Textbooks of Military Medicine

COMBAT ANESTHESIA: THE FIRST 24 HOURS

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1818
Medical Department of the Army

A 1976 etching by Vassil Ekimov of an
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COMBAT ANESTHESIA: THE FIRST 24 HOURS

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Foreword

I am pleased to present this volume, entitled C*ombat Anesthesia: The First 24 Hours*, published by the Army Medical Department's Borden Institute. The Borden Institute, part of the Army Medical Department Center and School, is the primary outlet for scholarly and peer-reviewed publications describing observations made and science conducted by the healthcare providers who take care of our Nation's Service Members and Veterans. The Institute's publications do not necessarily represent Army doctrine or the opinion of the Department of Defense or the Army; nevertheless, they represent our providers best work as they seek to inform future policy and decision-making.

This book focuses on anesthesia care during the 24 hours following battle wounds. It is written by British and American physicians who began this collaboration while providing acute care to injured Soldiers of both countries at Camp Bastion and Fort Leatherneck in the Helmand province of Afghanistan. These authors, having deployed throughout Afghanistan and Iraq, address the ways in which care was delivered by U.S. and British trauma teams working together and sharing their competence. This is a story of how these expert physicians organized care and improved in-hospital patient outcomes. The principles presented in this book are also relevant to trauma care in non-military hospitals in the United States, Britain, and beyond.

Looking back, the start of modern military anesthesia can be linked to the expansion of the role of anesthesiology in the post–Vietnam War era. Since then, many of the medical tools have evolved, enhancing the way we care for the trauma patient today. Airway management, vasopressor drug therapy and initiatives in resuscitation, and an array of antibiotic regimens are examples of advancement in acute trauma care over these years. Surgery and postoperative care are now safer and more reliably associated with better patient outcomes. Continued development in these clinical areas has allowed anesthesia providers to provide wounded Soldiers a level of care previously unattainable.

No one individual or group of practitioners is solely responsible for improved survival rates over the course of the war in Iraq and Afghanistan. Combat casualty care begins at the point of injury with the Combat Medic and continues with the collaborative teamwork of all military medical personnel, including technicians, nurses, physicians, and other medical specialists. With the growing experience in the acute care of wounded patients and the implementation of newer technologies during the wars in Iraq and Afghanistan, the survival rates of wounded Service Members has dramatically improved, from 83% (in 2002) to 92% (in 2014). This achievement is a direct result of jointly coordinated efforts by an entire team of military medical personnel.

I congratulate the authors on this collaborative effort, and I admire them for building a strong professional bridge between our countries through the practice of medicine. These officers recognized the importance of continuing, and then strengthening, the relationship formed in the operating rooms during the last 13 years of conflict. I recognize this effort as an important part of the tradition of military medicine, that is, presenting the lessons learned and preserving this knowledge for the next generation of military providers caring for our Service Members injured in combat. Whether the trauma care provider is a physician, nurse, or Combat Medic, the ensuing chapters of this text will serve as a valuable resource in documenting these lessons.

Serving to Heal . . . Honored to Serve!

Patricia D. Horoho
Lieutenant General, US Army
The Surgeon General and
Commanding General,
US Army Medical Command

Washington, DC
December 2014

Prologue

Surgeons rely on the capabilities of their anesthetist colleagues to ensure that complex procedures can be undertaken safely and successfully. This is no less the case during care for critically wounded patients from the battlefield, when surgical prowess requires the highest caliber of anesthesia. The advances achieved by multinational anesthesia teams in Iraq and Afghanistan over the last dozen years have changed thinking not only in the military but also in civilian practice. The authors of this text have drawn on their experiences in sustaining the physiology of the severely injured during prehospital transit, delivering a stabilized patient to the waiting surgical teams and improving survival chances. They have developed direct theater access and novel resuscitation and transfusion protocols aided by thrombo-elastography, which have become accepted civilian protocols. The military patient, however, may need to travel many thousands of miles to return home. Ensuring en-route pain relief with local anesthetic infusions that tolerate in-flight pressure changes has helped deliver patients pain free to the final hospital destination in the home country. Such practice is supported by research to develop an evidence base, and the authors have drawn on research from both sides of the Atlantic to underpin their knowledge. This ongoing research must remain vital for the future development of military anesthesia, even after the end of major conflict.

I have operated on the almost moribund at the multinational trauma hospital in Camp Bastion, Afghanistan. Patients who might not have survived 10 years ago, some with multiple injuries including triple limb amputation, are now carefully resuscitated and deftly anesthetised while their disrupted physiology is gradually restored. The depth of experience of our dedicated military anesthetic teams is distilled in this book, which will be of benefit to our civilian colleagues working in trauma hospitals as well as military providers in future conflicts. I would like to take the opportunity, on behalf of all military surgeons, to thank our anesthetist colleagues for the superb support they have provided to the great benefit of our patients.

Surgeon Rear Admiral Alasdair J. Walker, OBE, QHS, FRCS
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Lichfield, United Kingdom
October 2014

Preface

The genesis for this book began as a conversation between medical officers and anesthesiologists from different coalition countries in a tent in Camp Bastion, Afghanistan. The officers were discussing the advances in battlefield anesthetic care that had been achieved in the Iraq and Afghanistan conflicts, and the need to preserve this knowledge for the next generation of military anesthesia providers serving in upcoming wars. In short, it was felt to be ethically indefensible not to collect, organize, and record the advances in anesthetic practice that military anesthesia providers have achieved in the last 13 years of conflict. It was determined that the text would be a collaborative effort between military anesthesia providers of both the United States and United Kingdom, leveraging the experiences of the countries that provided the largest military medical response to the recent conflicts. The majority of chapters are products of this collaboration and naturally contain different perspectives of the two countries.

If anything positive can be said of war, it would be that it serves as a catalyst for rapid improvements in medical understanding and care. In the conflicts in Afghanistan and Iraq, coalition medical forces achieved a died-of-wounds rate below 10%. This statistic is historic and unprecedented in armed conflict. Many factors have contributed to this achievement, including improvements in body armor, highly trained medics, greater availability of blood and blood products, improved medical imaging far forward, faster evacuation with improved en-route care, and enhanced surgical approaches to wounds and trauma. Advances in battlefield anesthesia have made modern battlefield trauma resuscitation and surgery possible and thus have contributed greatly to enhanced survival of the injured.

The goal of this book is to document recent lessons learned in the anesthetic care of combat casualties and serve as a training foundation for anesthesia providers tasked with or contemplating providing anesthetic and analgesic care in future conflicts and disasters. The majority of its authors have deployed in the recent conflicts and are recognized authorities in the areas their chapters cover. This text is a tribute to their efforts and the patients they cared for and a gift to the next generation of combat anesthesia providers. We also take the opportunity to thank Mr Raul Gordon, of the Henry Jackson Foundation and Defense & Veteran Center for Integrative Pain Management, and Ms Alison Bess, of the Royal Centre of Defence Medicine. They have chased down authors, kept editors honest, and organized complex contributions. Without them there would be no book.

It was no easy task editorially; truly the United States and United Kingdom are two historical allies separated by a common language.

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Chapter 1

PHYSIOLOGY OF INJURY AND EARLY MANAGEMENT OF COMBAT CASUALTIES

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INTRODUCTION

The recent conflicts in Iraq and Afghanistan have resulted in a marked change in both the injury patterns sustained by wounded personnel and the subsequent management of these injuries. Survival of combat casualties (CCs) despite severe polytrauma is largely attributable to improvements in body armor,[1] prompt initiation of emergency medical care in accordance with Tactical Combat Casualty Care (TCCC) guidelines in proximity to point of wounding,[2,3] tactical damage control surgery and resuscitation, and rapid transport to escalating levels of care with "critical care in the air" intensive monitoring and care capability.

GOLDEN HOUR

Improvised explosive devices (IED) have been the most common mechanism of CC injury in these conflicts.[4–9] Troops in mine-resistant ambush-protected (MRAP) vehicles involved in IED blasts are well-protected, and injuries are usually limited to lower extremity and axial skeleton-loading fractures.[10] However, the most severe blast injuries are endured by troops on foot patrol who are involved in dismounted IED blasts in which they are exposed to primary, secondary, tertiary, and quaternary blast trauma.[11] Modern advancements in body armor worn by troops (including Kevlar [DuPont, Wilmington, DE] helmets and Kevlar vests with ceramic plates) have improved survival by decreasing secondary blast injuries to the head and thorax. Despite widespread use of body armor, dismounted troops involved in IED blasts can still suffer traumatic brain injury (TBI), as well as burns, soft tissue injury, fractures, and neurovascular damage to the pelvis, perineum, and extremities.[5,8,9,12,13] These insults manifest clinically as mangled or amputated appendages, altered mental status, pain, cardiovascular collapse, respiratory failure, loss of airway, visceral injury, and acute hemorrhage.

