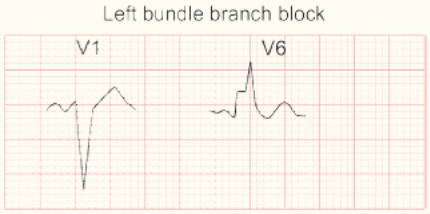
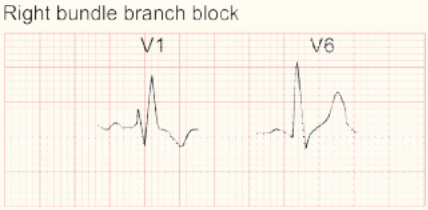
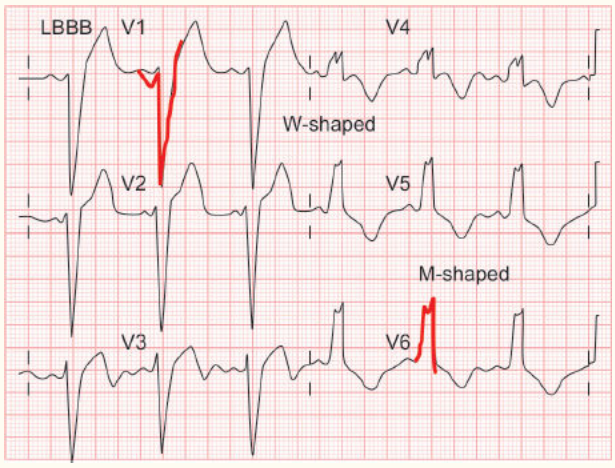
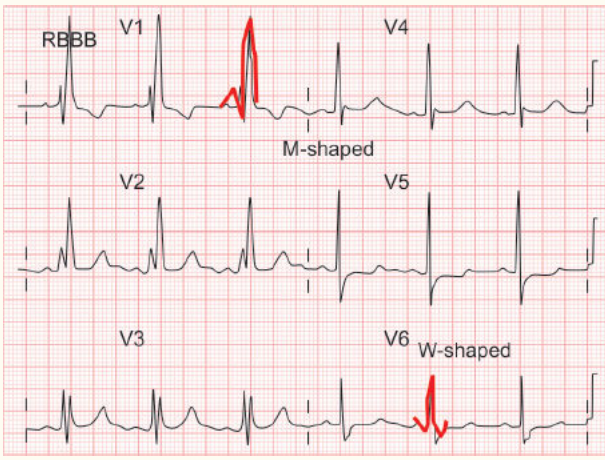
Prolonged QTc (>440 ms)—a prolonged QT can be very dangerous. It can predispose an individual to a type of ventricular tachycardia—Torsades de pointes.

- Hypokalemia
- Hypomagnesemia
- Hypocalcemia
- Hypothermia
- Myocardial ischemia
- Raised intracranial pressure

- Congenital long QT syndrome—Example: Jervell and Lange–Nielsen syndrome or Romano-Ward syndrome
- Drugs—chlorpromazine, haloperidol, quetiapine, quinidine, procainamide, disopyramide, flecainide, sotalol, amiodarone, amitriptyline, diphenhydramine, astemizole, loratadine, terfenadine, chloroquine, quinine, and macrolides.

Short QTc (<350 ms)
- Hypercalcemia
- Digoxin effect.

Bundle branch blocks:

Left bundle branch block (LBBB)—indirect activation causes left ventricle contracts later than the right ventricle.	**Right bundle branch block (RBBB)**—indirect activation causes right ventricle contracts later than the left ventricle
QS or rS complex in V1—W-shaped RsR' wave in V6—M-shaped 	Terminal R wave (rSR') in V1—M-shaped Slurred S wave in V6—W-shaped 
Mnemonic: **W**ILL**L**IA**M**	Mnemonic: **M**AR**R**O**W**
	

Step 6: Assess for Hypertrophy

Right Ventricular Hypertrophy (RVH)

Criteria of RVH
- Tall R in V1 with R >S, or R/S ratio >1
- Deep S waves in V4, V5, and V6
- Associated right axis deviation, right atrial enlargement (RAE)
- Deep T inversion in V1, V2, and V3.

Cause of RVH
- Long-standing mitral stenosis
- Pulmonary hypertension of any cause
- Ventricular septal defect (VSD) or atrial septal defect (ASD) with initial L to R shunt
- Congenital heart with RV over load

- Tricuspid regurgitation, pulmonary stenosis.

Left Ventricular Hypertrophy (LVH)

Causes of LVH

- Pressure overload—systemic hypertension and aortic stenosis
- Volume overload—AR or MR-dilated cardiomyopathy
- Ventricular septal defect—cause both right and left ventricular volume overload
- Hypertrophic cardiomyopathy.

Criteria of LVH

- High QRS voltages in limb leads:
  - Sokolow and Lyon criteria: S (V1) + R (V5 or V6) >35 mm
  - Cornell criteria: S (V3) + R (aVL) >28 mm (men) or >20 mm (women)
  - Others: R (aVL) >13 mm.
- Deep symmetric T inversion in V4, V5, and V6
- QRS duration >0.09 sec, associated left axis deviation, left atrial enlargement (LAE).

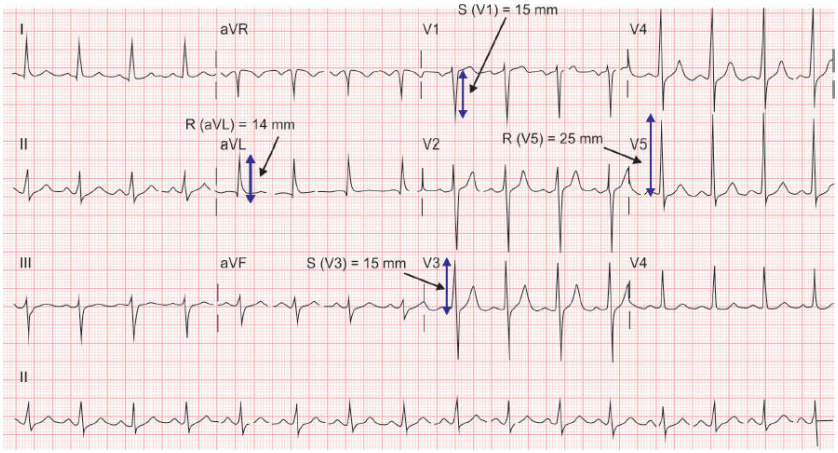


Fig. 11.7: ECG showing voltage criteria for LVH.

Romhilt–Estes Score: Score >5—definite LVH, <3 LVH unlikely

ECG criteria	Points
Voltage criteria (any of) **(Fig. 11.7)**: 1. R or S in limb leads ≥20 mm 2. S in V1 or V2 ≥30 mm 3. R in V5 or V6 ≥30 mm	3
ST-T abnormalities: • ST-T vector opposite to QRS without digitalis • ST-T vector opposite to QRS with digitalis	 3 1
Negative terminal P mode in V1, 1 mm in depth and 0.04 sec in duration (indicates left atrial enlargement)	3
Left axis deviation (QRS of –30° or more)	2
QRS duration ≥0.09 sec	1
Delayed intrinsicoid deflection in V5 or V6 (>0.05 sec)	1

TYPES OF LVH

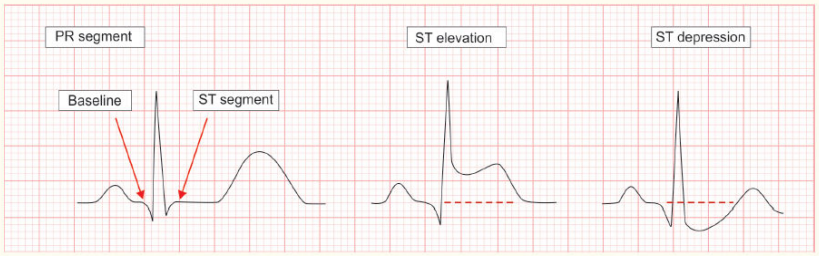
Pressure over load	Volume over load
• Like in hypertension, ischemic heart disease (IHD) • LV strain pattern—ST depression with T inversion in V5, V6, L1, and aVL leads	• Like in mitral or aortic regurgitation • Shows prominent Q waves, positive T waves in V5, V6, L1, and aVL

Step 7: Look for Evidence of Infarction/St Segment Abnormalities

ST Segment

- ST segment is isoelectric and at the same level as subsequent PR-interval
- The length between the end of the S wave (end of ventricular depolarization) and the beginning of repolarization
- From J point on the end of QRS complex, to inclination of T wave.

Causes of ST segment elevation
• Ischemia • Early repolarization • Acute pericarditis: ST elevation in all leads except aVR • Pulmonary embolism • Hypothermia • Hypertrophic cardiomyopathy • High potassium • Cerebrovascular accident • Acute sympathetic stress • Brugada syndrome • Cardiac aneurysm • Left ventricular hypertrophy • Idioventricular rhythm including paced rhythm.



Causes of ST segment depression
• Myocardial ischemia/non-ST-elevation myocardial infarction (NSTEMI) • Reciprocal change in STEMI • Posterior MI • Digoxin effect (reverse tick mark/“sagging” morphology, resembling Salvador Dali’s moustache) • Hypokalemia • Bundle branch block • Ventricular hypertrophy • Ventricular pacing.

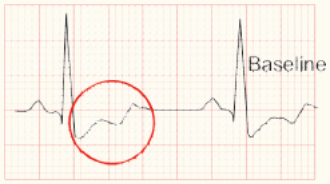
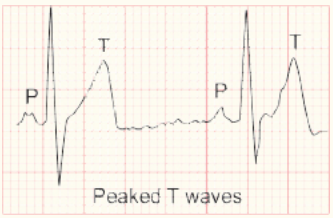
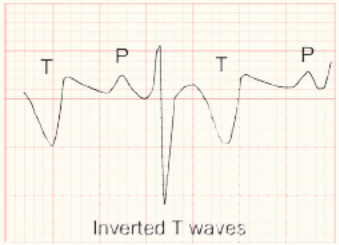
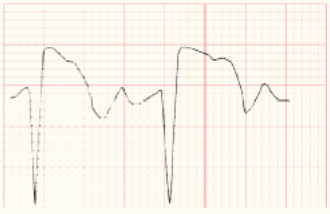
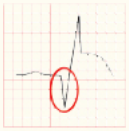
ECG CHANGES IN MYOCARDIAL INFARCTION (MI)

There are two types of MI. ST segment elevation myocardial infarction (STEMI) and non-STEMI (NSTEMI). ST elevation myocardial infarction criteria:

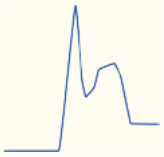
- ST elevation in >2 chest leads >2 mm elevation
- ST elevation in >2 limb leads >1 mm elevation

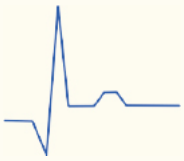
- Q wave >0.04 s (1 small square).

Location of MI	Lead with ST changes	Affected coronary artery
Anterior	V1, V2, V3, V4	Left anterior descending (LAD) artery
Septum	V1, V2	LAD
Left Lateral	I, aVL, V5, V6	Left circumflex
Inferior	II, III, aVF	Right coronary artery (RCA)
Right atrium	aVR, V1	RCA
Posterior	Posterior chest leads	RCA
Right ventricle	Right sided leads	RCA

Ischemia	Injury	Infarct
• T-wave inversion (flipped T) • ST segment depression • T wave flattening • Biphasic T waves	• ST segment elevation of greater than 1 mm in at least 2 contiguous leads • Heightened or peaked T waves • Directly related to portions of myocardium rendered electrically inactive	• Significant Q wave where none previously existed • Why? • Impulse traveling away from the positive lead • Necrotic tissue is electrically dead
 Depressed ST segment	 Peaked T waves	
 Inverted T waves	 Elevated ST segment	 Q wave

Sequential ECG changes in STEMI

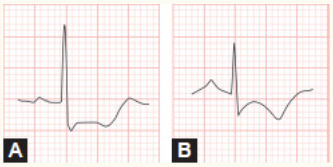
0 hour		Pronounced/hyperacute Tall T wave initially ST elevation (convex type)
1–24 hours		Depressed R wave, and pronounced T wave. Pathological Q waves may appear within hours or may take greater than 24 hours indicating full-thickness MI. Q wave is pathological if it is wider than 40 ms or deeper than a third of the height of the entire QRS complex
Days		Exaggeration of T wave continues for 24 hours

1–2		
Days later		T wave inverts as the ST elevation begins to resolve. Persistent ST elevation is rare except in the presence of a ventricular aneurysm
Weeks later		ECG returns to normal T wave, but retains pronounced Q wave

Non-ST-Elevation MI

Non-ST-elevation MI is also known as subendocardial or non-Q-wave MI.

In a PT with acute coronary syndrome (ACS) in which the ECG does not show ST elevation, NSTEMI (subendocardial MI) is suspected if

ST depression (A) T wave inversion with or without ST depression (B) Q wave and ST elevation will never happen	

A **ST depression is more suggestive of myocardial ischemia** than infarction.

ELECTROLYTES AND ECG

Hypocalcemia: Prolonged ST segment and QT intervals.

Hypercalcemia

- Shortened ST segment
- Widened T wave and short QT

Hypokalemia

- ST depression
- Shallow, flat, and inverted T wave
- Prominent U wave and P waves.

Hyperkalemia

- Tall, peaked T waves
- Flat P waves
- Widened QRS complex
- Prolonged PR interval
- Sine wave.

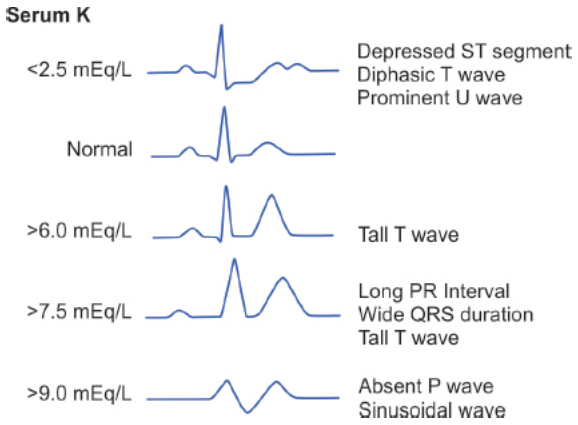


Fig. 11.8: ECG changes in seen with potassium.

Hypomagnesemia

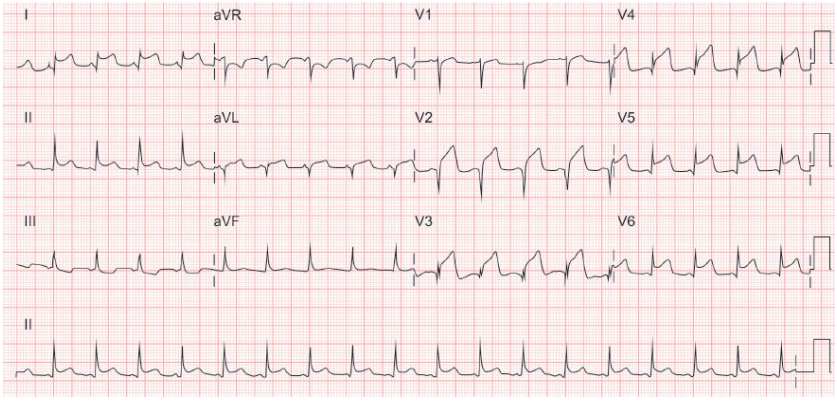
- Tall T waves
- Depressed ST segment.

Hypermagnesemia

- Prolonged PR interval
- Widened QRS complexes.

EXAMPLES

Example 1

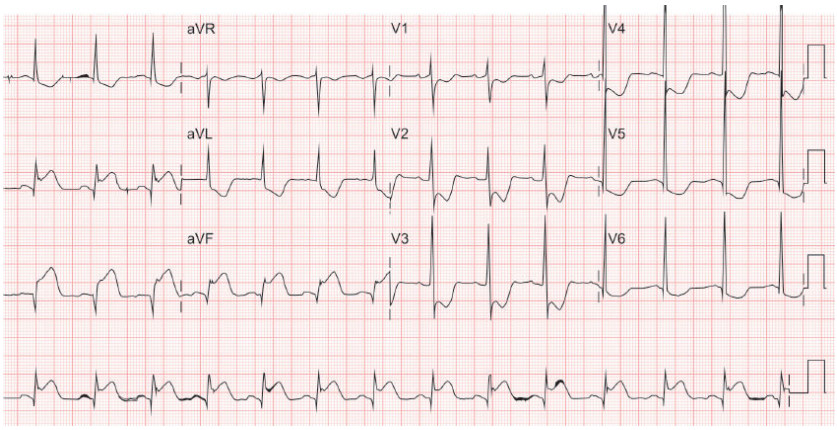


12-lead ECG showing

Rate	110 bpm
Rhythm	Sinus rhythm
Axis	Normal
P wave	Duration 0.08 sec and normal morphology
PR interval/segment	• 0.12 sec • PR segment elevation in aVR
QRS	0.08 sec

ST segment	• Elevation in V2-V6, I, aVL • Depression in aVR
T wave	Normal
QT interval	0.32 sec
Final diagnosis	Acute pericarditis

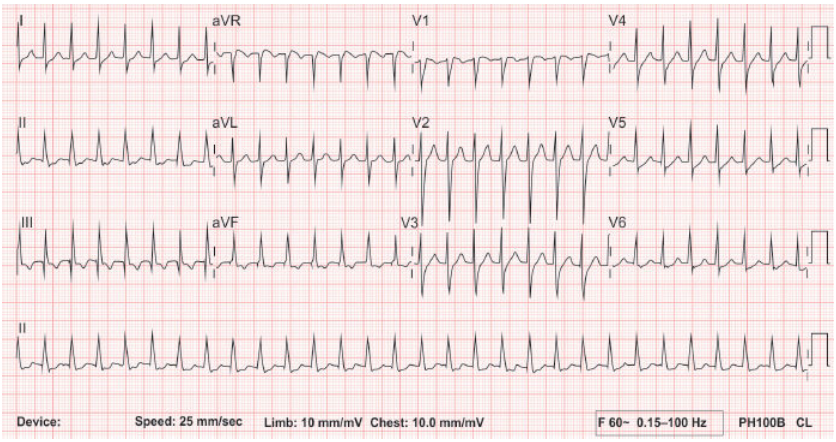
Example 2



12-lead ECG showing

Rate	85 bpm
Rhythm	Sinus
Axis	Normal
P wave	Duration 0.12 sec and normal morphology
PR interval/segment	0.16 sec
QRS	0.08 sec
ST segment	• Elevation in II, III, aVF (elevation in Lead III > II) • Depression in V1-V6, I, aVL
T wave	Corresponds to ST–T changes.
QT interval	0.36 sec
Final diagnosis	Inferior wall MI with signs of RV infarction

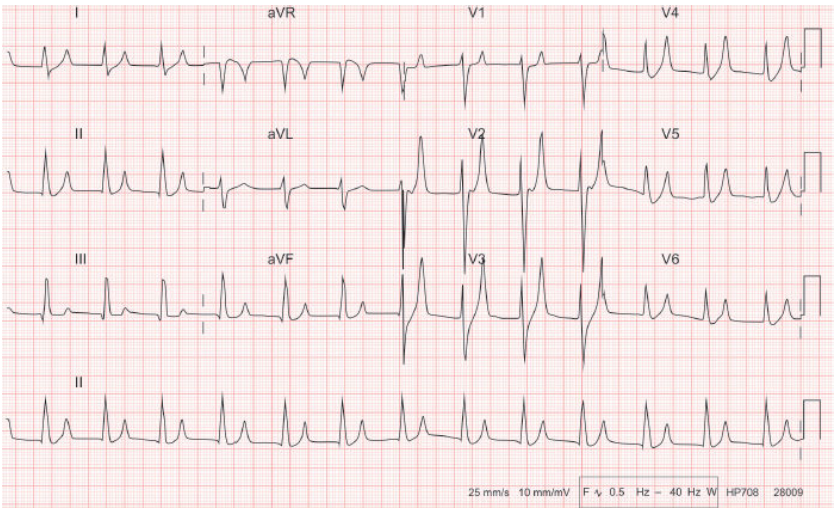
Example 3



12-lead ECG showing

Rate	200 bpm
Rhythm	Regular
Axis	Normal
P wave	Retrograde
PR interval/segment	
QRS	0.08 sec (narrow complex)
ST segment	Normal
T wave	Normal
QT interval	0.28 sec
Final diagnosis	Supraventricular tachycardia-atrioventricular nodal reentry tachycardia (SVT-AVNRT)

Example 4

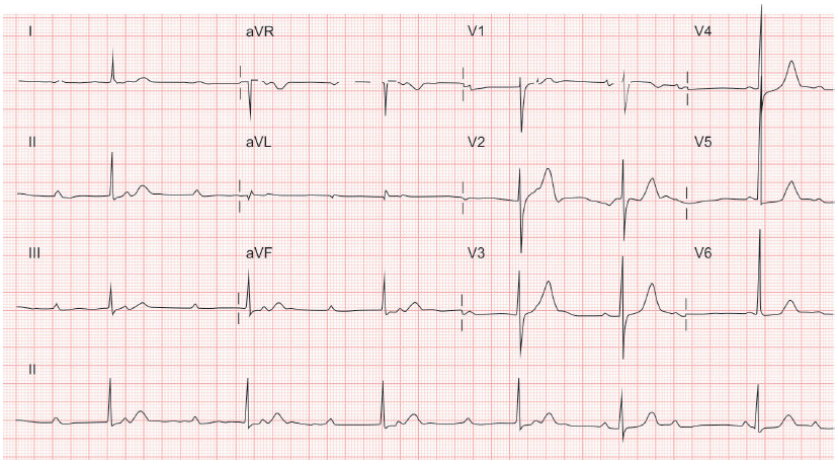


12-lead ECG showing

Rate	75 bpm

Rhythm	Junctional
Axis	Normal
P wave	Absent
PR interval/segment	–
QRS	0.14 sec notching at J point (V2)
ST segment	• Minimal elevation in V3-V5 • No reciprocal changes
T wave	Tall T steeple waves in precordial leads, concordant with QRS
QT interval	0.36 sec
Final diagnosis	Hyperkalemia

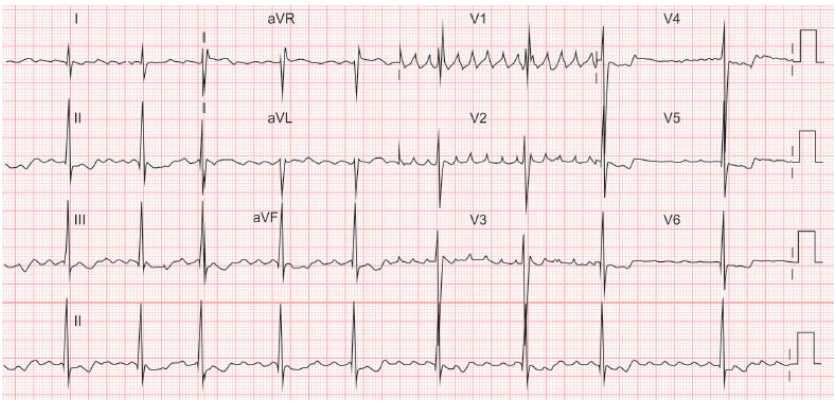
Example 5



12-lead ECG showing

Rate	Atrial—80 bpm; Ventricular—50 bpm
Rhythm	Junctional escape
Axis	Normal
P wave	Present
PR interval/segment	–
QRS	0.08 sec independent of P waves
ST segment	Normal
T wave	Normal
QT interval	0.36 sec
Final diagnosis	Complete heart block

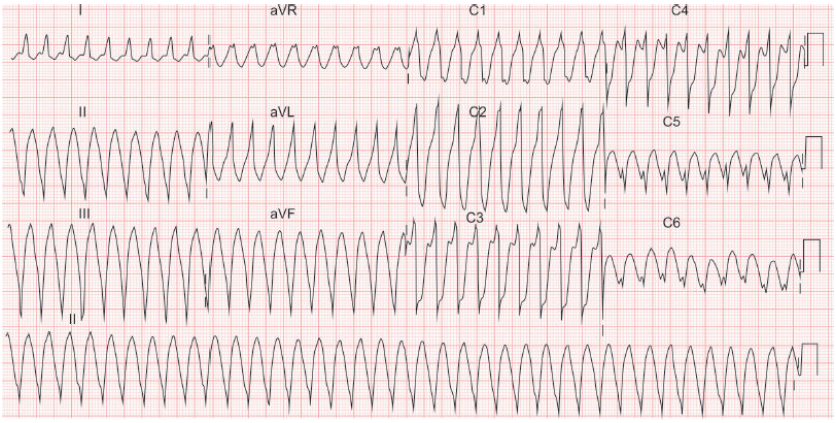
Example 6



12-lead ECG showing

Rate	70 bpm (6 sec rule)
Rhythm	Irregular
Axis	Normal
P wave	Absent, presence of fibrillary waves
PR interval/segment	–
QRS	0.08 sec varying RR interval
ST segment	Normal
T wave	Normal
QT interval	0.32 sec
Final diagnosis	Atrial fibrillation

Example 7



12-lead ECG showing

Rate	250 bpm
Rhythm	Regular
Axis	Left-northwest
P wave	AV dissociation

PR interval/segment	–
QRS	0.28 sec (Broad complex) Positive concordance
ST segment	–
T wave	-
QT interval	-
Final diagnosis	Monomorphic ventricular tachycardia (VT)

A Systematic Approach to Chest X-rays

CHAPTER 12

RADIOLOGY

We shall discuss practical aspects of radiology under following sections:

1. Approach to chest X-rays
2. Approach to CT scans
3. Approach to MRI scans
4. Contrast agents

APPROACH TO CHEST X-RAYS

Reading into the Chest Radiograph

The 10 Step Program

1. What type of view
2. Exposure/penetration
3. Inspiratory versus expiratory film
4. Rotation
5. Angulation
6. Soft tissues and bony structures
7. Trachea
8. Hilum/mediastinum
9. Diaphragm
10. Lung fields
11. Cardia

Type of View

Chest X-ray

1. PA view
2. AP view
3. Lateral view

1. **PA view (posteroanterior view) (Fig. 12.1)**
   The ray of beam is from posteroanteriorly with the film in front of the patient.
2. **AP view (anteroposterior view) (Fig. 12.2)**
   The ray of beam is from anteroposteriorly with the film behind the patient.

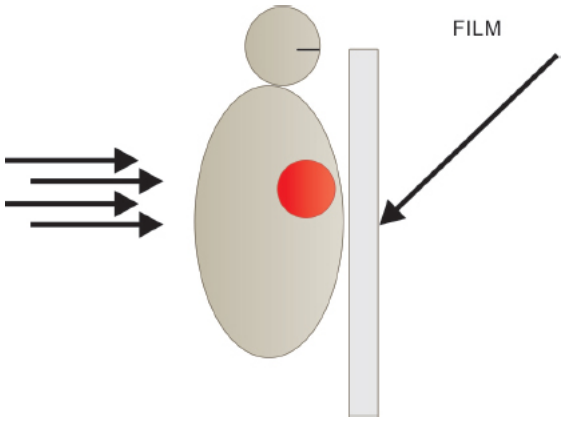


Fig. 12.1: Posteroanterior view.

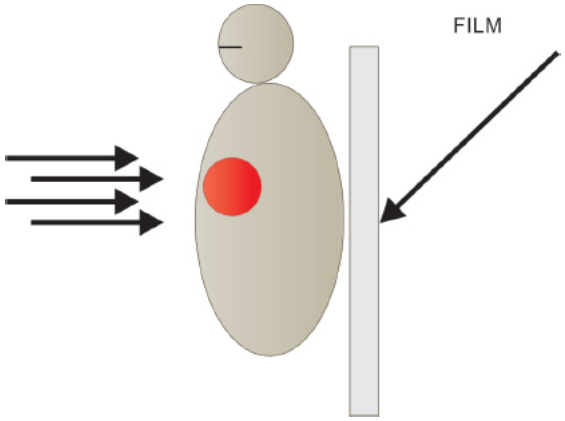


Fig. 12.2: Anteroposterior view.

3. **Lateral view (Fig. 12.3)**
   The ray of beam is from one side with the film placed on the opposite side of the patient.

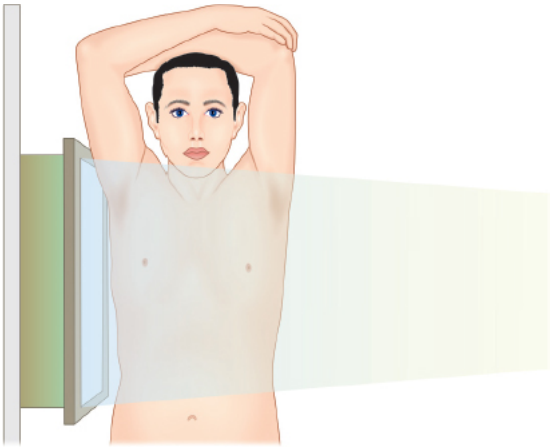


Fig. 12.3: Lateral view.

Differences between PA view and AP view of chest X-ray

	PA view (Fig. 12.4)	AP view (Fig. 12.5)
Fundic gas shadow	Usually present	Absent
Clavicles	Seen over the lung fields and more horizontal	Seen above the apex of lung field and more oblique
Scapula	Inner borders are away form the lung fields	Inner borders are seen over the lung fields
Ribs	Posterior ribs are better seen and more oblique	Anterior ribs are better seen
Apparent cardiomegaly	Not seen	Seen
Spine	Better seen	Not seen
The distance between the projector and the patient	6 feet	40 inches

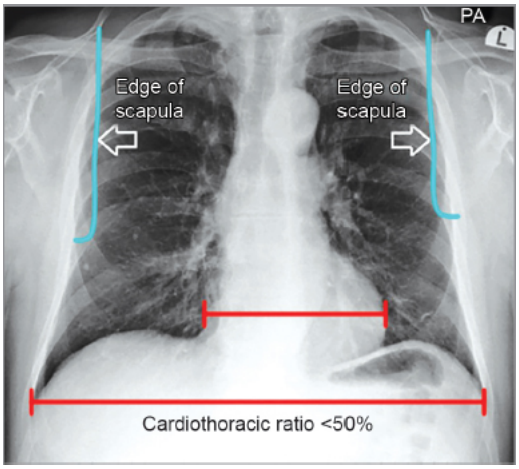


Fig. 12.4: PA view.

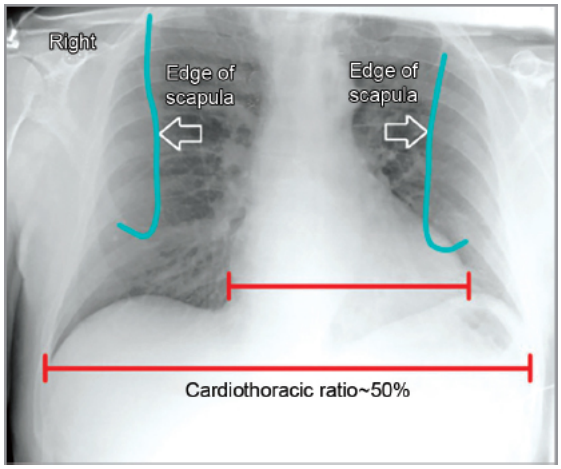


Fig. 12.5: AP view.

Exposure/Penetration

Penetration is the degree to which X-ray passes through the body. **Figure 12.6** depicts the grading of shadow in X-ray film.

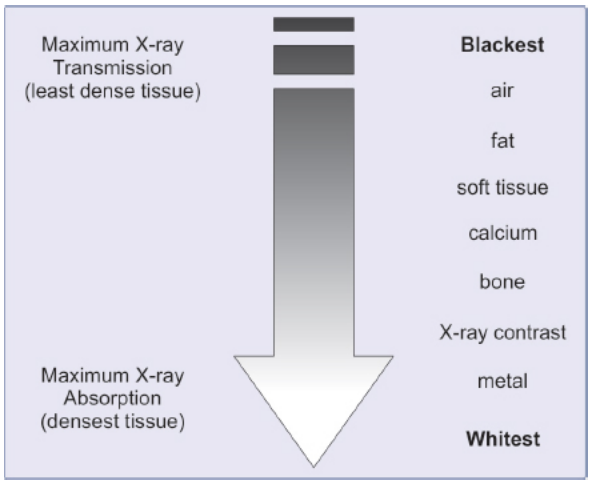


Fig. 12.6: Black and white areas in X-ray.

Criteria of well-penetrated chest X-ray:

- A well-penetrated X-ray is one where the thoracic vertebrae are just visible through the heart shadow, but bony details of spine are not usually seen.

Overpenetrated radiograph (Fig. 12.7)

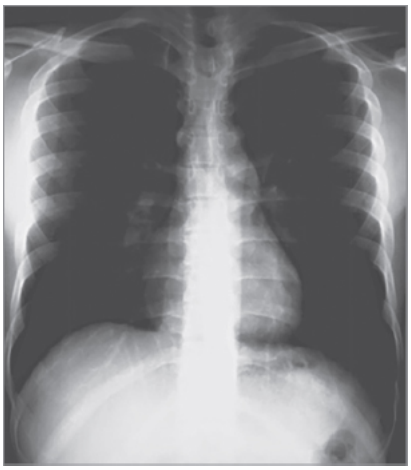


Fig. 12.7: Overpenetrated radiograph.

- In this radiograph, all thoracic vertebrae visible through the heart shadow.
- Lung field darker than normal; may obscure subtle pathologies.
- Inadequate lung detail.

Underpenetrated radiograph (Fig. 12.8)

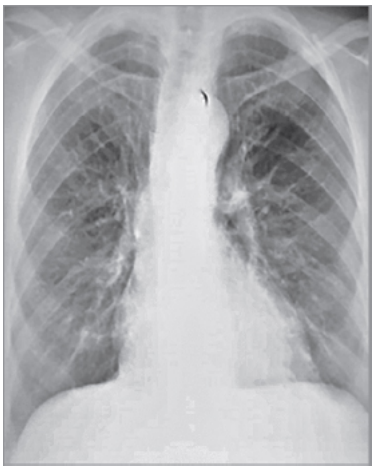


Fig. 12.8: Underpenetrated radiograph.

- In underpenetrated radiograph you will not able to see thoracic vertebrae through the heart shadow.
- Lung tissue behind the heart cannot be assessed.
- Hemidiaphragm is obscured.

Inspiratory versus Expiratory film

Inspiratory film (Fig. 12.9)	**Expiratory film (Fig. 12.10)**

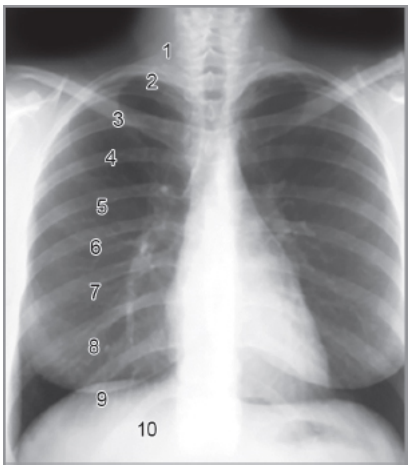


Fig. 12.9: Inspiratory film.

- Should be able to count 9–10 posterior ribs.
- Heart shadow should not be hidden by the diaphragm.

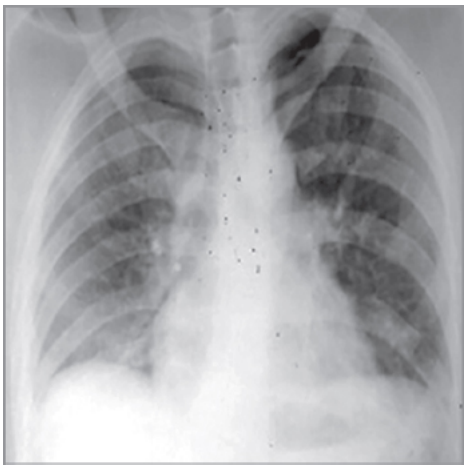


Fig. 12.10: Expiratory film.

- Poor inspiration can crowd lung markings producing pseudo-airspace disease.
- Expiration reduces lung volume, making a small pneumothorax easier to see.

Rotation

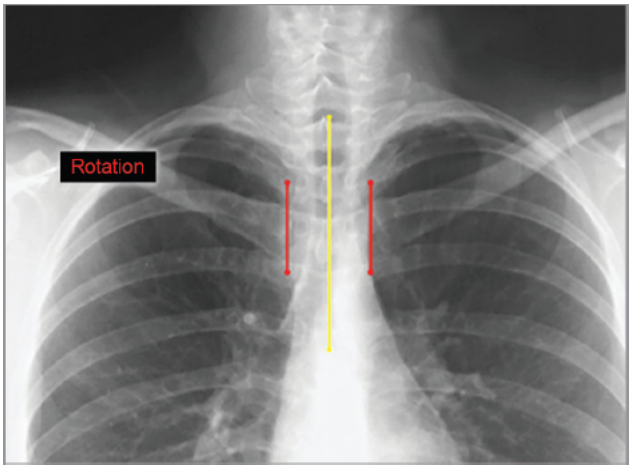


Fig. 12.11: Normal rotation.

- **Normal rotation (Fig. 12.11):** Medial ends of bilateral clavicles are equidistant from the midline or vertebral bodies.

Left-rotated film (Fig. 12.12)

Right-rotated film (Fig. 12.13)

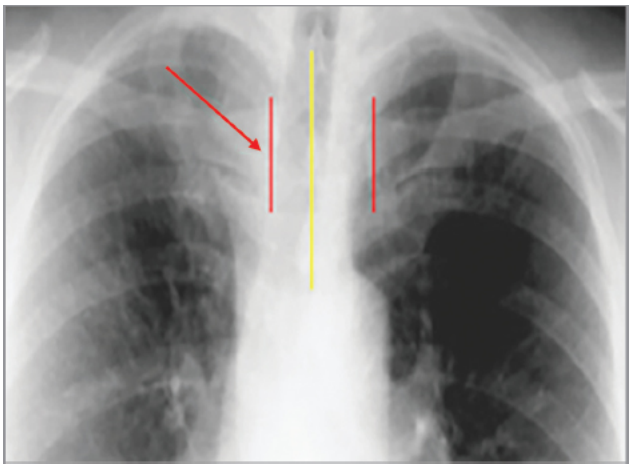


Fig. 12.12: Left-rotated film.

If spinous process appears closer to the right clavicle (red arrow), the patient is rotated toward their own left side.

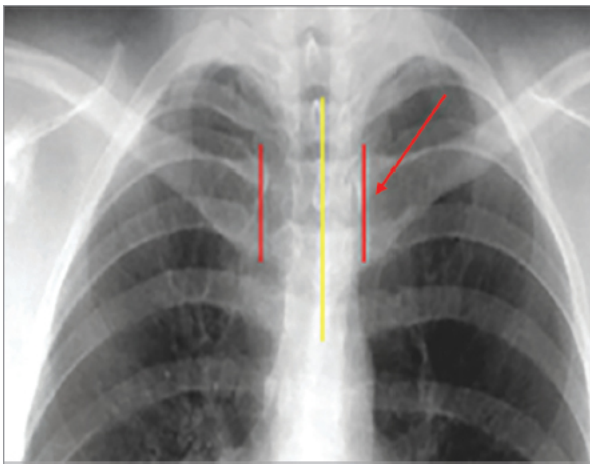


Fig. 12.13: Right-rotated film.

If spinous process appears closer to the left clavicle (red arrow), patient is rotated toward their own right side.

Angulation

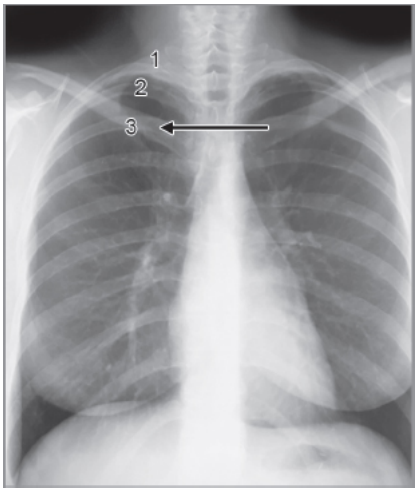


Fig. 12.14: Normal angulation.

Normal angulation (Fig. 12.14): Clavicle should lie over the 3rd rib (posterior end). With proper angulation the apex of lungs are clearly visualized.