The severe blood loss associated with blast injuries decreases oxygen delivery, and the CC enters a state of physiological shock in which the supply of oxygen fails to meet the demand of the tissues, resulting in end-organ dysfunction. The body promptly responds to the hemorrhage by inducing intense vasoconstriction to shunt blood centrally to the heart and brain.[14,15] Decreased perfusion to the peripheral tissues induces anaerobic respiration, which produces lactic acid, ultimately leading to metabolic acidosis. Metabolic acidosis, in turn, alters the function of multiple critical enzymes[16–18] and leads to coagulopathy.[19] The state of shock, coupled with exposure to the elements, can induce hypothermia, thereby further exacerbating the coagulopathy, which leads to continued hemorrhage.[20–22] The *lethal triad* (Figure 1-1) of acidosis, hypothermia, and coagulopathy becomes a vicious cycle, which, if uninterrupted, rapidly leads to death.

The polytraumatic nature of the injuries from these blasts[23] exposes the CC not only to the immediate threat of hemorrhagic, obstructive, cardiogenic, and neurogenic shock, but also to a severe posttraumatic inflammatory response,[24,25] which can lead to traumatic shock.[26,27] The extent of injuries and time to medical care dictate the magnitude of shock,[28] as measured by oxygen debt, which correlates significantly with the risk of mortality.[29–31] Furthermore, the magnitude of shock is also closely associated with a potentially overwhelming immune response, which can cause acute respiratory distress syndrome (ARDS) and multiple organ dysfunction syndrome.[32–36] This cascade of physiologic events illustrates the importance of minimizing time to medical intervention as defined by the principle of the "golden hour" of trauma. This time is vital to the care of trauma victims and is embodied in the prehospital principles of military trauma care by first responders in TCCC as well as rapid transfer to higher levels of surgical and resuscitative care.

Tactical Combat Casualty Care

TCCC is initiated at the time of the blast and is implemented during care under fire, through tactical field care, and finally during tactical evacuation care (Figure 1-2).[3] The emergent needs for the CC include airway and ventilation maintenance, hemorrhage control with rapid application of tourniquets[37–39] and

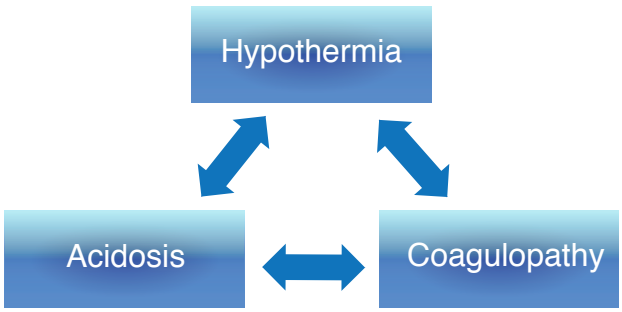

Figure 1-1. Lethal triad.

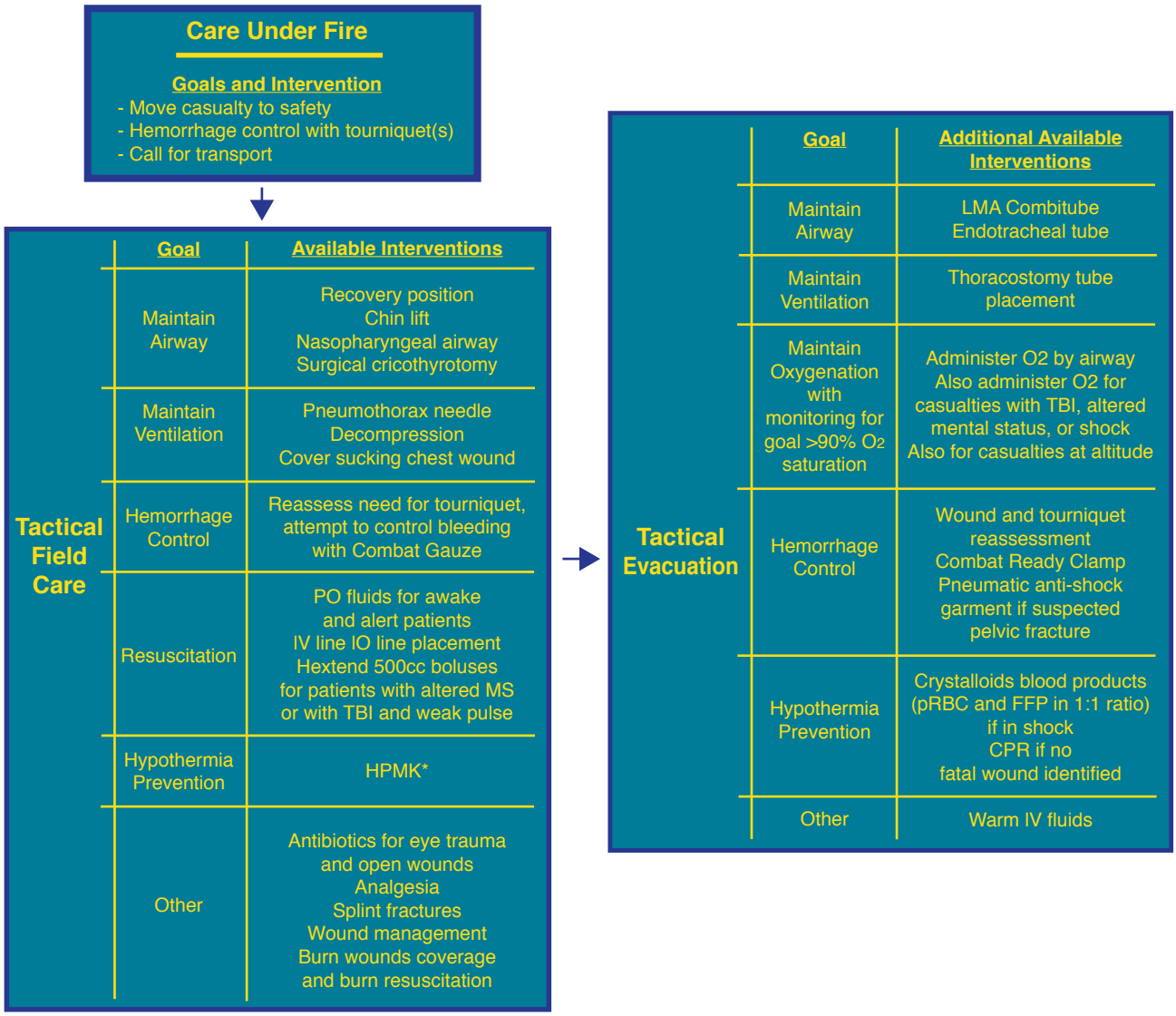

Figure 1-2. Tactical Combat Casualty Care (TCCC). As the combat casualty progresses through the stages of TCCC, he or she undergoes serial evaluations and repeated interventions as necessary. More resources and interventions are available at higher stages.
cc: cubic centimeters; HPMK: Hypothermia Prevention/Management Kits (North American Rescue, Greer, SC; Part Number: 80-0027; NSN: 6515-01-532-8056.); LMA: laryngeal mask airway; O_2: oxygen TBI: traumatic brain injury; FFP: fresh frozen plasma; pRBC: packed red blood cells; CPR: cardiopulmonary resuscitation; IV: intravenous
Adapted from: Military Health System website. Tactical Combat Casualty Care Guidelines. August 8, 2011. http://www.health.mil/Education_And_Training/TCCC.aspx. Accessed September 26, 2012.

hemostatic agents,[40,41] judicious infusion of resuscitative fluids for CCs in shock with Hextend (BioTime, Inc, Berkeley, CA),[42–46] and prevention of hypothermia. These initial management steps—performed by self-aid, buddy aid, and medics in austere environments—and the rapid transport to higher levels of care are designed to prevent progression to shock and mitigate the effects of shock by improving oxygen delivery and tissue perfusion.

Aeromedical Transport and Roles of Care

One of the most important advances in combat casualty care in these recent conflicts is prompt aeromedical evacuation to higher roles of care (Figure 1-3). In a combat theater, the severity of injuries and proximity to medical care determine which role of care the CC will be taken to. Combat casualties sustaining major injuries will undergo casualty evacuation (CASEVAC)