Soft tissues and Bony Structures

Soft Tissues (Fig. 12.15)

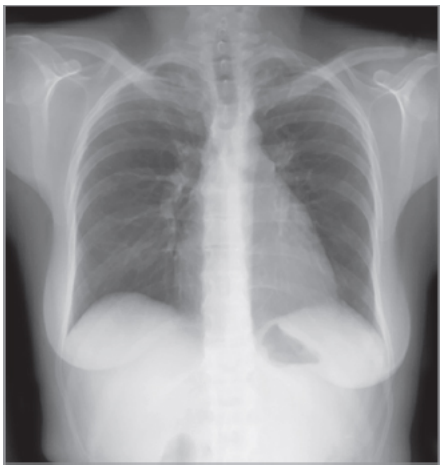


Fig. 12.15: Soft tissues.

Soft Tissues
- Breast shadows
- Supraclavicular areas
- Axillae
- Tissues along the side of breasts

Bony structures (Fig. 12.16)

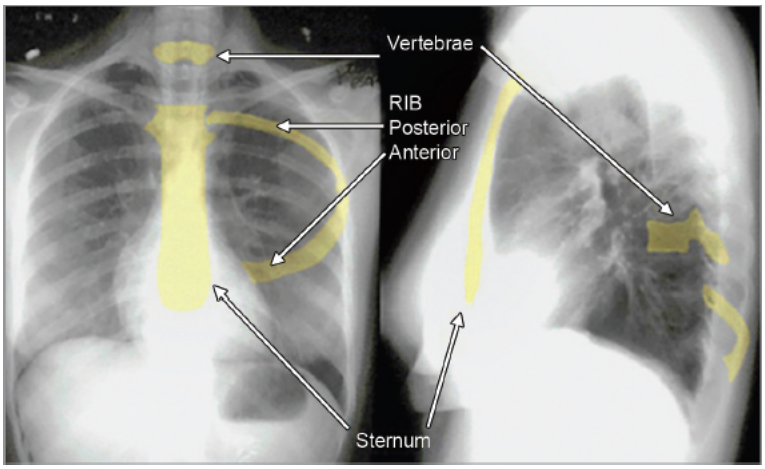


Fig. 12.16: Bony structures.

Bony Structures
- Ribs
- Sternum
- Spine
- Shoulder girdle
- Clavicles

Trachea (Figs. 12.17 A and B)

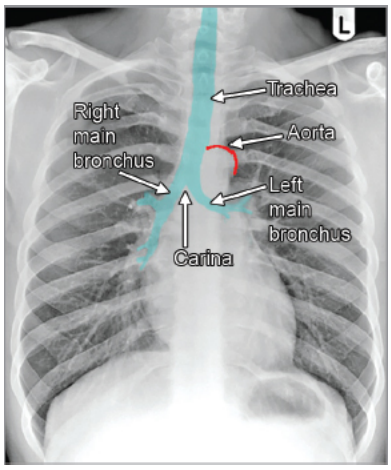


Fig. 12.17A: Trachea (PA view).

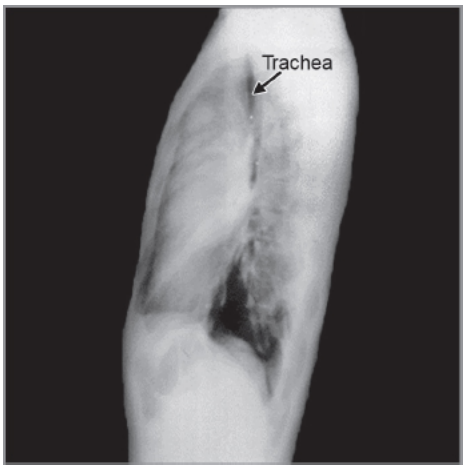


Fig. 12.17B: Trachea (lateral view).

Hilum/mediastinum (Fig. 12.18)

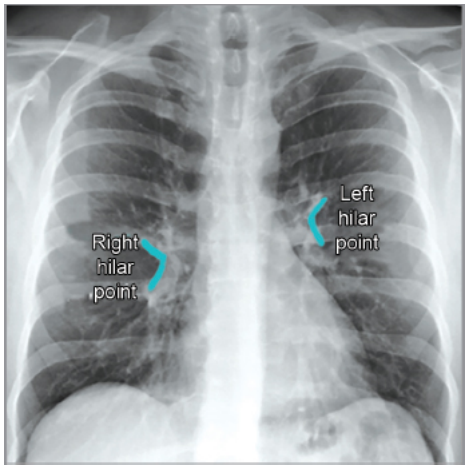


Fig. 12.18: Hilum.

Hilum is the wedge-shaped area on the central portion of each lung where the following structures leave the lung.

- Bronchi
- Pulmonary—arteries, veins and nerves.

Important point:

- Left hilar point is usually higher than right.

Diaphragm (Fig. 12.19)

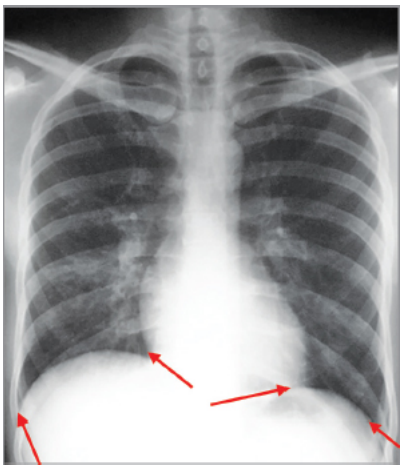


Fig. 12.19: Diaphragm.

Diaphragm
Dome-shaped
- Position:
  - Right hemidiaphragm is located at 9th–10th rib posteriorly or 6th rib anteriorly
  - Right hemidiaphragm is higher than the left by 2 cm because the cardia keeps the left hemidiaphragm down
- Costophrenic angles
- Cardiophrenic angles
- Normally the costophrenic and cardiophrenic angles are clear, they are obliterated due to fluid, fat or fibrosis
- Height—normally 2.5 cm

When do you say diaphragm is flattened (Figs. 12.20A and B)?

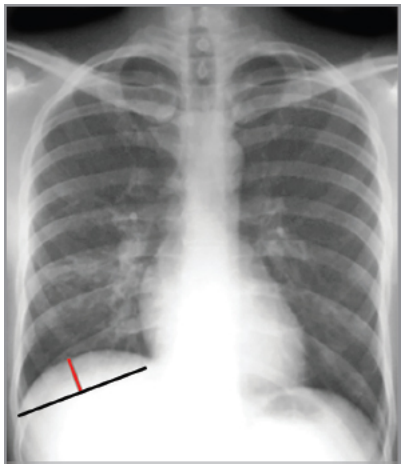


Fig. 12.20A: Normal height of diaphragm.

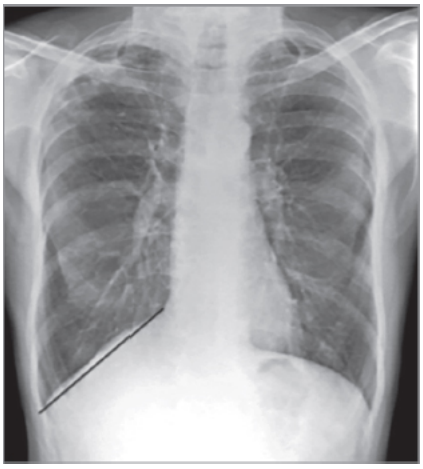


Fig. 12.20B: Flattening of diaphragm.

Draw a line from cardiophrenic angle to costophrenic angle. Now draw a perpendicular onto the line from the highest point of dome of diaphragm. Measure the height of the perpendicular (red line). If the height is <2.5 cm it suggest flattened diaphragm.

Lung Fields

Lung fields and hilum

- Hilum

  - Pulmonary arteries
  - Pulmonary veins
- Lungs
  - Linear and fine nodular shadows of pulmonary vessels
- Blood vessels
- 40% obscured by other tissue

Segments of the Lung	
Right lung	***Left lung***
Superior lobe: Apical, posterior, and anterior **Middle lobe:** Lateral and medial **Inferior lobe:** Superior (apical), medial basal, anterior basal, lateral basal, and posterior basal **Total: 10 segments on right.**	**Superior lobe:** Apicoposterior, anterior, superior lingular, and inferior lingular **Inferior lobe:** Superior (apical), anterior basal, lateral basal, and posterior basal **Total: 8 segments on left side.**

Zones of Lung (Fig. 12.21)

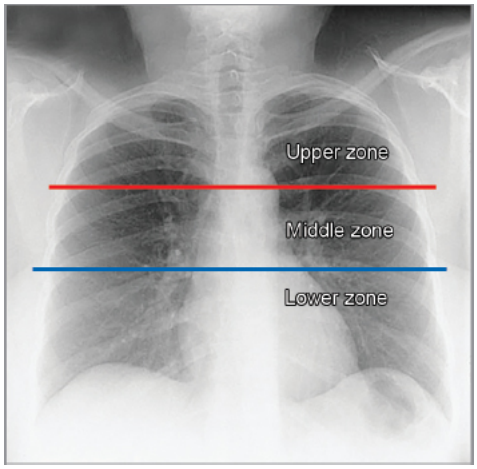


Fig. 12.21: Two lines are drawn one connecting the anteroinferior end of 2nd rib on both sides and 2nd connecting the anteroinferior ends of the 4th rib on both sides.

Note: Zones do not correspond to lobes.

Silhouette sign (Fig. 12.22)

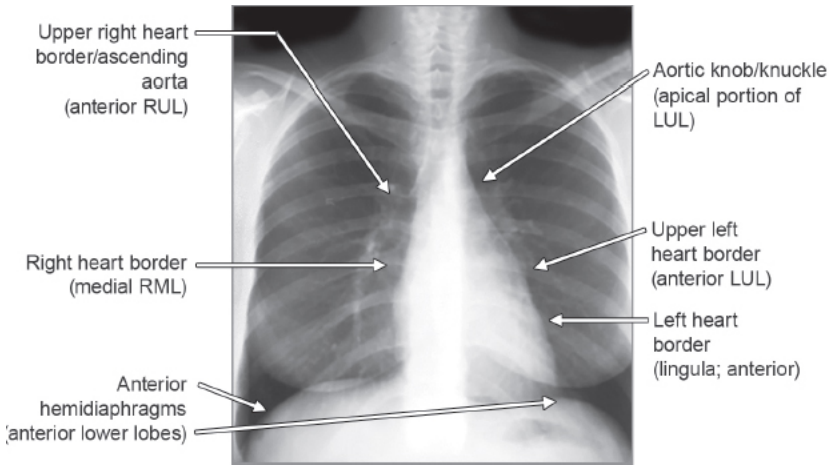


Fig. 12.22: Silhouette sign.

Silhouette sign: It actually denotes the loss of a silhouette; thus, it is sometimes also known as loss of silhouette sign or loss of outline sign.

Felson defined it as "An intrathoracic lesion touching a border of the heart, aorta, or diaphragm will obliterate that border on the roentgenogram. An intrathoracic lesion not anatomically contiguous with a border of one of these structures will not obliterate that border".

Loss of the anatomic border is described as a positive silhouette sign.

Recognition of this sign is useful in localizing areas of consolidation, atelectasis or mass within the lung, with the loss of these normal silhouettes on a PA chest X-ray.

- Right paratracheal stripe: Right upper lobe
- Right heart border: Right middle lobe or medial right lower lobe
- Right hemidiaphragm: Right lower lobe
- Aortic knuckle: Left upper lobe
- Left heart border: Lingular segments of the left upper lobe
- Left hemidiaphragm or descending aorta: Left lower lobe.

Cardia (Fig. 12.23)

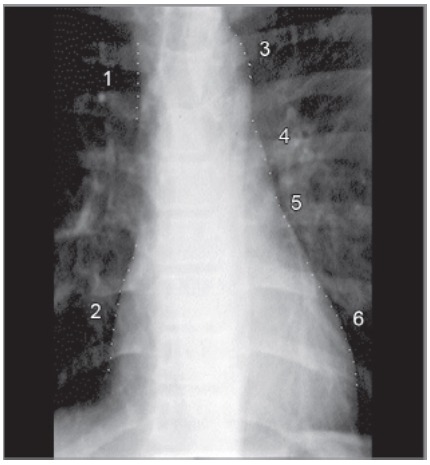


Fig. 12.23: Cardia. 1: Edge of superior vena cava; 2: Right atrium; 3: Aortic arch; 4: Edge of main pulmonary artery; 5: Left atrial appendage; 6: Left ventricle.

Cardiomegaly (Fig. 12.24)

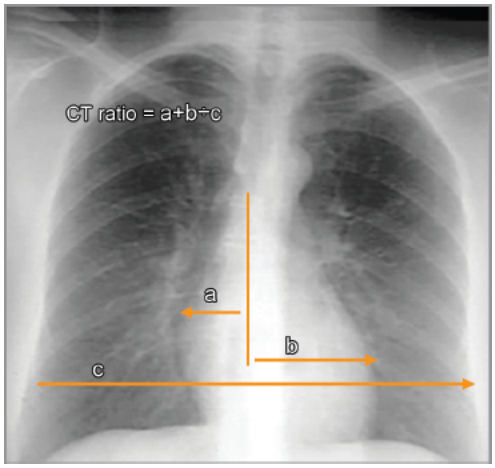


Fig. 12.24: Cardiomegaly.

The cardiothoracic ratio (CTR) is obtained by dividing the transverse cardiac diameter [sum of the horizontal distances from the right and left lateral-most margins of the heart to the midline (spinous processes of the vertebral bodies)] by the maximum internal thoracic diameter.

Cardiomegaly (Fig. 12.24):

- Adults: >0.50

	• Neonates and elderly: >0.60 **Chicken heart:** Cardiothoracic ratio less than 25%. Small sized heart. Causes are: • Bilateral emphysema • Anorexia nervosa • Addison's disease

Silhouette		Apex (left cardiophrenic angle)	
Clear	Not clear	Acute (RV contour)	Obtuse (LV contour)
Intrinsic cardiac disease (valvular/muscle)	Extrinsic problem (pericardial effusion)	Mitral stenosis Atrial septal defect Chronic obstructine pulmonary disease	Mitral regurgitation Aortic stenosis Aortic regurgitation Hypertension Cardiomyopathy

Differential diagnosis for gross cardiomegaly (wall-to-wall heart)

1. Pericardial effusion
2. Multivalvular heart disease
3. Severe aortic regurgitation (cor bovinum)
4. Ebstein's anomaly
5. Dilated cardiomyopathy

Chamber/vessel enlargement	Condition seen
Left atrial enlargement	• Enlarged left atrial appendage causes filling up of normal concavity between pulmonary artery shadow and the left ventricle. • **Double atrial shadow:** Border of enlarged left atrium together with right atrial border gives an appearance like atrium within an atrium. • **Straightening of left heart border:** Mitralization of heart. • Pushing of left main bronchus upwards causing wide carinal angle (**splaying of carina**). • Pushing esophagus backwards visible in lateral view of chest X-ray. • Left shift of aorta (**Bedford sign**). • **walking man** sign in lateral xray.
Pulmonary venous/capillary hypertension	• **Grade 1:** Cephalization (prominence of veins of upper lobe of lung) of pulmonary vasculature (pulmonary venous pressure ≤20 mm Hg) (reverse moustache sign or Stag's antler sign). • **Grade 2:** Kerley's lines (A, B, C) (pulmonary venous pressure 20–25 mm Hg), peribronchial, perivascular cuffing. – **Kerley A line:** Linear opacities extending from the periphery to hilum; they are caused by distension of anastomotic channels between periphery and central lymphatic's. – **Kerley B line:** Short horizontal lines situated perpendicularly to the pleural surface at the lung base; they represent edema of interlobar septa. – **Kerley c line:** Reticular opacities at lung base, representing Kerley's B line. • **Grade 3:** Batwing opacities (pulmonary venous pressure >25 mm Hg).
Pulmonary arterial hypertension	Prominent pulmonary outflow tract: enlarged pulmonary arteries (diameter of right descending pulmonary artery >14 mm in women and >16 mm in men) + pruning of peripheral pulmonary vessels.
Right ventricle	• Apex forms an acute angle with diaphragm • **Right ventricular hypertrophy:** In presence of cardiomegaly, acute angle is observed between apex of enlarged heart and diaphragm. • **Sternal contact sign:** Earliest and most sensitive sign in the lateral X-ray is obliteration of Holtzneck's space, i.e. retrosternal space.
Right atrial enlargement	• Right border >5.5 cm from midline or 3.5 cm from sternal border. • 2½ intercostal space in its vertical extent. • >50% vertical height compared with mediastinal height.
Left ventricular enlargement	• Left ventricular enlargement results in cardiomegaly with obtuse left cardiophrenic angle.

Differential diagnosis of consolidation

Based on the chronicity		
Acute	***Chronic***	
Pneumonia Aspiration Edema	Organizing pneumonia Malignancy Alveolar proteinosis Sarcoidosis Eosinophilic pneumonia	
Based on the content		
Water filled	***Pus filled***	***Blood filled***
Heart failure ARDS Renal failure	Pneumonia	Trauma Vasculitis (good pasture disease, HSP, SLE)
Based on the pattern of involvement		
Diffuse disease	Pulmonary edema ARDS Bronchopneumonia Diffuse alveolar hemorrhage Malignancy Organizing pneumonia Hypersensitive pneumonitis	
Lobar disease	Lobar pneumonia Infarction Contusion/hemorrhage Lymphomas	
Multiple ill defined	Bronchopneumonia Septic emboli	
	Metastasis Lymphomas Wegener's granulomatosis	
Bat wing appearance	Pulmonary edema Pneumocystis carinii pneumonia	
Reverse bat wing appearance	Bronchoalveolar carcinoma Radiation induced BOOP Eosinophilic pneumonia	

Differential diagnosis of atelectasis

Resorption atelectasis	Relaxation atelectasis
Mucus plug Tumor block Foreign body obstruction	Pleural effusion Pneumothorax

Differential diagnosis of Nodule-Mass

Solitary		Multiple
Nodule <3 cm	***Mass >3 cm***	
Granulomas Lung carcinoma	Lung carcinoma Metastatic lesions	Infections (TB/septic emboli/histoplasmosis) Metastasis

Metastatic lesions Hamartomas	Hamartomas	Sarcoidosis Wegener's granulomatosis Rheumatoid nodules

Differential diagnosis of interstitial disease

Based on the pattern

Reticular			Nodular		
Smooth septal	***Irregular septal***	***Honeycombing***	***Perilymphatic***	***Centrilobular***	***Random***
Pulmonary edema Lymphangitis carcinomatosis	Fibrosis Lymphangitis carcinomatosis	UIP Hypersensitive pneumonitis Sarcoidosis	Sarcoidosis Silicosis Pneumoconiosis Lymphangitis carcinomatosis	Endobronchial infection Pulmonary edema Tuberculosis and MAC infections	Miliary TB Metastases Fungal infection

Based on the attenuation

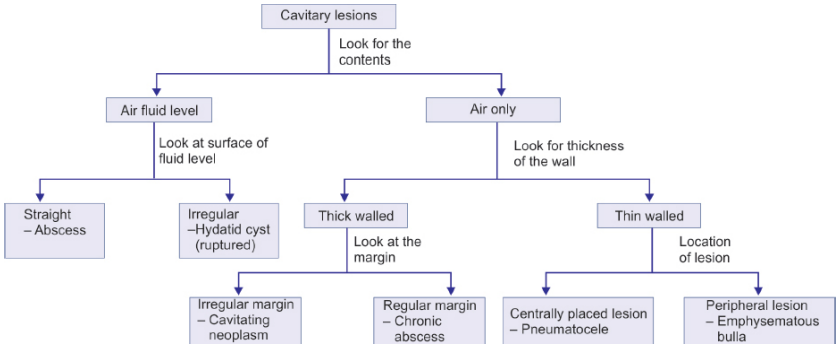
Low attenuation		High attenuation (ground glass appearance)	
Emphysema	***Cystic disease***	***Acute***	***Chronic***
Centrilobular Paraseptal Panlobular	Langerhans cell histiocytosis Pneumatoceles Lymphangioleiomyomatosis (LAM) Lymphocytic interstitial pneumonia (LIP)	Pulmonary edema Pulmonary hemorrhage Pneumocystis pneumonia	Fibrosis Alveolar proteinosis

Differential diagnosis of pleural Opacities

Solitary	Multiple
Loculated pleural effusion Loculated empyema Malignancy	Pleural plaques (asbestosis) Loculated pockets of effusions Sarcoidosis Silicosis Metastasis

Differential diagnosis of cavitary lesions (Flowchart 12.1)

Flowchart 12.1: Diagnosis of cavity lesions.



Differential diagnosis of mediastinal masses (Fig. 12.25)

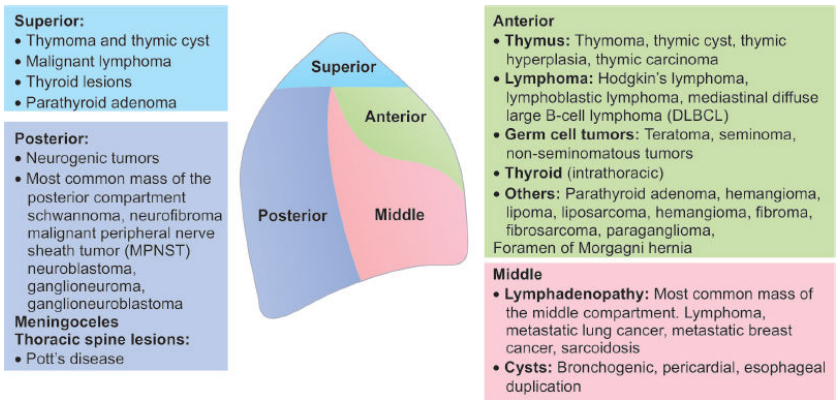
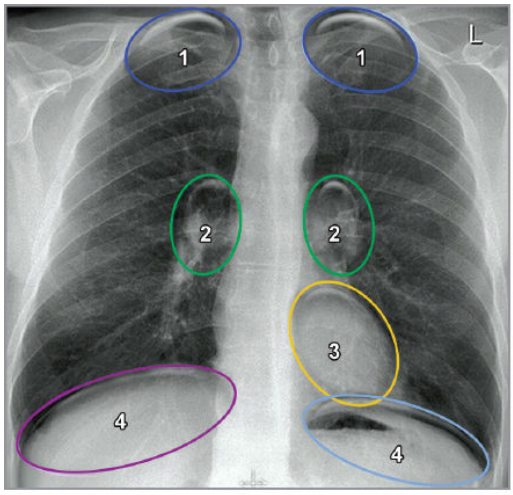


Fig. 12.25: differential diagnosis of mediastinal masses.

Differential diagnosis of hilar mass

Unilateral	Bilateral
Infections Tumors Vascular aneurysm	Sarcoidosis Silicosis Lymphomas Pulmonary artery hypertension

Hidden areas of lung (Fig. 12.26)



1. Apical zones
2. Hilar zones
3. Retrocardial zone
4. Zone below the dome of diaphragm

Fig. 12.26: Hidden areas of lung.

Discussion of Common X-rays (Figs. 12.27 to 12.59)

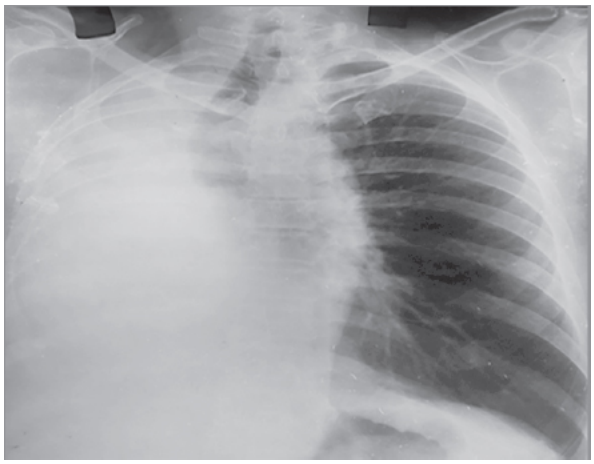


Fig. 12.27: Chest X-ray PA view showing homogeneous opacity on the right hemithorax with trachea shifted to same side suggestive of **right-sided collapse/pneumonectomy**.

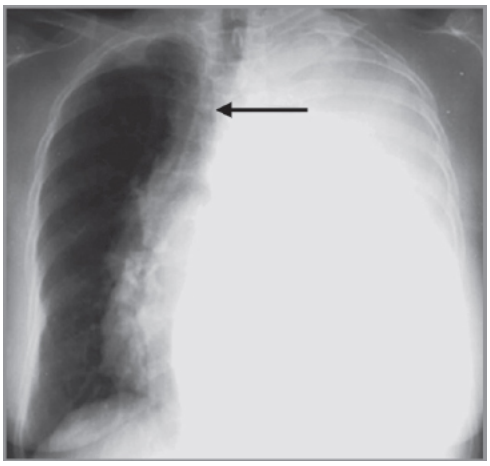


Fig. 12.28: Chest X-ray PA view showing homogeneous opacity on the left hemithorax with trachea shifted to opposite side suggestive of **left-sided massive pleural effusion (arrow)**.

Causes of hemithorax white homogeneous opacity/white-out lung:

a. With no mediastinal shift
   1. Consolidation
   2. Mesothelioma
   3. Fibrothorax
b. With mediastinal shift to opposite side
   1. Pleural effusion (moderate to large)
   2. Diaphragmatic hernia
c. With mediastinal shift to same side
   1. Collapse
   2. Postpneumonectomy

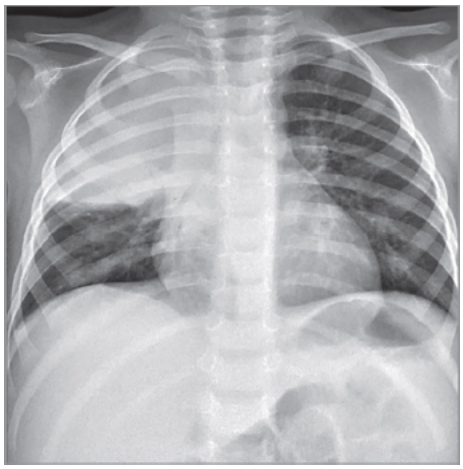


Fig. 12.29: Chest X-ray PA view showing trachea central, cardiophrenic and costophrenic angles are normal, homogeneous opacity in right upper zone with air bronchogram suggestive of **right upper lobe pneumonia**.

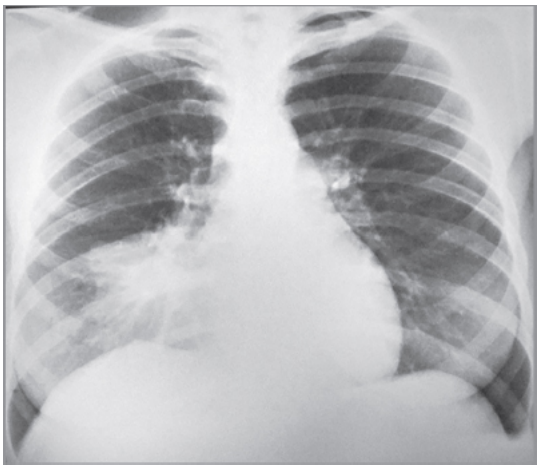


Fig. 12.30: Chest X-ray PA view showing trachea central, cardiophrenic and costophrenic angles are normal, homogeneous opacity in right mid and lower zone with air bronchogram, right heart border is not clear (silhouette sign) suggestive of **right middle lobe pneumonia**.

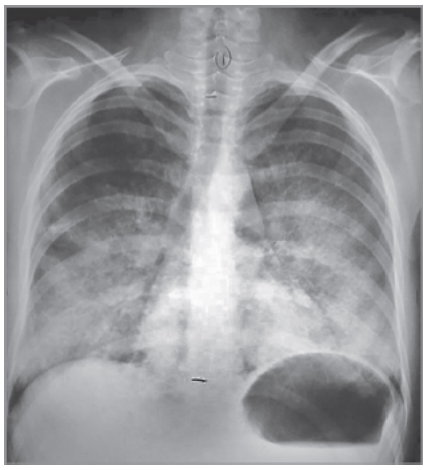


Fig. 12.31: Chest X-ray PA view showing trachea central, cardiophrenic and costophrenic angles are normal, non-homogeneous opacity in bilateral mid and lower zones with air bronchogram suggestive of **bilateral/atypical pneumonia**.

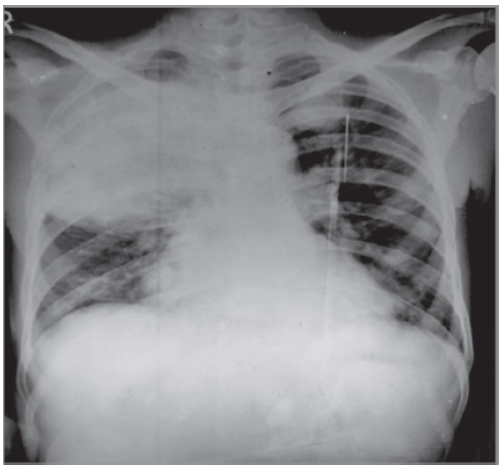


Fig. 12.32: Chest X-ray PA view showing trachea central, cardiophrenic and costophrenic angles are normal, non-homogeneous opacity in right upper zone with air bronchogram and bulging horizontal fissure suggestive of **right upper lobe pneumonia** due to *Klebsiella*.

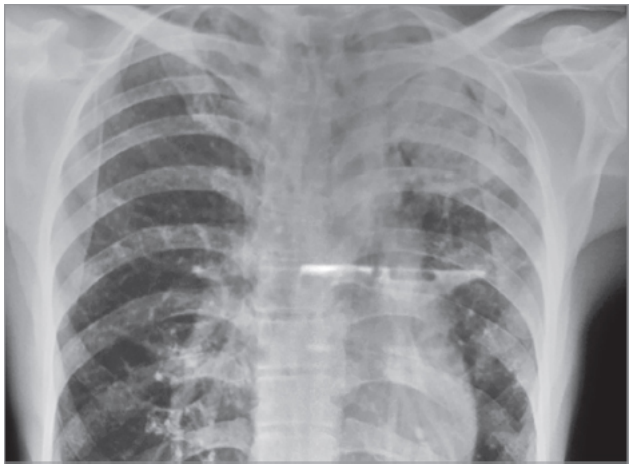


Fig. 12.33: Chest X-ray PA view showing trachea central, cardiophrenic and costophrenic angles are normal, nonhomogeneous opacity in left upper zone with cavity with air crescent sign suggestive of **aspergilloma—crescent sign of Monad**.

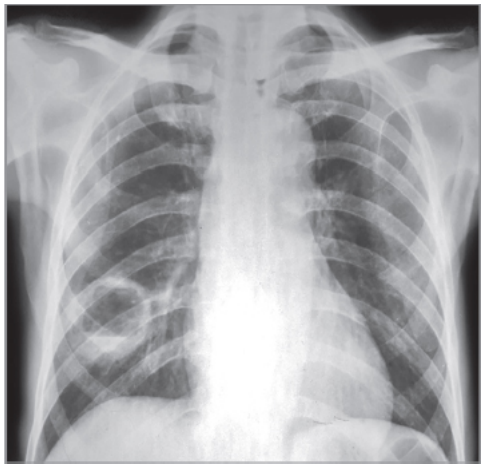


Fig. 12.34: Chest X-ray PA view showing trachea central, cardiophrenic and costophrenic angles are normal, thick walled cavity with air fluid level suggestive of **lung abscess**.

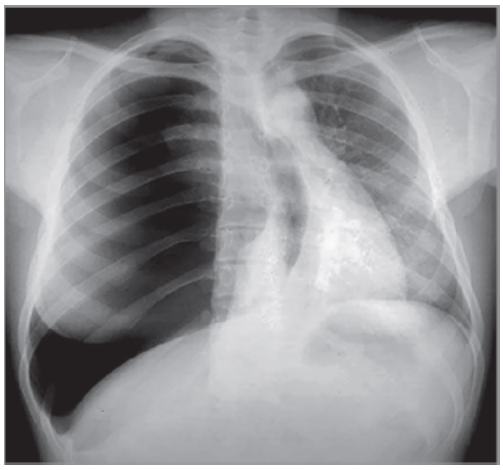


Fig. 12.35: Chest X-ray PA view showing trachea and mediastinum deviated to left, cardiophrenic and costophrenic angles are normal, homogenous hyperlucency in right hemithorax suggestive of **right-sided pneumothorax.**

Causes of unilateral hypertranslucency on chest x-ray

- **Technical**
  - Patient rotation
  - Incorrect centering of X-ray beam to grid
- Chest wall abnormality
  - Asymmetric soft tissues
  - **Mastectomy**
  - Absent or underdeveloped pectoral muscles **(Poland syndrome**)
- **Skeletal abnormality**
  - Scoliosis
- **Airway disease**
  - Large pneumothorax
  - Asymmetric emphysema
  - Bronchial obstruction
  - Previous bronchiolitis obliterans (**Swyer–James syndrome = Macleods syndrome)**
- **Vascular disease**
  - Pulmonary embolism

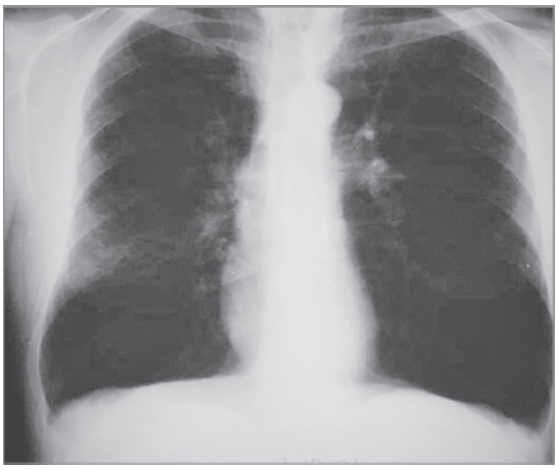
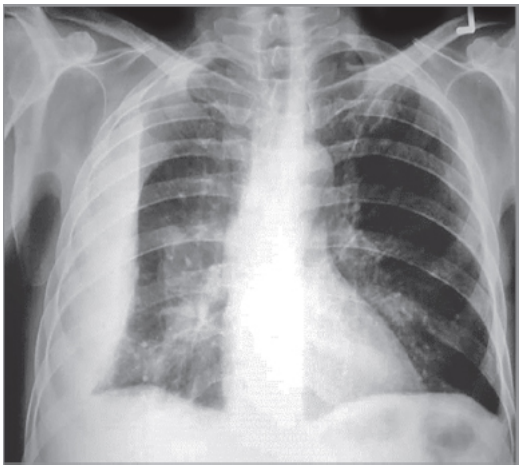


Fig. 12.36: Chest X-ray PA view showing trachea central, cardiophrenic and costophrenic angles are normal, bilateral hyperlucent lung fields with hyperinlation, flattened diaphragm and tubular heart suggestive of **bilateral emphysema**.

Causes of bilateral hyperlucent lung fields

- **Pulmonary emphysema**
- **Pulmonary overinflation**
- **Bilateral pneumothorax**
- Over exposure
- Bilateral congenital lobar emphysema
- Chronic bronchitis
- Cystic fibrosis
- Bronchiectasis
- Asthma



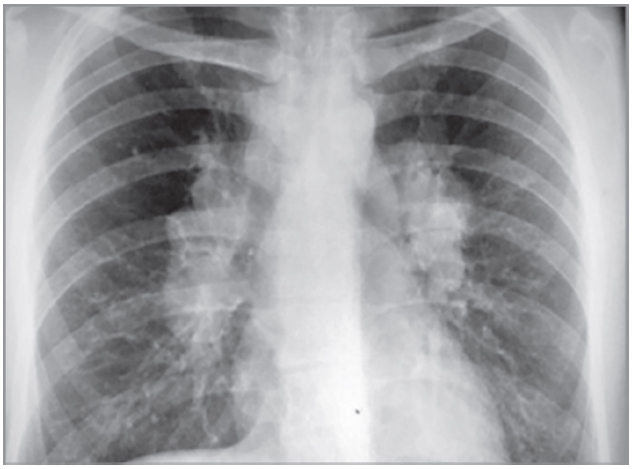
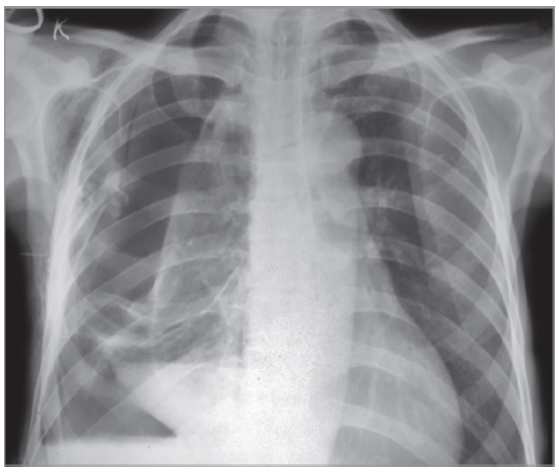


Fig. 12.37: Chest X-ray PA view showing homogeneous opacity in the right hemithorax obliterating the costophrenic angle, pleural based suggestive of **loculated pleural effusion**.

Fig. 12.38: Chest X-ray PA view showing bilateral hilar shadows, lobulated (also subcarinal shadow) suggestive of **lymphadenopathy**. Possible sarcoidosis.

Diffrential diagnosis—pleural mass/mesothelioma.



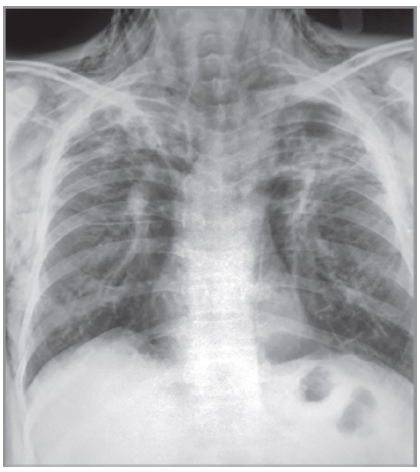
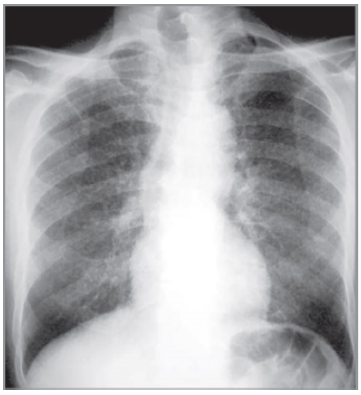
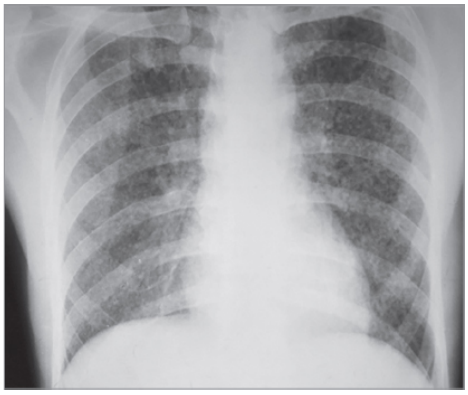


Fig. 12.39: Chest X-ray PA view showing tracheal shift to left, hyperlucency in right hemithorax with collapse lung margin (visceral pleural line) with obliteration of costophrenic angle with mulitiple air fluid levels suggestive of **hydropneumothorax**.

Fig. 12.40: Chest X-ray PA view showing air shadows in the subcutaneous plane in the neck, axilla, anterior chest wall, muscles suggestive of **subcutaneous emphysema**.





Figs. 12.41A and B: Chest X-ray PA view showing small millet sized (1–3 mm) shadows in bilateral lung fields suggestive of **miliary mottling**.

Differential diagnosis for miliary mottling:

- Miliary tuberculosis
- Tropical pulmonary eosinophilia
- Sarcoidosis
- Pneumocystis
- Fungal diseases: Histoplasmosis, coccidioidomycosis, blastomycosis, cryptococcosis
- Coal miner's pneumoconiosis
- Acute extrinsic allergic alveolitis
- Fibrosing alveolitis.
- Varicella pneumonia

Opacities (2–5 mm) tending to remain discrete:

- Miliary/lymphangitis carcinomatosis
- Lymphoma
- Sarcoidosis.

Opacities (2–5 mm) tending to coalasce:

- Multifocal pneumonia
- Pulmonary edema
- Extrinsic allergic alveolitis
- Fat emboli.

Those opacities having greater than-soft-tissue density:

- Pulmonary hemosiderosis
- Silicosis

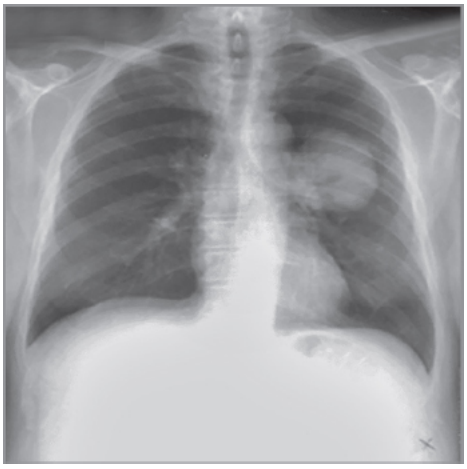


Fig. 12.42: Chest X-ray PA view showing rounded homogeneous lesion in the right mid zone—**solitary pulmonary nodule.**

For more details refer to page number 360 of Exam Preparatory Manual of Medicine for Undergraduates by the same author.

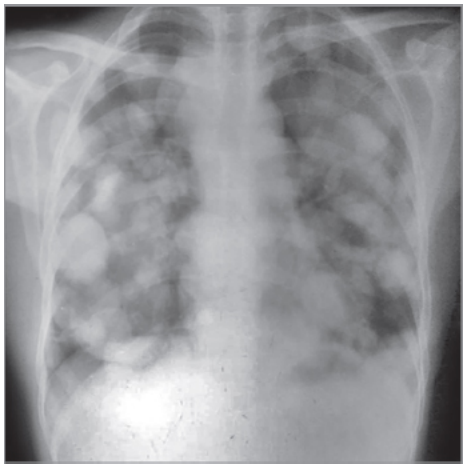


Fig. 12.43: Chest X-ray PA view showing multiple rounded nodular opacities in bilateral lung fields—**cannonball metastasis.**

Possible primary: Breast, thyroid, bowel, testes, renal cell carcinoma (RCC), choriocarcinoma

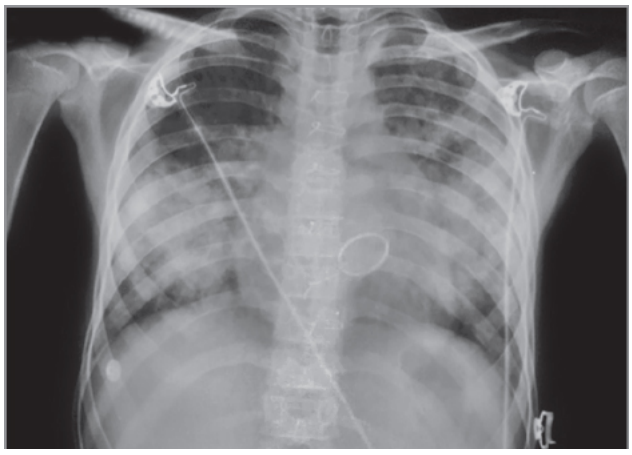


Fig. 12.44: Chest X-ray PA view showing cardiomegaly with bilateral nonhomogeneous opacity in mid and lower zones (bat wing appearance) suggestive of **pulmonary edema**. Also patient has **metallic mitral valve prosthesis.**

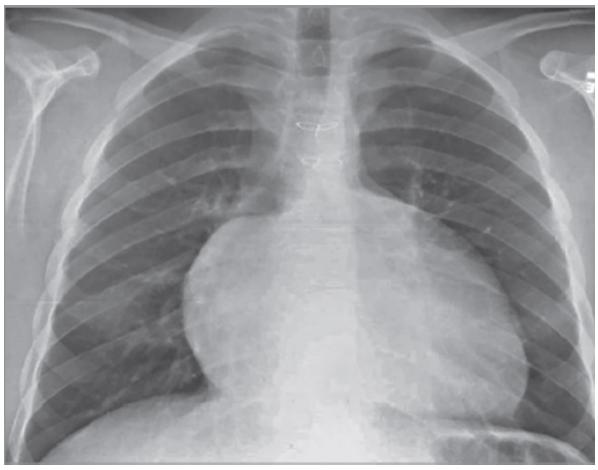


Fig. 12.45: Chest X-ray PA view showing gross cardiomegaly w stenciled heart borders, lungs clear. Suggestive of **pericardia effusion.**

Differential diagnosis—Ebstein's anomaly

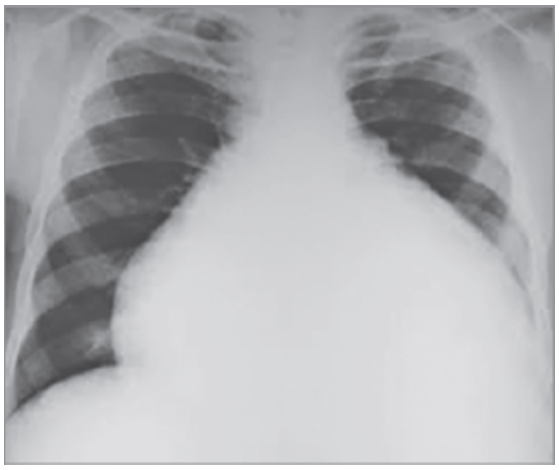


Fig. 12.46: Chest X-ray PA view showing gross cardiomegaly with stenciled heart borders, lungs clear. Suggestive of **pericardial effusion.**

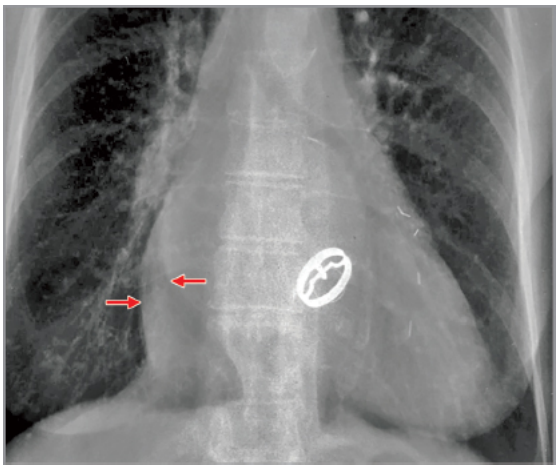


Fig. 12.47: Chest X-ray PA view showing cardiomegaly with **features of mitral valve disease**—splaying of carina, double atrial shadow (red arrows), straightening of left heart border, mitral valve metallic prosthesis.

Differfential diagnosis—Ebstein's anomaly

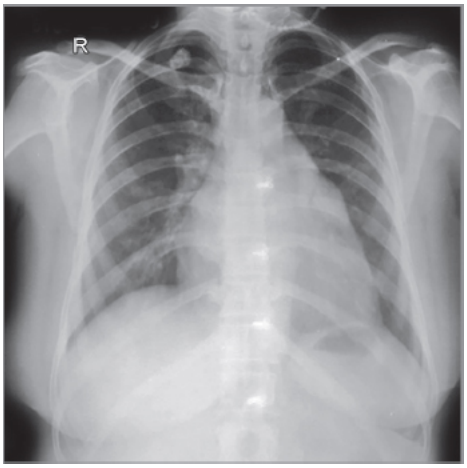


Fig. 12.48: Chest X-ray PA view showing cardiomegaly with **features of mitral valve disease**—splaying of carina, double atrial shadow, straightening of left heart border, enlarged left atrial appendage, prominent pulmonary artery.

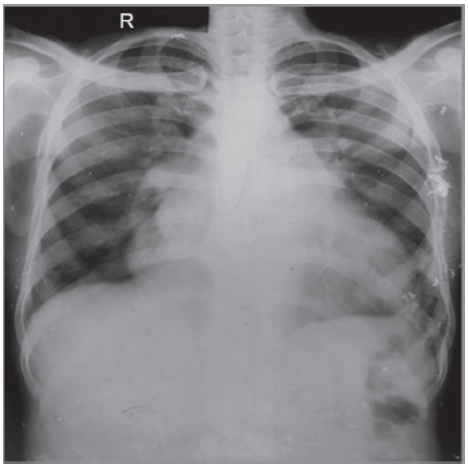


Fig. 12.49: Chest X-ray PA view showing cardiomegaly with **features of mitral valve disease**—splaying of carina, double atrial shadow, straightening of left heart border, mitral valve metallic prosthesis, enlarged left atrial appendage, prominent pulmonary artery, prominent upper lobe veins (stag's antler sign).

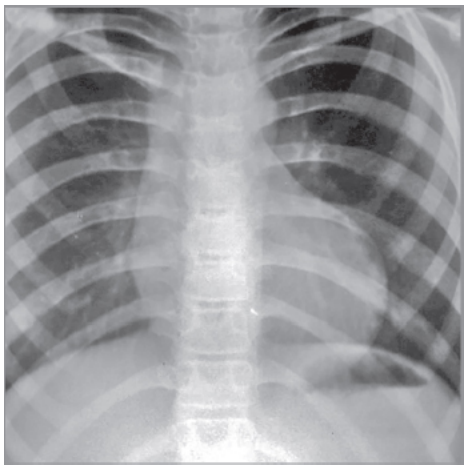


Fig. 12.50: Chest X-ray PA view showing pulmonary oligemia with upturned apex (right ventricle) suggestive of **tetralogy of Fallot (coeur-en-sabot)**.

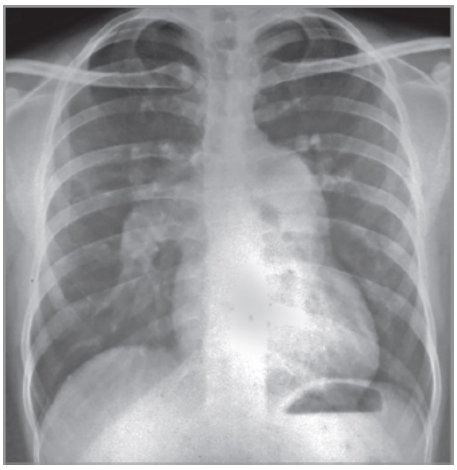


Fig. 12.51: Chest X-ray PA view showing mild cardiomegaly, prominent pulmonary artery, pulmonary plethora, prominent right atrium. Suggestive of **atrial septal defect—jug handle appearance.**

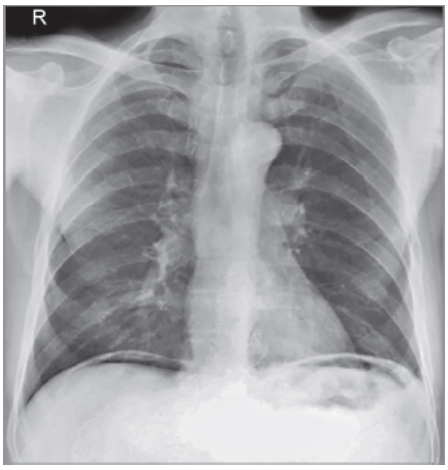


Fig. 12.52: Chest X-ray PA view showing free air under bilateral hemidiaphragm—**pneumoperitoneum**.

Causes:

- Hollow viscus perforation
- Postlaparotomy/laparoscopy
- Subphrenic abscess
- Tubal insufflation (Rubin's test)

Minimum amount of air needed to produce this is 1 cc.

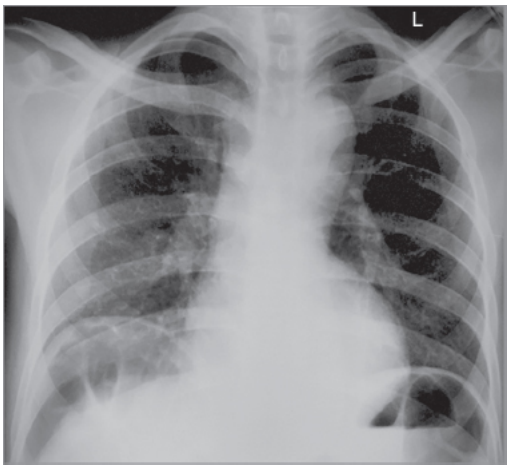


Fig. 12.53: Chest X-ray PA view showing interposition of transverse colon between liver and right hemidiaphragm—**Chilaiditi syndrome.**

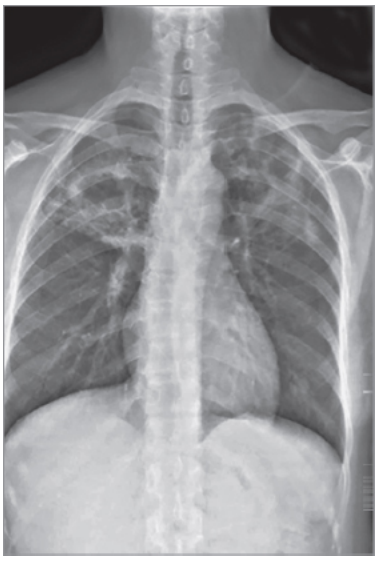


Fig. 12.54: Chest X-ray PA view showing trachea central, cardiophrenic and costophrenic angles are normal, nonhomogeneous opacity in bilateral upper zone with multiple cavities suggestive of **bilateral upper lobe active tuberculosis.**

X-ray signs of active tuberculosis—thin-walled cavities, pleural effusion, interstitial fluffy shadows.

X-ray signs of healed tuberculosis—thick-walled cavities, fibrosis, calcification, pleural thickening.

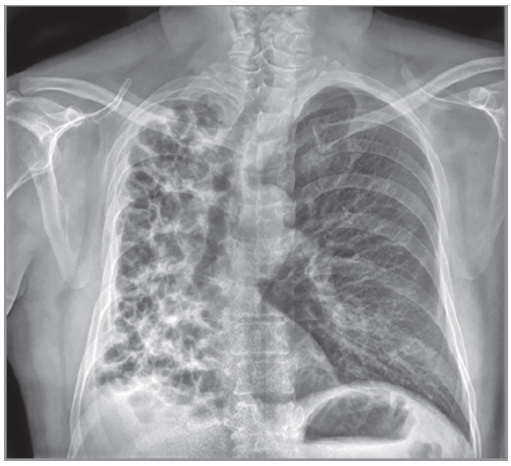


Fig. 12.55: Chest X-ray PA view showing trachea deviated to right, mediastinum pulled to right, decreased size of right hemithorax with rib crowding. Nonhomogeneous opacity in right hemithorax with multiple cystic shadows suggestive of **right-sided fibrosis with cystic bronchiectasis** possibly sequele of tuberculosis.

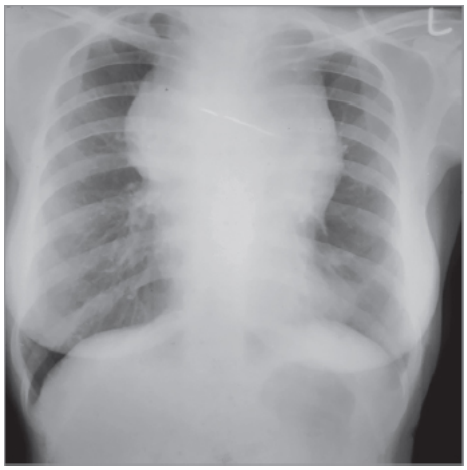


Fig. 12.56: Chest X-ray PA view showing trachea central, cardiophrenic and costophrenic angles are normal, mediastinal widening suggestive of **superior mediastinal mass**.

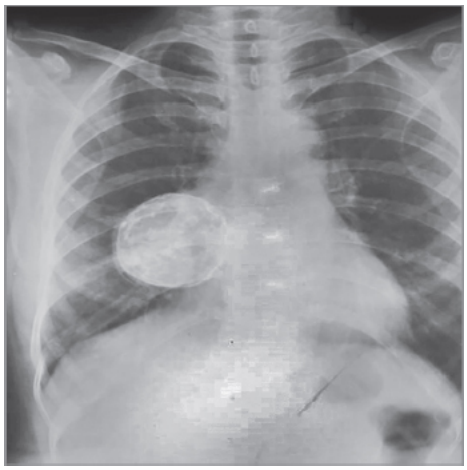


Fig. 12.57: Chest X-ray PA view showing trachea central, cardiophrenic and costophrenic angles are normal, rounded opacity arising from the anterior mediastinum which is **calcified—mediastinal cyst**.

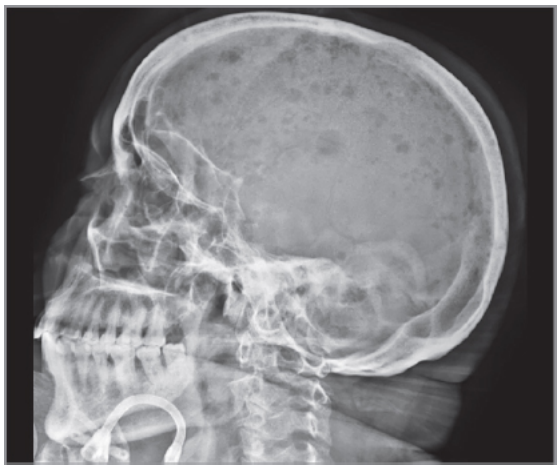


Fig. 12.58: Lateral X-ray of skull showing **multiple punched out lesions**.

Differential diagnosis: Myleoma, metastasis, rarely Langerhans cell histiocytosis.

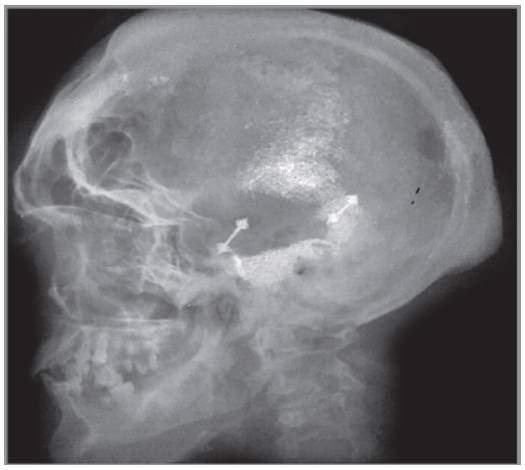


Fig. 12.59: Lateral skull X-ray showing prognathism, thickened skull vault, prominent air sinuses, enlarged sella turcica—**suggestive of acromegaly.**

COMPUTED TOMOGRAPHY (FIGS. 12.60 TO 12.64)

Computed Tomography

Types

1. Spiral CT
2. Multislice CT—coronary CT angiography and calcium score
3. Electron beam CT—faster, used for cardiac application
4. High resolution CT (HRCT)—1–2 mm slices, investigation of choice for ILD and bronchiectasis.

CT density scale—Hounsfield units—range from −1,000 (black) to +1,000 (white).

0—attenuation value of water (considered as reference)

−1,000	Air
−100	Fat
0	Water
+60	Hemorrhage
+1,000	Calcification

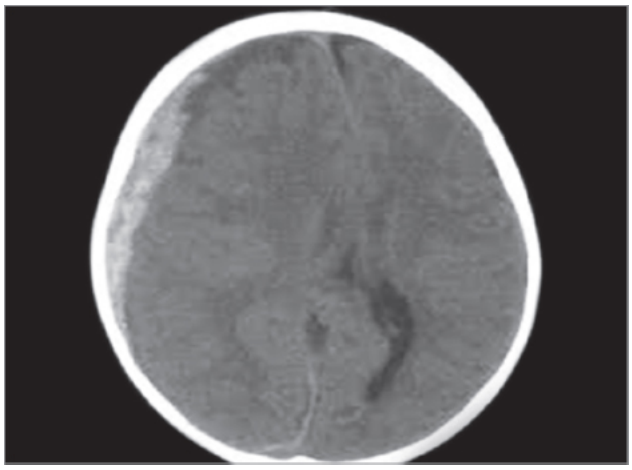


Fig. 12.60: Plain CT head showing hyperdense shadow which is **concavo-convex** in appearance suggestive of **acute right subdural hematoma**.

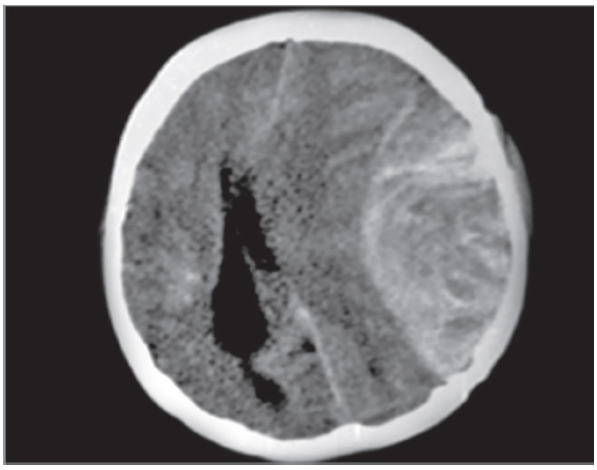


Fig. 12.61: Plain CT head showing hyperdense shadow whicl **biconvex** in appearance suggestive of **acute left extradural hematoma.**

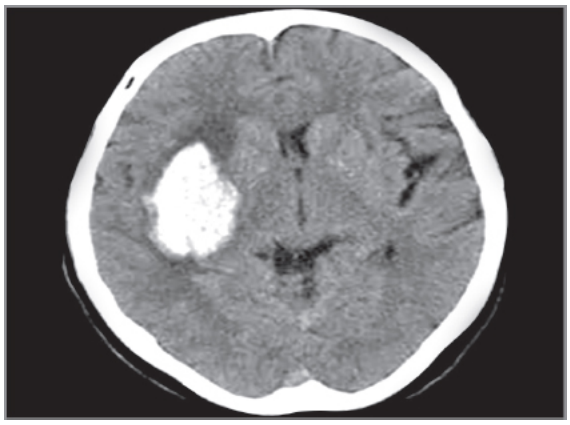


Fig. 12.62: Plain CT head showing hyperdense shadow in the right basal ganglia suggestive of **acute intraparenchymal hemorrhage**.

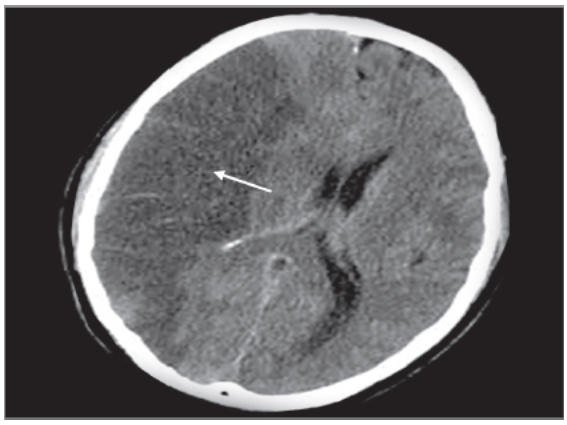


Fig. 12.63: Plain CT head showing hypodense shadow in the ri parietotemporal cortex suggestive of **acute infarct** (arrow).

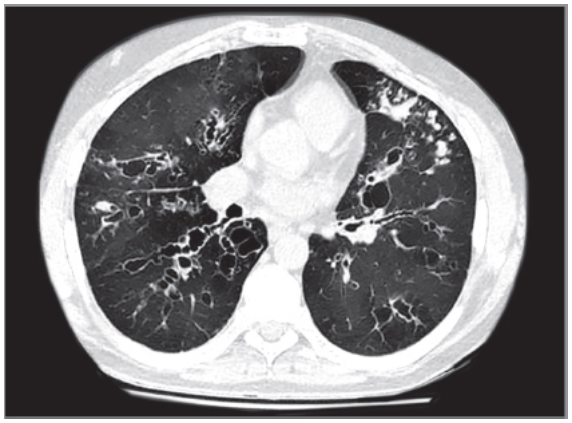


Fig. 12.64: High-resolution computed tomography (HRCT) of chest. Varicose and cystic **bronchiectasis** with mucus plugging in upper lobes.

MAGNETIC RESONANCE IMAGING (FIGS. 12.65 AND 12.66)

Proton acts as a dipole with magnetic dipole movement and gyromagnetic properties.

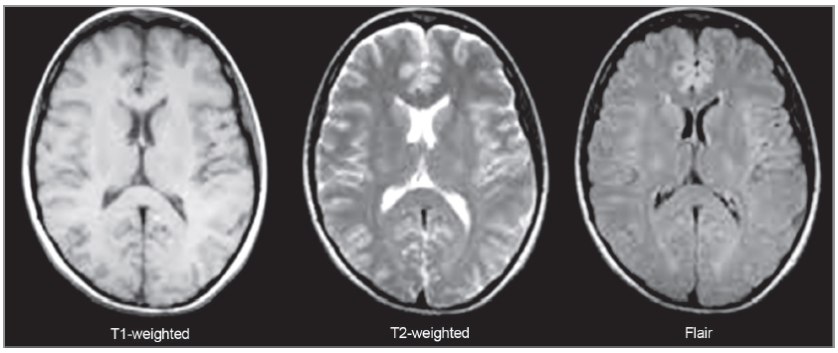


Fig. 12.65: Magnetic resonance imaging.

Types of MRI sequences

T1—Spin lattice relaxation time

T2—Spin-spin relaxation time

Flair—Fluid attenuated inversion recovery—preferred in CNS demyelinating diseases like multiple sclerosis

DWI/Diffusion weighted images—for detection of early infarcts

ADC (Apparent diffusion coefficient)

MR signal characteristics:

	T1	T2
CSF	Hypointense	Hyperintense
Gray matter	Gray	White
White matter	White	Gray
Fat	Hyperintense	Less hyperintense
Tumors (most)	--	Hyperintense
Melanoma	Hyperintense	Hypointense

Differential diagnosis

- Cerebral abscess
- Tuberculoma
- Neurocysticercosis
- Metastasis
- Glioblastoma
- Subacute infarct/hemorrhage
- Demyelination

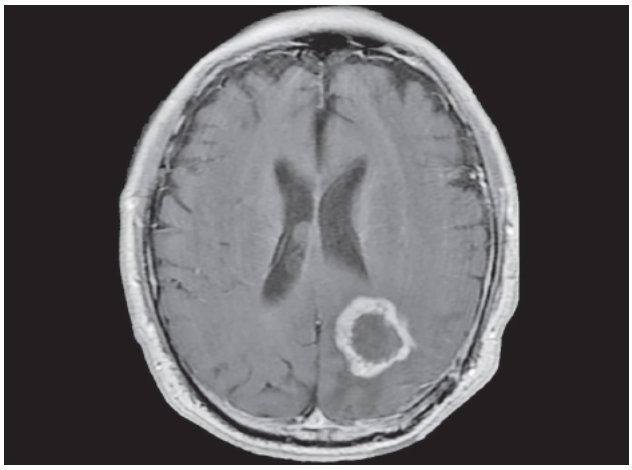


Fig. 12.66: MRI brain showing ring enhancing lesion.

- Radiation necrosis
- Lymphoma

CONTRAST AGENTS

Contrast for X-ray/CT

Positive contrast agents		Negative contrast agents
Water soluble (Iodine containing agents)	**Water insoluble** (Barium containing agents)	Air Water
High osmolar: Urografin, Diatrizoate sodium, Conray **Low osmolar:** Optiray, Iodixanol		
Note: Low osmolar agents are safer.		

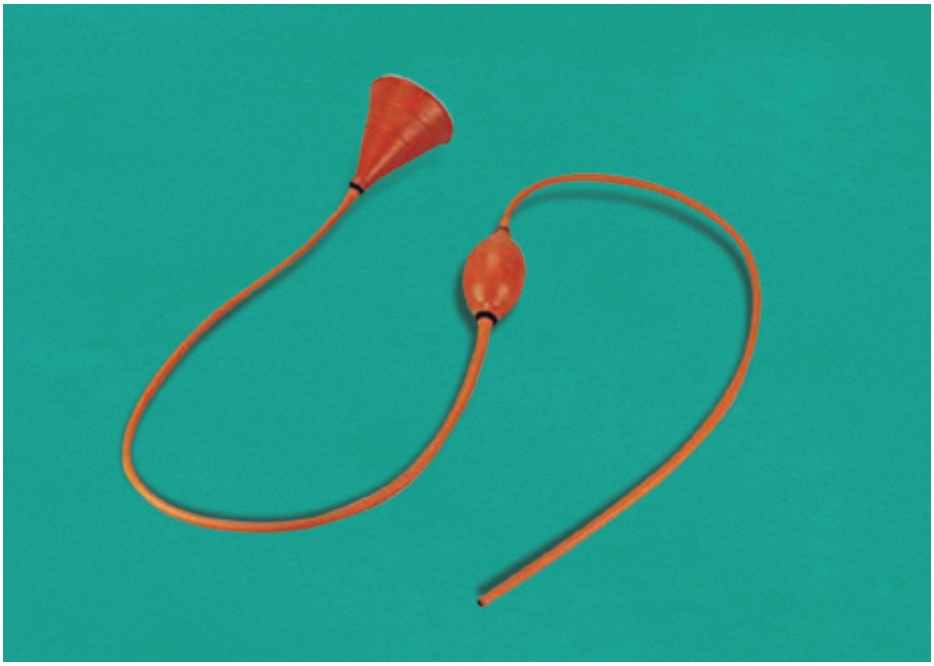
MRI Contrast Agents

Contain paramagnetic metal ions. For example: gadolinium ligated to diethylenetriaminepentaacetic (DTPA).

Basic Instruments in Viva

CHAPTER 13

GASTRIC LAVAGE TUBE



Description

Used for gastric decontamination by removing toxic substances from the stomach by sequential administration and reaspiration of small volumes of fluid through this tube.

Other names—Ewald's tube/boes tube.

Indications

For decontamination after oral consumption of poison.

Contraindications

- Corrosive ingestions or esophageal disease
- Hydrocarbon or petroleum distillate ingestion
- Convulsion
- Cardiac dysrhythmias
- The poison ingested is not toxic at any dose
- The poison ingested is adsorbed by charcoal and adsorption is not exceeded by the quantity of ingestion
- Presented several (4-6) hours after consumption of the poison
- A highly efficient antidote, such as N-acetylcysteine (NAC) is available.

Technique of Performing Orogastric Lavage (Table 13.1)

Table 13.1: The technique of performing orogastric lavage.

Select the correct tube size **Adults and adolescents:** 36–40 French **Children:** 22–28 French	
Procedure	
1.	If there is a potential airway compromise, endotracheal intubation should precede orogastric lavage.
2.	The patient should be kept in the left lateral decubitus position. Because the pylorus points upward in this orientation, this positioning theoretically helps prevent the xenobiotic from passing through the pylorus during the procedure.
3.	Before insertion, the proper length of tubing to be passed should be measured and marked on the tube. The length should allow the most promixal tube opening to be passed beyond the lower esophageal sphincter.
4.	After the tube is inserted, it is essential to confirm that the distal end of the tube is in the stomach.
5.	Any material present in the stomach should be withdrawn and immediate instillation of activated charcoal should be considered for large ingestions of xenobiotics that are known to be adsorbed by activated charcoal.
6.	In adults, 250-mL aliquots of a room temperature saline lavage solution is instilled via a funnel or lavage syringe. In children, aliquots should be 10 to 15 mL/kg to a

	maximum of 250 mL.
7.	Orogastric lavage should continue for at least several liters in an adult and for at least 0.5 to 1.0 L in a child or until no particulate matter returns and the effuent lavage solution is clear.
8.	After orogastric lavage, the same tube should be used to instill activated charcoal if indicated.

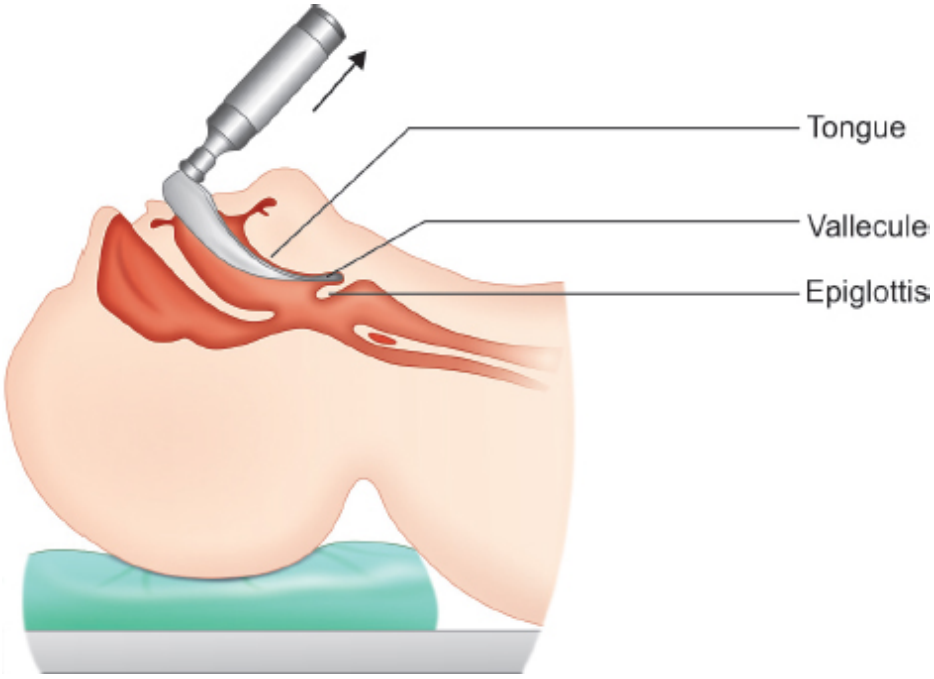
Complications

- Aspiration of gastric contents (3% of patients)
- Esophageal rupture (rare)
- Laryngospasm
- Bradycardia.

LARYNGOSCOPE

Description

Laryngoscopes are usually left-handed tools designed to facilitate visualization of the larynx. A laryngoscope consists of a handle, a blade, and a light source. The most commonly used blades include the curved Macintosh and the straight Miller blades.



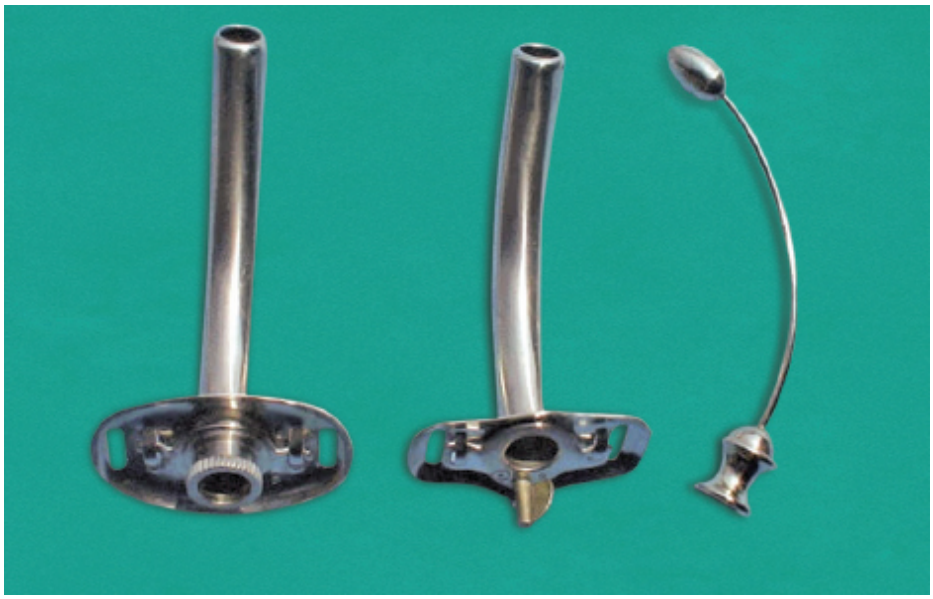
Indications

- Patients requiring emergent intubation in conditions like acute respiratory failure with inadequate oxygenation and ventilation.
- In patients with altered sensorium for airway protection.
- Nonemergent intubation occurs in the perioperative setting as patients may require general anesthesia.

Contraindications

- Suspected cervical spine injuries
- Patients who have supraglottic or glottic pathology.
- A relative contraindication to laryngoscopy includes patients with anatomy that does not allow successful laryngoscopy use, injuries to the area, or physiologic status that is not conducive to the procedure.

METAL TRACHEOSTOMY TUBE



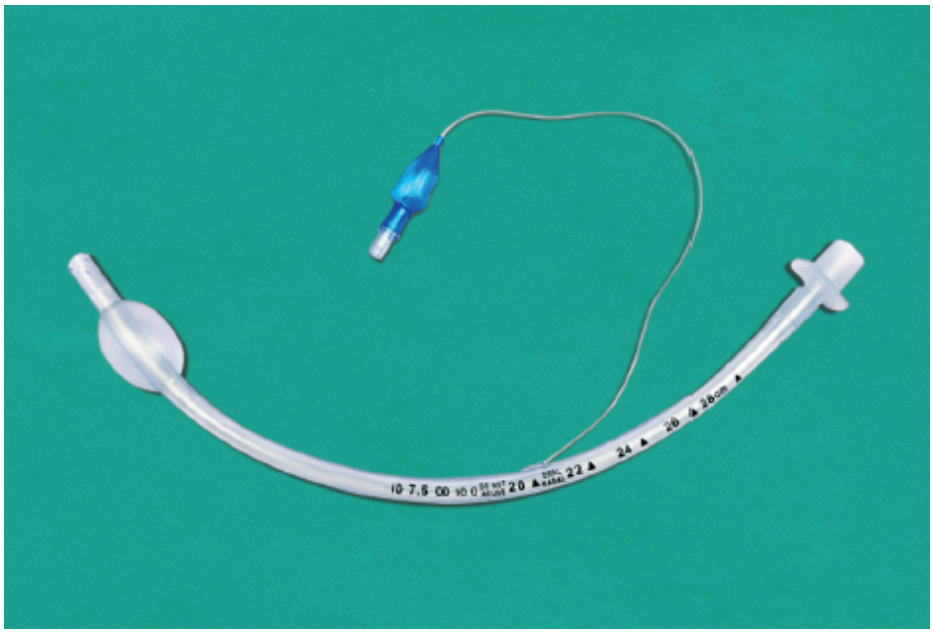
Description

- It consists of three parts—(1) outer cannula with flange (neck plate), (2) inner cannula, and (3) an obturator.

Indications

- Upper airway obstruction (e.g. stridor)
- Prolonged intubation
- Facilitation of ventilation support
- For management of pulmonary secretions.

ENDOTRACHEAL TUBE



Description

It is a tube constructed of polyvinylchloride (PVC) that is placed between the vocal cords into the trachea to provide oxygen and inhaled gases to the lungs. It also serves to protect the lungs from contamination, such as gastric contents and blood parts of endotracheal (ET) tube.

The Tube

The endotracheal tube (ETT) has a length and diameter. The endotracheal tubes size refers to its internal diameter in millimeters (mm). PVC is not radio-opaque, and thus a radio-opaque linear material is included throughout the length of the tube to make it easier to visualize the placement on X-ray. Ideally, the distal tip of the ETT is 4 cm (+/−2 cm) above the carina on chest X-ray in adults.

The Cuff

A cuff is an inflatable balloon at the distal end of the ETT. The inflated cuff produces a seal against the tracheal wall; this prevents gastric contents from entering the trachea and facilitates the execution of positive pressure ventilation. The cuff inflates by attaching an appropriate size syringe (10–20 mL for adult ETT) to the pilot balloon.

The Bevel

To facilitate placement through the vocal cords and to provide improved visualization ahead of the tip, the ETT has an angle or slant known as a bevel.

The Murphy's Eye

Endotracheal tubes have a built-in safety mechanism at the distal tip known as Murphy's eye, which is another opening in the tube positioned in the distal lateral wall.

The Connector

Endotracheal tube connectors attach the ETT to the mechanical ventilator tubing or an Ambu bag.

Indications

- Acute respiratory failure, inadequate oxygenation, or ventilation,
- Airway protection in a patient with depressed mental status.
- In the perioperative setting, endotracheal tubes may be placed in many clinical circumstances including patients receiving general anesthesia, surgery involving
- Less frequently to manage increased intracranial pressure or to manage copious secretions or bleeding from the airway.

Contraindications

- Severe airway trauma or obstruction that does not allow safe placement of the tube
- Severe cervical spine injury which requires complete immobilization, and
- Those patients with Mallampati III/IV classification suggesting potentially difficult airway management.