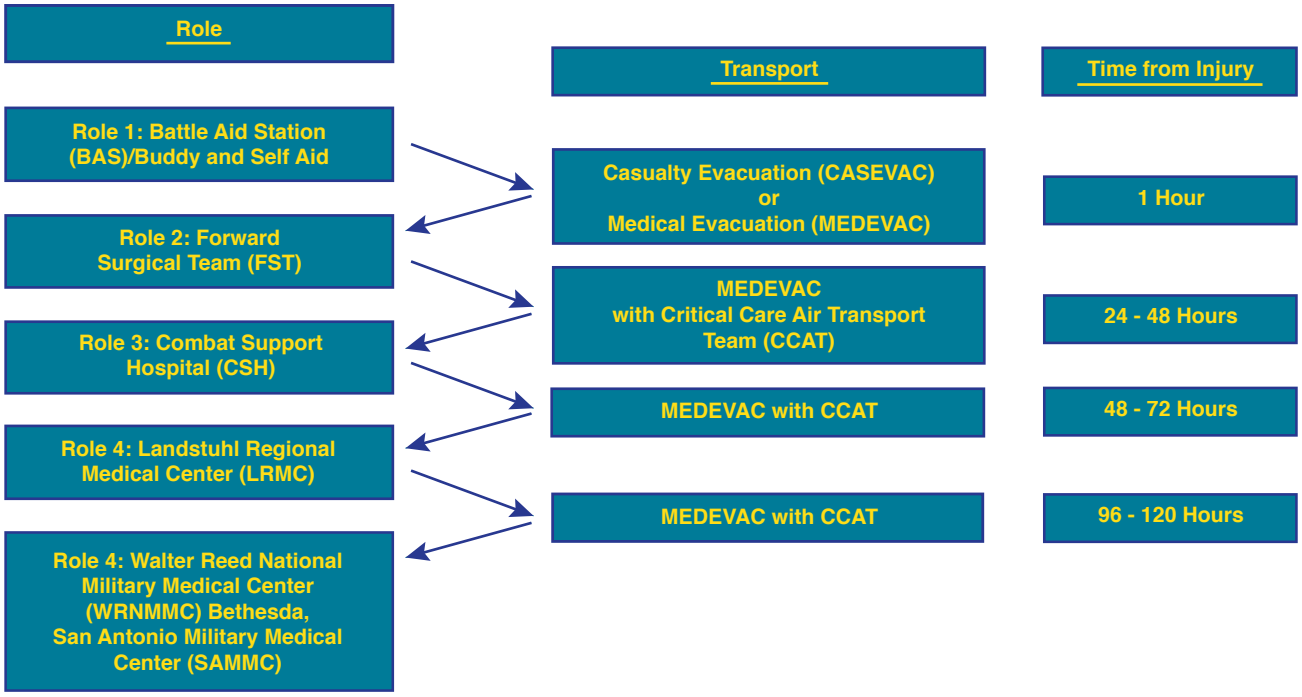

Figure 1-3. Roles of combat casualty care, transport, and time from injury.

from the point of injury or battle aid station (BAS) to a forward surgical team (FST) within one hour. Combat casualties who suffer dismounted IED blasts likely require emergent surgical management,[8] and hence should be transported to a Role 2 (FST) or Role 3 (combat support hospital [CSH]) facility in theater, if practicable.

Although time is one of the most critical components in the initial transport of the CC to advanced medical care to prevent or minimize the duration and severity of shock, several factors must be taken into account for intratheater transfers between Role 2 and Role 3 facilities.[47] Prior to transfer, the CC should be well resuscitated with a heart rate less than 120 beats per minute and systolic blood pressure (SBP) greater than 90 mm Hg. To prevent coagulopathy, the CC's temperature should be over 35° C.[20–22] Furthermore, laboratory values should show that the CC is stable for transport, with hematocrit greater than 24%, platelet count over 50/mm^3, international normalized ratio (INR) less than 2.0, pH greater than 7.3, and base deficit less than 5 mEq/L.

The CC should be prepared to receive appropriate medical care in flight with pain medications, two large-bore peripheral intravenous (IV) lines, and proper warming measures. If the CC has altered mental status (eg, Glasgow coma scale [GCS] <9), then he or she should be intubated and mechanically ventilated, with a nasogastric or orogastric tube placed to prevent aspiration during transport. All CCs on ventilator support should have a recent arterial blood gas demonstrating adequate oxygenation and ventilation. If the CC is sedated, a presedation neurologic exam should be documented and sent with the CC. Air should be removed from IV lines, IV fluid bags should be placed on pressure bags, and all maintenance fluids should be increased 25% to 50% to account for evaporative losses in flight.

CCs at risk for extremity compartment syndrome[48–51] (Exhibit 1-1) must have frequent and diligent evaluations in flight with serial physical exams and possible intracompartment pressure monitoring. Prophylactic fasciotomy should be performed in those CCs at risk if appropriate monitoring is not available, given the significant morbidity and mortality associated with delayed diagnosis of compartment syndrome.[52]

A critical care air transport (CCAT) team—consisting of an intensivist physician, critical care or emergency room nurse, and cardiopulmonary technician—will care for the casualty in flight. Particular attention must be paid to physiologic variations caused by changes in atmospheric pressure and altitude. Cabin altitude restrictions should be in place for intraocular air, pneumothorax, severe pulmonary disease, arte-

rial air embolism, and/or pneumocephalus. External air-containing devices such as air splints, will require adjustment in flight. CCs who require supplemental oxygen may require increased oxygen in-flight, given the decrease in the partial pressure of oxygen with altitude.[47]

MANAGEMENT OF COMBAT CASUALTIES AT ROLE 2 AND 3 FACILITIES

CCs who survive the initial injury have an over 96% survival rate after arrival at a Role 2 or 3 facility.[53] Prompt triage will be performed at these facilities, including assessment for airway, breathing, ongoing hemorrhage, pulse characteristics, and mental status.[54] If the CC is not an emergent surgical candidate, he or she may undergo further evaluation with advanced imaging.

Combat casualties who have sustained a dismounted IED blast will routinely present with an injury severity score (ISS) greater than 16,[54–66] and will generally have some degree of hemorrhagic shock with cardiovascular collapse (heart rate >105 beats per minute, and SBP <110 mm Hg),[54,67] hypothermia (temperature < 36° C),[54,68] acidosis (pH < 7.25),[69] and coagulopathy (INR > 1.5). [69,70] These CCs have emergent surgical needs, and should be taken immediately to the operating room for resuscitation and tactical damage control surgery to interrupt the lethal triad.[71]

EXHIBIT 1-1

RISK FACTORS FOR EXTREMITY COMPARTMENT SYNDROME

- Lower extremity fracture
- Open fracture
- Elbow or knee dislocation
- Gunshot wound
- Vascular injury
- High limb abbreviated injury score
- Injury severity score higher than 16
- Shock
- Tourniquet
- Packed red blood cells transfusion
- Resuscitation with more than 5 L of crystalloid

Adapted from: US Army Institute of Surgical Research website. Joint Theater Trauma System Clinical Practice Guideline: Compartment Syndrome (CS) and the Role of Fasciotomy in Extermity War Wounds. March 9, 2012. http://www.usaisr.amedd.army.mil/assets/cpgs/Compartment_Syndrome_and_Fasciotomy_9_Mar_12.pdf. Accessed October 10, 2012.

Damage Control Resuscitation

Damage control resuscitation in these emergent settings requires a delicately balanced and goal-directed infusion of both crystalloid fluids and blood products.[72,73] Aggressive fluid resuscitation without blood products can exacerbate coagulopathy, while blood-product only resuscitation will fail to replace interstitial fluid lost during the initial phase of hemorrhage.[74] The optimal ratio of blood products and the addition of hemostatic agents are critical in the resuscitation (as discussed further in the hematologic section below). Insufficient resuscitation can be complicated by renal failure and persistent tissue hypoperfusion, exacerbating oxygen debt. Overly aggressive resuscitation, on the other hand, can be complicated by iatrogenic compartment syndrome,[75,76] ARDS,[77–83] intracranial hypertension,[84] and infectious complications.[81]

Initially, resuscitation for CCs in hemorrhagic shock should be guided by the rate of hemorrhage and signs of shock (weak pulse, tachycardia, hypotension, slow capillary refill, and anxious or confused mental status).[85,86] Communication with medics involved in the TCCC is critical to determine if the CC responded to initial fluid boluses. CCs who do not respond to initial resuscitation are more likely have emergent need for blood products and surgical intervention. Once bleeding is controlled, the endpoints for resuscitation following combat trauma should be targeted towards resolving oxygen debt as measured by lactate[31,87–89] and base deficit.[31,89–95]

Tactical Damage Control Surgery

The goal of tactical damage control surgery is to obtain hemostasis and remove contamination while trying to minimize operating time in order to interrupt the lethal triad in accordance with the "golden hour" concept in CCs with emergent surgical needs per resources available.

Traumatic Brain Injury

Operative intervention with decompressive craniectomy,[60,96–98] placement of ventriculostomy, or intracranial pressure (ICP) monitor may be needed for CCs with closed or penetrating TBI and altered mental status, particularly with a GCS less than 9.[99–105]

Thoracic Trauma

CCs penetrating thoracic injury may require a pulmonary resection or pulmonary tractotomy with selective ligation of vessels,[106,107] ligation of chest wall vessels, repair of great vessels, and/or repair of heart trauma with pledgeted sutures,[108] with clamping of the superior vena cava and inferior vena cava (IVC) for larger heart wounds.[109] CCs with penetrating thoracic injuries who present in extremis or with recent loss of vital signs to a Role 2 or 3 facility may require a resuscitative thoracotomy for release of pericardial tamponade, hemorrhage control, aortic cross clamp, control of massive air embolism or bronchopleural fistula, or open cardiac massage (Figure 1-4).[110] CCs with similar presentation and blunt thoracic trauma have extremely low survival rates, and the selective use of resuscitative thoracotomy should be based on clinical judgment.[111–114]

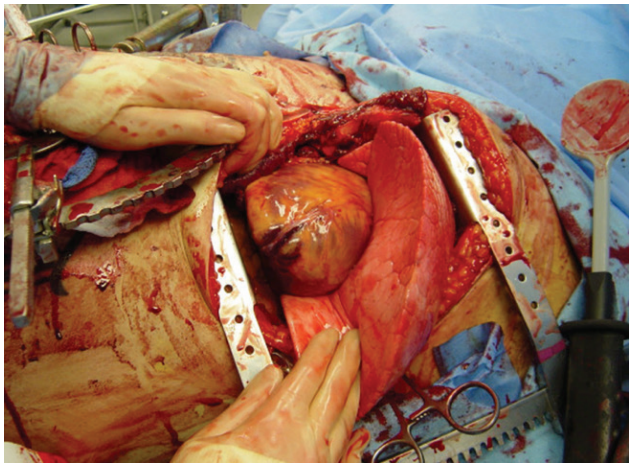

Figure 1-4. Emergency department thoracotomy can be performed for the following reasons: (1) release of pericardial tamponade, (2) hemorrhage control, (3) aortic cross clamp, (4) control or massive air embolism or bronchopleural fistula, or (5) open cardiac massage.