AMBU BAG

Description

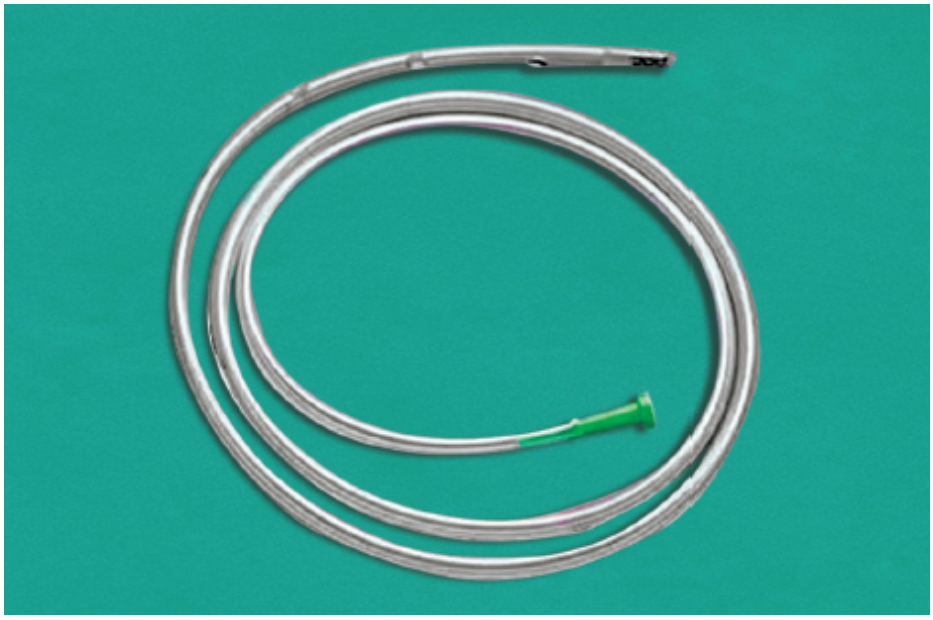
A **bag valve mask (BVM)**, **Ambu bag** or generically as a **manual resuscitator** or “self-inflating bag”, is a hand-held device commonly used to provide positive pressure ventilation to patients who are not breathing or not breathing adequately.

The BVM consists of a flexible air chamber (the “bag”, roughly a foot in length), attached to a face mask via a shutter valve.

Complications

- Air inflating the stomach;
- Lung injury from overstretching (called volutrauma); and/or
- Lung injury from overpressurization (called barotrauma).

RYLES TUBE—NASOGASTRIC TUBE



Description

It is a flexible tube made of rubber or non-toxic, medical grade PVC compound, and it has bidirectional potential. It can be used either to feed or remove the contents of the stomach including air to decompress the stomach or to remove small solid objects and fluid, such as poison from the stomach.

Indications

Diagnostic indications for nasogastric tube (NG) intubation include the following:

- Evaluation of upper gastrointestinal (GI) bleeding (i.e. presence and volume)
- Aspiration of gastric fluid content
- Identification of the esophagus and stomach on a chest radiograph
- Administration of radiographic contrast to the GI tract.

Therapeutic indications for NG intubation include the following:

- Gastric decompression including maintenance of a decompressed state after endotracheal intubation, often via the oropharynx
- Relief of symptoms and bowel-rest in the setting of small bowel obstruction

- Aspiration of gastric content from recent ingestion of toxic material
- Administration of medications in comatose patients
- Feeding when patient is unconscious or when the patient is conscious but unable to swallow voluntarily
- Bowel irrigation.

Contraindications

Absolute contraindications for NG intubation include the following:

- Severe midface trauma
- Recent nasal surgery.

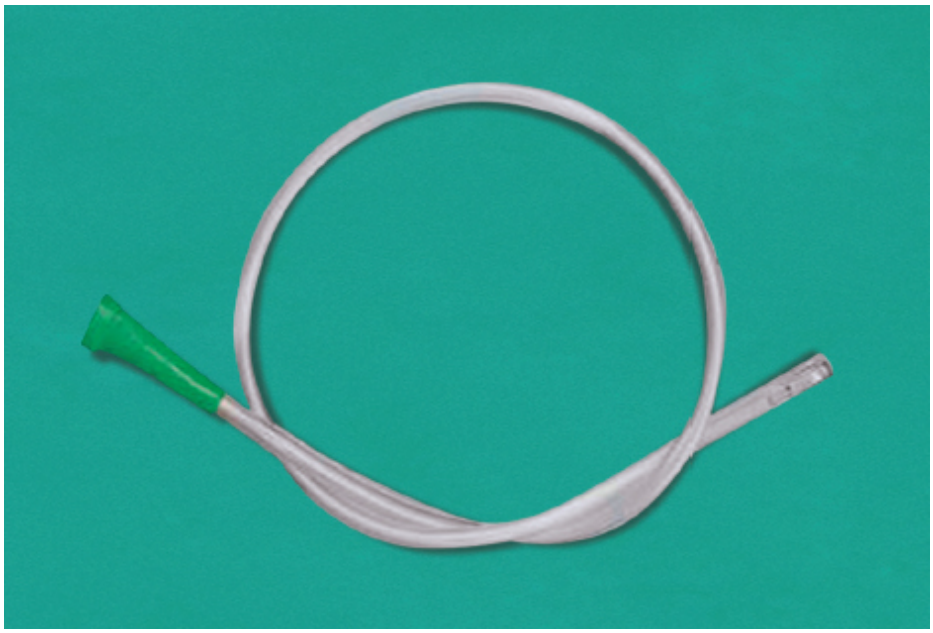
Relative contraindications for NG intubation include the following:

- Coagulation abnormality
- Esophageal varices
- Recent banding of esophageal varices
- Alkaline ingestion (the tube may be kept if the injury is not severe).

Verification of Position of Ryles Tube

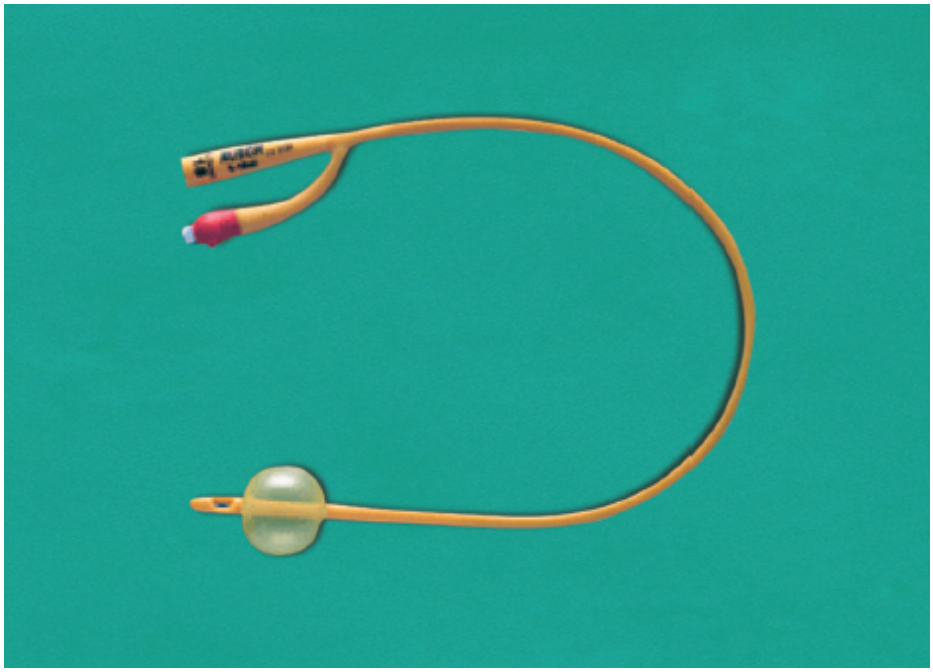
- Verify proper placement of the NG tube by auscultating a rush of air over the stomach using the 60 mL Toomey syringe or by aspirating gastric content
- Obtaining a chest radiograph
- Colorimetric capnography is another valid method for verifying NG tube positioning in mechanically ventilated patients.

SUCTION CATHETER



A **suction catheter** is a medical device used to extract bodily secretions, such as mucus or saliva from the upper airway. A *suction catheter* connects to a **suction machine** or **collection canister**.

FOLEYS CATHETER



Description

Foley catheter (named for Frederic Foley, who produced the original design in 1929), the tube has two separated channels, or *lumens* running down its length. One lumen open at both ends, drains urine into a collection bag. The other has a valve on the outside end and connects to a balloon at the inside tip. The balloon is inflated with sterile water when it lies inside the bladder to stop it from slipping out. Saline should not be used to inflate the bulb, as it can crystallize within. Air must not be used to inflate as it will float over the urine. Coatings include polytetrafluoroethylene, hydrogel, or a silicon elastomer—the different properties of these surface coatings determine whether the catheter is suitable for 28-day or 3-month indwelling duration.

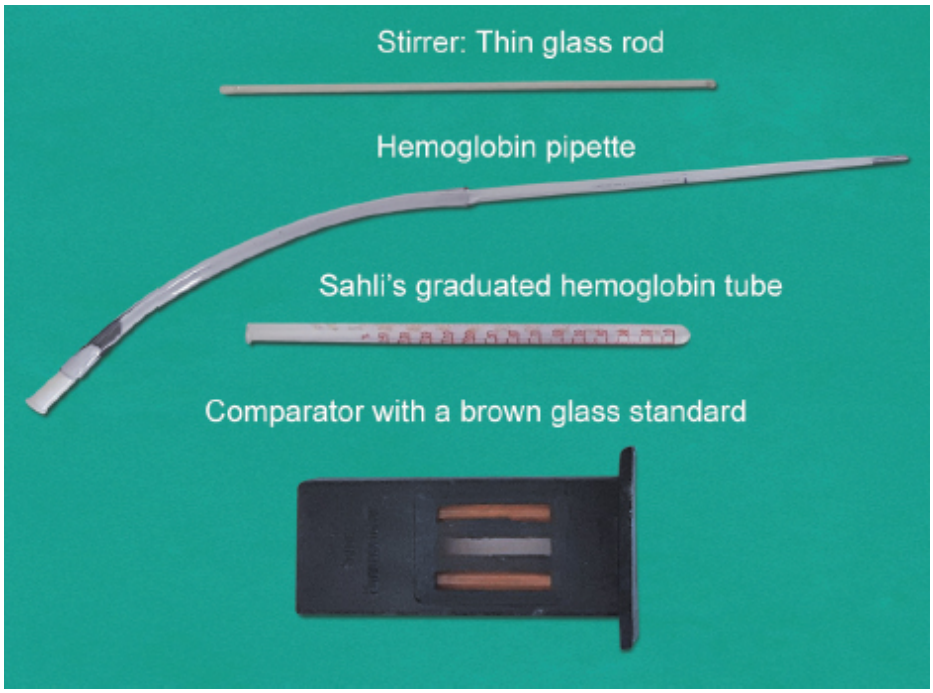
Indications

- Acute retention of urine
- Chronic retention of urine with overflow
- In cases of neurogenic bladder
- In surgery involving bladder and prostrate
- In all perineal operations
- Intravesical chemotherapy
- To carry out urethrography
- To monitor urine output.

Contraindication

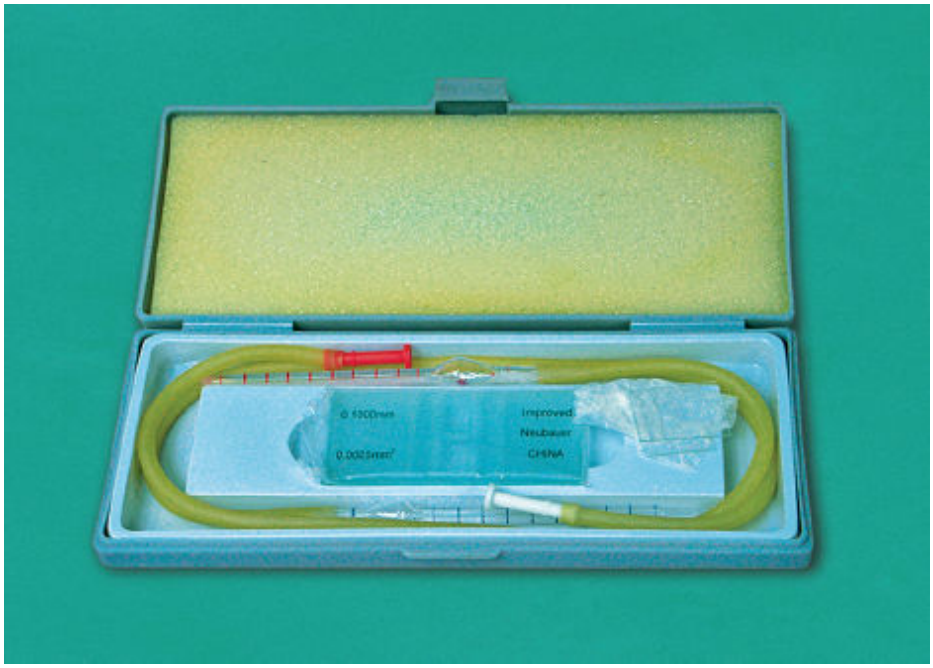
Urethral trauma is the only absolute contraindication to placement of a urinary catheter.

SAHLI'S HEMOGLOBINOMETER



Used to estimate hemoglobin: Method used is acid hematin method.

NEUBAUER CHAMBER/HEMOCYTOMETER

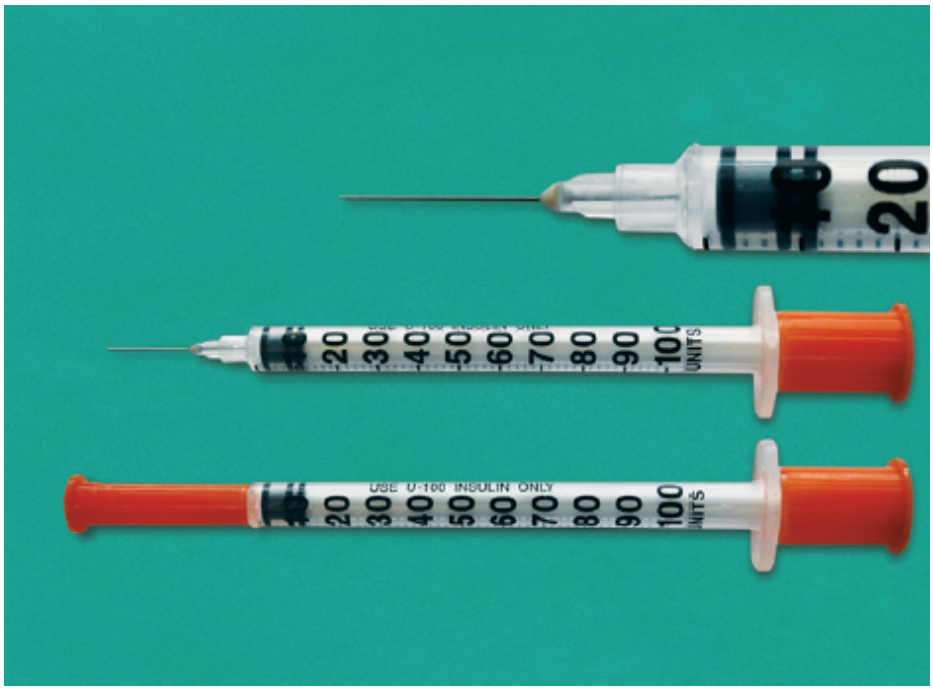


Description

The Neubauer chamber is a thick crystal slide with the size of a glass slide (30 × 70 mm and 4 mm thickness). In a simple counting chamber, the central area is where the cell counts are performed.

Use: Used to count red blood cell/white blood cell (RBC/WBC).

INSULIN SYRINGE



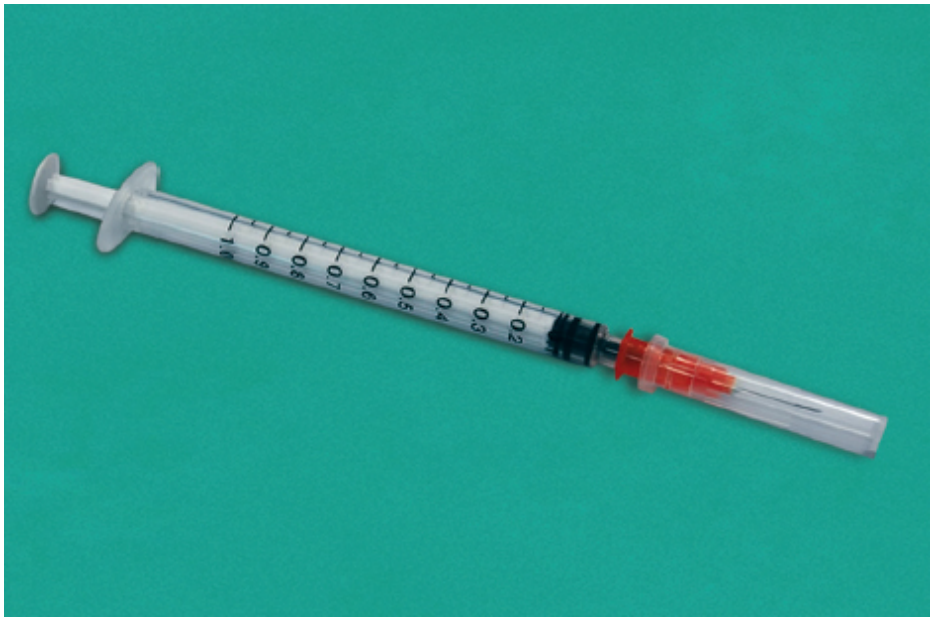
Description

Syringes for insulin users are designed for standard U-100 insulin. The dilution of insulin is such that 1 mL of insulin fluid has 100 standard “units” of insulin. Even 40 IU syringes are available.

Use

It is used for subcutaneous insulin administration.

TUBERCULIN SYRINGE

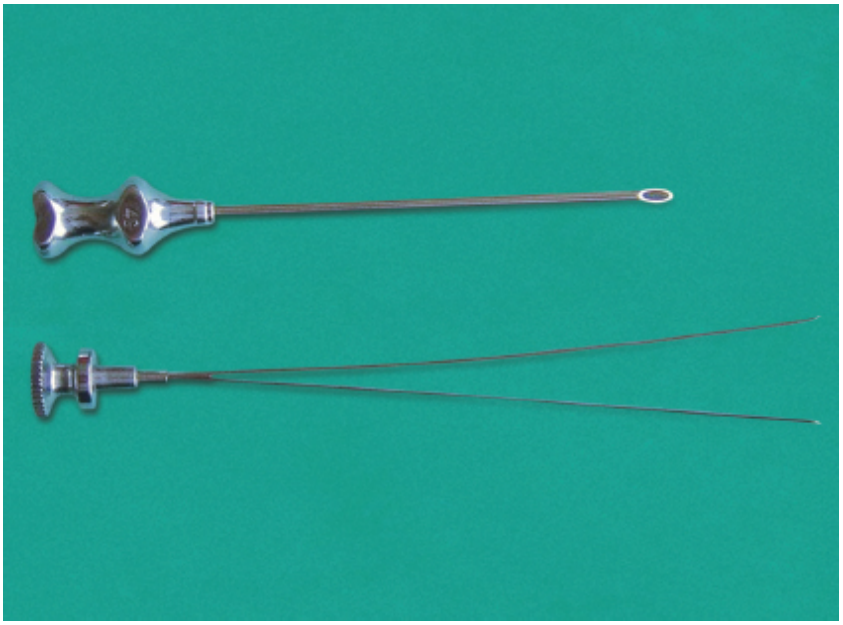


Tuberculin syringes are small syringes with fine needles that hold up to one-half to one cubic centimeter of fluid, used to administer medication (antigen) under the skin and perform a tuberculosis test called purified protein derivative (PPD)/Mantoux test.

Insulin 40 Versus Insulin 100 Versus Tuberculin Syringe

U-40 insulin syringes markings on the barrel are up to 40 units, while in U-100 markings are up to 100 units. While in case of 1 mL tuberculin syringes the markings are at zero (0) and each 0.05 mL, e.g., 0.05, 0.1, 0.15, 0.2, 0.25, 0.3, etc.

VIM SILVERMAN LIVER BIOPSY NEEDLE



Description

It has **three parts:**

1. Cannula
2. Stylet/trocar
3. Prong/fork/bifid needle—longer than needle and it protrudes out of the needle. It has a very sharp cutting edge and has longitudinal groove. This retains the tissue when the needle and cannula are withdrawn.

Indications for Liver Biopsy

- In evaluation of jaundice
- Liver cirrhosis
- Storage disorders: Glycogen storage disease, hemochromatosis, and Wilson's disease
- Granulomatous lesions like tuberculosis and sarcoidosis
- Infections: Viral [cytomegalovirus (CMV), herpes, and parasitic (amoebic liver abscess where it is both diagnostic and therapeutic)]
- To diagnose Benign and malignant neoplasms.

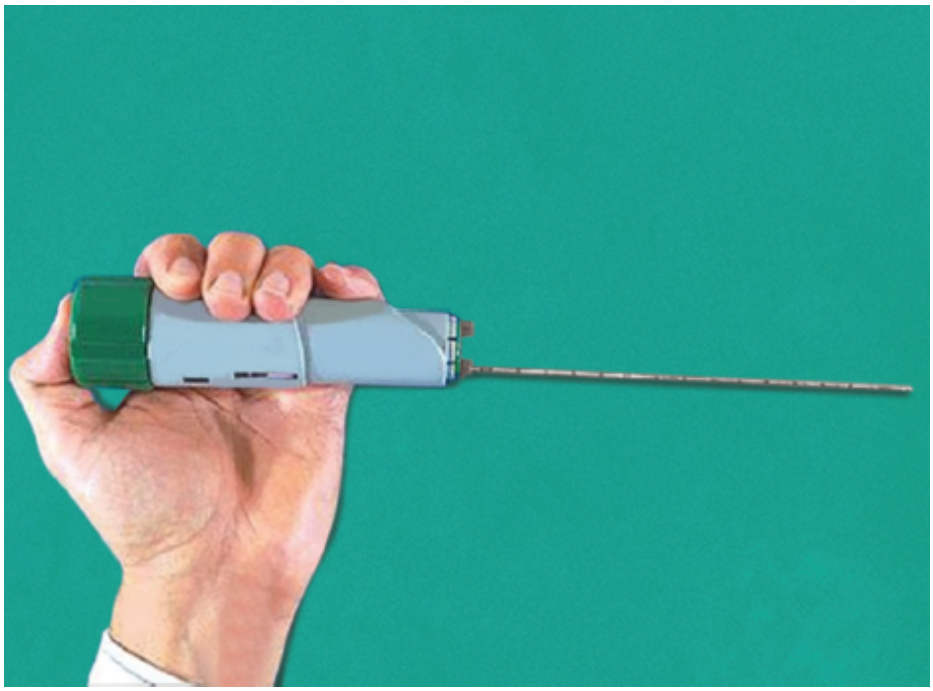
Contraindications of Liver Biopsy

- Bleeding diathesis
- Hemangiomas
- Hydatid cyst
- Severe ascites.

Complications of Liver Biopsy

- Hemorrhage
- Infection
- Adjacent structures can be injured (gallbladder, colon, and blood vessels)
- Rarely there can be precipitation of hepatic coma.

TRUCUT BIOPSY GUN



Description

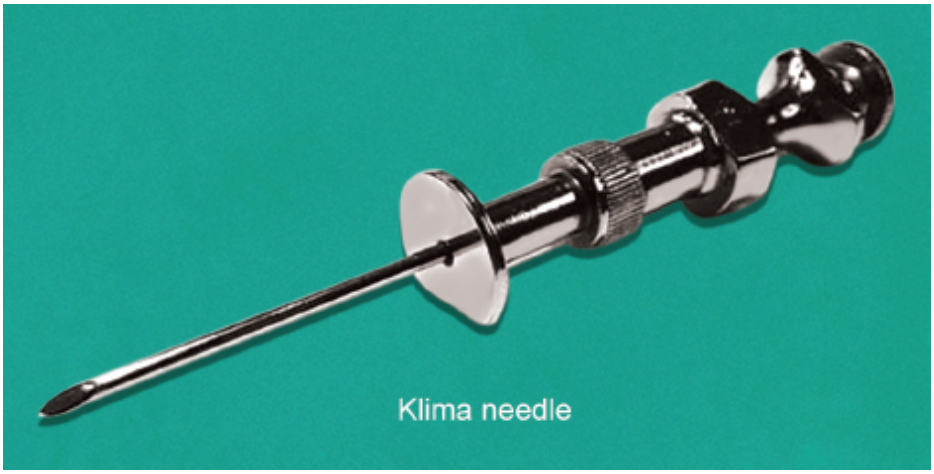
A needle with a gap near its tip is passed into the lesion. A surrounding sheath with a cutting tip is passed down the needle. The sheath cuts a specimen corresponding to the gap in the needle. The

needle and sheath with the specimen are then removed from the patient.

Use: For tissue biopsy—liver/kidney.

BONE MARROW ASPIRATION NEEDLE

Salah's bone marrow aspiration needle



Klima needle

Indications

The diagnosis of acute leukemia staging for lymphoma, evaluation of pancytopenia, thrombocytopenia, investigation of anemia, fever (pyrexia of unknown origin), lymphadenopathy, and hepatosplenomegaly.

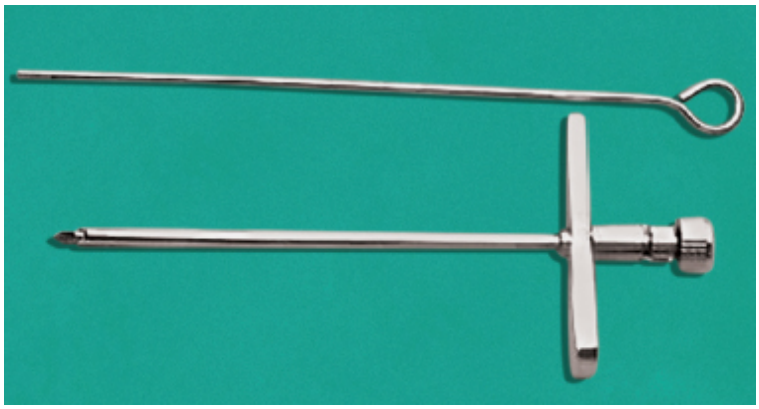
Contraindications

- Bleeding disorders and coagulopathy
- Local skin infection/osteomyelitis.

Sites

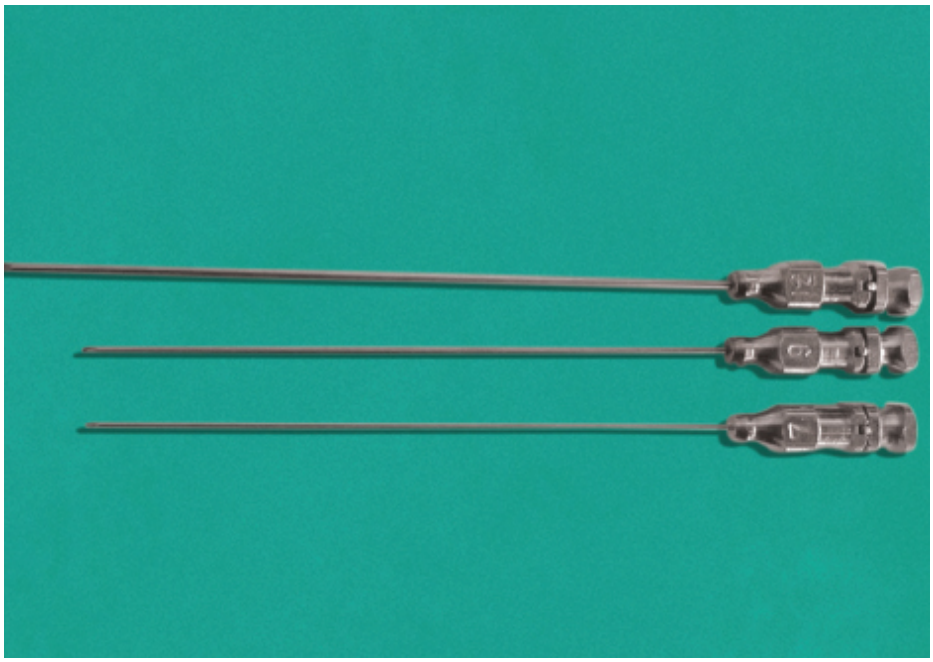
Posterior superior iliac spine, anterior superior iliac spine. Sternum, tibial tuberosity.

BONE MARROW BIOPSY NEEDLE (JAMSHIDI NEEDLE)



- Biopsy done when bone marrow tap is dry
- Also for infiltrative disorders.

LUMBAR PUNCTURE NEEDLE



Description

Lumbar puncture is a technique done to obtain cerebrospinal fluid (CSF) sample.

It also provides an indirect measure of intracranial pressure (ICP). It is usually done between L3 and L4 (3rd lumbar space) through the dura and into the spinal canal..

Indications for Lumbar Puncture

Diagnostic Indications

- Meningitis
- Encephalitis
- Subarachnoid hemorrhage
- Primary or metastatic malignancy (e.g. acute leukemias and lymphoma)
- Demyelinating diseases: Multiple sclerosis and
- Subacute sclerosing panencephalitis (SSPE)
- Guillain–Barré syndrome
- Injecting the radio-opaque dye for myelography.

Therapeutic Indications

- Spinal anesthesia and epidural analgesia
- Intrathecal injection of chemotherapeutic drugs for CNS prophylaxis/relapse of acute lymphoblastic leukemia (ALL), lymphomas
- Therapeutic CSF drainage in cases of normal pressure hydrocephalus.

Contraindications for Lumbar Puncture

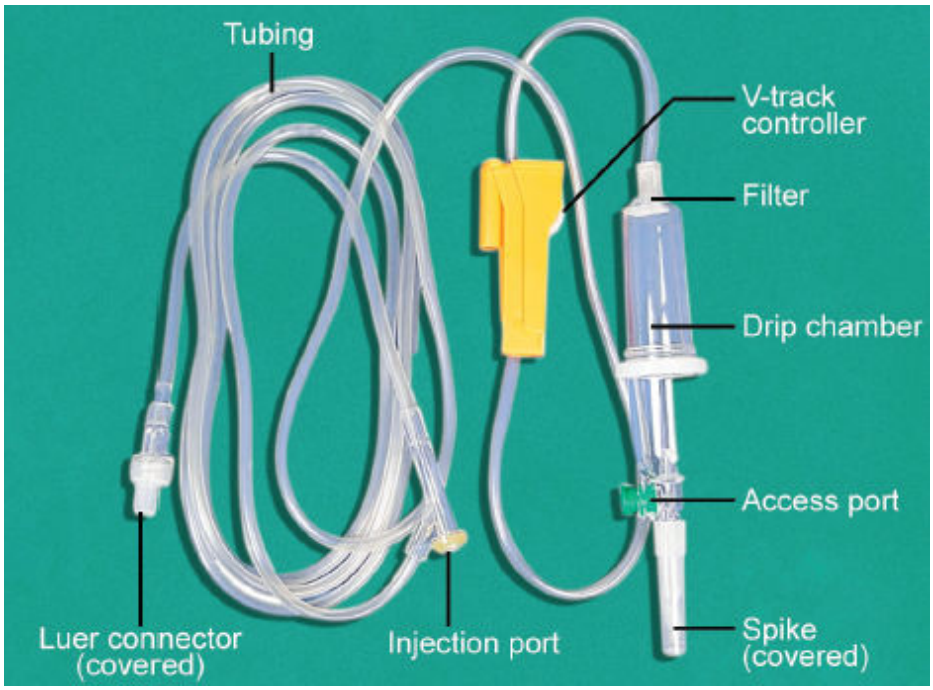
- Raised intracranial pressure, coagulopathy
- Local infective lesion
- Bony deformities at site of puncture.

Complications of Lumbar Puncture

- **Postspinal headache**.
- Herniation of cerebellum through the foramen magnum due to raised intracranial pressure.
- Introduction of infection by the lumbar puncture needle through the infected skin or subcutaneous tissue.

For further details on lumbar puncture analysis, findings of CSF analysis refer to page number 978 for Exam Preparatory Mannual of Medicine for Undergraduates by the same author.

INTRAVENOUS DRIP SET



IV Drip Set

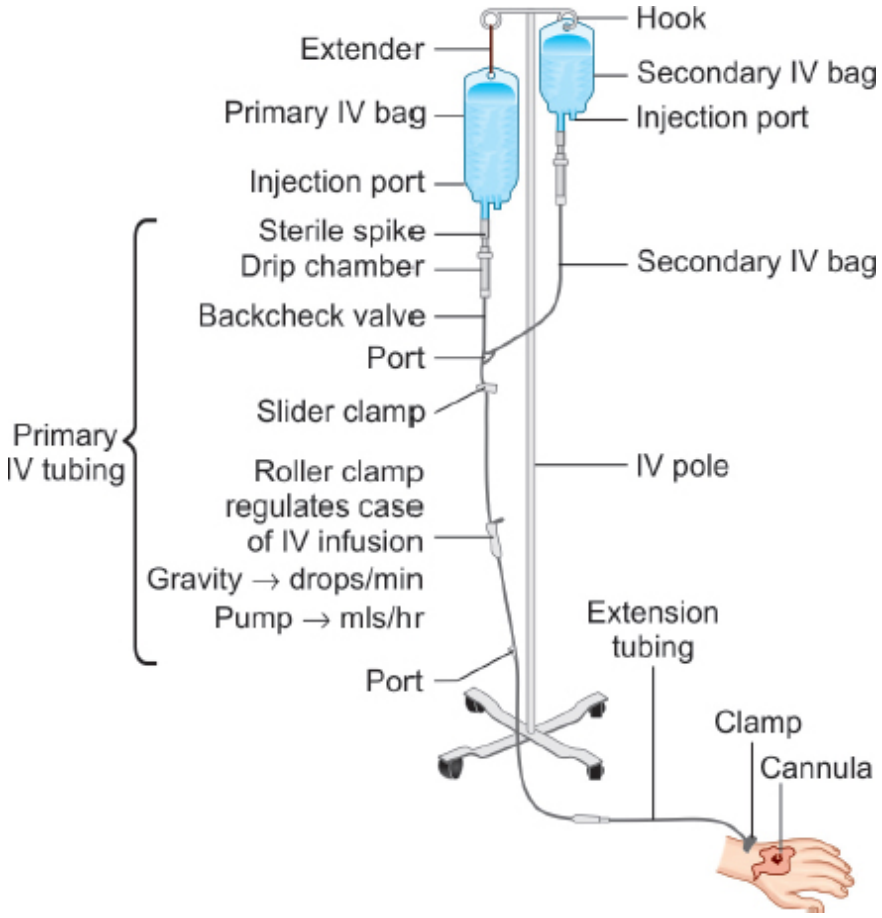
Used for administering intravenous fluids, drugs, and blood products.

Intravenous (IV) fluids are administered through thin, flexible plastic tubing called an *infusion set* or **primary infusion tubing/administration set** (Perry et al. 2014). The infusion tubing/administration set connects to the bag of IV solution. Primary IV tubing is either a macrodrip solution administration set that delivers 10, 15, or 20 drops/mL, or a microdrip set that delivers 60 drops/mL. Macrodrip sets are used for routine primary infusions. Microdrip IV tubing is used mostly in pediatric or neonatal care, when small amounts of fluids are to be administered over a long period of time (Perry et al. 2014). The drop factor can be located on the packaging of the IV tubing.

Primary IV tubing is used to infuse continuous or intermittent fluids or medication. It consists of the following parts:

- Backcheck valve: Prevents fluid or medication from traveling up the IV
- Access ports: Used to infuse secondary medications and give IV push medications

- Roller clamp: Used to regulate the speed of, or to stop or start, a gravity infusion
- Secondary IV tubing: Shorter in length than primary tubing with no access ports or backcheck valve; when connected to a primary line via an access port used to infuse intermittent medications or fluids. A **secondary tubing administration set** is used for secondary IV medication.



Flow Rate Calculation

When calculating the flow rate of IV solutions, remember that the number of drops required to deliver 1 mL varies with the type of administration set. Administration sets are of two types:

1. Macrodrip set (delivers 10–20 drops/mL)
2. Microdrip set (60 drops/mL).

Flow rate = Volume of infusion in mL × Drip factor (in drops/mL)/Time of infusion in minutes.

INTRAVENOUS CANNULA

Used for administering intravenous fluids, drugs, and blood products.

Size	Color	Length mm	Flow rate (mL/min)	Uses
14G	Orange	45	250–300	• Used for adolescent and adult major surgery and trauma • Infusion of large amount of fluids and colloids
16G	Gray	45	150–240	• Adolescent and adult major surgery and trauma • Infusion of large amount of fluids or colloids
18G	Green	45	100–120	• Adolescent and adult major surgery and trauma • Infusion of large amount of fluids or colloids
20G	Pink	32	55–80	• Older children, adolescent, and adult • Ideal for IV Infusion or blood infusion • Medication administration • Emergency management
22G	Blue	25	22–50	• Older children, adolescent, and elderly adult • IV Infusion with moderate flow rate • Medication administration
24G	Yellow	19	23	• Infant, toddler, and older children • Major surgery and trauma among children • Can administer fluid and medications
26G	Violet	19	10–15	• Neonate, infants, and elderly adults • Suitable for infusion but infusion rate is low

OXYGEN MASK

Used for administering oxygen.

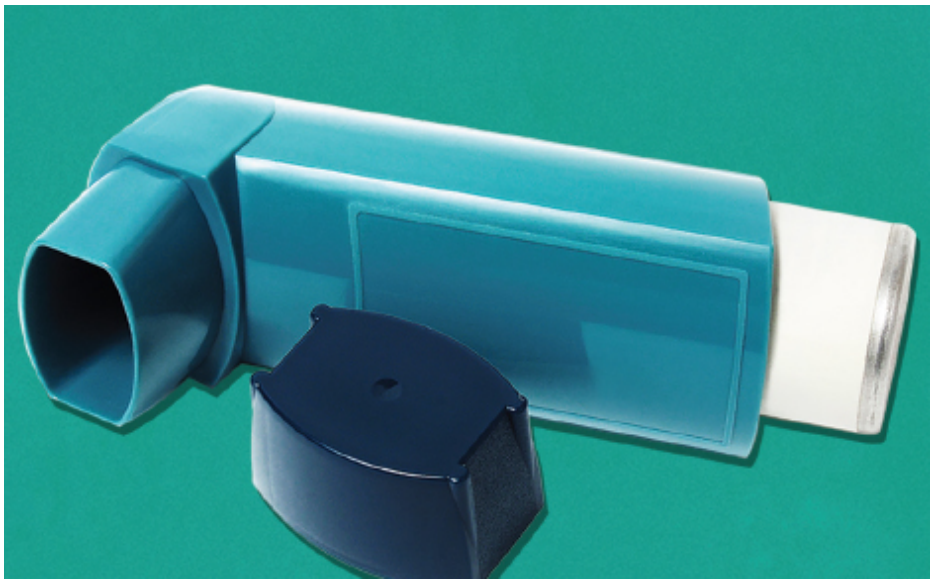
An **oxygen mask** provides a method to transfer breathing oxygen gas from a storage tank to the lungs. Oxygen masks may cover only the nose and mouth (oral nasal mask) or the entire face (full-face mask). They may be made of plastic, silicone, or rubber.

INHALER DEVICES

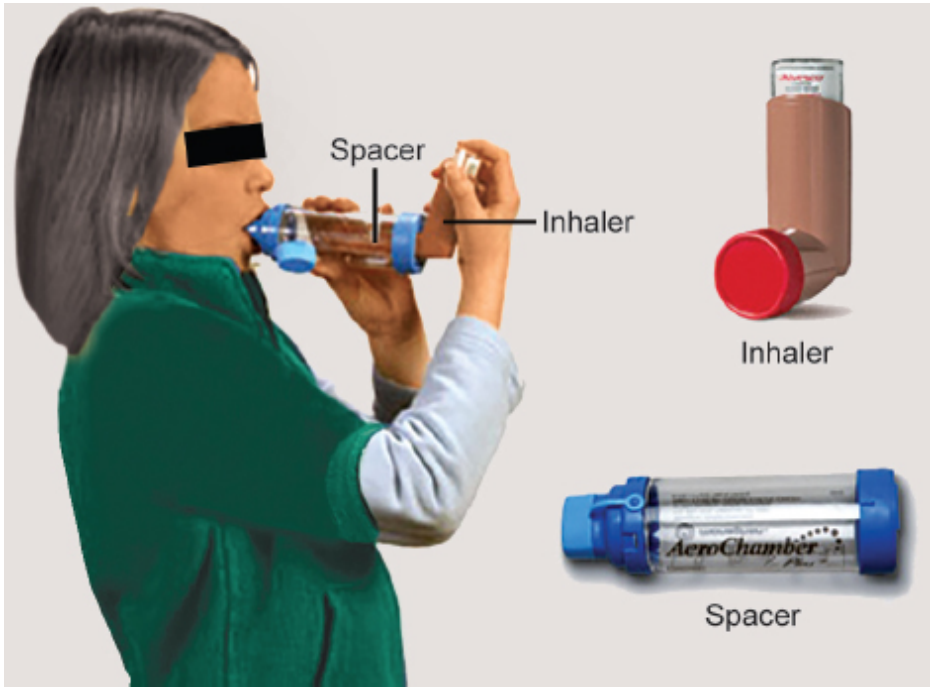
It can be meter dose inhaler, dry powder inhalers, or nebulizers.