Abdominal Trauma

CCs with intraabdominal injuries may require a damage control celiotomy, which includes resection of injured bowel with or without formal diversion, vascular clamps, temporary intravascular shunts, packing of diffusely bleeding surfaces (eg, liver), and temporary abdominal closure.[115—119]

Extremity Injury and Wounds

Damage control orthopedics strategies focus on the reduction of fractures and splinting or placement of provisional external fixation or pelvic resuscitation frames,[120,121] with definitive open reduction and internal fixation, if indicated, delayed until wounds are stable and greater resources are available at higher echelons of care.

Damage control strategies for wounds include irrigation,[122] removal of exposed foreign bodies, aggressive and rapid debridement of nonviable tissue, fasciotomies for ischemia time greater than 6 hours[123] or overt or probable impending extremity compartment syndrome, and ligation or temporary shunting of vascular injuries.[124,125] The vast majority of combat wounds are not closed primarily at this operation, but rather packed with gauze or sealed with negative pressure wound therapy.[122,126–128] Treatment of these wounds with antibiotic beads may be indicated for open fractures and traumatic amputations.[129–133]

CARDIOVASCULAR INJURY

Several potential injuries resulting from dismounted IED blast can immediately compromise the cardiovascular system from hemorrhagic, posttraumatic, neurogenic, obstructive, and cardiogenic shock.

Hemorrhagic and Traumatic Shock

Hemorrhage remains the most common cause of death in CCs.[134] As the intravascular volume drops with hemorrhage, the cardiac pre-load decreases, decreasing cardiac output and subsequently decreasing blood pressure, which undermines tissue perfusion.[114] In the early postinjury phase, known as the ebb phase, the body attempts to preserve critical organ perfusion and reduce the metabolic rate by inducing peripheral vasoconstriction (Figure 1-5) and decreasing body temperature, respectively.[24,71,135]

These compensatory mechanisms are initiated after blast injury via afferent pain nerve signals from the wound, which then trigger the sympathetic efferent nerves and stimulate the hypothalamus-pituitary-adrenal axis.[14,136] The increase in sympathetic tone directly induces tachycardia and arteriolar vasoconstriction, as well as triggering the release of catecholamines from the adrenal glands. The stimulation of the hypothalamic-pituitary-adrenal axis activates production of cortisol from the adrenal glands, which then sensitizes the vasculature to the peripheral effects of sympathetic tone, further increasing peripheral vasoconstriction.[137]

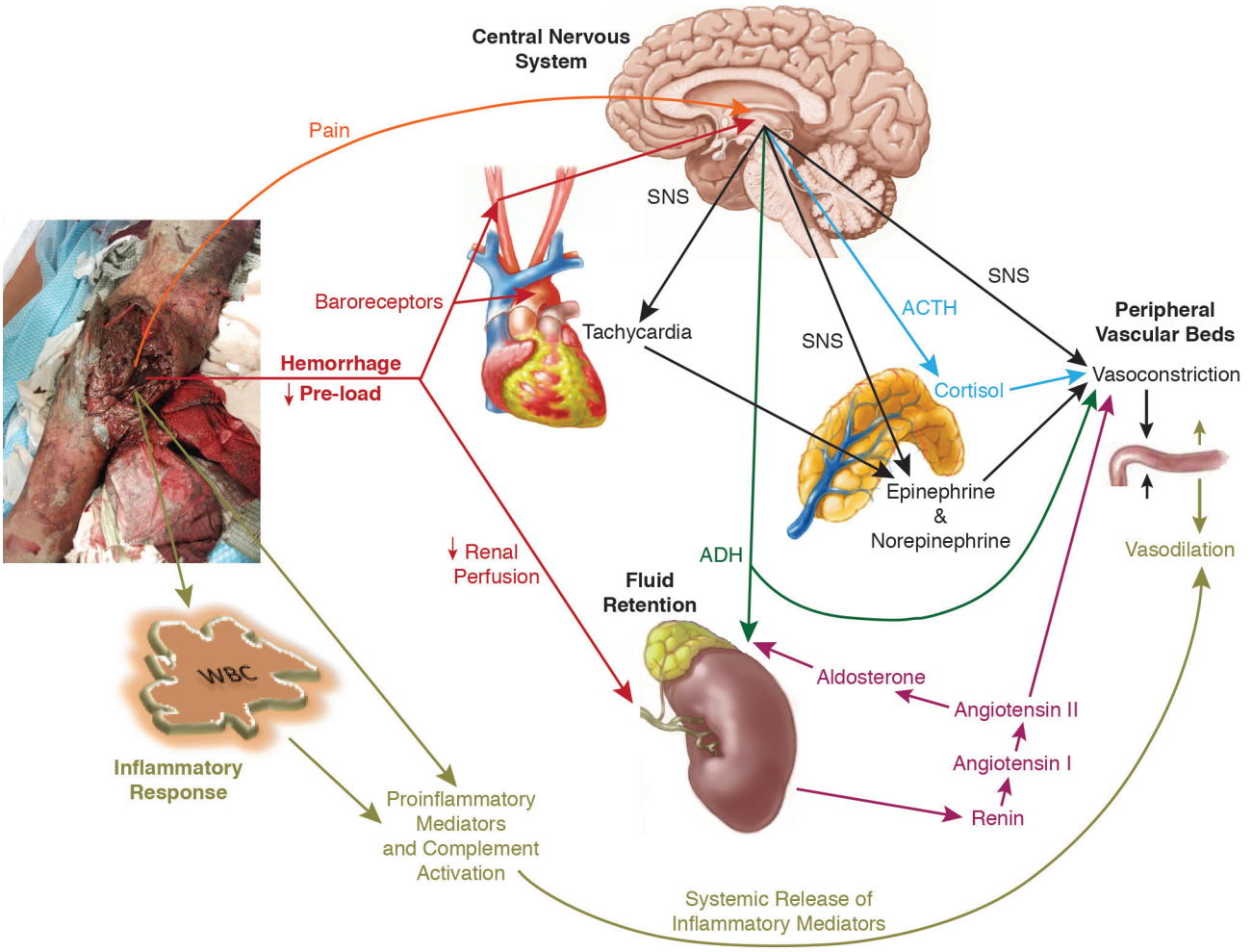

Figure 1-5. Effect of neurohormonal and inflammatory response to trauma on cardiovascular system.
ACTH: adrenocorticotropic hormone; ADH: antidiuretic hormone; SNS: sympathetic nervous system; WBC: white blood cell
Images adapted with permission from Vesalius: http://www.vesalius.com/welcome.asp. Accessed October 16, 2012.

Drops in blood pressure associated with hemorrhage are sensed by baroreceptors in the aorta and carotid bodies, which stimulates production of vasopressin (antidiuretic hormone) in the pituitary gland.[14,138] Vasopressin, a powerful vasoconstrictor, also preserves the intravascular volume via its antidiuretic effect. Decreased renal perfusion activates the renin-angiotensin-aldosterone system, producing angiotensin II and aldosterone, which promote vasoconstriction and fluid retention, respectively.[14]

This collective surge in vasoconstriction works primarily at the level of the arterioles, and acts to shunt blood away from peripheral tissues and toward the heart and brain, where blood flow is autoregulated.

In addition to the physiologic challenges presented by decreased intravascular volume in hemorrhage, the posttraumatic inflammatory response from injury can overcome the compensatory mechanisms to preserve perfusion and lead to profound hypotension in these CCs.[139] Prolonged time to surgical intervention and resuscitation, polytrauma, the use of tourniquets, and global hypoperfusion from hemorrhagic shock can lead to ischemia-reperfusion injury (IRI) of both traumatized and nontraumatized tissues and the subsequent systemic release of inflammatory mediators from these tissues once resuscitation is initiated and tourniquets are released (see Figure 1-5).[35,36,140–143] This response can overcome vasoconstriction and increase capillary permeability, leading to further loss of intravascular volume, irreversible shock, and death.[26,27] Hence, adherence to the "golden hour" concept with prompt initiation of damage control resuscitation and tactical damage control surgery is crucial for survival of severely injured CCs.

Neurogenic Shock

Neurogenic shock is associated with traumatic injury to the cervical or high thoracic spine in which loss of sympathetic impulses causes distributive shock and, more rarely, decreased cardiac output with bradycardia.[143] These sequelae of neurogenic shock should be managed initially with aggressive fluid resuscitation for a goal mean arterial pressure (MAP) greater than 85 mm Hg,[144] but the patient may ultimately require vasopressor support. Invasive blood pressure monitoring is required for appropriate management, and hence the CC with signs of neurogenic shock would optimally be treated at a Role 3 facility.

Obstructive Shock

Another immediate cause of cardiovascular compromise in the CC is obstructive shock from tension pneumothorax or cardiac tamponade. If obstructive shock is suspected, evaluation for pneumothorax should be performed on presentation of the CC to the trauma bay, given that tension pneumothorax can compromise right heart filling and cause fulminant cardiovascular collapse. Placement of a needle decompression catheter or thoracostomy tube may be performed as part of the TCCC,[3,145] or immediately upon diagnosis in the trauma bay. Cardiac tamponade can also cause obstructive shock and should be evaluated by assessing for muffled heart sounds, distended neck veins, and unexplained hypotension. Pericardial fluid may be evident on Focused Assessment with Sonography for Trauma (FAST) exam. If pericardial fluid is identified, immediate thoracotomy and repair of cardiac or great vessel injury should be pursued.

Cardiogenic Shock

Cardiogenic shock is rare after blunt chest trauma,[146] but can be associated with deadly arrhythmias and myocardial failure. If pump failure is suspected, further evaluation with electrocardiography, cardiac enzymes, pulmonary artery catheter, and transthoracic or transesophageal echocardiography is indicated[147] at a Role 3 facility. Support of myocardial contractility should include resolving hypothermia[148,149] and acidosis,[150,151] as well as ionotropic support. Mechanical assistance with an intraaortic balloon pump for medically refractory cases of traumatic cardiogenic shock may be considered if the procedure is available at a Role 4 facility (Landstuhl Regional Medical Center, Walter Reed National Military Medical Center, or San Antonio Military Medical Center),[152] but it is generally unavailable in the combat theater.

PULMONARY INJURY

The injuries associated with IED blasts present several immediate challenges to the respiratory system, including apnea from neurologic compromise, respiratory obstruction from laryngeal and/or oral maxillofacial trauma, ineffective ventilation from chest wall trauma, pulmonary contusion, hemothorax, pneumothorax, and/or blast lung. Overwhelming systemic inflammatory responses associated with the trauma, hemorrhage, and IRI in these injuries have been well associated with the development of ARDS, which can further compromise pulmonary function.

Airway

The first step in the management of these injuries is to secure the airway; even in cases of severe oral and maxillofacial trauma, an endotracheal intubation should be attempted (Figure 1-6). If an endotracheal intubation fails, then a cricothyrotomy[153,154] should be performed. A CC with laryngeal injury, particularly due to blunt trauma, will likely require an emergent airway with cricothyrotomy or tracheostomy.[155]

Chest Wall and Pulmonary Trauma

Once the airway is secure, ventilation can be assessed, and further evaluation for hemothorax, pneumothorax, and chest wall trauma can be performed. If found, these conditions should be expeditiously treated to avoid the sequelae of obstructive shock. CCs may develop pneumothorax or hemothorax from chest wall or lung injury. The degree of respiratory compromise from fluid or air in the pleural space depends on the CC's baseline pulmonary function and physiological reserve. Most cases of hemothorax and pneumothorax can be managed effectively with tube thoracostomy; however, placement of a second tube thoracostomy or operative intervention may be required if the hemothorax cannot be completely drained or if intrathoracic hemorrhage continues after decompression. Specifically, operative intervention for chest trauma patients with thoracostomy tubes may also be required if any of the following occur:

- more than 1,500 mL of blood is found on placement of chest tube,

Figure 1-6. Endotracheal intubation in setting of severe oral and maxillofacial trauma.

- the chest tube drains more than 200 mL per hour over 4 hours,
- a large air leak suggestive of bronchoplueral fistula is encountered, or
- the patient remains hemodynamically unstable following primary survey and resuscitation without another identifiable and correctable cause.[86]

Chest wall trauma can cause significant respiratory compromise by inhibited generation of negative pressure in a chest wall defect ("sucking wound"); by paradoxical movement of the chest wall associated with multiple rib fractures in flail chest[156]; or, most commonly, by the associated underlying pulmonary contusions that impede alveolar filling and gas exchange. In addition, flail chest and pulmonary contusion lead to atelectasis, mucus plugging, and decreased functional residual capacity. Aggressive lung recruitment strategies—physiotherapy and appropriate ventilator strategies—are required to improve respiratory status. Pressure support ventilation is not recommended in these CCs because generation of negative pressure can destabilize the chest wall.[156] Volume control ventilation with synchronized intermittent mandatory ventilation or inverse ratio ventilation with airway pressure release ventilation are preferred to help maintain positive pressure ventilation. (These ventilators are available at Role 3 facilities).

Blast Lung

The CC is also at immediate threat from blast lung as part of the primary blast injury. As the pressure wave from the blast travels through tissue, it causes massive damage to the air-filled organs, particularly at the air–fluid interface.[135] The shearing stress of the blast wave causes alveolar hemorrhage, which damages the endothelium and epithelium and ultimately leads to ARDS.[157,158] CCs will typically present with hemoptysis, tachypnea, cough, and shortness of breath.[159] CCs with blast lung may also have blood seen in the airway on intubation or in the endotracheal tube during ventilation (Figure 1-7). The clinical effects on the lung and the subsequent management are similar to those for pulmonary contusion.

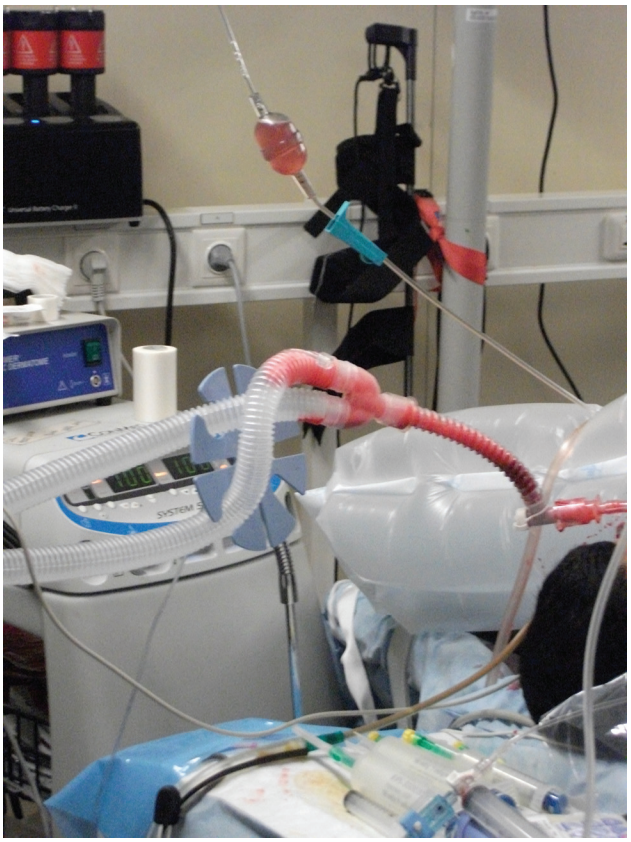

Figure 1-7. Blood in endotracheal tube of combat casualty with blast lung injury.

Acute Respiratory Distress Syndrome

The massive inflammatory response associated with polytrauma as well as direct lung injury can trigger a diffuse pulmonary inflammatory response resulting in alveolar injury and impediment of alveolar gas exchange, which can cause ARDS. Typically, ARDS presents within 12 to 48 hours of the inciting event and is particularly common in CCs with pulmonary contusion.[160] The diagnosis of ARDS can be made with evidence of bilateral patchy infiltrates on chest x-ray (Figure 1-8) and PaO_2:FIO_2 ratio less than 200. Risk factors for ARDS in CCs involved in blasts include high ISS,[161] blood transfusion,[162] and direct pulmonary injury (contusion, aspiration).[163] Protective ventilator settings for these CCs should be maintained at a tidal volume between 6 and 8 mL/kg and FIO_2 less than 60%.[164] To reduce FIO_2, peak end-expiratory pressure (PEEP) can be increased while keeping peak pressure at less than 35 cm to 40 cm H_2O to prevent lung injury.[165] It may be difficult to obtain eucapnia (PCO_2 40–45 mm Hg)[14] with these settings; permissive hypercapnia (PCO_2 > 46 mm Hg)[166] may be required and is generally well-tolerated once the patient's metabolic acidosis is corrected.[167]

NEUROLOGIC INJURY

CCs who experience IED blasts are at risk for TBI from penetrating fragments of secondary blast injury and blunt forces associated with tertiary blast injury. Moderate (GCS 9–13) or severe (GCS 3–8) TBI presents significant management challenges that require head computed tomography (CT) scan and interventions by neurosurgery subspecialty care; hence, the CC must be taken to a Role 3 facility as rapidly as feasible.