Inhalant Drugs

- Broncodilators—salbutamol, formeterol, ipratropium, tiotropium
- Corticosteroids—beclomethasone, budesonide, and fluticasone
- Mucolytic agents—acetylcysteine
- Antimicrobials—ribavirin and tobramycin
- Immune modulators—cyclosporine and interferon α
- Anesthetics—opioids.



Metered Dose Inhaler



Spacer

A **spacer** is a device used to increase the ease of administering aerosolized medication from a metered dose inhaler (MDI). It adds space in the form of a tube or “chamber” between the mouth and canister of medication. Most spacers have a one-way valve that

allows the person to inhale the medication while inhaling and exhaling normally; these are often referred to as **valved holding chambers** (VHC).

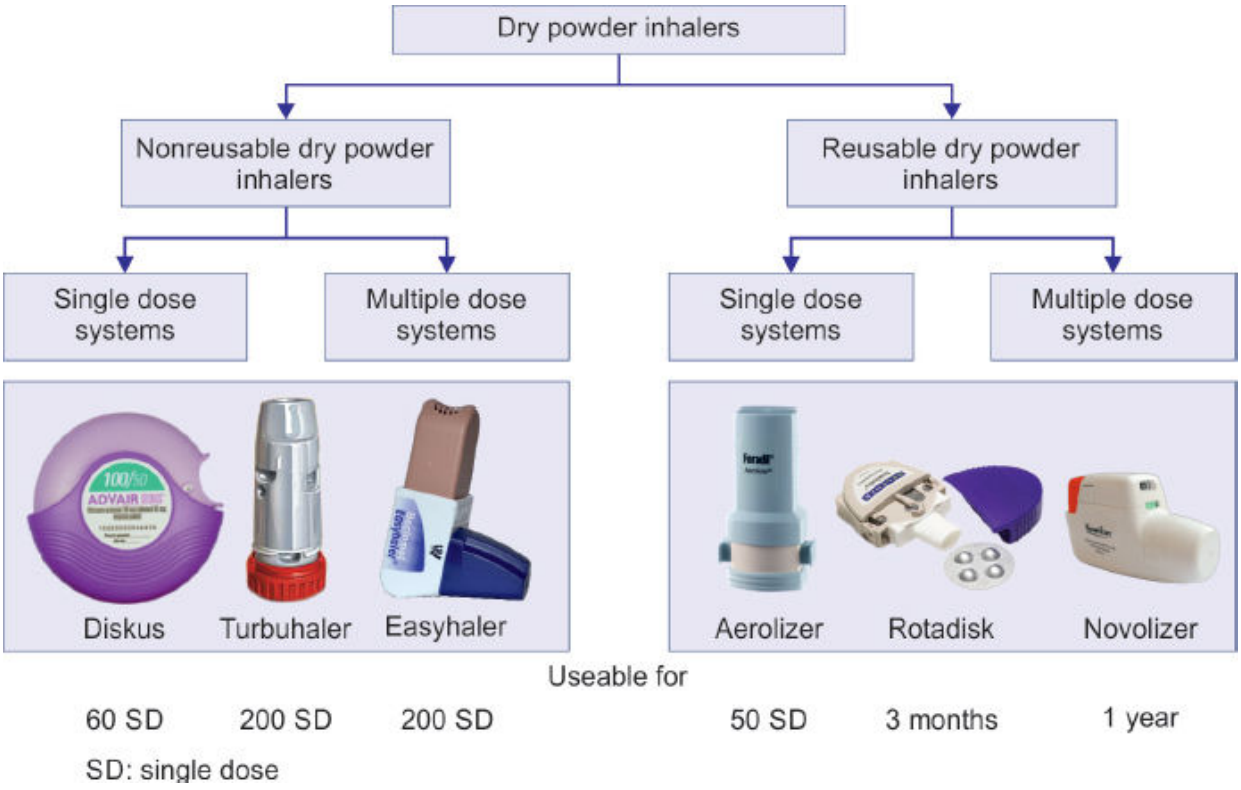
Metered dose inhaler	
Advantages	***Disadvantages***
• Rapid application • Handling • Multidose	• Hand-breathe coordination • Ineffective use in poor ventilated patients • Oropharyngeal deposition and local side effects

Dry Powder Inhalers

Dry powder inhalers	
Advantages	***Disadvantages***
• Less patient coordination required • Spacer not necessary • Compact portable • No propellant • Usually, higher lung deposition than a pressurized metered dose inhaler (pMDI)	• Work poorly if inhalation is not forceful enough • Many patients cannot use them correctly • Most types are moisture sensitive • Need to reload capsule each time

NEBULIZERS

Nebulizers	
Advantages	***Disadvantages***
• Provide therapy for patients who cannot use other inhalation modalities (e.g. MDI and DPI) • Allow administration of large doses of medicine • Patient coordination not required • Effective with tidal breathing • Dose modification possible • Can be used with supplemental oxygen	• Decreased portability • Longer set-up and • Administration time • Higher cost • Electrical power source required • Contamination possible



URINOMETER

Urinometer is an instrument used to measure the specific gravity of urine.

There are three parts of urinometer. They are as illustrated in the Figure above:

1. ***The float:*** It is the air containing part
2. ***Weight:*** The lower end of urinometer
3. ***Stem:*** It has calibrations with numbers marked to measure the specific gravity.

Normal values of specific gravity are 1.003–1.030. It signifies the relative mass density. Specific gravity of urine is a measure of concentrating ability of kidneys and is determined to get information about its tubular function.

Increased-Specific Gravity in Urine

Diabetes mellitus, nephritic syndrome, fever, and dehydration.

Decreased-specific gravity in urine

Diabetes insipidus, chronic renal failure (low and fixed at 1.010) due to loss of concentrating ability of tubules, and compulsive water drinking.

Isosthenuria

This is condition where there is fixed specific gravity. The specific gravity of the urine remains at 1.010 regardless of the volume of water consumption by the person. It occurs specifically in chronic renal disease.

WESTERGREN TUBE

The Westergren method requires collecting 2 mL of venous blood into a tube containing 0.5 mL of sodium citrate. It should be stored no longer than 2 hours at room temperature or 6 hours at 4°C. The blood is drawn into a Westergren-Katz tube to the 200 mm mark. The tube

is placed in a rack in a strictly vertical position for 1 hour at room temperature, at which time the distance from the lowest point of the surface meniscus to the upper limit of the red cell sediment is measured. The distance of fall of erythrocytes, expressed as millimeters in 1 hour, is the erythrocyte sedimentation rate (ESR).

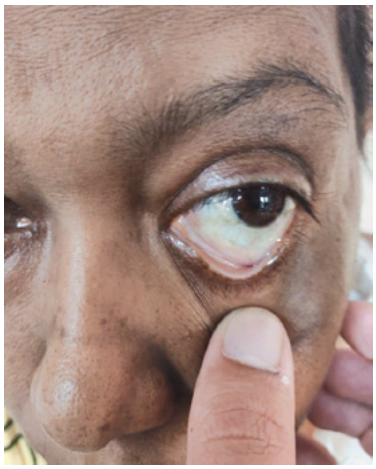


Fig. 14.1: Pallor.

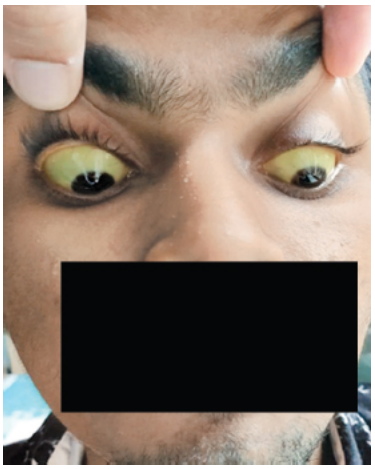


Fig. 14.2: Icterus.

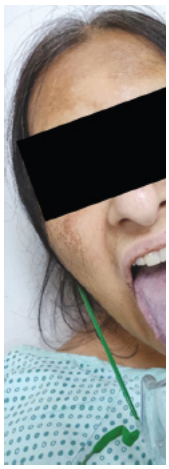


Fig. 14.

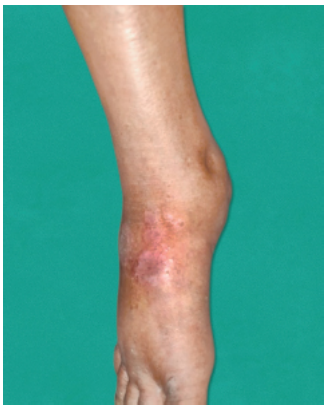


Fig. 14.4: Pitting edema.

Fig. 14.5: Clubbing.

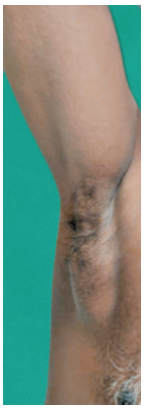
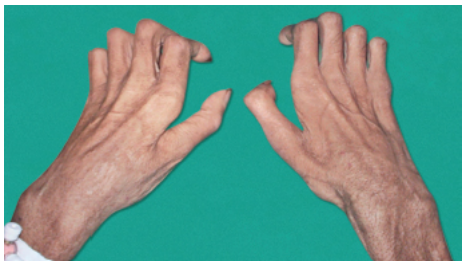


Fig. 14.6: Axilla



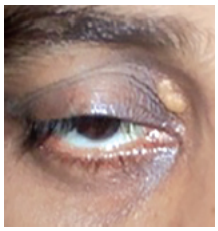


Fig. 14.9:

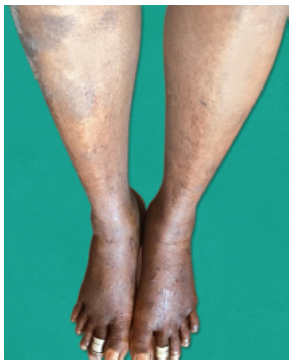


Fig. 14.7: Nonpitting type of pedal edema.

Fig. 14.8: Claw hand.

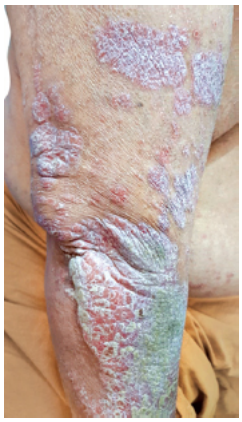


Fig. 14.10: Psoriasis.

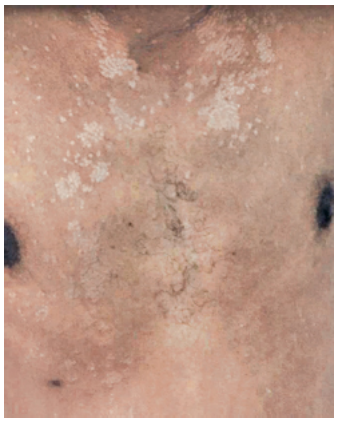


Fig. 14.11: Pityriasis versicolor (tinea versicolor).

Fig. 14

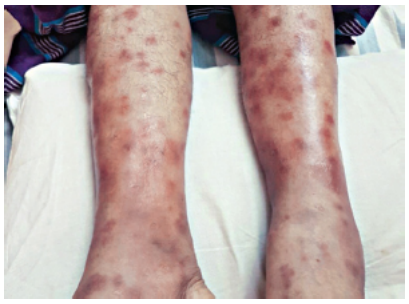


Fig. 14.13: Erythema nodosum.

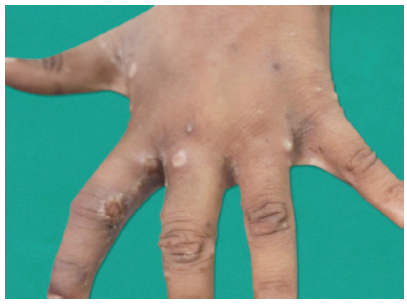


Fig. 14.14: Scabies.

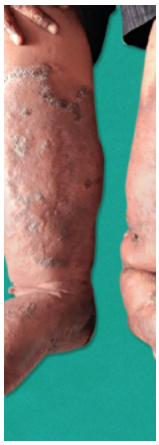


Fig. 14.1

Fig. 14.16: Acanthosis nigricans and skin tags.

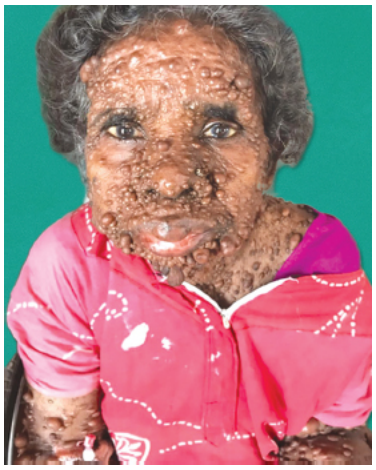


Fig. 14.17: Neurofibromatosis.

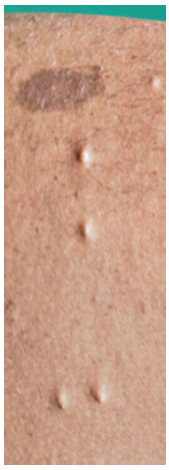
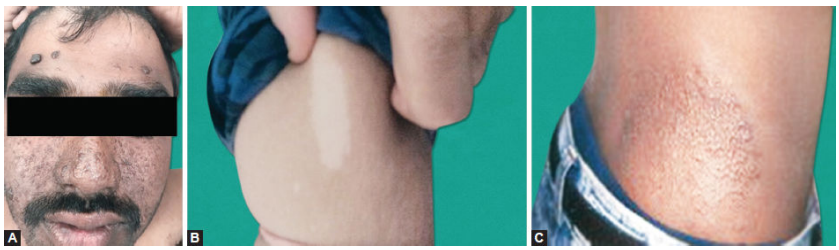
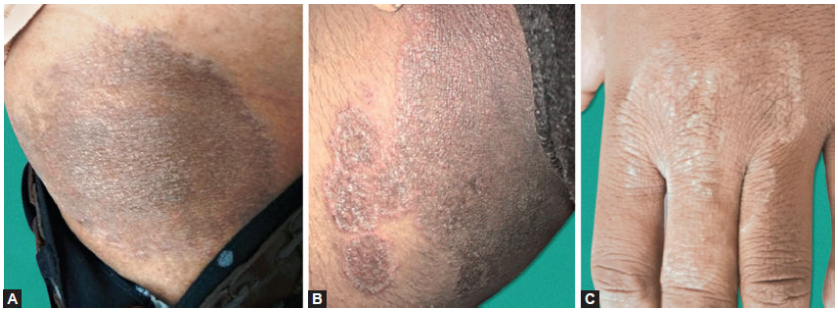


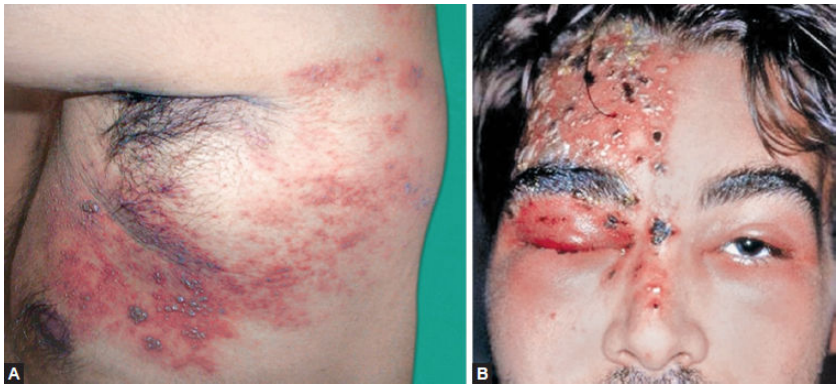
Fig. 14.18: Café-a



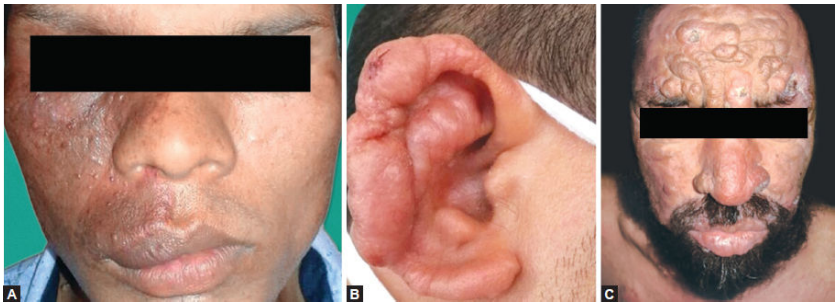
Figs. 14.19A to C: (A) Adenoma sebaceum; (B) Ash leaf-shaped macule is a hypopigmented macule—oval at one end and pointed at patches—tuberous sclerosis.



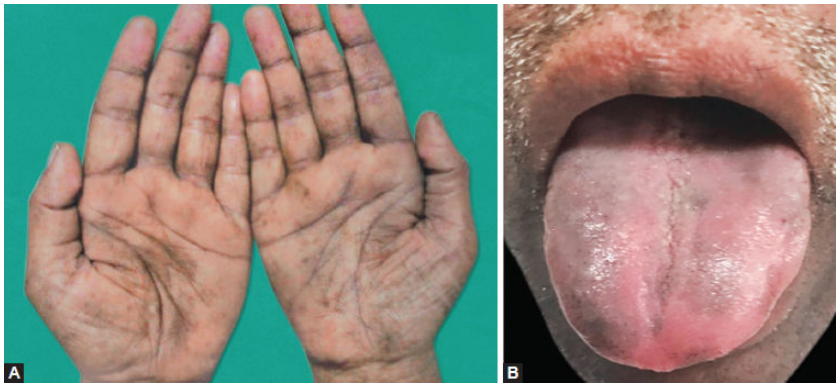
Figs. 14.20A to C: (A) Tinea corporis; (B) Tinea cruris; (C) Tinea manuum.



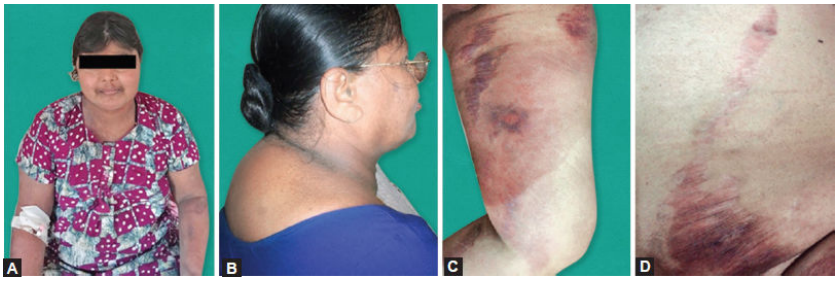
Figs. 14.21A and B: (A) Herpes zoster—dermatomal involvement; (B) Herpes zoster ophthalmicus.



Figs. 14.22A to C: Lesions of lepromatous leprosy. (A) Facial involvement; (B) Nodular lesions on ear; (C) Leon



Figs. 14.23A and B: (A) Pigmentation of palms; (B) Oral pigmentation in Addison's disease.



Figs. 14.24A to D: Features of Cushing's syndrome. (A) Cushing's habitus, obesity, and moon facies; (B) Buffalo hump; (C a

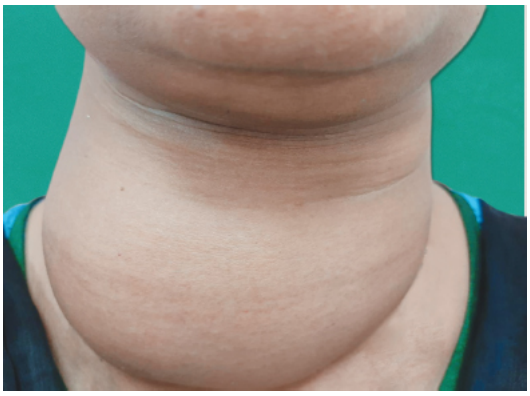
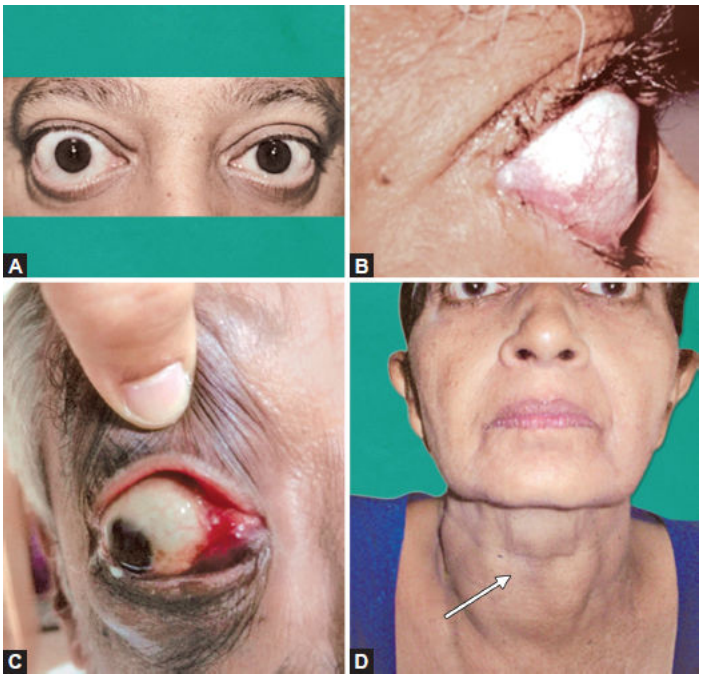
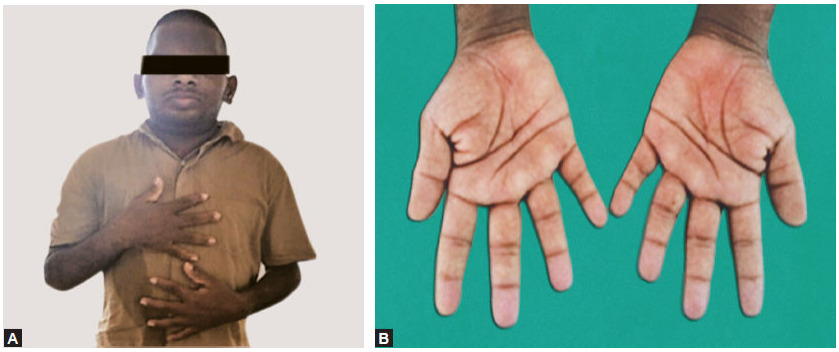


Fig. 14.25: Thyromegaly.



Figs. 14.26A to D: (A and B) Exophthalmos (front and side view); (C) Infiltration of extraocular muscles in hyperthyroidism; (D) Eye si
(arrow).



Figs. 14.27A and B: (A) Acromegalic facies; (B) Thick and spade-shaped hands.

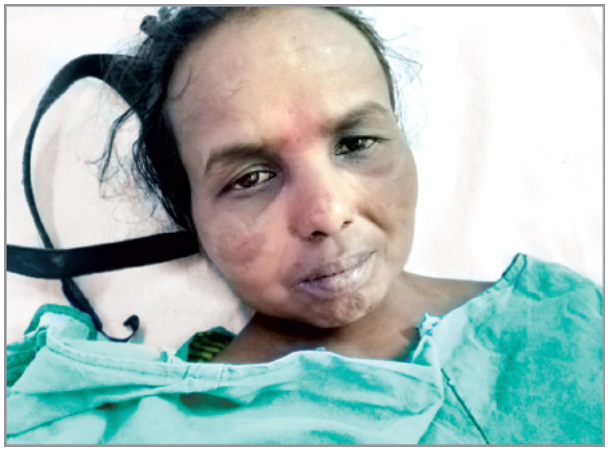


Fig. 14.28: Systemic lupus erythematosus— malar rash, alopecia.

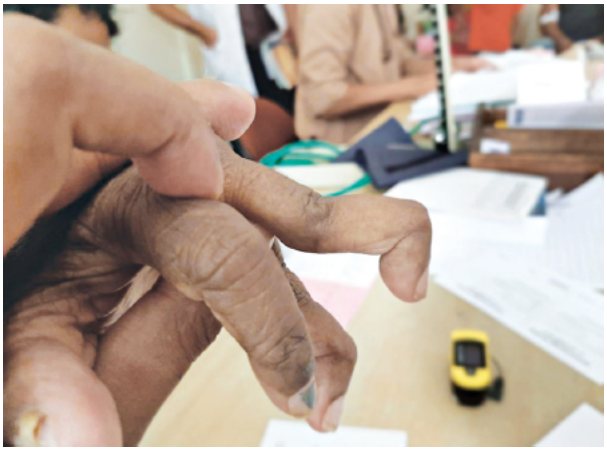


Fig. 14.29: Rheumatoid hand.

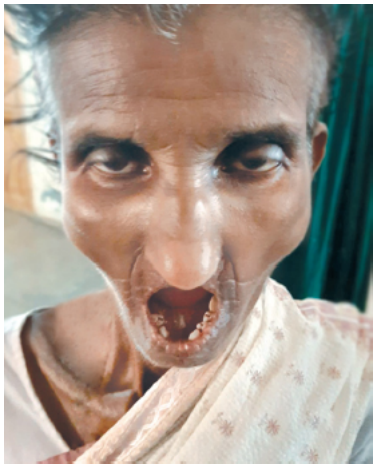
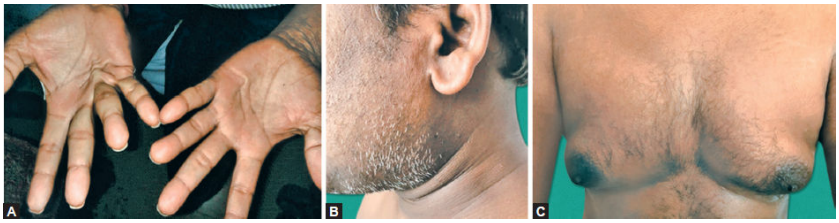


Fig. 14.30: Scleroderma facies.



Figs. 14.31A to C: Features of cirrhosis. (A) Palmar erythema with Dupuytren's contracture; (B) Diminished facial hair with parotid enlargement; (C) Gynecomastia.

Fig. 14.32: Parkinson's hand tremors.

Fig. 14.33: Parkinson's facies.

CHAPTER 15

Annexures

A. MISCELLANEOUS TOPICS

List of miscellaneous topics which are discussed include:

1. History taking framework
2. Pedigree charts
3. Alcohol usage
4. Smoking

HISTORY TAKING FRAMEWORK

Eliciting a history from a patient is an essential part of being an effective doctor. Books have been written about how to consult with patients and there is no set “right way”. Eliciting a patient’s history does not need to be done in a set order. Taking a history is not just going down a checklist of symptoms.

It does include active listening to the patient, awareness of nonverbal communication, with respect and support of their feelings. It is not just information gathering from the patient; it is a two-way communication in which you need to be aware of what and how you are communicating and its impact on the patient.

Ineffective communication is the most common reason for complaints against doctors. The majority of malpractice allegations arise from communication errors. History taking:

- Establishes the doctor-patient relationship
- Explores the patient’s ideas and concerns about the illness and their expectations of the doctor
- Identifies the patient’s physical, psychological, and social environment
- Often leads to diagnosis.

Respect Patient’s Confidentiality

Remember to introduce yourself and state the purpose of the interview and approximate time needed.

What the patient will be discussing with you may be very personal to them, so remember to stress that the interview is confidential.

Presenting Complaint

This is the opening question.

Some options include:

- What has the problem been?
- What made you go to the doctor?
- Can you tell me how you came to be in hospital?

History of Presenting Complaint

This is the main part of the history and you will need to spend time discussing this with the patient. Usually has two parts:

1. A description and exploration of the patient's problem
2. How the problem affects the patients personally.

1. **A description and exploration of the patient's problems:** Allow the patient to tell you in his/her words; this can take a couple of minutes of uninterrupted talk from the patient.

   Allowing the patient to talk without interruption enhances patient satisfaction and efficacy of the interview. They are likely to need verbal and nonverbal encouragement from you to maintain the flow.

   Next, think about trying to direct your line of questioning to test diagnostic hypotheses at this stage. For example, with a patient who has a chest pain, it is important to assess if the pain comes with exercise (if you suspect they have angina).

   You can ask some leading questions for clarification like:

   – When did the problem start?
   – Is it a new or old problem?
   – How often does it occur?
   – What starts it off?
   – How long does it last?
   – What makes is worse?
   – What makes it better?
   – Does anything else happen to you at the same time, before or after?
   – What medicines have you tried?
   – What effect have they had?

2. **How the problem affects the patient personally:** This should not be forgotten! This also connects with the assessment of mental state, particularly inquiring about symptoms of depression.

   You can ask:

– How has this illness affected you generally?
– How does this make you feel overall?

It is important to find out about the patient's own interpretation of their illness. Of equal importance, patients will not be satisfied if their concerns have not been listened to and addressed.

Systemic (or Functional) Enquiry

As illnesses affect different parts of the body and many illnesses may be multisystemic, it is important to ask about connected symptoms.

You need to cover the following areas:

- Respiratory systems: Dyspnea, wheeze, cough, sputum, hemoptysis, and chest pain.
- Cardiovascular systems: Chest pain, orthopnea, paroxysmal nocturnal dyspnea, ankle swelling, palpitations, and intermittent claudication.
- Gastrointestinal system: Abdominal pain, nausea, vomiting, hematemesis, bowel habit, bleeding per rectum, and melena.
- Urogenital system: Frequency, nocturia, polydipsia, loin pain, hematuria, menarche, menopause, cycle, intermenstrual bleeding, and postcoital bleeding.
- Central nervous system: Headaches, visual disturbances, sleep, hearing, tinnitus, lightheadedness, blackouts, fits, unsteady gait, weakness, and paresthesiae.
- Musculoskeletal: Myalgia, arthralgia, back pain, and joint swelling.
- Psychiatric: The mental state examination will be taught more formally in your psychiatric attachment. Remember, depression is common and may often coexist with physical ill health.

Past Medical History

Always ask the patient if they have or have had any serious illnesses. It includes:

- Ask specifically about hypertension, ischemic heart disease, strokes or TIAs, diabetes, asthma, jaundice, TB, and rheumatic fever
- Surgeries
- Blood transfusion
- Hospital admissions.

Family History (FH)

This gives a clue to any predisposition to any illnesses and may highlight specific concerns the patient may have about a certain disease. Ask the patient if their parent(s) suffered from any illness, if not alive then ask what they died from and at what age. Then ask similar questions about brothers and sisters and children.

Social History/Personal History

This is a very important part of the patient's history. Remember to ask about:

- Social life
- Diet
- Alcohol
- Smoking
- Recreational drug use
- Occupation.

It is also important to assess a patient's "activities of daily living". This is an assessment of how much support a patient requires to live on a day-to-day basis.

It includes asking about:

- Help with dressing
- Help with washing/toileting
- Heal with eating
- Help with walking
- Help with shopping.

Drug History

List all of the patient's drugs and doses. Remember over the counter and alternative medicines. Some patients can be quite vague about their tablets-try and persevere.

Drug Allergy

Identify any drug allergies the patient may have and details of what happens, for example, rash or anaphylaxis.

For female patients—menstrual history and obstetric history.

Summary

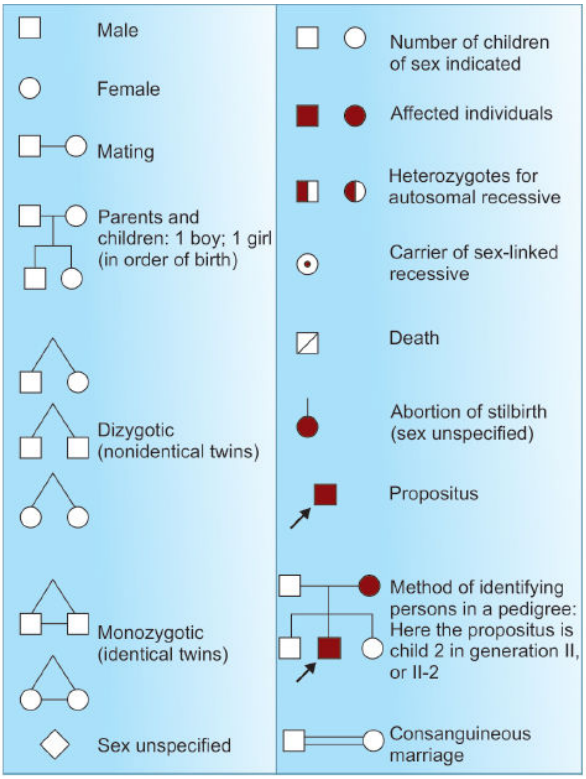
At the end of presenting a history, you will often be asked to give a summary. Prepare two to three sentences to summarize the patient's

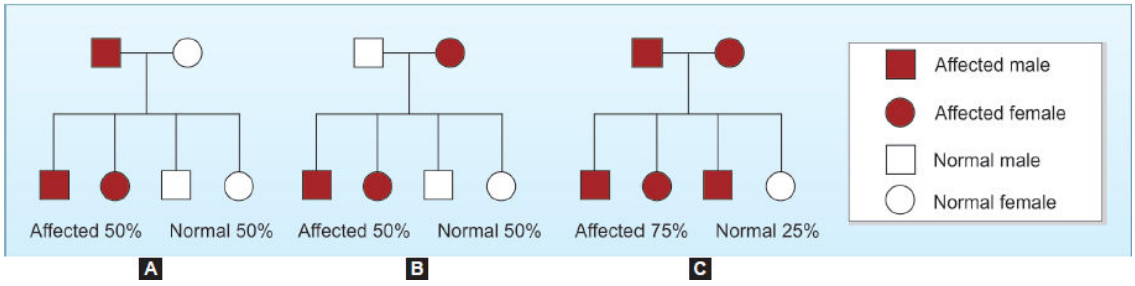
problems including, if you can give a provisional diagnosis or differential diagnosis.

PEDIGREE ANALYSIS (FIGS. 15A.1 TO 15A.4, AND TABLES 15A.1 AND 15A.2)

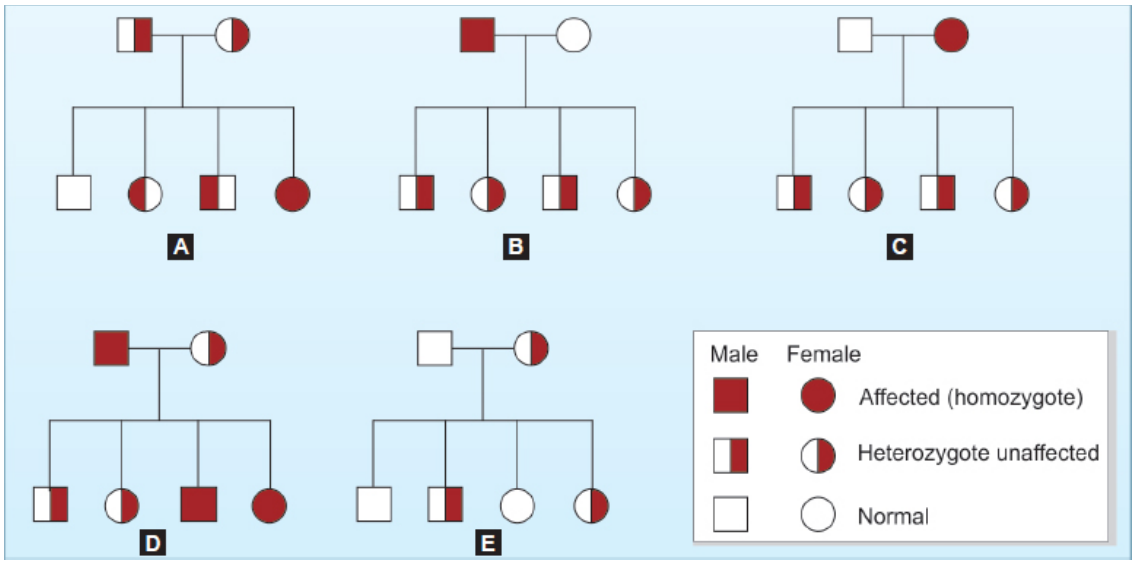
A pedigree chart displays a family tree, and shows the members of the family who are affected by a genetic trait.

- Circles represent females and squares represent males.
- Each individual is represented by: A Roman Numeral, which stands for the generation in the family and a Digit, which stands for the individual within the generation.
- A darkened circle or square represents an individual affected by the trait.
- A male and female directly connected by a horizontal line have mated and have children.
- Vertical lines connect parents to their children.
- The “founding family” consists of the two founding parents and their children.

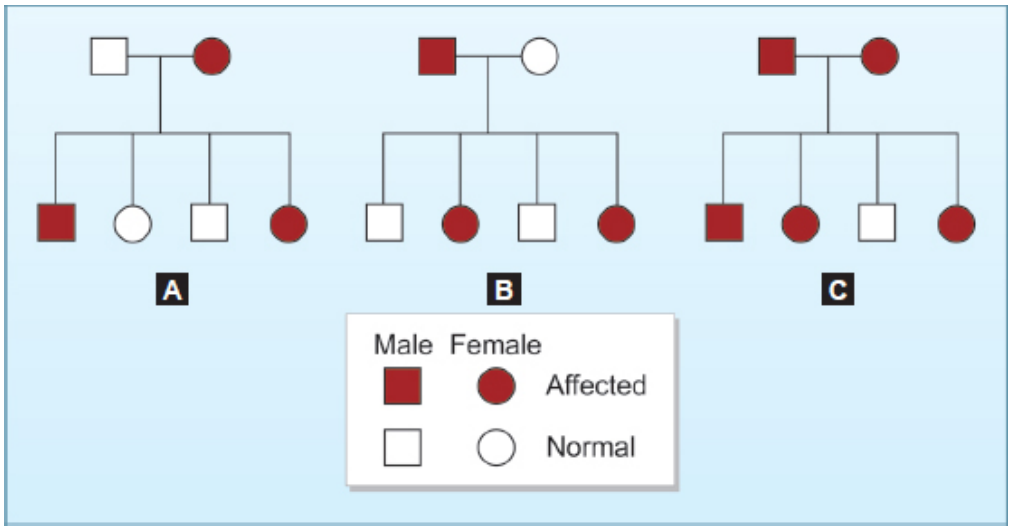




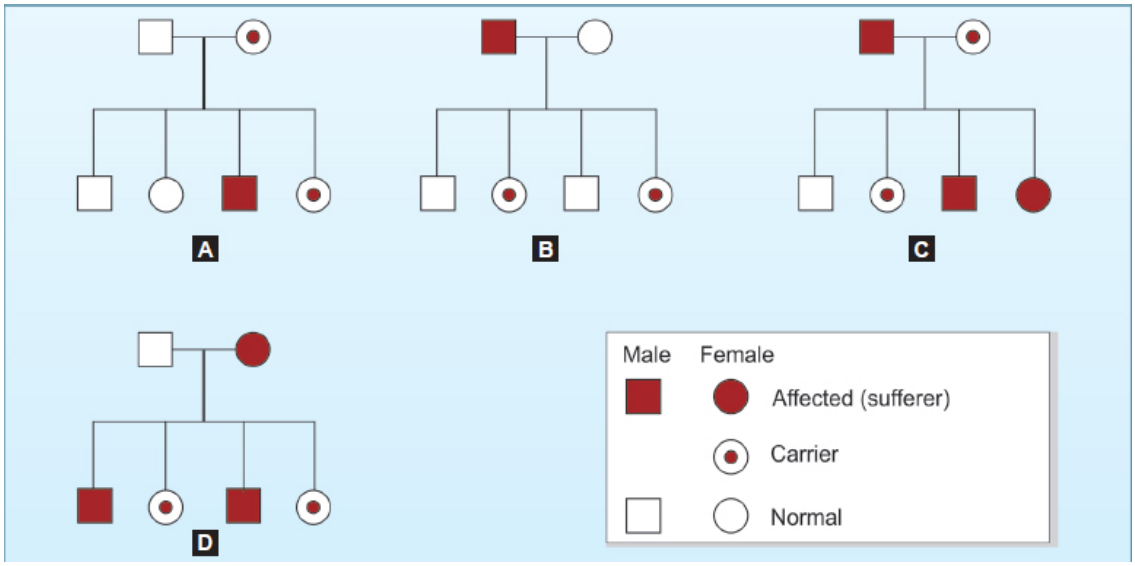
Figs. 15A.4A to C: Pedigree illustrating **autosomal dominant transmission**. (A and B) One parent is affected; (C) Both parents are affected. Note that both males and females are affected equally.