Intracranial Pressure and Cerebral Perfusion Pressure

TBI can cause increased ICP from hematomas, edema from inflammation at the site of injury, and cytotoxic edema. Increased ICP can lead to decreased cerebral perfusion pressure (CPP), herniation, and, ultimately, death or brain death. Although little can be done about primary TBIs that occur in IED blasts, appropriate management of these CCs in accordance with clinical practice guidelines can help prevent secondary injuries associated with hypotension (SBP < 90 mm Hg), decreased CPP (< 60 mm Hg), elevated intracranial pressure (ICP > 20 mm Hg), hypoxia (PaO_2 < 60 mm Hg or SaO_2 < 93%), hypothermia or hyperthermia, and hypoglycemia or hyperglycemia.[168–176] Hypotension is the most significant of these factors, and even a single episode negatively impacts cognitive and functional outcomes.[172] Hence, permissive hypotension strategies in damage control resuscitation must be balanced with preventing secondary injury in CCs with TBI.

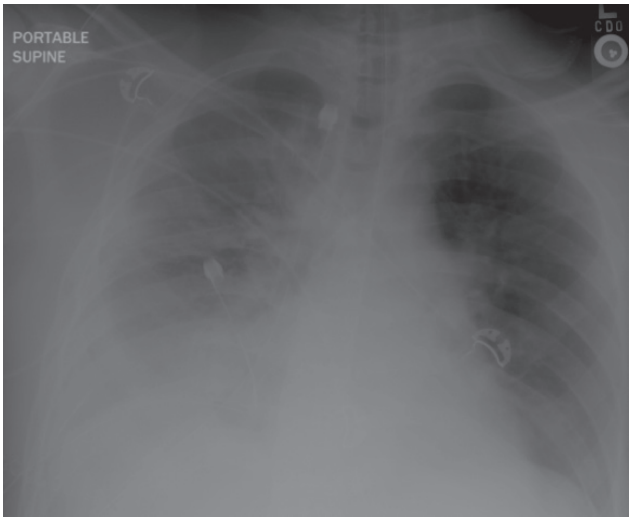

Figure 1-8. Patient with acute respiratory distress syndrome with bilateral patchy infiltrates on chest x-ray.

Prompt initiation of medical management with intubation, appropriate oxygenation (goal PaO_2 > 60 mm Hg), ventilation (goal $PaCO_2$ between 35 and 40 mm Hg), elevation of head of bed to 15 to 30 degrees, and resuscitation to appropriate blood pressure will help preserve autoregulated cerebral blood flow and maintain oxygen delivery.[177]

To ensure adequate CPP, current military clinical practice guidelines indicate evaluations with head CT scans on admission and at 24 hours postinjury, as well as ICP monitoring in CCs with severe TBI and an abnormal CT scan.[177] ICP monitoring may also be considered for CCs with severe TBI but normal CT scans if two or more of the following are present: age greater than 40, unilateral or bilateral motor posturing (decorticate or decerebrate), and hypotension. ICP monitoring requires at least a Role 3 facility. Unique to military populations, ICP monitoring should be considered for CCs with TBIs who will undergo aeromedical evacuation and cannot awaken for hourly neurological exams, as well as CCs with polytrauma and severe burns who are at high risk for cerebral edema.[178]

To further optimize CPP and oxygen delivery, decrease vasospasm, and prevent intracranial bleeding, goal-directed therapies should include the targets defined in Table 1-1.[178–180] CCs with TBI who have re-

TABLE 1-1

NEUROPROTECTIVE MEASURES IN THE MANAGEMENT OF SEVERE TRAUMATIC BRAIN INJURY

	Goal	Management
Neurologic	ICP < 20 mm Hg	Sedation (short-acting agent, ie, propofol) $Paco_2 \geq 35$ mm Hg Hypertonic saline (3%) to goal Na 138–165 Paralysis (first line agent: vecuronium) CSF drainage with ventriculostomy
	CPP > 60 mm Hg	Maintain euvolemia (per CVP or pulmonary artery catheter) Vasoactive medications (first line agent: vasopressin)
Hemodynamic	SBP > 90 mm Hg CVP > 5 mm Hg	Damage control resuscitation, isotonic or hypertonic saline, vasoactive medications
Pulmonary	Spo_2 > 93%	Aggressive oxygenation strategies on ventilator
	co_2 35–40 mm Hg in first 24 h, and 30-35 mm Hg the next 7 days	Avoid routine hyperventilation
Hematologic	INR < 1.3	Administer fresh frozen plasma
	Platelets > 100,000/mm^3	Administer platelets
	Hemoglobin >10 g/dL	Administer packed red blood cells
	Normalized TEG values	Administer appropriate blood product per result
Metabolic	80 > serum glucose < 150 mg/dL	Administer glucose and insulin as needed, consider insulin drip
Renal	280 > serum osmolarity < 320 mOsm 135 > serum sodium < 165 mEq/L	Hypertonic saline bolus and infusion, evaluate for sodium disorders (SIADH, cerebral salt wasting, mannitol use, diabetes insipidus)

CPP: cerebral perfusion pressure; CSF: cerebrospinal fluid; CVP: central venous pressure; Hb: hemoglobin; ICP: intracranial pressure; INR: international normalized ratio; SBP: systolic blood pressure; SIADH: syndrome of inappropriate secretion of antidiuretic hormone; TEG: thromboelastography

Adapted from: US Army Institute of Surgical Research website. Joint Theater Trauma System Clinical Practice Guideline: Management of Patients with Severe Head Trauma. March 6, 2012. http://www.usaisr.amedd.army.mil/assets/cpgs/Mgmt_of_Patients_with_%20Severe_Head_Trauma_7_Mar_12.pdf . Accessed October 10, 2012.

fractory elevated ICP (> 20 mm Hg) despite optimal therapy, high velocity transcranial gunshot wounds, or space-occupying lesions may benefit from formal decompressive craniectomy.[60,96,98,181]

Prophylaxis for Severe Traumatic Brain Injury

All CCs with severe TBI should be considered at increased risk for venous thromboemoblism (VTE),[182] gastric ulcers, and epilepsy; prophylaxis should be initiated as soon as practicable.[178] Thromboprophylaxis should include placement of graduated sequential compression devices and chemical VTE prophylaxis. Chemical VTE prophylaxis in these CCs should be coordinated with consultation of a neurosurgeon and considered on postoperative day 1, unless there are risk factors for hemorrhagic complications (eg, increased blood on CT scan) or prohibitive contraindication for anticoagulation (eg, high-grade liver injury with ongoing coagulopathy).[182,183] CCs with TBI who cannot undergo chemical VTE prophylaxis should be considered for IVC filter placement.[184]

RENAL INJURY

Renal failure remains a significant problem in severe burn and polytrauma CCs. Risk factors for acute renal failure include an ISS greater than 17, hemoperitoneum, shock, long-bone or pelvic fractures, acute lung injury, GCS less than 10, a need for mechanical ventilation with PEEP greater than 6, and rhabdomyolysis with creatinine phosophokinase more than 10,000 IU.[185]

Acute Kidney Injury

When acute renal failure does occur in CCs involved in IED blasts, it most frequently occurs secondary to ischemic insult from global hypoperfusion associated with hemorrhagic shock. While the renal cortex is well perfused to optimize glomerular filtration rate, the renal medulla has a low blood flow at baseline in order to maintain osmotic gradients. During hemorrhagic shock, decreased renal medullary perfusion leads to ischemic injury of tubular cells, subsequent acute tubular necrosis, and acute kidney injury.[186] Other causes of acute kidney injury in CCs include rhabdomyolysis from crush injuries or extremity compartment syndrome as well as decreased renal perfusion in abdominal compartment syndrome. As renal function declines, the critical functions of regulating fluid and electrolyte balance, clearing nitrogen waste products, and correcting metabolic acidosis are lost. The degree of acute renal failure can be graded by increases in serum creatinine and decreases in urine output per the "RIFLE" criteria (Figure 1-9).[187]

Fluid overload from renal failure can pose a threat to the respiratory system by impeding alveolar gas exchange, causing mucosal edema, and exacerbating the restrictive effects of edema on the work of breathing.[188] Furthermore, acidosis can also increase the work of breathing by intensifying the respiratory drive. The combination of these two physiologic alterations can lead to rapid ventilator failure in the polytrauma CC with renal compromise.

Acidosis from renal failure and shock can also result in globally decreased enzyme function and cardiac output when pH is less than 7.2.[150] This acidosis can be difficult to correct with IV bicarbonate and hyperventilation if the patient also has impeded CO_2 clearance with ARDS.[16,150,189] Accumulation of nitrogen waste products in renal failure can cause uremia, which can lead to altered mental status, platelet dysfunction, and pericarditis.

Compartment Syndrome

The first critical step in treating renal failure complications in CCs involved in dismounted IED blasts is preventing further renal injury with appropriate resuscitation, vigilant monitoring, expeditious management of abdominal compartment syndrome (ACS),[190,191] and prevention of rhabdomyolysis from extremity compartment syndrome.[123,192,193]

Abdominal Compartment Syndrome

The risk factors for abdominal compartment syndrome[190,191] in CCs include:

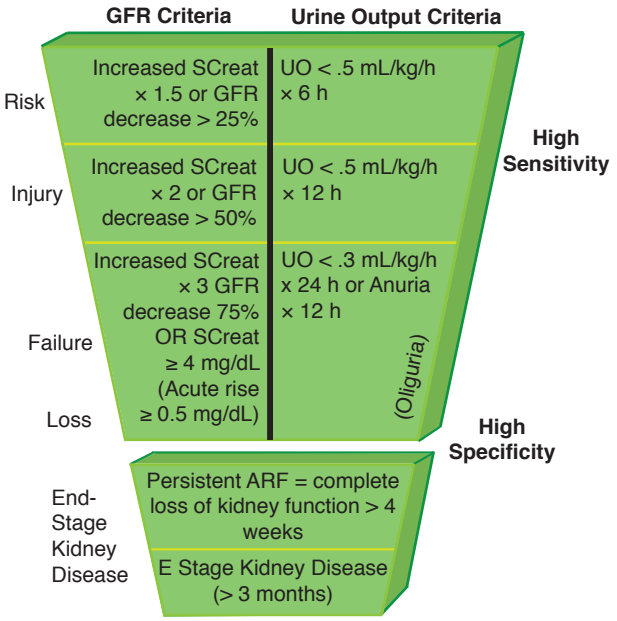

Figure 1-9. "RIFLE" criteria for acute renal failure.
ARF: acute renal failure
E: end
GFR: glomerular filtration rate
SCreat: serum creatinine
UO: urine output
Adapted with permission from: Bellomo R, Ronco C, Kellum JA, Mehta RL, Palevsky P; Acute Dialysis Quality Initiative Workgroup. Acute renal failure—definition, outcome measures, animal models, fluid therapy and information technology needs: the Second International Consensus Conference of the Acute Dialysis Quality Initiative (ADQI) Group. *Crit Care*. 2004;8(4):R206.
Original publisher: BioMed Central.

- Diminished abdominal wall compliance: acute respiratory failure (especially with elevated intrathoracic pressure); abdominal surgery with primary fascial or tight closure, major trauma / burns, prone positioning, head of bed > 30 degrees, high body mass index), AND/OR central obesity.
- Increased abdominal contents: hemoperitoneum, pneumoperitoneum, AND/OR ascites.
- Capillary leak / fluid resuscitation: acidosis (pH < 7.2), hypotension, hypothermia (core temperature < 33°C), polytransfusion (>10 units of blood / 24 h), coagulopathy (platelets < 55,000 / mm^3 OR prothrombin time > 15 seconds OR partial thromboplastin time > 2 times normal OR international standardized ratio > 1.5), massive fluid resuscitation (> 5 L / 24 h), oliguria, sepsis, AND/OR major trauma / burns.

CCs with at least two risk factors for ACS should undergo serial evaluations of intraabdominal pressure (IAP) with bladder pressure and abdominal perfusion pressure (APP). Sustained IAP greater than 12 mm HG can impede venous return, leading to decreased blood pressure and decreased APP (APP = MAP - IAP), and ultimately to end-organ damage. Appropriate interventions to improve abdominal wall compliance, decompress the gastrointestinal tract, evacuate intraabdominal space-occupying lesions, and optimize systemic and regional perfusion should be considered to reduce IAP and increase APP to over 60 mm Hg; however, if the IAP remains elevated (> 25 mm Hg) and there is evidence of new organ failure, a decompressive laparotomy should be considered.

Extremity Compartment Syndrome

CCs at risk for extremity compartment syndrome (see Exhibit 1-1)[48–51] who do not undergo early fasciotomies should have serial clinical evaluation. Physicians monitoring these CCs should remember that delayed presentation of compartment syndrome is possible, with tissue edema maximizing at 1 to 2 days postinjury.[49,194,195] Clinical criteria for extremity compartment syndrome includes paralysis, paresthesias or sensory deficit, palpably tense muscle compartments, pulselessness, pain on passive movement of distal appendage, or pain out of proportion to injury. The clinical exam can be supplemented with the use of the Stryker Intra-Compartmental Pressure Monitor System (Stryker Instruments, Kalamazoo, MI) or arterial slit-cath pressure monitor.[197,196–198] Elevation in extremity compartmental pressure impedes tissue perfusion, particularly when the compartment pressure rises to within 10 to 30 mm Hg of diastolic blood pressure,[196] which ultimately leads to compartment tissue destruction and systemic release of cell contents, including myoglobin. The released myoglobin can then obstruct renal tubules and induce renal vasoconstriction, leading to acute kidney injury as part of rhabdomyolysis.[199]

Management of rhabdomyolysis with mannitol to promote washout of tubules and scavenge free radicals is effective in ameliorating myglobinuria-induced renal failure[200,201]; however, it is not recommended in the under-resuscitated polytrauma CC due to its diuretic effects.[178] Similarly, bicarbonate therapy can be used to alkalinize the urine[202] to promote tubule clearance, but it should not be used in acidotic and hypercapneic CCs because it will worsen the acidosis.

Renal Replacement Therapy

Polytrauma CCs with renal failure will require renal replacement therapy if they have uncompensated and persistent metabolic acidosis with pH less than 7.2, significant electrolyte abnormalities such as hyperkalemia with electrocardiomyography changes, fluid overload with respiratory compromise, or uremia with pericarditis, mental status changes, or bleeding complications.

HEPATIC INJURY

The liver plays a key role in maintaining homeostasis in CCs with polytrauma and hemorrhagic shock, and it is at direct risk of injury from trauma as well as IRI from hemorrhage.

Liver Trauma

The liver is the most commonly injured organ in abdominal trauma. The degree of liver injury is graded by size and depth of laceration or hematoma, as well as the involvement of inflow and outflow vascular structures.[203,204] Evaluation for liver trauma begins by determining if the CC is hemodynamically unstable and a candidate for immediate damage control surgery. Prompt evaluation should include the FAST exam or diagnostic peritoneal lavage; demonstration of hemoperitoneum on either of these tests indicates that operative intervention is required in the hypotensive CC. The hemodynamically stable CC should undergo a contrast-enhanced CT scan to help determine the grade of the injury. CCs with active blush on CT scan demonstrating arterial bleeding may be managed with arterial embolization,[205,206] although this procedure is not widely available prior to reaching a Role 4 facility.

Most liver injuries are managed nonoperatively according to current guidelines. While the CC remains hemodynamically stable, even grade V injuries can be managed with observation, intensive care unit resuscitation, and follow-up imaging.[207–210] Complications of nonoperative management of liver trauma, particularly in grade V liver injury, are associated with coagulopathy and include bile leak, abscess, hemorrhage, devascularization, and hemobilia.[211]

If operative management of the liver is required, minor liver injuries respond well to packing, while larger liver injuries may require more involved intervention with ligation or repair of bleeding vessels, hepatic arterial ligation, hepatic resection, and extra-hepatic biliary repair.[212]

Liver Ischemia Reperfusion Injury and Shock Liver

CCs who are in shock are at risk for direct hypoxic injury to the liver[215,216] as well as IRI from hemorrhagic shock and splanchnic hypoperfusion,[215,216] which releases hepatotoxic cytokines to the liver.[216–224] This leads to shock liver and a resulting rise in liver enzymes, elevated bilirubin, coagulopathy, thrombocytopenia, lactic acidosis, and hypoglycemia.[225] In most cases, appropriate resuscitation will be accompanied by return of liver function; however, in the interim the clinician must account for coagulopathy and thrombocytopenia during blood product replacement as well as lactic acidosis and hypoglycemia during resuscitation.[213,225]

HEMATOLOGIC INJURY

CCs can rapidly exsanguinate from mangled extremities, thoracic great vessel injuries, large abdominal or pelvic vessel injury, or visceral pedicle injury. Prompt application of tourniquets, antishock garments, and transport to Role 2 or 3 facilities can be life-saving for these casualties.

Massive Transfusion and Damage Control Resuscitation

CCs with external bleeding who present hypotensive will require blood transfusion.[86] It is likely that these CCs will require massive transfusion (>10 U blood within 24 hours), and it is not uncommon for CCs with similar presentation and multiple mangled extremities to require over 30 units of blood. Coagulopathy in CCs requiring massive transfusion is a major concern that can exacerbate hemorrhage by causing microvascular, nonsurgical bleeding.[69] These CCs are at risk for dilutional coagulopathy,[19] consumptive coagulopathy,[19,226] and coagulopathy from acidosis[69,85,227] and hypothermia.[20–22,227] Rapid correction of hypothermia and damage control resuscitation are key for the management of CCs requiring massive transfusion.[228]

Damage control resuscitation with component transfusions of packed red blood cells (pRBCs), platelets, and fresh frozen plasma in a 1:1:1 ratio helps prevent dilutional coagulopathy and improves survival compared with older crystalloid and pRBC methods.[229] An example of this massive transfusion protocol is shown in Table 1-2.[230] Aged blood is known to develop storage lesions with decreased 2,3-diphosphoglycerate, decreased pH, increased potassium, and increased proinflammatory factors.[231,232] Rapid infusion of these products can exacerbate the lethal triad, as well as causing or exacerbating hyperkalemia or hypocalcemia. Hence, prior to massive transfusion, clinicians should