Figs. 15A.2A to E: Pedigree illustrating mechanism of **autosomal recessive transmission**. (A) Both parents are unaffected heterozygotes; (B and C) One parent is sufferer (homozygous) and other is normal; (D) One parent is sufferer and other is unaffected heterozygote; (E) One parent is normal and other is an unaffected heterozygote.



Figs. 15A.3A to C: X-linked dominant transmission. Only females are affected. Usually males who inherit the mutant allele die in utero. (A) Normal male and affected female (sufferer); (B) Affected male and female; (C) Both male and female are affected.



Figs. 15A.4A to D: Mode of **X-linked recessive transmission**. Note the absence of male-to-male transmission. (A) Male is normal and female is a carrier; (B) Male is sufferer and female is normal; (C) Male is a sufferer and female is a carrier; (D) Male is normal and female is sufferer.

Table 15A.1: Examples of automosal dominant and autosomal recessive disorders.

System	*Autosomal dominant disorder*	*Autosomal recessive disorder*
Nervous	Huntington disease Neurofibromatosis Tuberous sclerosis	Neurogenic muscular atrophies Friedreich's ataxia Spinal muscular atrophy
Skeletal	Marfan syndrome Achondroplasia Noonan syndrome	Alkaptonuria Ehlers-Danlos syndrome
Metabolic	Familial hypercholesterolemia Intermittent porphyria	Cystic fibrosis, phenylketonuria, lysosomal storage diseases, galactosemia, hemochromatosis, glycogen storage diseases
Hematopoietic	Hereditary spherocytosis von Willebrand disease	Sickle cell anemia, thalassemia
Renal	Polycystic kidney disease	Congenital adrenal hyperplasia
Gastrointestinal	Familial polyposis	Wilson's disease

	coli	

Table 15A.2: Examples of x-linked recessive disorders.	
System	***Related x-linked recessive disease***
Musculoskeletal	Duchenne muscular dystrophy
Blood	Hemophilia A and B
	Glucose-6-phosphate dehydrogenase deficiency
Immune	Agammaglobulinemia
Metabolic	Diabetes insipidus
Nervous	Fragile-X syndrome

ALCOHOL USE (TABLE 15A.3)

1 unit of alcohol contains 8 g of ethanol.

A conservative threshold of 14 units/week for both men and women is considered safe.

The risk threshold for developing ALD is variable but begins at 30 g/day of ethanol.

The average alcohol consumption of a man with cirrhosis is 160 g/day for over 8 years.

Some of the risk factors for ALD are:

- **Drinking pattern**: Liver damage is more likely to occur in continuous rather than intermittent or “binge” drinkers, as this pattern gives the liver a chance to recover. It is therefore recommended that people should have at least two alcohol-free days each week.
- **Gender:** The incidence of ALD is increasing in women, who have higher blood ethanol levels than men after consuming the same amount of alcohol. This may be related to the reduced volume of distribution of alcohol.
- **Genetics:** Alcoholism is more concordant in monozygotic than dizygotic twins. The patatin-like *phospholipase domain-containing protein 3 (PNPLA3)* gene, also known as adiponutrin, has been implicated in the pathogenesis of both ALD and NAFLD.
- **Nutrition**: Obesity increases the incidence of liver-related mortality by over five-fold in heavy drinkers. Ethanol itself produces 7 kcal/g (29.3

kJ/g) and many alcoholic drinks also contain sugar, which further increases the calorific value and may contribute to weight gain.

Units of alcohol explained:

A UK unit is 10 milliliters (8 g) of pure alcohol

For example, most whisky has an ABV (alcohol by volume) of 40%.

1 liter (1,000 mL) bottle of this whisky therefore contains 400 mL of pure alcohol. This is 40 units (as 10 mL of pure alcohol = one unit).

So, in 100 mL of the whisky, there would be 4 units.

And hence, a 25 mL single measure of whisky would contain 1 unit.

The maths is straightforward. To calculate units, take the quantity in milliliters, multiply it by the ABV (expressed as a percentage) and divide by 1,000.

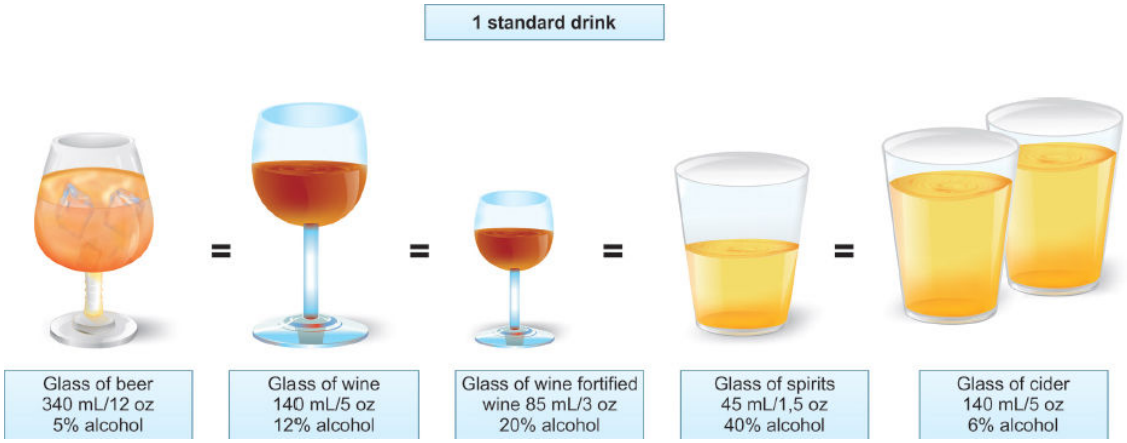


Fig. 15A.5: Description of one standard drink based on different beverages.

Table 15A.3: Amount of alcohol in an average drink.

Alcohol type	*% Alcohol by volume*	*Amount*	*Units**
Beer	3.5 9	568 mL (1 pint) 568 mL (1 pint)	2 4
Wine	10 12	125 mL 750 mL	1 9
'Alcopops'	6	330 mL	2
Sherry	17.5	750 mL	13
Vodka/rum/gin	37.5	25 mL	1

Whisky/brandy	40	700 mL	28

*1 unit = 8 g

In the example of a glass of whisky (above), the calculation would be: **25 mL × 40% divided by 1,000 = 1 unit** Or for a 250 mL glass of wine with ABV 12%, the number of units is: **250 mL × 12% divided by 1,000 = 3 units.** A 330 mL bottle of lager (ABV 5%) contains: **330 mL × 5% divided by 1,000 = 1.65 units.**

Complications of Alcohol

Neurologic
- Blackouts
- Withdrawal syndromes (e.g. tremors, hallucinations, rum fits, and delirium tremens)
- Cerebellar degeneration
- Alcoholic dementia
- Alcoholic myopathy
- Autonomic neuropathy
- Peripheral neuropathy
- Marchiafava–Bignami disease (demyelination of corpus callosum)
- Central pontine myelinolysis
- Traumatic brain injury
- Hepatic encephalopathy
- Hemorrhagic stroke
- Seizures

Cardiovascular Cardiomyopathy Cardiac arrhythmias (holiday heart syndrome), and atrial fibrillation Hypertension	**Gastrointestinal** Acute gastric erosions GI bleeding—Mallory–Weiss tears, gastric erosions, esophageal varices, and peptic ulcers Pancreatitis (acute, recurrent or chronic) Diarrhea • Watery diarrhea due to alcohol itself • Steatorrhea due to pancreatitis or alcoholic liver disease Hepatomegaly (alcoholic hepatitis, fatty liver, and chronic liver disease) Chronic liver disease and associated complications
Respiratory Increased susceptibility to pneumonia and tuberculosis	**Musculoskeletal** Increased risk of fractures and osteonecrosis of femoral head Increased risk of fall Myopathy Osteoporosis

Cancers Oral cavity Oropharynx Esophageal Colorectal Breast Hepatocellular carcinoma Pancreatic	**Metabolic** Hyponatremia Hypoglycemia Hypokalemia Hypomagnesemia Hypocalcemia Hypophosphatemia Gout Hypercholesterolemia Ketoacidosis
Psychiatric Unipolar depressive disorders Anxiety Chronic suicidality Amnestic disorder Psychosis Cognitive impairment Impulsivity	**Behavioral and psychosocial** Injuries Violence Crime Partner or child abuse Tobacco and other drug abuse Unemployment Legal problems Poor hygiene
Hematologic Anemia Iron deficiency from blood loss Dietary folate deficiency B_{12} deficiency with pancreatitis Direct toxic suppression of bone marrow Sideroblastic anemia Zieve's syndrome (hemolytic anemia) Thrombocytopenia due to bone marrow suppression or hypersplenism Leukopenia	**Nutritional** Thiamine deficiency—Wernicke's encephalopathy, Korsakoff psychosis, and peripheral neuropathy Niacin deficiency—Pellagra Folate deficiency B_{12} deficiency Vitamin D deficiency Zinc deficiency
Endocrine Diabetes mellitus Gynecomastia Testicular atrophy Amenorrhea Infertility	**Miscellaneous** Erectile dysfunction Fetal alcohol syndrome Spontaneous abortions Increased susceptibility to infections like HIV

SMOKING

- Cigarette smoking is the leading preventable cause of mortality, responsible for nearly 6 million deaths worldwide.
- The three major causes of smoking-related mortality are atherosclerotic cardiovascular disease, lung cancer, and chronic obstructive pulmonary disease (COPD).

Pack years = number of packs of cigarettes smoked per day × number of years the patient has smoked

More pack years correlates with higher lung disease risk including lung cancer.

Patients should be considered for screening with low-dose CT if they are ≥55 years with ≥30 pack years history.

Pack years = No. of packs of cigarettes/day × No. of years smoked

Smoking index is defined as the product of average number of cigarettes smoked per day and the total duration of smoking in years.

Example: If a patient is smoking 1 cigarette per day for 10 years the smoking index will be 10.

Smoking index (si) = No. of cigarettes/day × No. of years smoked
SI <100 = Mild smoker
SI <101–300 = Moderate smoker
SI <300 = Heavy smoker

Lung cancer is common if Smoking index more than 300

Complications of Tobacco Use

Cardiovascular disease • Premature coronary artery disease • Peripheral vascular disease and erectile dysfunction • Cerebrovascular disease • Aortic aneurysm	**Respiratory disease** • Chronic obstructive pulmonary disease • Cancer of lung, bronchus, and trachea • Increased incidence of postoperative respiratory complications • Increased incidence of respiratory infections including tuberculosis • ILD • Pneumothorax
Gastrointestinal • GERD	**Pregnancy** • Spontaneous abortion

• Peptic ulceration • Gallstones and cholecystitis in women • Pancreatitis • Crohn's disease	• Abruptio placentae • Premature rupture of membranes • Fetal death • Neonatal death • Sudden infant death syndrome • Postpartum venous thromboembolism
Renal • Increased risk of CKD	**Endocrine** • Increased risk of diabetes mellitus
Infections—increased risk of several types of infection including tuberculosis, pneumococcal pneumonia, Legionnaires' disease, meningococcal disease, influenza, and the common cold	**Osteoporosis and hip fracture**—smoking accelerates bone loss and is a risk factor for hip fracture in women
Neurological • Dementia and cognitive decline • Increased risk of amyotrophic lateral sclerosis	**Ophthalmological** • Age-related macular degeneration • Increased risk of cataract
Drug interactions • Induces hepatic microsomal enzyme systems, e.g. increased metabolism of propranolol and theophylline	
Other cancers • Larynx • Oral cavity and lip • Nasopharynx, oropharynx, and hypopharynx • Nasal cavity and paranasal sinus • Esophagus • Stomach • Pancreas • Colorectal • Kidney • Bladder • Uterine • Cervix • Acute myeloid leukemia	

B. DEFINITIONS

PULSE

Pulse is the pressure distension wave produced by contraction of left ventricle against a partially filled aorta, which is transmitted to peripheries and is felt on a peripheral artery against a bony prominence.

BLOOD PRESSURE

Arterial blood pressure (BP) can be defined as the lateral pressure exerted by the moving column of blood on the walls of the arteries **(Table 15B.1)**.

BP = Cardiac output × Peripheral resistance

Systolic BP (SBP) Defined as the maximum BP in the arteries Attainable during systole Normal 120+/–20 mm Hg	**Diastolic BP (DBP)** Defined as the minimum pressure that is obtained at the end of the ventricular diastole. Normal range 60–90 mm Hg
Pulse pressure (PP) Denotes the difference between systolic and diastolic pressure. PP = SBP − DBP = 40 mm Hg	**Mean arterial pressure (MAP)** DBP + 1/3 pulse pressure normal = 95 mm Hg

Table 15B.1: Blood pressure measurement and definitions.

BP measurement	*Definition*
SBP	First Korotkoff sound
DBP	Fifth Korotkoff sound
Pulse pressure	SBP minus DBP
Mean arterial pressure	DBP pulse one-third pulse pressure
Mid-BP	Sum of SBP and DBP, divided by 2

Reference

Williams B, Mancia G, Spiering W, et al. 2018 ESC/ESH Guidelines for the management of arterial hypertension. Eur Heart J. 2018;39(33):3021-104.

HYPERTENSION

"Hypertension" is defined as the level of BP at which the benefits of treatment (either with lifestyle interventions or drugs) unequivocally outweigh the risks of treatment, as documented by clinical trials.

Reference

Williams B, Mancia G, Spiering W, et al. 2018 ESC/ESH Guidelines for the management of arterial hypertension. Eur Heart J. 2018 1;39(33):3021-104

Hypertension is most commonly defined as systolic blood pressure (SBP) ≥140 mm Hg or diastolic blood pressure (DBP) ≥90 mm Hg, but definitions vary by professional organization.

ACC/AHA - >130/80

ESC/ESH - >140/90.

RESISTANT HYPERTENSION

Elevated blood pressure despite concurrent use of three antihypertensive drugs of different classes including a diuretic.

Reference

Williams B, Mancia G, Spiering W, et al. 2018 ESC/ESH Guidelines for the management of arterial hypertension. Eur Heart J. 2018;39(33):3021-104.

REFRACTORY HYPERTENSION

A subgroup of patients with resistant hypertension that remains uncontrolled despite maximal medical therapy, often with four or more antihypertensive drugs.

Reference

Rimoldi SF, Scherrer U, Messerli FH. Secondary arterial hypertension: when, who, and how to screen? Eur Heart J. 2014;35(19):1245-54.

PSEUDORESISTANT HYPERTENSION

- Elevated blood pressure measurements due to inaccurate blood pressure measurement techniques such as:
  - Failure to have patient sit quietly for ≥5 minutes before measurement
  - Too small cuff size.
- Poor adherence to medical therapy
- White coat hypertension
- Marked brachial artery calcification
- Clinician inertia.

References:

- *Rimoldi SF, Scherrer U, Messerli FH. Secondary arterial hypertension: when, who, and how to screen? Eur Heart J. 2014;35(19):1245-54.*
- *Williams B, Mancia G, Spiering W, et al. 2018 ESC/ESH Guidelines for the management of arterial hypertension. Eur Heart J. 2018;39(33):3021-104.*

PSEUDOHYPERTENSION

- Defined as cuff diastolic blood pressure ≥15 mm Hg higher than simultaneously measured intra-arterial blood pressure.
- Elevated blood pressure due to arterial stiffening in elderly patients.

Reference

Rimoldi SF, Scherrer U, Messerli FH. Secondary arterial hypertension: when, who, and how to screen? Eur Heart J. 2014;35(19):1245-54.

SECONDARY HYPERTENSION

Hypertension due to an identifiable and potentially curable cause.

MASKED HYPERTENSION

Elevated blood pressure at home or on ambulatory blood pressure monitoring but normal office blood pressure.

WHITE COAT HYPERTENSION

Normal blood pressure at home or on ambulatory blood pressure monitoring but elevated office blood pressure.

HYPERTENSIVE CRISIS

Severe elevations in blood pressure (systolic blood pressure ≥180 mm Hg or diastolic blood pressure ≥120 mm Hg) with impending complications including target end-organ dysfunction.

HYPERTENSIVE EMERGENCY

Severe elevation in blood pressure which is accompanied by end-organ damage.

MALIGNANT HYPERTENSION

Malignant hypertension is term used for patients with severely elevated blood pressure and ischemic end-organ damage usually involving the retina, but may also include the kidneys, heart, arteries, and/or brain.

HYPERTENSIVE URGENCY

Severe elevation in blood pressure which occurs without end-organ damage.

References

- *Whelton PK, Carey RM, Aronow WS, et al. 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA Guideline for the Prevention, Detection, Evaluation, and Management of High Blood Pressure in Adults: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. Hypertension. 2018;71(6):e13.*

JVP

Defined as undulating top of oscillating column of blood in right internal jugular vein that faithfully represents the pressure and volumetric changes in the right side of heart which changes with various stages of cardiac cycle and respiration.

ANEMIA

Anemia is a condition in which the number of red blood cells or their oxygen-carrying capacity is insufficient to meet physiologic needs, which vary by age, sex, altitude, smoking, and pregnancy status.

World Health Organization (WHO) definition of anemia at sea level **(Table 15B.2)**:

- Hemoglobin <13 g/dL (130 g/L) in men ≥15 years old
- Hemoglobin <12 g/dL (120 g/L) in nonpregnant women ≥15 years old or adolescents aged 12–14 years
- Hemoglobin <11.5 g/dL (115 g/L) in children aged 5–11 years
- Hemoglobin <11 g/dL (110 g/L) in pregnant women, or children aged 6–59 months.

ERYTHROCYTOSIS AND POLYCYTHEMIA

Erythrocytosis is an increase in the number of red blood cells (relative to the plasma volume), manifested by a persistent increase in the venous hematocrit, and associated with increased blood viscosity and risk of thrombosis.

Erythrocytosis and polycythemia are often used interchangeably; however, erythrocytosis refers exclusively to an increase in erythrocytes, whereas polycythemia more accurately refers to pan-myeloproliferation (as seen in some patients with polycythemia vera).

Table 15B.2: Hemoglobin levels to diagnose anemia at sea level (g/L)±.

	*Non-anemia**		*Anemia**	
Population		***Mild[a]***	***Moderate***	***Severe***
Children 6–59 months of age	110 or higher	100–109	70–99	Lower than 70
Children 5–11 years of age	115 or higher	110–114	80–109	Lower than 80
Children 12–14 years of age	120 or higher	110–119	80–109	Lower than 80
Nonpregnant women (15 years of age and above)	120 or higher	110–119	80–109	Lower than 80
Pregnant women	110 or higher	100–109	70–99	Lower than 70

Men (15 years of age and above)	130 or higher	110–129	80–109	Lower than 80

± *Adapted from* references 5 and 6

* Hemoglobin in grams per liter

a “Mild” is a misnomer: iron deficiency is already advanced by the time anemia is detected. The deficiency has consequences even when no anemia is clinically apparent.

Reference: WHO.

References:

- *Lee G, Arcasoy MO. The clinical and laboratory evaluation of the patient with erythrocytosis. Eur J Intern Med. 2015;26(5):297-302.*
- *McMullin MF, Bareford D, Campbell P, et al. Guidelines for the diagnosis, investigation and management of polycythaemia/erythrocytosis. Br J Haematol. 2005;130(2):174-95.*

JAUNDICE

Jaundice (also termed icterus) is a condition of yellow discoloration of the skin, conjunctivae, and mucous membranes, resulting from widespread tissue deposition of the pigmented metabolite bilirubin.

Reference

Feldman M, Friedman L, Brandt L. Sleisenger and Fordtran’s Gastrointestinal and Liver Disease. Philadelphia: Saunders; 2015.

CYANOSIS

Cyanosis refers to a bluish discoloration of the skin that is caused by increased amounts of reduced hemoglobin in the subpapillary venous plexus.

Reference

Fishman’s Pulmonary Diseases and Disorders.

CLUBBING

Clubbing of the fingers designates the selective bulbous enlargement of the distal segments of the digits due to an increase in soft tissue.

Reference

Fishman’s Pulmonary Diseases and Disorders.

FEVER

Fever is “a state of elevated core temperature, which is often, but not necessarily, part of the defensive responses of multicellular organisms (host) to the invasion of live (microorganisms) or inanimate matter recognized as pathogenic or alien by the host.”

Reference

Commission for Thermal Physiology of the International Union of Physiological Sciences (IUPS Thermal Commission): Glossary of terms for thermal physiology (3rd ed.). Jpn J Physiol. 2001;51:245-80.

FUO

Petersdorf and Beeson—“fever higher than 38.3°C (100.9°F) on several occasions, persisting without diagnosis for at least 3 weeks in spite of at least 1 week’s investigation in hospital”.

REVISED DEFINITION OF FUO

- Requires fever >38.3°C (101°F)
- Subcategorized by patient immune status and clinical setting
  - Classic fever of unknown origin (FUO):
    - » Fever duration >3 weeks
    - » No diagnosis after ≥3 visits or 3 days of hospitalization.
  - Nosocomial (healthcare-associated) FUO:
    - » Fever duration >3 days
    - » Fever acquired after ≥24 hours in hospital (not present or incubating on admission)
    - » No diagnosis after 3 days of appropriate in-hospital investigation.
  - Neutropenic (or immunodeficient) FUO:
    - » Fever duration >3 days
    - » Neutrophil count ≤500 cells/mm^3 with negative cultures after 48 hours
    - » No diagnosis after 3 days of appropriate in-hospital investigation.
  - HIV-associated FUO:
    - » Confirmed HIV infection

» Fever duration >3 weeks for outpatients and >3 days for inpatients.

References:

- *Wright WF, Mackowiak PA. Fever of unknown origin. In: Mandell GL, Bennett JE, Dolin R, (Eds). Mandell, Douglas, and Bennett's Principles and Practice of Infectious Diseases. 8th edition. New York, NY: Saunders; 2014:721-31.*

HYPERPYREXIA

A fever of >41.5°C is called hyperpyrexia.

Reference

Harrison's Principles of Internal Medicine.

HYPERTHERMIA

An uncontrolled increase in body temperature that exceeds the body's ability to lose heat without a change in the hypothalamic set point. Hyperthermia does not involve pyrogenic molecules.

Reference

Harrison's Principles of Internal Medicine.

HEATSTROKE

Core body temperature ≥104°F (40°C) with central nervous system dysfunction; can progress to multiple system organ failure.

Reference

Atha WF. Heat-related illness. Emerg Med Clin North Am. 2013;31(4):1097-108.

DYSPNEA

A subjective experience of breathing discomfort that consists of qualitatively distinct sensations that vary in intensity.

Reference

Parshall MB, Schwartzstein RM, Adams L, et al. An official American Thoracic Society statement: update on the mechanisms, assessment, and management of dyspnea. Am J Respir Crit Care Med. 2012;185(4):435-52.

ORTHOPNEA

Orthopnea signifies dyspnea in the recumbent, but not in the upright or semi-upright position.

Reference
Fishman's Pulmonary Diseases and Disorders.

PAROXYSMAL NOCTURNAL DYSPNEA

Acute episodes of severe shortness of breath and coughing that generally occur at night and awaken the patient from sleep, usually 1–3 hours after the patient retires.

Reference
Harrison's Principles of Internal Medicine.

PLATYPNEA

Platypnea signifies dyspnea induced by assuming the upright position and relieved by recumbency.

Reference
Fishman's Pulmonary Diseases and Disorders.

ORTHODEOXIA

Desaturation of arterial blood when the patient is upright.

Reference
Fishman's Pulmonary Diseases and Disorders.

TREPOPNEA

Dyspnea when the affected side of the chest is in the dependent position, thereby promoting ventilation–perfusion mismatch and resultant hypoxemia.

Reference
Fishman's Pulmonary Diseases and Disorders.

BENDOPNEA

Shortness of breath may be particularly noticeable when bending forward, termed bendopnea.

Reference
Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine.

PALPITATIONS

Palpitations are the awareness of the heartbeat that may be caused by a rapid heart rate, irregularities in heart rhythm, or an increase in the force of cardiac contraction, as occurs with a postextrasystolic beat; however, this perception can also exist in the setting of a completely normal cardiac rhythm.

Reference
Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine.

TACHYCARDIA

An abnormally rapid heartbeat, usually applied to a heart rate above 100 per minute.

Reference
ICD-10.

BRADYCARDIA

The National Institutes of Health defines bradycardia as a heart rate <60 bpm in adults other than well trained athletes.

Reference
National Institutes of Health. Pulse. [online] Available from https://medlineplus.gov/ency/article/003399.htm [Last accessed November, 2019].

APEX BEAT

The apex beat or apical impulse is the palpable cardiac impulse farthest away from the sternum and farthest down on the chest wall, usually caused by the LV and located near the midclavicular line (MCL) in the fifth intercostal space.

Reference

McGee S. Palpation of the Heart. Evidence-Based Physical Diagnosis. Netherlands: Elsevier; 2018. pp. 317-26.

ACUTE CORONARY SYNDROME

Definition of Acute Coronary Syndrome(s)

- Acute coronary syndrome includes spectrum of ST-elevation myocardial infarction (STEMI), non-STEMI (NSTEMI), and unstable angina (UA).
- UA/NSTEMI are defined in an appropriate clinical setting (chest discomfort or anginal equivalent), often accompanied by:
  - Electrocardiographic (ECG), ST-segment depression or prominent T-wave inversion and/or
  - Positive biomarkers of necrosis (for example, troponin) in the absence of ST-segment elevation.
- NSTEMI is differenciated from UA by the presence of myocardial necrosis.
- STEMI is diagnosed by ECG in the absence of left ventricular hypertrophy or left bundle branch block (LBBB) in the presence of new ST elevation (at J point) and either of:
  - ≥2 mm [0.2 millivolts (mV)] in men or ≥1.5 mm (0.15 mV) in women in leads V2–V3
  - ≥1 mm (0.1 mV) in 2 other contiguous chest leads or limb leads.
- Criteria for acute myocardial infarction:
  - Evidence of acute myocardial injury in clinical setting consistent with acute myocardial ischemia, as evidenced by any of:
- Detection of rise and/or fall of cardiac troponin (cTn) values with ≥1 value >99th percentile of upper reference limit.
- Symptoms of ischemia:
  - New ischemic ECG changes
  - Development of pathological q waves on electrocardiogram (ECG)
  - Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality
  - Identification of intracoronary thrombus by angiography or autopsy.

- Postmortem finding of acute atherothrombosis in artery supplying the infarcted myocardium, regardless of cTn values.
- Detection of rise and/or fall of cardiac troponin (cTn) values with ≥1 value >99th percentile of upper reference limit PLUS atleast 1 of the following:
  - Symptoms of ischemia
  - New ischemic ECG changes
  - Development of pathological q waves on electrocardiogram (ECG)
  - Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality.
- Cardiac death with symptoms suggestive of myocardial ischemia and presumed new ischemic ECG changes, but death occurring before blood samples obtained or before increases in cardiac biomarkers in blood can be identified.

References:

- *European Society of Cardiology, American College of Cardiology, American Heart Association, and World Heart Federation (ESC/ACC/AHA/WHF) 2018 universal definition of myocardial infarction.*

PULMONARY HYPERTENSION

Pulmonary hypertension refers to a group of conditions with increased mean pulmonary arterial pressure (mPAP) >20 mm Hg with a PVR ≥3 Wood units (isolated postcapillary PH may have PVR <3 Wood units) as measured by right heart catheterization in supine position at rest.

Reference

Simonneau G, Montani D, Celermajer DS, et al. Haemodynamic definitions and updated clinical classification of pulmonary hypertension. Eur Respir J. 2019;53(1).

HEART FAILURE

Heart failure is a complex clinical syndrome caused by structural or functional impairment of ventricular filling or ejection of blood, resulting in insufficient perfusion to meet metabolic demands.

Reference

Yancy CW, Jessup M, Bozkurt B, et al. 2013 ACCF/AHA Guideline for the Management of Heart Failure: A Report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. Circulation. 2013;128(16):e240-319,

DILATED CARDIOMYOPATHY

Dilated cardiomyopathy (DCM) refers to a large group of heterogeneous myocardial disorders that are characterized by ventricular dilation and depressed myocardial contractility in the absence of abnormal loading conditions such as hypertension or valvular disease.

Reference

Yancy CW, Jessup M, Bozkurt B, et al. 2013 ACCF/AHA Guideline for the Management of Heart Failure: A Report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. Circulation. 2013;128(16):e240-319.

COUGH

A cough is an explosive expiration that protects the lungs against aspiration and promotes the movement of secretions and other airway constituents upward toward the mouth.

Reference

Fishman's Pulmonary Diseases and Disorders.

MASSIVE HEMOPTYSIS

No clear consensus for definition of massive hemoptysis and criteria have ranged from 100 mL to 1,000 mL of expectorated blood within 24 hours.

Blood loss of 400 mL in 24 hours or 100–150 mL expectorated at one time are considered massive hemoptysis.

References:

- *Larici AR, Franchi P, Occhipinti M, et al. Diagnosis and management of hemoptysis. Diagn Interv Radiol. 2014;20(4):299-309.*

LUNG SOUNDS (TABLE 15B.3)

Table 15B.3: Classification of common lung sounds.

Acoustic characteristics	*American Thoracic Society nomenclature*	*Common synonyms*

Discontinuous, interrupted explosive sounds; loud, low in pitch	**Coarse crackle**	Coarse rale
Discontinuous, interrupted explosive sounds; less loud than above and of shorter duration; higher in pitch than coarse crackles or rales	**Fine crackle**	Fine rale, crepitation
Continous sounds longer than 250 ms, high-pitched; dominant frequency of 400 Hz or more, hissing sound	**Wheeze**	Sibilant rhonchus
Continous sounds longer than 250 ms, low-pitched; dominant frequency about 200 Hz or less, snoring sound	**Rhonchus**	Sonorous rhonchus

Source: Adapted with permission from Loudon R, Murphy RLH. Lung sounds. Am Rev Respir Dis. 1984;130(4):663-73.

CHRONIC OBSTRUCTIVE PULMONARY DISEASE

Chronic obstructive pulmonary disease (COPD) is a common, preventable, and treatable disease that is characterized by persistent respiratory symptoms and airflow limitation that is due to airway and/or alveolar abnormalities usually caused by significant exposure to noxious particles or gases.

Reference
GOLD,2018.

CHRONIC BRONCHITIS

Cough and excess sputum production for ≥3 months per year in each of 2 consecutive years.

Reference
GOLD,2018.

EMPHYSEMA

Pathological term describing destruction of gas exchanging surfaces of lung (alveoli).

Reference
GOLD,2018.

CHRONIC COR PULMONALE

Right ventricular hypertrophy, dilatation or both as a result of pulmonary hypertension [defined as pulmonary artery mean pressure (PAP) >20 mm Hg] resulting from pulmonary disorders involving lung parenchyma, impaired bellows function or altered ventilatory drive.

Reference

Budev MM, Arroliga AC, Wiedemann HP, et al. Cor pulmonale: an overview. Semin Respir Crit Care Med. 2003;24(3):233-44.

ASTHMA

Asthma is a heterogeneous disease, usually characterized by chronic airway inflammation. It is defined by the history of respiratory symptoms such as wheeze, shortness of breath, chest tightness, and cough that vary over time and in intensity, together with variable airflow limitation.

Reference

GINA 2019

BRONCHIECTASIS

Persistent or progressive suppurative lung disease characterized by irreversibly dilated bronchi and chronic or recurrent bronchial inflammation and infection.

Reference

Pasteur MC, Bilton D, Hill AT. British Thoracic Society guideline for non-CF bronchiectasis. Thorax. 2010;65 Suppl 1:i1-58.

UNINTENTIONAL WEIGHT LOSS

Clinical entity whereby the patient does not purposefully set out to lose weight for any reason and when weight loss as a consequence of advanced chronic diseases or their treatments (e.g. diuretics for heart failure) is excluded.

Definition criteria were numerical verification of >5% reduction in usual body weight over the preceding 6–12 months, or, for subjects without

numerical documentation, at least two of the following: evidence of change in clothing size, corroboration of the reported weight loss by a relative or friend, and ability to give a numerical estimate of the amount of weight loss.

Reference

Bosch X, Monclús E, Escoda O, et al. Unintentional weight loss: Clinical characteristics and outcomes in a prospective cohort of 2677 patients. PLoS One. 2017;12(4):e0175125.

DYSPHAGIA

Dysphagia is sensation of impaired passage of food from the mouth to stomach.

Reference

- *Lind CD. Dysphagia: evaluation and treatment. Gastroenterol Clin North Am. 2003;32(2):553-75.*

DYSPEPSIA

Dyspepsia is often broadly defined as pain or discomfort centered in the upper abdomen but may include varying symptoms like epigastric pain, postprandial fullness, early satiation, anorexia, belching, nausea and vomiting, upper abdominal bloating, and even heartburn and regurgitation.

Reference

Feldman M, Friedman L, Brandt L. Sleisenger and Fordtran's Gastrointestinal and Liver Disease. Philadelphia: Saunders; 2015.

NAUSEA

Nausea is an unpleasant subjective sensation, most people have experienced at some point in their lives and usually recognize as a feeling of impending vomiting in the epigastrium or throat.

Reference

Feldman M, Friedman L, Brandt L. Sleisenger and Fordtran's Gastrointestinal and Liver Disease. Philadelphia: Saunders; 2015.

RETCHING

Retching consists of spasmodic and abortive respiratory movements with the glottis closed. When part of the emetic sequence, retching is associated with intense nausea and usually, but not invariably, culminates in the act of vomiting.

Reference:

Feldman M, Friedman L, Brandt L. Sleisenger and Fordtran's Gastrointestinal and Liver Disease. Philadelphia: Saunders; 2015.

VOMITING

Vomiting is a partially voluntary act of forcefully expelling gastric or intestinal content through the mouth.

Reference

Feldman M, Friedman L, Brandt L. Sleisenger and Fordtran's Gastrointestinal and Liver Disease. Philadelphia: Saunders; 2015.

REGURGITATION

An effortless reflux of gastric contents into the esophagus that sometimes reaches the mouth but is not usually associated with the forceful ejection typical of vomiting.

Reference

Feldman M, Friedman L, Brandt L. Sleisenger and Fordtran's Gastrointestinal and Liver Disease. Philadelphia: Saunders; 2015.

DIARRHEA

Change in normal bowel movement characterized by passage of unusually soft or liquid stools ≥3 times in 24 hours (or >250 g unformed stool/day)

- Acute diarrhea—duration <14 days
- Persistent diarrhea—duration 14–29 days
- Chronic diarrhea—duration ≥30 days.

Reference

DuPont HL. Acute infectious diarrhea in immunocompetent adults. N Engl J Med. 2014;370(16):1532-40.

CONSTIPATION

Constipation defined as unsatisfactory defecation characterized by infrequent stools (fewer than 3 in a week), hard stools, excessive straining or a sense of incomplete evacuation.

Functional Constipation—ROME III Criteria

- ≥2 of the following:
  - Straining during ≥25% of defecations
  - Lumpy or hard stools during ≥25% of defecations
  - Feeling of incomplete evacuation during ≥25% of defecations
  - Feeling of anorectal obstruction or blockage during ≥25% of defecations
  - Manually facilitating defecation during ≥25% of defecations
  - <3 unassisted bowel movements/week.
- Loose stools rarely present without laxatives
- Criteria for irritable bowel syndrome not sufficiently met (although abdominal pain and/or bloating may be present, they are not predominant symptoms)
- Symptoms present for past 3 months with symptom onset ≥6 months before diagnosis.

Reference

Feldman M, Friedman L, Brandt L. Sleisenger and Fordtran's Gastrointestinal and Liver Disease. Philadelphia: Saunders; 2015.

FECAL INCONTINENCE

Fecal incontinence is defined as involuntary passage of fecal matter through the anus or inability to control the discharge of bowel contents.

Reference

Feldman M, Friedman L, Brandt L. Sleisenger and Fordtran's Gastrointestinal and Liver Disease. Philadelphia: Saunders; 2015.

HEMATEMESIS

Hematemesis is defined as vomiting of blood, which is indicative of bleeding from the esophagus, stomach, or duodenum.

Hematemesis includes vomiting of bright red blood, which suggests recent or ongoing bleeding, and dark material (coffee-ground emesis) which suggests bleeding that stopped some time ago.

Reference

Feldman M, Friedman L, Brandt L. Sleisenger and Fordtran's Gastrointestinal and Liver Disease. Philadelphia: Saunders; 2015.

MALENA

Melena is defined as black tarry stool and results from degradation of blood to hematin or other hemochromes by intestinal bacteria. Melena can signify bleeding that originates from a UGI, small bowel, or proximal colonic source and generally occurs when 50–100 mL or more of blood is delivered into the GI tract (usually the upper tract), with passage of characteristic stool occurring several hours after the bleeding event.

Reference

Feldman M, Friedman L, Brandt L. Sleisenger and Fordtran's Gastrointestinal and Liver Disease. Philadelphia: Saunders; 2015.

HEMATOCHEZIA

Hematochezia refers to bright red blood per rectum and suggests active UGI or small bowel bleeding or distal colonic or anorectal bleeding.

Reference

Feldman M, Friedman L, Brandt L. Sleisenger and Fordtran's Gastrointestinal and Liver Disease. Philadelphia: Saunders; 2015.

SEVERE GI BLEEDING

Severe GI bleeding is defined as documented GI bleeding (hematemesis, melena, hematochezia, or positive nasogastric lavage) accompanied by shock or orthostatic hypotension, a decrease in the hematocrit value by at least 6% (or a decrease in the hemoglobin level of at least 2 g/dL), or transfusion of at least 2 units of packed red blood cells.

Reference

Feldman M, Friedman L, Brandt L. Sleisenger and Fordtran's Gastrointestinal and Liver Disease. Philadelphia: Saunders; 2015.

OCCULT GI BLEEDING

Occult GI bleeding refers to subacute bleeding that is not clinically visible.

Reference

Feldman M, Friedman L, Brandt L. Sleisenger and Fordtran's Gastrointestinal and Liver Disease. Philadelphia: Saunders; 2015.

OBSCURE GI BLEEDING

Obscure GI bleeding is bleeding from a site that is not apparent after routine endoscopic evaluation with esophagogastroduodenoscopy (upper endoscopy) and colonoscopy, and possibly small bowel radiography.

Reference

Feldman M, Friedman L, Brandt L. Sleisenger and Fordtran's Gastrointestinal and Liver Disease. Philadelphia: Saunders; 2015.

ACUTE LIVER FAILURE

Acute liver failure is the clinical syndrome of liver dysfunction, coagulopathy and encephalopathy developing within 26 weeks of onset of symptoms in patients without pre-existing liver failure.

Reference

Sherlock's diseases of the liver and biliary system.

Note: One categorization based on clinical patterns and outcome described three groups based on the time interval between the onset of jaundice and encephalopathy:

- Hyperacute liver failure (7 days or less)
- Acute liver failure (ALF) (8–28 days), and
- Subacute liver failure (4–24 weeks).

Reference

Feldman M, Friedman L, Brandt L. Sleisenger and Fordtran's Gastrointestinal and Liver Disease. Philadelphia: Saunders; 2015.

CIRRHOSIS OF LIVER

Cirrhosis is defined as a diffuse disruption of the normal architecture of the liver with fibrosis and nodule formation.

Reference

Sherlock's diseases of the liver and biliary system

PORTAL HYPERTENSION

Syndrome of increased pressure (>5 mm Hg) in portal venous system due to increased vascular resistance plus increased blood flow.

Reference

Bloom S, Kemp W, Lubel J. Portal Hypertension—Pathophysiology, Diagnosis and Management. Intern Med J. 2015;45(1):16-26.

HEPATIC ENCEPHALOPATHY

Hepatic encephalopathy is a potentially reversible neuropsychiatric complication of liver failure with a wide variety of clinical manifestations from minimal changes in cognitive function to severe complications of stupor and coma.

Reference

Vilstrup H, Amodio P, Bajaj J, et al. Hepatic encephalopathy in chronic liver disease: 2014 Practice Guideline by the American Association for the Study of Liver Diseases and the European Association for the Study of the Liver. Hepatology. 2014;60(2):715-35.

POLYURIA

The conventional definition of polyuria is a urine volume that is more than 2.5 L/day or

Polyuria is present if the urine flow rate is higher than what is expected in a specific setting.

Reference

Brenner and Rector's The Kidney.

NOCTURIA

The International Continence Society defines nocturia as a urinary storage symptom with the complaint that the individual has to wake one or more times at night to void, with each void being preceded and followed by sleep.

OLIGURIA

Decreased urine output <300 cc/m^2/24 hours
<0.5 cc/kg/hour in children
<1 cc/kg/hour in infants
Usually <500 cc/day in adults.

Reference
CDC.

ANURIA

No or minimal urine output
Usually <100 mL/day

Reference
CDC.

HEMATURIA

Hematuria is defined as three or more erythrocytes per high-power field.

Reference
Brenner and Rector's The Kidney.

MODERATELY INCREASED ALBUMINURIA

Urine albumin levels between 30 mg/day and 300 mg/day. This was previously referred to as microalbuminuria.

Reference
National Kidney Foundation Primer on Kidney Diseases.

SEVERELY INCREASED ALBUMINURIA

Urine albumin levels greater than 300 mg/day. This was previously referred to as macroalbuminuria.

Reference
National Kidney Foundation Primer on Kidney Diseases.

ACUTE KIDNEY INJURY

Acute kidney injury (AKI) is defined as any of the following:

- Increase in SCr by >0.3 mg/dL (>26.5 µmol/L) within 48 hours; or
- Increase in SCr to 1.5 times baseline, which is known or presumed to have occurred within the prior 7 days; or
- Urine volume <0.5 mL/kg/h for 6 hours.

Reference
KDIGO 2012 Guidelines on CKD.

CHRONIC KIDNEY DISEASE

Chronic kidney disease (CKD) is defined as abnormalities of kidney structure or function, present for >3 months with implications for health.

Criteria for CKD (either of the following present for >3 months)	
Markers of kidney damage (one or more)	Albuminuria (AER >/= 30 mg/24 hours; ACR >/= 30 mg/g Urine sediment abnormalities Electrolyte and other abnormalities due to tubular disorders Abnormalities detected by histology Structural abnormalities detected by imaging History of kidney transplantation
Decreased GFR	GFR <60 mL/min/1.73 m^2 (GFR categories G3a–G5)

Reference

KDIGO 2012 Guidelines on CKD.

NEPHROTIC SYNDROME

Nephrotic syndrome is a clinical syndrome characterized by:

- Proteinuria—adult >3.5 g/day, child >40 mg/h per m^2
- Hypoalbuminemia— <3.5 g/dL
- Edema
- Hypercholesterolemia
- Lipiduria.

Reference

Comprehensive clinical nephrology, John Feehally.

UNCOMPLICATED UTI AND COMPLICATED UTI

Uncomplicated UTI

Uncomplicated urinary tract infection refers to acute cystitis or pyelonephritis in nonpregnant outpatient women without anatomic abnormalities or instrumentation of the urinary tract.

Complicated UTI

The term complicated UTI encompasses all other types of UTI.

Reference

Jameson JL, Fauci AS, Kasper DL, et al. Harrison's Principles of Internal Medicine, 20th edition. United States of America: McGraw-Hill Education; 2018.

ASYMPTOMATIC BACTERIURIA

Asymptomatic bacteriuria is defined as the presence of two separate consecutive clean-voided urine specimens, both with 10^5 or more colony-forming units per milliliter (cfu/mL) of the same uropathogen in the absence of symptoms referable to the urinary tract.

Reference

Johnson RJ, Feehally J. Comprehensive clinical nephrology. US: Mosby; 2000.

NEUTROPENIA AND AGRANULOCYTOSIS

Neutropenia is defined as absolute neutrophil count (ANC) ≤1.5 × 109/L

Agranulocytosis defined as ANC ≤0.2 × 109/L which carries a risk of severe infections with susceptibility to opportunistic organisms.

Reference

Newburger PE, Dale DC. Evaluation and management of patients with isolated neutropenia. Semin Hematol. 2013;50(3):198-206.

FEBRILE NEUTROPENIA

Febrile neutropenia is defined as a single fever [101°F (38.3°C)] or sustained elevated temperature [100.4°F (38°C)] in a patient with a current or anticipated absolute neutrophil count (ANC) <500 cells/mm.

Reference

Freifeld AG, Bow EJ, Sepkowitz KA, et al. Clinical practice guideline for the use of antimicrobial agents in neutropenic patients with cancer: 2010 update by the Infectious Diseases Society of America. Clin Infect Dis. 2011;52(4):e56-93.

LYMPHADENOPATHY

Lymphadenopathy is defined as lymph nodes of:

- Abnormal size, generally >1 cm, although definition of normal size range varies by lymph node regions and age of patient;
  - Jugulodigastric lymph nodes (often the largest of cervical lymph nodes) >1.5 cm are considered abnormal
  - Epitrochlear lymph nodes >5 mm are considered abnormal
  - Any palpable supraclavicular, popliteal, or iliac lymph nodes are considered abnormal
  - Abdominal lymph nodes vary from 6–10 mm; retrocrural lymph nodes >6 mm, retroperitoneal lymph nodes >10 mm, and pelvic lymph nodes >8–10 mm are considered abnormal
  - Inguinal lymph nodes >1.5 cm in diameter are considered abnormal.
- Abnormal dimensions, consistency or mobility.

Reference

Gaddey HL, Riegel AM. Unexplained Lymphadenopathy: Evaluation and Differential Diagnosis. Am Fam Physician. 2016;94(11):896-903.

GENERALIZED LYMPHADENOPATHY

Generalized lymphadenopathy is defined as involvement of ≥2 noncontiguous lymph node groups and is typically indicative of systemic disease.

Reference

Gaddey HL, Riegel AM. Unexplained Lymphadenopathy: Evaluation and Differential Diagnosis. Am Fam Physician. 2016;94(11):896-903.

MASSIVE SPLENOMEGALY

Spleen is massively enlarged when it is palpable >8 cm below the left costal margin or its drained weight is ≥1,000 g.

Reference

Harrison's Principles of internal medicine.

HYPERSPLENISM

Hypersplenism defined as a syndrome comprised of:

- Splenomegaly
- Anemia, leukopenia, and/or thrombocytopenia
- Compensatory bone marrow hyperplasia
- Improvement after splenectomy (if performed).

Reference

Pozo AL, Godfrey EM, Bowles KM. Splenomegaly: investigation, diagnosis and management. Blood rev. 2009;23(3):105-11.

STUPOR

Stupor is a state of baseline unresponsiveness that requires repeated application of vigorous stimuli to achieve arousal.

Reference

Bradley's Neurology in Clinical Practice, 5, 34-50.e1

COMA

Coma is a state of complete unresponsiveness to arousal, in which the patient lies with the eyes closed.

Reference

- *Bradley's Neurology in Clinical Practice, 5, 34-50.e1*

CONFUSION

Confusion is a general term denoting the patient's incapacity to think with customary speed, clarity, and coherence.

Reference

Ropper AH, Samuels MA, Klein JP, et al. Adams and Victor's Principles of Neurology, 11th edition. United States of America: McGraw-Hill Education; 2019.

DEMENTIA

Dementia denotes a deterioration of all intellectual or cognitive functions with little or no disturbance of consciousness or perception.

Reference

Ropper AH, Samuels MA, Klein JP, et al. Adams and Victor's Principles of Neurology, 11th edition. United States of America: McGraw-Hill Education; 2019.

DELIRIUM

The American Psychiatric Association's Diagnostic and Statistical Manual, 5th edition (DSM-5) defines delirium as:

- Disturbance of consciousness with reduced ability to focus, sustain, or shift attention.
- The disturbance develops over a short period of time (usually hours to days) and tends to fluctuate during the course of a day.
- An additional disturbance in cognition (e.g. memory deficit, disorganization, language, visuospatial ability, or perception).

- A change in cognition or development of a perceptual disturbance that is not better accounted for by a pre-existing, established, or evolving dementia.
- There is evidence from history, physical examination, or laboratory findings that the disturbance is caused by medical condition, substance intoxication or withdrawal (i.e. due to a drug of abuse or to a medication), or exposure to a toxin, or is due to multiple etiologies.

AKINETIC MUTISM

Akinetic mutism refers to a state in which the patient, although seemingly awake remains silent and motionless.

Reference

Brazis P, Masdeu JC, Biller J. Localization in clinical neurology. Philadelphia: Wolters Kluwer Health; 2016.

LOCKED IN SYNDROME

The locked in syndrome refers to a condition in which the patient is mute and motionless but remains awake, alert, aware of self and capable of perceiving sensory stimuli.

Reference

Brazis P, Masdeu JC, Biller J. Localization in clinical neurology. Philadelphia: Wolters Kluwer Health; 2016.

ABULIA

Abulia refers to difficulty in initiating and sustaining spontaneous movements and reduction in emotional responsiveness, spontaneous speech and social interactions.

Reference

Campbell WW. De Jong's The Neurological Examination. Philadelphia: Lippincott Williams & Wilkins; 2012.

ATTENTION AND CONCENTRATION

Attention is the ability to focus on a particular sensory stimulus to the exclusion of others.

Concentration is sustained attention.

Reference

Neurologic History & Examination. In: Simon RP, Aminoff MJ, Greenberg DA (Eds). Clinical Neurology, 10th edition. New York, NY: McGraw-Hill; 2017.

MEMORY

Memory is the ability to register, store, and retrieve information.

Reference

Neurologic History & Examination. In: Simon RP, Aminoff MJ, Greenberg DA (Eds). Clinical Neurology, 10th edition. New York, NY: McGraw-Hill; 2017.

AMNESIA

The amnesic state, defined by Ribot possesses two salient features that may vary in severity but are always conjoined:

1. An impaired ability to recall events and other information that has been firmly established before the onset of illness (retrograde amnesia).
2. An inability to acquire new information, learn or form new memories (anterograde amnesia).

Reference:

Ropper AH, Samuels MA, Klein JP, et al. Adams and Victor's Principles of Neurology, 11th edition. United States of America: McGraw-Hill Education; 2019.

AGNOSIA

A conceptual inability to recognize objects, persons or sensory stimuli in the absence of a primary deficit in the sensory modality.

Reference

Ropper AH, Samuels MA, Klein JP, et al. Adams and Victor's Principles of Neurology, 11th edition. United States of America: McGraw-Hill Education; 2019.

INSOMNIA

A chronic inability to sleep despite adequate opportunity to do so. It indicates any impairment in the duration, depth or restorative properties of sleep.

Reference

Ropper AH, Samuels MA, Klein JP, et al. Adams and Victor's Principles of Neurology, 11th edition. United States of America: McGraw-Hill Education; 2019.

APHASIA

Loss of the production or comprehension of spoken or written language because of an acquired lesion in the brain.

Reference

Ropper AH, Samuels MA, Klein JP, et al. Adams and Victor's Principles of Neurology, 11th edition. United States of America: McGraw-Hill Education; 2019.

DYSARTHRIA

A defect in articulation of speech with intact mental functions, and comprehension of spoken and written language and normal syntax (grammatical construction of sentences).

Reference

Ropper AH, Samuels MA, Klein JP, et al. Adams and Victor's Principles of Neurology, 11th edition. United States of America: McGraw-Hill Education; 2019.

APHONIA AND DYSPHONIA

A loss (aphonia) or alteration (dysphonia) of voice due to a disorder of the larynx or its innervation.

Reference

Ropper AH, Samuels MA, Klein JP, et al. Adams and Victor's Principles of Neurology, 11th edition. United States of America: McGraw-Hill Education; 2019.

AGRAPHIA/DYSGRAPHIA

Loss of the ability to write not due to weakness, incoordination, or other neurologic dysfunction of the arm or hand is called agraphia.

Milder involvement may be referred to as dysgraphia.

Reference
Campbell WW. De Jong's The Neurological Examination. Philadelphia: Lippincott Williams & Wilkins; 2012.

ALEXIA

Loss of the ability to read in the absence of actual loss of vision is alexia.

Reference
Campbell WW. De Jong's The Neurological Examination. Philadelphia: Lippincott Williams & Wilkins; 2012.

ECHOLALIA

Echolalia is the meaningless repetition of heard words.

Reference
Campbell WW. De Jong's The Neurological Examination. Philadelphia: Lippincott Williams & Wilkins; 2012.

PALILALIA

Palilalia is the repetition of one's own speech.

Reference
Campbell WW. De Jong's The Neurological Examination. Philadelphia: Lippincott Williams & Wilkins; 2012.

PERSEVERATION

Perseveration is the persistence of one reply or one idea in response to various questions.

Reference
Campbell WW. De Jong's The Neurological Examination. Philadelphia: Lippincott Williams & Wilkins; 2012.

NEOLOGISMS

Neologisms are new words, usually meaningless, coined by the patient and usually heard in psychotic states or in aphasic patients.

Reference

Campbell WW. De Jong's The Neurological Examination. Philadelphia: Lippincott Williams & Wilkins; 2012.

IDIOGLOSSIA

Idioglossia is the imperfect articulation with utterance of meaningless sounds; the individual may speak with a vocabulary all his own.

Reference

Campbell WW. De Jong's The Neurological Examination. Philadelphia: Lippincott Williams & Wilkins; 2012.

DYSLOGIA

Dyslogia refers to abnormal speech due to mental disease, and it is most often used to refer to abnormal speech in dementia.

Reference

Campbell WW. De Jong's The Neurological Examination. Philadelphia: Lippincott Williams & Wilkins; 2012.

CONFABULATION

The creative falsification of memory in an alert, responsive individual.

Reference

Ropper AH, Samuels MA, Klein JP, et al. Adams and Victor's Principles of Neurology, 11th edition. United States of America: McGraw-Hill Education; 2019.

TONE

Tone is resistance of a muscle to passive movement at a joint.

Reference

Neurologic History & Examination. In: Simon RP, Aminoff MJ, Greenberg DA, (Eds). Clinical Neurology, 10th edition. New York, NY: McGraw-Hill; 2017.

RIGIDITY

Rigidity is characterized by a plastic resistance to passive movements that affects both agonist and antagonist muscles to a similar extent and that is constant throughout the entire range of movement.

Reference

Brazis P, Masdeu JC, Biller J. Localization in clinical neurology. Philadelphia: Wolters Kluwer Health; 2016.

COGWHEEL RIGIDITY

Cogwheel rigidity is characterized by periodic modifications of muscle tone due to the superimposed tremor that can be seen and felt when passively moving the extremity.

Reference

Brazis P, Masdeu JC, Biller J. Localization in clinical neurology. Philadelphia: Wolters Kluwer Health; 2016.

AKATHISIA

Akathisia refers to a feeling of inner restlessness that is often relieved by movement.

Reference

Brazis P, Masdeu JC, Biller J. Localization in clinical neurology. Philadelphia: Wolters Kluwer Health; 2016.

ASTERIXIS

Sudden loss of muscle tone during sustained contraction of an outstretched limb.

Reference

Talley and O'Connor's Clinical examination.

ATHETOSIS

Athetosis is characterized by slow, uncoordinated, twisting, writhing, and involuntary movements of wide amplitude. These predominantly involve the distal appendicular musculature, especially the upper extremities, although face and axial muscles may also be involved.

Reference

Brazis P, Masdeu JC, Biller J. Localization in clinical neurology. Philadelphia: Wolters Kluwer Health; 2016.

CHOREA

Chorea is characterized by sudden, brief, spontaneous, involuntary, purposeless, continuous, irregular, and unpredictable jerks that randomly involve the appendicular, facial, or truncal musculature.

Reference

Brazis P, Masdeu JC, Biller J. Localization in clinical neurology. Philadelphia: Wolters Kluwer Health; 2016.

DYSTONIA

Dystonia is characterized by slow, long sustained, contorting, involuntary movements, and postures involving proximal appendicular and axial muscles.

Reference

Brazis P, Masdeu JC, Biller J. Localization in clinical neurology. Philadelphia: Wolters Kluwer Health; 2016.

HEMIBALLISMUS

Hemiballismus is characterized by occurrence of sudden, paroxysmal, large amplitude, flinging, throwing movements of the arm, and leg contralateral to a lesion in or near the subthalamic nucleus.

Reference

Brazis P, Masdeu JC, Biller J. Localization in clinical neurology. Philadelphia: Wolters Kluwer Health; 2016.

MYOCLONUS

Myoclonus is a movement disorder characterized by unexpected, brief, brisk, shock-like, involuntary, repetitive, synchronous, or asynchronous contractions of a muscle or group of axial or appendicular muscles.

Reference

Brazis P, Masdeu JC, Biller J. Localization in clinical neurology. Philadelphia: Wolters Kluwer Health; 2016.

MYOKYMIA

A repeated contraction of a small muscle group; often involves the orbicularis oculi muscle.

Reference

Talley and O'Connor's Clinical examination.

RESTLESS LEG SYNDROME

Restless leg syndrome refers to a condition in which the patient notes unpleasant crawling sensations of the legs, particularly when sitting and relaxing in the evening which disappear on walking.

Criteria for diagnosis include:

- An intense irresistible urge to move the legs, usually associated with sensory complaints including paresthesia and dysesthesias.
- Motor restlessness.
- Worsening of the symptoms with rest and relief with motor activity.
- Increased severity of symptoms in the evening or at night.

Reference

Brazis P, Masdeu JC, Biller J. Localization in clinical neurology. Philadelphia: Wolters Kluwer Health; 2016.

TICS

Tics are sudden, rapid, usually stereotyped, and predominantly colonic hyperkinesias which may be willfully suppressed for short periods of time

and disappear during sleep.

Reference

Brazis P, Masdeu JC, Biller J. Localization in clinical neurology. Philadelphia: Wolters Kluwer Health; 2016.

TREMOR

Involuntary, rhythmic, and oscillatory movements about a fixed point resulting from either alternating or synchronous contractions of reciprocally innervated antagonist muscles.

Reference

Brazis P, Masdeu JC, Biller J. Localization in clinical neurology. Philadelphia: Wolters Kluwer Health; 2016.

AGRAPHESTHESIA

Agraphesthesia is the inability to identify by touch a number written on the hand.

Reference

Neurologic History & Examination. In: Simon RP, Aminoff MJ, Greenberg DA. (Eds). Clinical Neurology, 10th edition. New York, NY: McGraw-Hill; 2017.

ALLODYNIA

Increase in sensibility to pain; pain in response to a stimulus not normally painful.

Reference

Campbell WW. De Jong's The Neurological Examination. Philadelphia: Lippincott Williams & Wilkins; 2012.

ALLOESTHESIA

Perception of a sensory stimulus at a site other than where it was delivered; Tactile allesthesia is feeling something other than at the site of the stimulus.

Visual allesthesia is seeing something other than where it actually is.

Reference

Campbell WW. De Jong's The Neurological Examination. Philadelphia: Lippincott Williams & Wilkins; 2012.

ANALGESIA

Absence of sensibility to pain.

Reference

Campbell WW. De Jong's The Neurological Examination. Philadelphia: Lippincott Williams & Wilkins; 2012.

ASTEROGNOSIS

Absence of spatial tactile sensibility; inability to identify objects by feel.

Reference

Campbell WW. De Jong's The Neurological Examination. Philadelphia: Lippincott Williams & Wilkins; 2012.

ANESTHESIA

Absence of all sensations.

Reference

Campbell WW. De Jong's The Neurological Examination. Philadelphia: Lippincott Williams & Wilkins; 2012.

DYSESTHESIAS

Unpleasant or painful abnormal perverted sensations, either spontaneous or after a normally nonpainful stimulus (e.g. burning in response to touch); often accompanies paresthesias.

Reference

Campbell WW. De Jong's The Neurological Examination. Philadelphia: Lippincott Williams & Wilkins; 2012.

EXTINCTION

Extinction is the failure to perceive a visual or tactile stimulus when it is applied bilaterally, even though it can be perceived when applied unilaterally.

Reference

Neurologic History & Examination. In: Simon RP, Aminoff MJ, Greenberg DA. (Eds). Clinical Neurology, 10th edition. New York, NY: McGraw-Hill; 2017.

HYPALGESIA

Decrease in sensibility to pain.

Reference

Campbell WW. De Jong's The Neurological Examination. Philadelphia: Lippincott Williams & Wilkins; 2012.

HYPERALGESIA

Increase in sensibility to pain; pain in response to a stimulus not normally painful.

Reference

Campbell WW. De Jong's The Neurological Examination. Philadelphia: Lippincott Williams & Wilkins; 2012.

HYPERPATHIA

Increase in sensibility to pain; pain in response to a stimulus not normally painful.

Reference

Campbell WW. De Jong's The Neurological Examination. Philadelphia: Lippincott Williams & Wilkins; 2012.

KINESTHESIA

The sense of movement.

Reference

Campbell WW. De Jong's The Neurological Examination. Philadelphia: Lippincott Williams & Wilkins; 2012.

PALLESTHESIA

Vibratory sensation.
Hypopallesthesia = decreased vibratory sensation
Apallesthesia = absent vibratory sensation

Reference

Campbell WW. De Jong's The Neurological Examination. Philadelphia: Lippincott Williams & Wilkins; 2012.

PARESTHESIAS

Abnormal spontaneous sensations experienced in the absence of specific stimulation (feelings of cold, warmth numbness, tingling, burning, prickling, crawling, heaviness, compression or itching).

Reference

Campbell WW. De Jong's The Neurological Examination. Philadelphia: Lippincott Williams & Wilkins; 2012.

NEGLECT

Neglect is failure to attend to space or use the limbs on one side of the body.

Reference

Neurologic History & Examination. In: Simon RP, Aminoff MJ, Greenberg DA (Eds). Clinical Neurology, 10th edition. New York, NY: McGraw-Hill; 2017.

ANOSOGNOSIA

Anosognosia is unawareness of a neurologic deficit.

Reference

Neurologic History & Examination. In: Simon RP, Aminoff MJ, Greenberg DA (Eds). Clinical Neurology, 10th edition. New York, NY: McGraw-Hill; 2017.

CONSTRUCTIONAL APRAXIA

Constructional apraxia is the inability to draw accurate representations of external space, such as filling in the numbers on a clock face or copying geometric figures.

Reference

Neurologic History & Examination. In: Simon RP, Aminoff MJ, Greenberg DA (Eds). Clinical Neurology, 10th edition. New York, NY: McGraw-Hill; 2017.

ATAXIA

Ataxia refers to a disturbance in the smooth performance of voluntary motor acts causing muscular incoordination or impaired balance.

Reference

Brazis P, Masdeu JC, Biller J. Localization in clinical neurology. Philadelphia: Wolters Kluwer Health; 2016.

PARALYSIS AND PARESIS

Paralysis means loss of voluntary movement as a result of interruption of one of the motor pathways at any point from the cerebrum to the muscle fiber. A lesser degree of weakness is spoken of as paresis.

Monoplegia refers to weakness or paralysis of all the muscles of one leg or arm.

Hemiplegia refers to weakness or paralysis involving the arm, the leg, and sometimes the face on one side of the body.

Paraplegia indicates weakness or paralysis of both legs.

Quadriplegia (tetraplegia) denotes weakness or paralysis of all four extremities.

Reference

Ropper AH, Samuels MA, Klein JP, et al. Adams and Victor's Principles of Neurology, 11th edition. United States of America: McGraw-Hill Education; 2019.

APRAXIA

The term apraxia denotes a disorder in which an attentive patient loses the ability to execute previously learned activities in the absence of weakness,

ataxia, sensory loss, or extrapyramidal derangement that would be adequate to explain the deficit.

Reference

Ropper AH, Samuels MA, Klein JP, et al. Adams and Victor's Principles of Neurology, 11th edition. United States of America: McGraw-Hill Education; 2019.

STROKE

Rapidly developing clinical signs of focal (or global) disturbance of cerebral function, lasting more than 24 hours or leading to death, with no apparent cause other than that of vascular origin.

Reference

WHO

TIA

Transient ischemic attack (TIA) is a transient episode of neurologic dysfunction caused by focal ischemia of the brain, spinal cord, or retina, and without detection of acute infarction on neuroimaging.

Reference

American Heart Association/American Stroke Association 2009 tissue-based definition of TIA

LACUNAR STROKE

Lacunar stroke (or lacunar infarct) is defined as stroke caused by occlusion of small vessels in the brain.

- Infarcts are generally rounded, ovoid, or tubular in shape, and <20 mm in axial diameter.
- Infarcts result in a small cavity, or lacune, which typically ranges from >3 mm to <15 mm.

Reference

Pantoni L. Cerebral small vessel disease: from pathogenesis and clinical characteristics to therapeutic challenges. Lancet Neurol. 2010;9(7):689-701.

EPILEPTIC SEIZURE

Epileptic seizure—transient occurrence of signs and/or symptoms due to abnormal excessive or synchronous neuronal activity in the brain.

Reference

Berg AT, Berkovic SF, Brodie MJ, et al. Revised terminology and concepts for organization of seizures and epilepsies: report of the ILAE Commission on Classification and Terminology, 2005-2009. Epilepsia. 2010;51(4):676-85.

EPILEPSY

International League Against Epilepsy defines epilepsy as disease of brain defined by any of the following:

- 2 or more unprovoked or reflex seizures occurring >24 hours apart.
- Single unprovoked (or reflex) seizure and high risk of recurrence over the next 10 years [similar high risk (≥60%) that occurs after 2 unprovoked seizures].
- Diagnosis of an epilepsy syndrome.

Reference

Berg AT, Berkovic SF, Brodie MJ, et al. Revised terminology and concepts for organization of seizures and epilepsies: report of the ILAE Commission on Classification and Terminology, 2005-2009. Epilepsia. 2010;51(4):676-85.

SYNCOPE

Syndrome of transient loss of consciousness secondary to cerebral hypoperfusion characterized by rapid onset, short duration, and complete spontaneous recovery.

Reference

Brignole M, Moya A, de Lange FJ, et al. 2018 ESC Guidelines for the diagnosis and management of syncope. Eur Heart J. 2018;39(21):1883-948.

METABOLIC SYNDROME

Metabolic syndrome is a cluster of commonly co-occurring metabolic risk factors associated with cardiovascular disease and type 2 diabetes mellitus, including elevated blood pressure, atherogenic dyslipidemia, insulin resistance, and central obesity.

Reference

Alberti KG, Eckel RH, Grundy SM, et al. Harmonizing the metabolic syndrome: a joint interim statement of the International Diabetes Federation Task Force on Epidemiology and Prevention; National Heart, Lung, and Blood Institute; American Heart Association; World Heart Federation; International Atherosclerosis Society; and International Association for the Study of Obesity. Circulation. 2009;120(16):1640-5.

SEPSIS

The Third International Consensus Definitions for Sepsis and Septic Shock (Sepsis-3)

- Sepsis—life-threatening organ dysfunction caused by dysregulated host response to infection.
- Organ dysfunction—acute change in total sequential organ failure assessment (SOFA) score ≥2 points consequent to infection:
  - Assume baseline SOFA score of 0 in patients without known preexisting organ dysfunction
  - SOFA score ≥2 points associated with overall mortality risk of about 10% in general hospital population with suspected infection.
- Septic shock:
  - Sepsis with underlying circulatory and cellular/metabolic abnormalities severe enough to substantially increase mortality
  - Clinically defined as persistent hypotension requiring vasopressors to maintain mean arterial pressure (MAP) ≥65 mm Hg and serum lactate level ≥2 mmol/L (18 mg/dL) despite adequate volume resuscitation
  - Associated with hospital mortality ≥40%.

SIRS

Systemic inflammatory response syndrome (SIRS):

- ≥2 of:
  - Temperature >38.3°C (100.9°F) or <36°C (96.8°F)
  - Heart rate >90 beats/minute
  - Respiratory rate >20 breaths/minute or arterial partial pressure of carbon dioxide ($PaCO_2$) <32 mm Hg
  - White blood cell count (WBC) >12,000/mm^3 or WBC <4,000/mm^3 or >10% immature neutrophils (bands).

- Above abnormalities should represent change from baseline without other known cause (such as leukopenia due to chemotherapy).

Reference

Levy MM, Fink MP, Marshall JC, et al. Society of Critical Care Medicine (SCCM), European Society of Intensive Care Medicine (ESICM), American College of Chest Physicians (ACCP), American Thoracic Society (ATS), and Surgical Infection Society (SIS) 2001 international Sepsis Definitions Conference. Intensive Care Med. 2003;29(4):530-8.

ARDS

Berlin definition of acute respiratory distress syndrome (ARDS):

- Onset within 1 week of known clinical insult or new or worsening respiratory symptoms.
- Bilateral opacities not fully explained by effusions, lobar/lung collapse, or nodules on chest X-ray or computed tomography.
- Respiratory failure not fully explained by cardiac failure or fluid overload (in the absence of risk factors for ARDS, an objective assessment such as echocardiography is required to exclude these causes of hydrostatic edema)
- Oxygenation status:
  - Mild ARDS defined as partial pressure of oxygen in arterial blood (PaO_2) to fraction of inspired oxygen (FiO_2) >200 mm Hg but ≤300 mm Hg with positive end-expiratory pressure (PEEP) or continuous positive airway pressure (CPAP) ≥5 cm H_2O
  - Moderate ARDS defined as PaO_2/FiO_2 >100 mm Hg but ≤200 mm Hg with PEEP ≥5 cm H_2O
  - Severe ARDS defined as PaO_2/FiO_2 ≤100 mm Hg with PEEP ≥5 cm H_2O
  - If altitude >1,000 meters, correction factor is PaO_2/FiO_2 × (barometric pressure/760).

MACULE

A flat, colored lesion, <2 cm in diameter, not raised above the surface of the surrounding skin. A “freckle,” or ephelid, is a prototypical pigmented macule.

Reference

Harrison's Principles of Internal Medicine.

PATCH

A large (>2 cm) flat lesion with a color different from the surrounding skin. This differs from a macule only in size.

Reference
Harrison's Principles of Internal Medicine.

PAPULE

A small, solid lesion, <0.5 cm in diameter, raised above the surface of the surrounding skin and thus palpable (e.g. a closed comedone, or whitehead, in acne).

Reference
Harrison's Principles of Internal Medicine.

NODULE

A larger (0.5–5.0 cm), firm lesion raised above the surface of the surrounding skin. This differs from a papule only in size (e.g. a large dermal nevomelanocytic nevus).

Reference
Harrison's Principles of Internal Medicine.

TUMOR

A solid, raised growth >5 cm in diameter.

Reference
Harrison's Principles of Internal Medicine.

PLAQUE

A large (>1 cm), flat-topped, and raised lesion; edges may either be distinct (e.g. in psoriasis) or gradually blend with surrounding skin (e.g. in eczematous dermatitis).

Reference
Harrison's Principles of Internal Medicine.

VESICLE

A small, fluid-filled lesion, <0.5 cm in diameter, raised above the plane of surrounding skin. Fluid is often visible, and the lesions are translucent [e.g. vesicles in allergic contact dermatitis caused by *Toxicodendron* (poison ivy)].

Reference
Harrison's Principles of Internal Medicine.

PUSTULE

A vesicle filled with leukocytes. Note: The presence of pustules does not necessarily signify the existence of an infection.

Reference
Harrison's Principles of Internal Medicine.

BULLA

A fluid-filled, raised, often translucent lesion >0.5 cm in diameter.

Reference
Harrison's Principles of Internal Medicine.

WHEAL

A raised, erythematous, edematous papule or plaque, usually representing short-lived vasodilation and vasopermeability.

Reference
Harrison's Principles of Internal Medicine.

TELANGIECTASIA

A dilated, superficial blood vessel.

Reference
Harrison's Principles of Internal Medicine.

LICHENIFICATION

A distinctive thickening of the skin that is characterized by accentuated skin-fold markings.

Reference
Harrison's Principles of Internal Medicine.

SCALE

Excessive accumulation of stratum corneum.

Reference
Harrison's Principles of Internal Medicine.

CRUST

Dried exudate of body fluids that may be either yellow (i.e. serous crust) or red (i.e. hemorrhagic crust).

Reference
Harrison's Principles of Internal Medicine.

EROSION

Loss of epidermis without an associated loss of dermis.

Reference
Harrison's Principles of Internal Medicine.

ULCER

Loss of epidermis and at least a portion of the underlying dermis.

Reference
Harrison's Principles of Internal Medicine.

EXCORIATION

Linear, angular erosions that may be covered by crust and are caused by scratching.

Reference
Harrison's Principles of Internal Medicine.

ATROPHY

An acquired loss of substance. In the skin, this may appear as a depression with intact epidermis (i.e. loss of dermal or subcutaneous tissue) or as sites of shiny, delicate, and wrinkled lesions (i.e. epidermal atrophy).

Reference
Harrison's Principles of Internal Medicine.

SCAR

A change in the skin secondary to trauma or inflammation. Sites may be erythematous, hypopigmented, or hyperpigmented depending on their age or character. Sites on hair-bearing areas may be characterized by destruction of hair follicles.

Reference
Harrison's Principles of Internal Medicine.

PURPURIC LESIONS

Small, nonblanching, red, or purple areas on skin caused by extravasation of blood from vasculature into skin or mucous membranes.

- Petechiae—spots usually <2 mm in diameter
- Purpura—larger areas of extravasated blood usually 2 mm to 1 cm in diameter

- Ecchymoses—purpuric lesions >1 cm in diameter.

Reference
Leung AK, Chan KW. Evaluating the child with purpura. Am Fam Physician. 2001;64(3):419-28.

GYNECOMASTIA

Gynecomastia refers to enlargement of the male breast

True gynecomastia is associated with glandular breast tissue that is >4 cm in diameter and often tender.

Reference
Harrison's Principles of Internal Medicine.

C. GRADING SYSTEMS

1952 MRC BREATHLESSNESS SCALE

Grade	Description
Grade 1	Is the patient's breath as good as that of other men of his age and build at work, on walking, and on climbing hills or stairs?
Grade 2	Is the patient able to walk with normal men of own age and build on the level but unable to keep up on hills or stairs?
Grade 3	Is the patient unable to keep up with normal men on the level, but able to walk about a mile or more at his own speed?
Grade 4	Is the patient unable to walk more than about 100 yards on the level without a rest?
Grade 5	Is the patient breathless on talking or undressing, or unable to leave his house because of breathlessness?

Note: "Used with the permission of the Medical Research Council"

MODIFIED MRC DYSPNEA SCALE

Grade	Description
Grade 0	I only get breathless with strenuous exercise
Grade 1	I get short of breath when hurrying on the level or walking up a slight hill
Grade 2	I walk slower than people of the same age on the level because of breathlessness, or I have to stop for breath when walking at my own pace on the level
Grade 3	I stop for breath after walking about 100 meters or after a few minutes on the level
Grade 4	I am too breathless to leave the house or I am breathless when dressing or undressing

Reference: GOLD, 2019.

MRC MUSCLE SCALE

Grade	Contraction
Grade 0	No contraction
Grade 1	Flicker or trace of contraction
Grade 2	Active movement with gravity eliminated
Grade 3	Active movement against gravity
Grade 4	Active movement against gravity and resistance
Grade 5	Normal power
Grades 4-, 4, and 4+ may be used to indicate movement against slight, moderate, and strong resistance, respectively. *"Used with the permission of the Medical Research Council"*	

NYHA BREATHLESSNESS

For symptoms or signs in patients with defined or presumed cardiac disease.

Grade	Contraction
Class I	Without limitations of physical activity. Ordinary physical activity does not cause undue fatigue, palpitations, or dyspnea
Class II	Slight limitation of physical activity. The patient is comfortable at rest. Ordinary physical activity results in fatigue, palpitations, or dyspnea
Class III	Marked limitation of physical activity. The patient is comfortable at rest. Less than ordinary activity causes fatigue, palpitations, or dyspnea
Class IV	Inability to carry on any physical activity without discomfort. Heart failure symptoms are present even at rest or with minimal exertion

Reference: Criteria Committee of the New York Heart Association. Nomenclature and Criteria for Diagnosis of Diseases of the Heart and Great Vessels. Boston: Little, Brown & Co; 1994.

CANADIAN CARDIOVASCULAR SOCIETY—GRADING OF ANGINA PECTORIS

Grade	Description
Grade 0	Ordinary physical activity does not cause angina, such as walking and climbing stairs. Angina with strenuous or rapid or prolonged exertion at work or recreation
Grade 1	Slight limitation of ordinary activity. Walking or climbing stairs rapidly, walking uphill, walking or stair climbing after meals, or in cold, or in wind, or under emotional stress, or only during the few hours after awakening. Walking more than two blocks on the level and climbing more than one flight of ordinary stairs at a normal pace and in normal conditions
Grade 2	I walk slower than people of the same age on the level because of breathlessness, or I have to stop for breath when walking on my own pace on the level
Grade 3	Marked limitation of ordinary physical activity. Walking one or two blocks on the level and climbing one flight of stairs in normal conditions and at normal pace
Grade 4	Inability to carry on any physical activity without discomfort, anginal syndrome may be present at rest

NINDS MYOTACTIC REFLEX SCALE

Reflex	Description
0	Reflex absent
1	Reflex small, less than normal; includes a trace response or a response brought out only with reinforcement
2	Reflex in lower half or normal range
3	Reflex in upper half of normal range
4	Reflex enhanced, more than normal; includes clonus if present, which optionally can be noted in an added verbal description of the reflex

Reference: Hallett M. NINDS myotatic reflex scale. Neurology. 1993;43(12):2723.

FREEMAN AND LEVINE GRADING OF SYSTOLIC MURMUR

Systolic Murmurs

Levine and Freeman grading of systolic murmurs:

Grade	Description	Thrill

Grade 1	Murmur so faint that it can be heard only with special effort. Heard only after a few seconds have elapsed	Absent
Grade 2	Murmur is faint, but is immediately audible	
Grade 3	Murmur that is moderately loud	
Grade 4	Murmur that is very loud	Present
Grade 5	A murmur is extremely loud and is audible with one edge of the stethoscope touching the chest wall	
Grade 6	A murmur is so loud that it is audible with the stethoscope just removed from contact with the chest wall	

Reference: Levine SA. The systolic murmur: its clinical significance. JAMA. 1933;101(6):436-8.

Diastolic Murmurs (by AIMS)

Grade	Description	Thrill
Grade 1	Very soft	Absent
Grade 2	Soft	
Grade 3	Loud	
Grade 4	Very loud	Present

Grading of Pulse

Grade	Description
0	Pulse not palpable
1+	Faint
2+	Slightly diminished pulse than normal
3+	Normal pulse
4+	Bounding pulse

ABCD AND ABCD2 SCORES (TABLE 15C.1)

Table 15C.1: ABCD and ABCD2 scores.		
	Value	***Score***
ABCD risk factor		
Age	≥60 years	1
Blood pressure	Systolic blood pressure >140 mm Hg or diastolic blood pressure >90 mm Hg	1
Clinical symptoms	Unilateral weakness	2
	Speech disturbance without weakness	1
Duration of symptoms	>60 minutes	2
	10–59 minutes	1
ABCD2 additional factor		
Diabetes	Oral medication or insulin	1

ABCD risk score		
Age	Age ≥60 years	1 point
Blood pressure	Systolic blood pressure >140 mm Hg Diastolic blood pressure >90 mm Hg	1 point
Clinical symptoms	Unilateral weakness, speech disturbance without weakness	2 points 1 point
Duration of symptoms	>60 minutes 10–59 minutes	2 points 1 point

ABCD2 risk score		
Age	Age ≥60 years	1 point
Blood pressure	Systolic blood pressure >140 mm Hg Diastolic blood pressure >90 mm Hg	1 point
Clinical symptoms	Unilateral weakness, speech disturbance without weakness	2 points 1 point
Duration of symptoms	>60 minutes 10–59 minutes	2 points 1 point

Diabetes	Diabetes	1 point

It is reasonable to hospitalize patients with transient ischemic attack who present within 72 hours of symptoms with:

- ABCD2 score of 3 points or higher
- ABCD2 score of 0–2 points with evidence of focal ischemia
- ABCD2 score of 0–2 points if uncertain that patient can obtain outpatient work-up within 2 days.

Reference: Easton JD, Saver JL, Albers GW, et al. Definition and evaluation of transient ischemic attack: a scientific statement for healthcare professionals from the American Heart Association/American Stroke Association Stroke Council; Council on Cardiovascular Surgery and Anesthesia; Council on Cardiovascular Radiology and Intervention; Council on Cardiovascular Nursing; and the Interdisciplinary Council on Peripheral Vascular Disease. The American Academy of Neurology affirms the value of this statement as an educational tool for neurologists. Stroke. 2009;40(6):2276-93.

BODE INDEX (TABLE 15C.2)

Table 15C.2: Variables and point values used for the computation of the body mass index, degree of airflow obstruction and dyspnea, and exercise capacity (BODE) index.*

Variable	*Points on BODE index*			
	0	**1**	**2**	**3**
FEV_1 (% of predicted)†	≥65	50–64	36–49	≤35
Distance walked in 6 minutes (m)	≥350	250–349	150–249	≤149
MMRC dyspnea scale‡	0–1	2	3	4
Body mass index§	>21	≤21		

* The cutoff values for the assignment of points are shown for each variable. The total possible values range from 0 to 10. FEV_1 denotes forced expiratory volume in 1 second.

† The FEV_1 categories are based on stages identified by the American Thoracic Society.

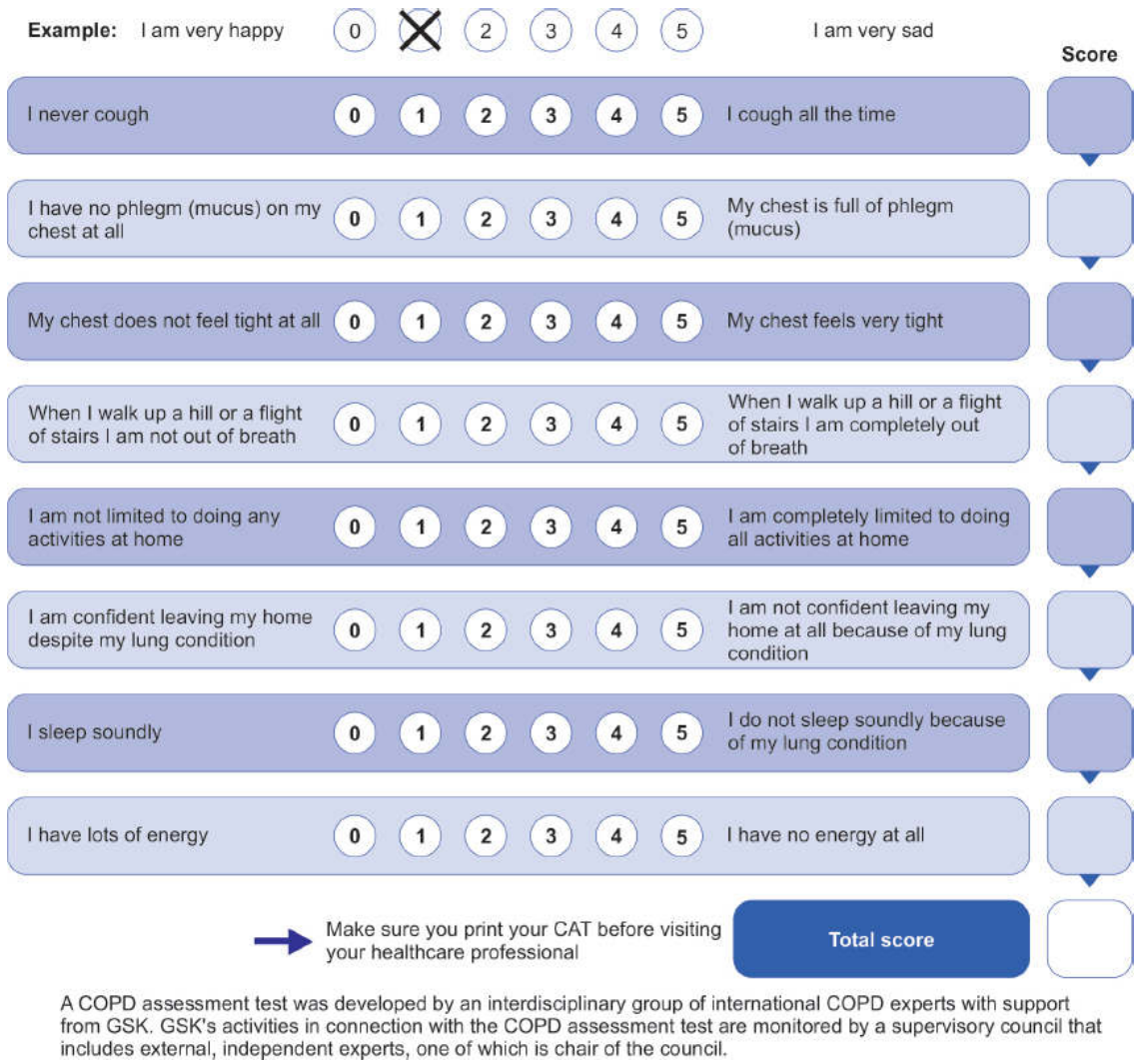
‡ Scores on the modified Medical Research Council (MMRC) dyspnea scale can range from 0 to 4, with a score of 4 indicating that the patient is too breathless to leave the house or becomes breathless when dressing or undressing.

§ The values for body mass index were 0 to 1 because of the infection point in the inverse relation between survival and body mass index at a value of 21.

- Body mass index
- Obstruction = FEV_1 (% of predicted)
- Dyspnea = MMRC dyspnea scale
- Exercise = Distance walked in 6 minutes

Reference: Celli BR, Cote CG, Marin JM, et al. The body-mass index, airflow obstruction, dyspnea, and exercise capacity index in chronic obstructive pulmonary disease. N Engl J Med. 2004;350(10):1005-12.

COPD ASSESSMENT TEST



A COPD assessment test was developed by an interdisciplinary group of international COPD experts with support from GSK. GSK's activities in connection with the COPD assessment test are monitored by a supervisory council that includes external, independent experts, one of which is chair of the council.

CHADS2

Risk factor	Score
Congestive heart failure	1
Hypertension	1
Age ≥75 years	1

Diabetes mellitus	1
Stroke/TIA/TE	2
Maximum score	6

The CHADS2 score for stroke risk in AF.

CHADS-VASC

CHADS-VASc clinical characteristic.

Risk factor	Score
Congestive heart failure	1
Hypertension	1
Age ≥75	2
Age 65–74	1
Diabetes mellitus	1
Stroke/TIA/thromboembolism	2
Vascular disease	1
Sex: Female	1

Reference: https://www.chadsvasc.org.

HAS-BLED

HAS-BLED clinical characteristic.

Clinical characteristic	Points awarded
Hypertension	1
Abnormal liver function	1
Abnormal renal function	1
Stroke	1
Bleeding	1

Labile INRs	1
Elderly (age >65)	1
Drugs	1
Alcohol	1
Your score	0

Reference: https://www.chadsvasc.org/.

EHRA SCORE

Classification of AF-related symptoms (EHRA score).

EHRA I	No symptoms
EHRA II	Mild symptoms; normal daily activity not affected
EHRA III	Severe symptoms; normal daily activity affected
EHRA IV	Disabling symptoms; normal daily activity discontinued

Reference: https://www.chadsvasc.org/.

CHILD-TURCOTTE-PUGH SCORE

Child-Turcotte-Pugh scoring system and Child-Pugh classification.

Parameter	Numerical score		
	1	**2**	**3**
Ascites	None	Slight	Moderate/severe
Encephalopathy	None	Slight	Moderate/severe
Bilirubin (mg/dL)	<2	2–3	>3
Albumin (g/dL)	>3	2.8–3.5	<2.8
Prothrombin time (seconds increased)	1–3	4–6	>6
Total numerical score		***Child-Pugh class***	
5–6		A	
7–9		B	

10–15	C

FRAMINGHAM HEART FAILURE CRITERIA

Diagnosis of CHF requires the simultaneous presence of at least 2 major criteria or 1 major criterion in conjunction with 2 minor criteria.

Major criteria:

- Paroxysmal nocturnal dyspnea
- Neck vein distention
- Rales
- Cardiomegaly
- Acute pulmonary edema
- S3 gallop
- Increased central venous pressure (>16 cm H_2O)
- Sustained hepatojugular reflux
- Circulation time ≥25 seconds.

Minor criteria:

- Ankle edema
- Nocturnal cough
- Dyspnea on ordinary exertion
- Hepatomegaly
- Pleural effusion
- Decrease in vital capacity by one-third from maximum recorded
- Tachycardia (heart rate >120 beats/min).
- Major or minor criterion: Weight loss ≥4.5 kg in 5 days in response to treatment.

Minor criteria are acceptable only if they cannot be attributed to another medical condition (such as pulmonary hypertension, chronic lung disease, cirrhosis, ascites, or the nephrotic syndrome).

GCS

Glasgow Coma Scale (GCS)		

Eye opening		*Best verbal response*		*Best motor response*	
				Obeys commands	**6**
		Oriented and converses	**5**	Localizes pain	**5**
Open spontaneously	**4**	Converses, but disoriented, confused	**4**	Exhibits flexion withdrawal	**4**
Open only to verbal stimuli	**3**	Uses inappropriate words	**3**	Decorticate rigidity	**3**
Open only to pain	**2**	Makes incomprehensible sounds	**2**	Decerebrate rigidity	**2**
Never open	**1**	No verbal response	**1**	No motor response	**1**
Maximum score = **15** Minimum score = **3** Coma is equal to GCS of less than **8 or less.**					

***Mnemonic (GCS → EVM = 4, 5, and 6)**

*In intubated patients, verbal response is denoted as V_T.

WEST HAVEN GRADING OF HEPATIC ENCEPHALOPATHY (TABLE 15C.3)

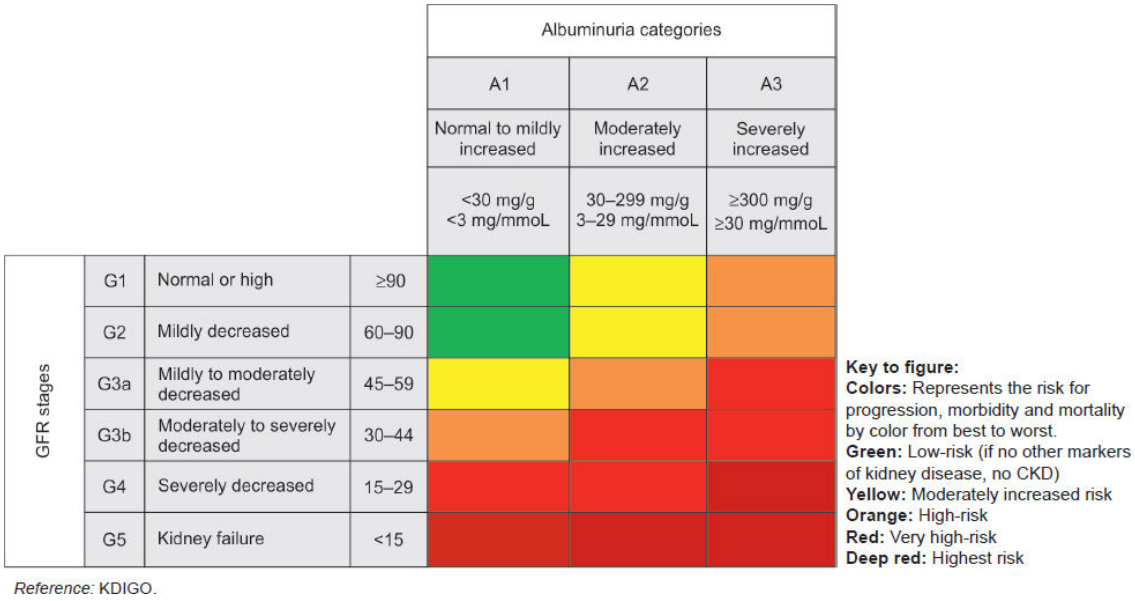
Table 15C.3: Clinical stages of hepatic encephalopathy (HE): The West Haven criteria and the proposed classification of the spectrum of neurocognitive impairment in cirrhosis (SONIC).

West haven criteria			*Sonic*			
Grade	*Intellectual function*	*Neuromuscular function*	*Classification*	*Mental status*	*Special tests*	*Asterixis*
0	***Normal***	***Normal***	***Unimpaired***	***Not impaired***	***Normal***	***Absent***
Minimal	Normal examination findings; sutable changes in work or driving	Minor abnormalities of visual perception or on psychometric or number tests	**Covert HE**	Not impaired	Abnormal	Absent
1	Personality changes, attention deficits,	Tremor and incoordination				

	irritability, depressed state					
2	Changes in sleep-wake cycle, lethargy, mood and behavioral changes, cognitive dysfunction	Asterixis, ataxic gait, speech abnormalities (slow and slurred)	**Overt HE**	Impaired	Abnormal	Present (absent in coma)
3	Altered level of consciousness (somnolence), confusion, disorientation, and amnesia	Muscular rigidity, nystagmus, clonus, babinski sign, hyporeflexia				
4	Stupor and coma	Oculocephalic reflex, unresponsiveness to noxious stimuli				

Reference: Ferenci P, Lockwood A, Mullen K, et al. Hepatic encephalopathy—definition, nomenclature, diagnosis, and quantification: final report of the working party at the 11th World Congress of Gastroenterology, Vienna, 1998. Hepatology. 2002;35(3):716-21; Bajaj JS, Cordoba J, Mullen KD, et al. Review article: the design of clinical trials in hepatic encephalopathy—an International Society for Hepatic Encephalopathy and Nitrogen Metabolism (ISHEN) consensus statement. Aliment Pharmacol Ther. 2011;33(7):739-47.

CKD STAGES



Reference: KDIGO.

Reference: KDIGO.

2015 REVISED JONES CRITERIA

2015 AHA-Revised jones criteria for diagnosis fo rheumatic fever*	
Major criteria	
Low-risk populations	**Moderate-and High-risk populations**
Carditis (clinical or subclinical†)	Carditis (clinical or subclinical)
Arthritis (polyarthritis only)	Arthritis (inculding polyarthritis, monoarthritis or polyarthalgia‡)
Chorea	Chorea
Erythema marginatum	Erythema marginatum
Subcutaneous nodules	Subcutaneous nodules
Minor criteria	
Low-risk populations	**Moderate-and High-risk populations**
Polyarthralgia	Monoarthralgia
Fever (>38.5°C)	Fever (>38°C)
ESR >60 mm in the first hour and/or CRP >3.0 mg/dL	ESR >30 mm in the first hour and/or CRP >3.0 mg/dL§

Prolonged PR interval, after for age variability (unless carditis is a major criterion)	Prolonged PR interval, after accounting for age variability (unless carditis is a major criterion)

Joint manifestations are only considered in either the major or the minor category, but not in both categories in the same patient.

*Annual acute rheumatic fever (ARF) incidence of <2 per 1,00,000 school-aged children or all-age rheumatic heart disease (RHD) prevalence of <1 per 1,000 people per year.

†Defined as echocardiographic valvulitis, table 15C.4.

‡Polyarthralgia should only be considered as a major manifestation in moderate and high-risk populations after exclusion of other causes.

§C-reactive protein (CRP) value must be greater than the normal laboratory upper limit. In addition, because the erythrocyte sedimentation rate (ESR) might evolve during the course of ARF, peak ESR values should be used.

Table 15C.4: The World Heart Federation minimum echocardiographic criteria for diagnosis of pathologic valvular regurgitation caused by rheumatic carditis.

Pathologic mitral regugitation*	Pathologic aortic regurgitation*
1. Seen in at least two views 2. In at least one view, jet length is >2 cm† 3. Peak velocity >3 meter/second 4. Pansystolic jet in at least one envelope	1. Seen in at least two views 2. In at least one view, jet length is >1 cm† 3. Peak velocity >3 meters/second 4. Pandiastolic jet in at least one envelope

*All four Doppler criteria must be met

†A regurgitant jet length should be measured from the vena contract to the last pixel of regurgitant color (blue or red) on nonmagnified (nonzoomed) images.

Reference: Reményi B, Wilson N, Steer A, et al. World Heart Federation criteria for echocardiographic diagnosis of rheumatic heart disease—an evidence-based guideline. Nat Rev Cardiol. 2012;9(5):297-309.

MODIFIED DUKE'S CRITERIA (TABLE 15C.5)

Table 15C.5: Definition of infective endocarditis (IE): Modified Duke criteria.

Definite infective endocarditis
Pathologic criteria • Microorganisms demonstrated by results of cultures or histologic examination of vegetation that has embolized, or an intracardiac abscess specimen; or • Pathologic lesions; vegetation, or intracardiac abscess confirmed by results of histologic examination showing active endocarditis
Clinical criteria • 2 major criteria, or

- 1 major criterion and 3 minor criteria, or
- 5 minor criteria

Possible infective endocarditis

- 1 major criterion and 1 minor critetion, or
- 3 minor criteria

Rejected diagnosis of infective endocarditis

- Firm alterante diagnosis explaining evidence of suspected IE, or
- Resolution of IE syndrome with antibiotic therapy for <4 days, or
- No evidence of IE at surgery of autopsy, on antibiotic therapy for <4 days, or
- Does not meet criteria for possible IE

Definition of terms used in the modified duke criteria for diagnosis of infective endocarditis

Major criteria

Blood culture findings positive for IE

Typical microorganisms consistent with IE from two separate blood cultures

- Viridans streptococci, *streptococcus gallolyticus* (formerly knowns as *S. bovis*), *Staphylococcus aureus*, HACEK group, or
- Community-acquired enterococci, in the absence of a primary focus, or

Microorganisms consistent with IE from persistently positive blood culture findings, defined as

- >2 positive culture findings of blood samples drawn >12 hours apart, or
- 3 or more of >4 separate culture findings of blood (with first and last sample drawn >1 hour apart)
- Single positive blood culture for coxiella burnetti or anti-phase I IgG liter >1:800

Evidence of endocardial involvement

Echocardiographic findings positive for IE [TEE recommended in patients with prosthetic valves, rated at least possible IE by clinical criteria or comlicated IE (paravalvular) abscess TTE as first test in other patients] defined as follow:

- Oscillating intracardiac mass on valve or supporting structures, in the path of regurgitant jets, or on implanted material in the absence of an alternative anatomic explanation, or
- Abscess, or
- New partial dehiscence of prosthetic valve

New valvular regurgitation; worsening or changing of pre-existing murmur not sufficient

Minor criteria

- Predisposition, predisposing heart condition, or intravenous drug use
- Fever—temperature >38°C
- Vascular phenomena, major arterial emboli, septic pulmonary infacts, mycotic aneurysm, intracranial hemorrhage, conjunctival hemorrhages, and Janeway lesions

- Immunologic phenomena: Glomerulonephritis, Osler nodes, roth spots, and rheumatoid factor
- Microbiologic evidence: Positive blood culture findings but does not meet a major criterion as noted above (excludes single positive culture findings for coagulase-negative staphylococci and organisms that do not cause endocarditis) or serologic evidence of active infection with organism consistent with IE

(TEE: transesophageal echocardiography; TTE: transthoracic echocardiography)

Reference:

Li JS, Sexton DJ, Mick N, et al. Proposed modifications to the Duke criteria for the diagnosis of infective endocarditis. Clin Infect Dis. 2000;30(4):633-8.

CAGE QUESTIONNAIRE

- Have you ever felt you should cut down on your drinking?
- Have people annoyed you by criticizing your drinking?
- Have you ever felt bad or guilty about your drinking?
- Have you ever had a drink first thing in the morning to steady your nerves or to get rid of a hangover (eye opener)?
- Scoring: Item responses on the CAGE are scored 0 or 1, with a higher score an indication of alcohol problems.
- A total score of 2 or greater is considered clinically significant.

Reference: Steinweg DL, Worth H. Alcoholism: the keys to the CAGE. Am J Med. 1993;94(5):520-3.

LIGHT'S CRITERIA

These criteria classify an effusion as exudate if one or more of the following are present:

1. The ratio of pleural fluid protein to serum protein is greater than 0.5
2. The ratio of pleural fluid lactate dehydrogenase (LDH) to serum LDH is greater than 0.6
3. The pleural fluid LDH level is greater than two-third of the upper limit of normal for serum LDH.

Reference:

Light RW. Clinical practice. Pleural effusion. N Engl J Med. 2002;346(25):1971-7.

qSOFA

A patient is said to have high-risk for developing adverse outcomes if two out of:

- Altered mental status (GCS <15)
- Hypotension (systolic BP ≤ 100 mm Hg), and
- Tachypnea (respiratory rate ≥22 breaths/min) are present.

SOFA

	Score				
System	0	1	2	3	4
Respiration PaO_2/FiO_2 mm Hg (kPa)	≥400 (53.3)	<400 (53.3)	<300 (40)	<200 (26.7) with respiratory support	<100 (13.3) with respiratory support
Coagulation platelets ($\times 10^3/\mu L$)	≥150	<150	<100	<50	<20
Liver bilirubin µmol/L (mg/dL)	<20 (1.2)	20–32 (1.2–1.9)	33–101 (2.0–5.9)	102–204 (6.0–11.9)	>204 (12.0)
Cardiovascular (catecholamine doses in µg/kg/min for at least 1 hour)	MAP ≥70 mm Hg	MAP <70 mm Hg	Dopamine <5 or dobutamine (any dose)	Dopamine—5.1–15 or adrenaline ≤0.1 or noradrenaline ≤0.1	Dopamine >15 or adrenaline >0.1 or noradrenaline >0.1
Central nervous system Glasgow coma scale score	15	13–14	10–12	6–9	<6
Renal creatinine µmol/L (mg/dL)	<110 (1.2)	110–170 (1.2–1.9)	171–299 (2.0–3.4)	300–440 (3.5–4.9)	>440 (5.0)
Urine output (mL/day)				<500	<200

CURB 65

Confusion of new onset (defined as an AMTS of 8 or less)	1 point
Blood **U**rea nitrogen greater than 7 mmol/L (19 mg/dL)	1 point
Respiratory rate of 30 breaths/min or greater	1 point
Blood pressure less than 90 mm Hg systolic or diastolic blood pressure 60 mm Hg or less	1 point
Age **65** years or older	1

	point

The risk of death at 30 days increases as the score increases:

- 0—0.6%
- 1—2.7%
- 2—6.8%
- 3—14.0%
- 4—27.8%
- 5—27.8%

Reference: Lim WS, van der Eerden MM, Laing R, et al. Defining community acquired pneumonia severity on presentation to hospital: an international derivation and validation study. Thorax. 2003;58(5):377-82.

FORREST GRADING OF GI ULCERS

Acute hemorrhage:

- Forrest I a (spurting hemorrhage)
- Forrest I b (oozing hemorrhage).

Signs of recent hemorrhage:

- Forrest II a (pigmented protuberance or nonbleeding visible vessel)
- Forrest II b (adherent clot)
- Forrest II c (flat pigmented spot).

Lesions without active bleeding:

- Forrest III (Clean-based ulcer).

Reference: Forrest JA, Finlayson ND, Shearman DJ. Endoscopy in gastrointestinal bleeding. Lancet. 1974;2(7877):394-7.

SEVERITY INDEX FOR ULCERATIVE COLITIS (TABLE 15C.6)

Table 15C.6: Truelove and Witts' severity index for ulcerative colitis.

Features	*Mild*	*Moderate*	*Severe*
Bowel movements (number per day)	Fewer than 4	4–6	6 or more plus at least one of the features of systemic upset (marked with * below)
Blood in stools	No more than	Between	Visible blood

	small amounts of blood	mild and severe	
Pyrexia (temperature greater than 37.8°C)*	No	No	Yes
Pulse rate greater than 90 bpm*	No	No	Yes
Anemia*	No	No	Yes
Erythrocyte sedimentation rate (mm/hour)*	30 or below	30 or below	Above 30

Comparison of JNC 7 and 2017 guidelines AHA classification of hypertension.				
SBP (mm Hg)	***And/Or***	***DBP (mm Hg)***	***JNC 7***	***2017 GL***
<120	and	<80	Normal BP	Normal BP
120–129	and	<80	Prehypertension	Elevated BP
130–139	or	80–89	Prehypertension	Stage 1 hypertension
140–159	or	90–99	Stage 1 hypertension	Stage 2 hypertension
≥160	or	≥100	Stage 2 hypertension	Stage 2 hypertension

BP should be based on an average of >2 careful readings on ≥2 occasions. Adults with systolic blood pressure (SBP) or diastolic blood pressure (DBP) in 2 categories should be designated to the higher BP category.

ESC guidelines for classification of hypertension.			
Category	***Systolic (mm Hg)***	***And/Or***	***Diastolic (mm Hg)***
Optimal	<120	and	<80
Normal	120–129	and/or	80–84
High normal	130–139	and/or	85–89
Grade 1 hypertension	140–159	and/or	90–99
Grade 2 hypertension	160–179	and/or	100–109
Grade 3 hypertension	>180	and/or	≥110
Isolated systolic hypertension*	≥140	and	<90

*Isolated systolic hypertension is graded 1, 2, or 3 according to SBP values in the ranges indicated.

D. LABORATORY VALUES OF CLINICAL IMPORTANCE

Hematology and Coagulation (Table 15D.1)

Table 15D.1: Hematology and coagulation.

Component (specimen)	*Reference value*	
	Conventional	*SI units*
RBCs and hemoglobin		
RBC count • **Males** • **Females**	4.5–5.5 × 10^{12}/L (mean 5.0 × 10^{12}/L) 3.8–4.8 × 10^{12}/L (mean 4.3 × 10^{12}/L)	
RBC diameter	6.7–7.7 µm (mean 7.2 µm)	
RBC indices (absolute values) • **Mean corpuscular volume (MCV)** • **Mean corpuscular hemoglobin (MCH)** • **Mean corpuscular hemoglobin concentration (MCHC)** • **Red cell distribution width (RDW)**	82–100 fL 27–32 pg 31–35 g/dL 11.5–14.0%	
RBC lifespan	120 days	
Erythrocyte sedimentation rate (ESR) (Whole blood) • **Westergren, 1st hour** – **Males** – **Females** – **Children**	0–15 mm 1st hour 0–20 mm 1st hour 0–10 mm 1st hour	
• **Wintrobe, 1st hour** – **Males** – **Females**	0–9 mm 1st hour 0–20 mm 1st hour	
Ferritin (serum) • **Males** • **Females**	20–300 ng/mL 15–200 ng/mL	20–300 µg/L 15–200

		µg/L
Folate (serum)	3–20 µg/L	3–20 ng/mL
Hematocrit (PCV) • **Males** • **Females** • **Infants (cord blood)**	38–47% 36–46% 45–70%	
Haptoglobin (serum)	40–240 mg/dL	0.4–2.4 g/L
Hemoglobin (Hb) • **Adult hemoglobin (HbA)** • **Males** • **Females** • **Hemoglobin A_2 (HbA_2)** • **Hemoglobin, fetal (HbF) in adults** • **HbF, children under 6 months**	95–98% 13.0–17.0 g/dL 12.0–15.0 g/dL 1.5–3.5% <0–2% <5%	
Iron, total (serum) • **Total iron binding capacity (TIBC)** • **Iron saturation**	50–150 µg/dL 310–340 µg/dL 20–45%	7–25 µmol/L 45–73 µmol/L 0.20–0.45
Osmotic fragility • **Slight hemolysis** • **Complete hemolysis** • **Mean corpuscular fragility**	at 0.45 to 0.39 g/dL NaCl at 0.33 to 0.36 g/dL NaCl 0.4–0.45 g/dL NaCl	
Reticulocytes • **Adults** • **Infants** • **Newborn (cord blood)**	0.5–2.5% 2–6% 1–7%	
Transferrin saturation • **Male** • **Female**	25–56% 14–51%	
Vitamin B_{12} (serum) • **Body stores** • **Daily requirement** • **Serum level**	10–12 mg 2–3 µg 280–1000 pg/mL	
Autohemolysis test (whole blood)	0.4–4.50%	0.004–

		0.045
Autohemolysis test with glucose (whole blood)	0.3–0.7%	0.003–0.007
Leukocytes		
Differential leukocyte count (DLC) • **P (polymorphs or neutrophils)** • **L (lymphocytes)** • **M (monocytes)** • **E (eosinophils)** • **B (basophils)**	40–70% (2,000–7,500/µL) 20–40% (1,500–4,000/µL) 2–10% (200–800/µL) 1–6% (40–450/µL) <1% (10–100/µL)	
Total leukocyte count (TLC) • **Adults** • **Infants (full term, at birth)** • **Infants (1 year)**	4,000–11,000/µL 10,000–25,000/µL 6,000–16,000/µL	
Platelets and coagulation		
Bleeding time (BT) • **Ivy's method** • **Template method**	2–7 minutes 2–9 minutes	
Clot retraction time (clotted blood) • **Qualitative** • **Quantitative**	Visible in 60 minutes (complete in <24 hours) 48–64% (55%)	
Clotting time (CT) • **Lee and White method**	4–11 minutes	
D-dimer (plasma)	220–740 ng/mL	
Fibrinogen (plasma)	200–400 mg/dL	
Fibrin split (or degradation) products (FSP or FDP)	<10 µg/mL	<10 mg/L
Partial thromboplastin time with kaolin (PTTK) or activated partial thromboplastin time (APTT/aAPTT)	30–40 seconds	
Platelet count	150,000–450,000/µL	
Prothrombin time (PT) (Quick's one stage method)	11–16 sec	
Thrombin time (TT)	15–19 sec (control ± 2 sec)	

Clinical Chemistry of Blood (Table 15D.2)

Table 15D.2: Clinical chemistry of blood.			
Component	***Specimen***	***Reference value***	
		Conventional	***SI units***
Alpha fetoprotein (AFP), adults	Serum	0–8.5 ng/mL	0–8.5 µg/L
Aminotransferases (transaminases) • **Aspartate (AST, SGOT)** • **Alanine (ALT, SGPT)**	Serum Serum	12–38 U/L 7–41 U/L	0.20–0.65 µkat/L 0.12–0.70 µkat/L
Amylase	Serum	20–96 U/L	0.34–1.6 µkat/L
Bilirubin • **Total** • **Direct (conjugated)** • **Indirect (unconjugated)**	Serum	0.3–1.3 mg/dL 0.1–0.4 mg/dL 0.2–0.9 mg/dL	5.1–22 µmol/L 1.7–6.8 µmol/L 3.4–15.2 µmol/L
CA 125	Serum	0–35 U/mL	0–35 Ku/L
Calcium—ionized	Whole blood	4.5–5.3 mg/dL	1.12–1.32 mmol/L
Calcium—total	Serum	8.7–10.2 mg/dL	2.2–2.6 mmol/L
Chloride	Serum	102–109 mEq/L	102–109 mmol/L
C-reactive proteins	Serum	0.2–3.0 mg/L	0.2–3.0 mg/L
Creatine kinase (CK), total • **Males** • **Females**	Serum	51–294 U/L 39–238 IU/L	0.87–5.0 µkat/L 0.66–4.0 µkat/L
Creatine kinase MB (CKMB)	Serum	0–5.5 ng/mL	0–5.5 µg/L
Gamma glutamyl transpeptidase (transferase) (γ-GT)	Serum	9–58 IU/L	0.15–1.00 µmol/L
Glucose (fasting) • **Normal** • **Impaired fasting glucose (IFG)** • **Diabetes mellitus**	Plasma	70–100 mg/dL 101–125 mg/dL >126 mg/dL	<5.6 mmol/L 5.6–6.9 mmol/L >7.0 mmol/L
Glucose (2-hour postprandial)	Plasma	<140 mg/dL	<7.8 mmol/L

• **Normal** • **Impaired glucose tolerance (IGT)** • **Diabetes mellitus**		140–200 mg/dL >200 mg/dL	7.8–11.1 mmol/L >11.1 mmol/L
Glycated hemoglobin (HbA_{1c})	Whole blood	4.0–6.0%	20–42 mmol/mol Hb
Lactate dehydrogenase (LDH)	Serum	115–221 U/L	2.0–3.8 μkat/L
Muramidase	Serum	5–20 μg/mL	
5-nucleotidase	Serum	0–11 U/L	0.02–0.19 μkat/L
Phosphatases • **Acid phosphatase** • **Alkaline phosphatase**	Serum	0–5.5 U/L 33–96 U/L	0.90 μkat/L 0.56–1.63 μkat/L
Prostate-specific antigen (PSA)	Serum	0–4.0 ng/mL	0–4.0 μg/L
Proteins—total • **Albumin** • **Globulins** • **Albumin/globulin ratio**	Serum	6.7–8.6 g/dL 3.5–5.5 g/dL 2.0–3.5 g/dL 1.5–3:1	67–86 g/L 35–55 g/L 20–35 g/L
Rheumatoid factor	Serum	<15 IU/mL	<15 kIU/L
Troponins, cardiac (cTn) • **Troponin I (cTnI)** • **Troponin T (cTnT)**	Serum Serum	0–0.08 ng/mL 0–0.01 ng/mL	0–0.8 μg/L 0–0.1 μg/L
Urea nitrogen (BUN)	Blood	7–20 mg/dL	2.5–7.1 mmol/L
Uric acid • **Males** • **Females**	Serum	3.1–7.0 mg/dL 2.5–5.6 mg/dL	0.18–0.41 μmol/L 0.15–0.33 μmol/L

Lipid Profile (Table 15D.3)

Table 15D.3: Lipid profile.

Component	*Reference value*	
	Conventional	*SI units*
Total serum cholesterol • **Desirable for adults** • **Borderline high**	<200 mg/dL 200–239 mg/dL >240 mg/dL	<5.17 mmol/L 5.17–6.18 mmol/L >6.21 mmol/L

• **High undesirable**		
LDL cholesterol • **Desirable range** • **Borderline high** • **High** • **Very high**	100–130 mg/dL 130–159 mg/dL 160–189 mg/dL >190 mg/dL	<3.34 mmol/L 3.36–4.11 mmol/L 4.11–4.20 mmol/L >4.21 mmol/L
HDL cholesterol • **Low** • **High, protective range**	<40 mg/dL >60 mg/dL	<1.03 mmol/L >1.55 mmol/L
Triglycerides	<160 mg/dL	<2.26 mmol/L

Urea and Electrolytes (Table 15D.4)

Table 15D.4: Urea and electrolytes.

Analyte	*Reference value*	
	Conventional	*SI units*
Sodium	136–146 mEq/L	136–146 mmol/L
Potassium	3.5–5.0 mEq/L	3.5–5.0 mmol/L
Chloride	95–107 mEq/L	95–107 mmol/L
Urea	20–40 mg/dL	3.3–6.6 mmol/L
Creatinine	0.6–1.2 mg/dL	53–106 µmol/L

Thyroid Function Tests (Table 15D.5)

Table 15D.5: Thyroid function tests.

Thyroid function tests	*Specimen*	*Reference value*	
		Conventional	*SI units*
Radioactive iodine uptake (RAIU) 24 hours		5–30%	
Thyroxine (T4) total	Serum	5.4–11.7 µg/dL	70–151 nmol/L
Triiodothyronine (T3) total	Serum	77–135 ng/dL	1.2–2.1 nmol/L
Thyroid stimulating hormone (TSH)	Serum	0.4–4.25 µU/mL	0.4–4.25 mU/L

Urine (Table 15D.6)

Table 15D.6: Normal urine values.	
Component	***Reference value***
Volume—24 hours	600–1800 mL (variable)
pH	5.0–9.0
Specific gravity, quantitative (random)	1.002–1.028 (average 1.018)
Protein—24 hours urine	<150 mg/day
Protein, qualitative (random)	Negative
Glucose, quantitative—24 hours urine	50–300 mg/day
Glucose, qualitative (random)	Negative
Urobilinogen—24 hours urine	1.0–3.5 mg/day
Microalbuminuria (24 hours)	0–30 mg/24 hours (0–0.03 g/day) (0–30 µg/mg creatinine) (0–0.03 g/g creatinine)

Cerebrospinal Fluid (Table 15D.7)

Table 15D.7: Normal values of cerebrospinal fluid.		
Component	***Reference value***	
	Conventional	***SI units***
CSF volume	120–150 mL	
Appearance	Clear and colorless	
CSF pressure	60–150 mm water	
pH	7.31–7.34	
Total proteins	20–40 mg/dL	0.14–0.45 g/L
Glucose	40–80 mg/dL	2.3–4.5 mmol/L
Chlorides	720–750 mg/dL	
Cells • **Polymorphs** • **Lymphocytes**	Usually absent 0–5/µL	

SHORT LIST OF ROUTINELY USED FORMULAS IN MEDICINE (TABLE 15D.8)

Table 15D.8: Short list of routinely used formulas in medicine.	
Electrolyte disorders	
Plasma osmolality	2 Na^+ (mEq/L) + Serum glucose (mg/dL)/18 + BUN (mg/dL)/2.8
Corrected sodium	Increase Na^+ by 1.6 mEq/L for each 100 mg% (when serum glucose >100 mg%)
Total body sodium deficit	(Desired sodium – measured sodium) × Body weight × [0.6 (men) or 0.5 (women)]
Potassium deficit	1 mmol/L decrease → approximately 200–400 mmol loss of total body K^+
Urine-plasma electrolyte ratio (in chronic hyponatremia)	Urinary (sodium + potassium)/plasma sodium • >1 (fluid restriction up to less than 500 mL/day) • =1 (500–700 mL/day) • <1 (fluid restriction up to 1 L)
Water deficit (in hypernatremia)	Water deficit = (plasma sodium – 140) × TBW/140
Transtubular potassium gradient (TTKG) in hypokalemia	Urinary potassium × plasma osmolality/serum potassium × urinary osmolality • >4 indicates renal loss of potassium
Corrected calcium	0.8 × (4 – serum albumin) + serum calcium
Acid base disorders	
Anion gap (serum)	(Sodium + potassium) – (bicarbonate + chloride) • 8–16 mEq/L (old methods) • 5–11 mEq/L (new techniques)
Urine anion gap	Urine sodium + potassium – chloride • –25 to –50 (normal range)
Delta ratio	(Serum anion gap – 12)/(24 – serum bicarbonate) • <0.4 hyperchloremic normal anion gap acidosis • <1 high AG and normal AG acidosis • >2 high AG acidosis and a concurrent metabolic alkalosis
Respiratory acidosis	Acute: 10 increase in pCO_2 → 1 increase in

	bicarbonate Chronic: 10 increase in pCO_2 → 4 increase in bicarbonate
Respiratory alkalosis	Acute: 10 decrease in pCO_2 → 2 decrease in bicarbonate Chronic: 10 decrease in pCO_2 → 5 decrease in bicarbonate
Metabolic acidosis	pCO_2 = 1.5 (bicarbonate) + 8 ± 2
Metabolic alkalosis	10 increase in bicarbonate → pCO_2 increases by 6
Nephrology	
Renal failure index	Urine Na/(Urine Cr/PCr)
Cockcroft-Gault GFR	(140 – Age) × (Body wt. in kg) × (0.85 if female)/(72 × Cr)
Fractional excretion of sodium (FENa)	(Serum Cr × Urine Na)/(Serum Na × Urine Cr)%
Hematology	
Corrected reticulocyte count	Reticulocyte % × (Hb/15)
Reticulocyte production index	• = Corrected reticulocyte count/maturation time • At a hemoglobin of 15, the maturation time = 1 day • At a hemoglobin of 12, the maturation time = 1.5 days • At a hemoglobin of 8, the maturation time = 2 days • At a hemoglobin of 5, the maturation time = 2.5 days
Mentzer index	(MCV, in fL) divided by (RBC, in millions per µL) • Less than 13, thalassemia is said to be more likely
Parenteral iron in iron deficiency anemia	[2.3 × body weight (kg) × Hb deficit (g/dL)] + 1000 mg (to replenish stores)
Respiratory system	
A-a gradient	$PAO_2 – PaO_2$ ($PAO_2 = (FiO_2 \times 713) – PaCO_2/0.8$; PaO_2 is obtained from the ABG)
Cardiology	
Corrected QT	QT/√ RR (Bazzett's formula)

MAP	Systolic BP + (2 × diastolic BP)/3
Miscellaneous	
BMI	W/H^2 (W = weight in kg and H = Height in meters)

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