TABLE 1-2

EXAMPLE OF MASSIVE TRANSFUSION

Initial Transfusion Procedure	
Pack One	4 U pRBC 4 U FFP 1 U apheresis plts 1 10-U bag cryo Strongly consider the early use of TXA
Pack Two	4 U pRBC 4 U FFP
Pack Three	4 U pRBC 4 U FFP 1 U apheresis plts 1 10-Unit bag of cryo +/- rFVIIa
Pack Four	4 U pRBC 4 U FFP
Pack Five	4 U pRBC 4 U FFP 1 U apheresis plts 1 10-U bag of cryo
Reassessment	
Pack Six	4 U pRBC 4 U FFP
Pack Seven	4 U pRBC 4 U FFP 1 U apheresis plts 1 10-U bag of cryo
Pack Eight	4 U pRBC 4 U FFP
Pack Nine	4 U pRBC 4 U FFP 1 U apheresis plts 1 10-U bag of cryo

cryo: cryoprecipitate; FFP: fresh frozen plasma; plts: platelents; rFVIIa: recombinant activated factor VII; pRBC: packed red blood cells; TXA: tranexamic acid; U: unit

Adapted from: US Army Institute of Surgical Research website. Joint Theater Trauma System Clinical Practice Guideline: Damage Control Resuscitation at Level IIb/III Treatment Facilities. August 10, 2011. http://www.usaisr.amedd.army.mil/assets/cpgs/Damage%20Control%20Resuscitation%20-%201%20Feb%202013.pdf. Accessed October 10, 2012.

coordinate with the blood bank to assure that these critically ill CCs are receiving "last in, first out" blood.

Although this damage control resuscitation technique is FDA-approved when using screened blood products and is the primary method of resuscitation for CCs, the blood product components may not always be available in austere environments. In order to rapidly obtain all blood components, Role 2 and 3 facilities have developed walking blood banks set up for the collection of matched warm fresh whole blood (WFWB) for CC care. When necessary, WFWB can be ready in 20 minutes to provide a blood product with more clotting factors, less anticlotting agent, and more platelets than component transfusion. As such, WFWB has also been associated with improved survival.[233]

Evaluation of transfused WFWB from Role 3 facilities demonstrated low rates of hepatitis C and lymphotropic virus, and zero evidence of HIV or hepatitis B.[234] These risks are minimal compared to the over 33% mortality from hemorrhagic shock; furthermore, active duty service members (ie, donors) are vaccinated against hepatitis B and serially screened for HIV, further improving the safety of this practice with regard to disease transmission.

The initial rate of transfusion in CCs with ongoing hemorrhage should be based on clinical judgment, rate of blood loss, and refractory blood pressure. Once surgery is complete and the CC has returned to the intensive care unit, resuscitation strategies should target ongoing oxygen debt—using base deficit and arterial lactate levels—and maintaining hemoglobin levels of 6 to 7 g/dL[235,236] unless otherwise dictated by associated injuries or comorbidities (eg, severe TBI).

Hemostatic Agents and Anticoagulation

CCs receiving massive transfusion should be considered for recombinant activated factor VII (rVIIa) or antifibrinolytic therapy with transaminic acid. Both products have been demonstrated to improve survival in selected CCs receiving massive transfusion,[56,237,238] and no increased incidence of thromboembolic events has been seen with the use of rVIIa in trauma patients.[237] Once coagulopathy resolves and there is no longer risk for catastrophic bleeding, all CCs should be started on chemical VTE prophylaxis. CCs with ongoing risk of bleeding, other major contraindications to anticoagulation, or a documented early thromboembolic event should be considered for retrievable IVC filter placement.[184]

ANESTHESIA

The anesthesia provider plays a critical role in the management of CCs. Early control of airway, goal-directed resuscitation, and adequate sedation and analgesia are life-saving interventions after a CC suffers a dismounted IED blast. Furthermore, in civilian populations, early aggressive management of pain with multimodal therapy (including regional anesthesia) can lead to improved surgical outcomes,[239,240] and recent studies demonstrate that early and multimodal therapy in CCs leads to improved pain control and decreased anxiety.[241] Adequate analgesia after trauma may also reduce the rate of posttraumatic stress disorder in CCs.[242]

SUMMARY

The treatment of critically ill CCs with polytrauma requires the coordination of a comprehensive trauma team to reach management goals and optimize patient outcomes. Improving tissue oxygenation and perfusion remains a fixed target of CC care; however, critically ill CCs are in a hyperdynamic physiologic state, and appropriate management requires rapid adjustments for these physiologic alterations. An understanding of the physiology of injury as well as adherence to TCCC and clinical practice guidelines with astute clinical judgment is crucial for meeting management goals and improving survival.

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Chapter 2

PREPARING THE TEAM

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INTRODUCTION

As an introduction to this chapter on preparing the team and environment for a deployment as a military anesthesiologist, it is important to point out that there are several differences between United Kingdom Defence Medical Services (UK-DMS) anesthetists and US military anesthesiologists.

UK-DMS anesthetists predominantly work in the National Health Service (NHS) and deploy on operations once every 6 to 18 months depending on their role. Both regular and reserve personnel contribute to a consultant cadre of about 180. A number of trainee anesthetists also deploy and complete a higher military training module in accordance with the Royal College of Anaesthetists program.[1] Hospital units tend to change at 3 or 6 monthly intervals, and individuals are often "trickle posted" as individual replacements deploying for 8 to 12 weeks.

US military anesthesiologists include both active duty and reserve component personnel. Active duty anesthesiologists are full time uniformed officers working within the military health system at military medical treatment facilities around the world at both operational and fixed facilities. The average deployment cycle is every 5 years across the services. This infrequent deployment cycle is mostly due to the rapid turnover of personnel retiring to civilian practice after their initial service obligation has been completed. Reserve personnel have a similar deployment cycle; however, when not on duty they work within the civilian healthcare sector. The average turnover for US forces is 6 months to 1 year, although specialists within the US Army and US Air Force may be reposted more frequently. Despite these differences, the two groups share similarities of practice, which will be discussed in terms of predeployment training.

Military hospitals in Iraq and Afghanistan managed considerably more severe trauma than an average UK or US hospital.[2] As a consequence, the injury patterns seen with military trauma (mainly blast and ballistic injury) are very different than the blunt trauma that predominates in UK and US civilian trauma practice.[3–5] Deployed anesthesiologists are required to work with equipment they are not familiar with and therefore must train to become competent with the equipment prior to deploying. Additionally, the military has unique guidelines or standard operating procedures (SOPs). In the UK these are written as Clinical Guidelines for Operations,[6] and the US Army Institute for Surgical Research publishes the Joint Theater Trauma System Clinical Practice Guidelines.[7] In military trauma, the traditional resuscitation guidelines of airway, breathing, and circulation (ABC) are modified to <C>ABC,[8] where <C> indicates the control of catastrophic hemorrhage. Other differences include the early and rapid use of blood and blood products as part of damage control resuscitation.[9,10]

Predeployment training allows individuals the opportunity to become fully immersed in the operational environment and familiar with the new equipment. The busy operating room schedule in the deployed environment necessitates that individuals are comfortable with the unfamiliar equipment and environment.[11] For instance, in a conflict environment an alarm bell might be a signal to drop down flat on the floor ("on your belt buckle") because of an incoming mortar attack, whereas in a civilian setting it might signify a fire alarm or a patient's cardiac arrest. First-rate human factors or nontechnical skills are important in this environment, and effective clinicians use them as part of their working routine.[12] The clinical tempo in theater precludes the potential for any significant "just-in-time" training. The whole medical system must be prepared to work in the deployed environment and rapidly integrate individuals into the team when they arrive.

HUMAN FACTORS IN DEFENSE ANESTHESIA

In the 1970s simulation in healthcare began gaining recognition as a means to limit human error and improve patient safety. The advantages of simulation training had been clearly demonstrated in the aviation and nuclear power industries as well as the National Aeronautics and Space Administration (NASA). Previously, NASA had shown that 70% of its errors were due to human factors such as failed interpersonal communication, decision-making, and leadership.[13] Similar figures have been seen in an analysis of adverse events in anesthesia[14] and also in the landmark report *To Err Is Human,* which showed that between 44,000 and 98,000 people die in the United States every year from medical errors.[15] Multiple case reports and a report from the UK National Patient Safety Agency also suggest that human factors contribute to the majority of medical errors.[16–19]

Research on human behaviors has led to the development of a set of behavioral principles initially termed Crew Resource Management (CRM). CRM, also called human factors, is defined as "the cognitive, social, and personal resource skills that complement

technical skills, and contribute to safe and efficient task performance."[20] As adapted for anesthesia, these principles are listed in Exhibit 2-1.

Carthey et al[21] reported that highly performing surgeons demonstrated nontechnical skills as an integral part of their surgical expertise, and these attributes were thought to play an equally significant role as technical skills. Ineffective communication was found to be a causal factor in 43% of errors by surgeons in three US teaching hospitals.[12] Human factors are also very important in the critical care environment, where patients have life-threatening illness, diagnostic uncertainties, and the potential for rapidly changing medical conditions, and are managed along variable treatment pathways. Patient care is carried out over a 24-hour period involving multiple team transitions and moves to different areas of the hospital, which can result in lapses and discontinuities in communication.[22] In the UK, the Houses of Parliament Health Committee has recently acknowledged that a paucity of nontechnical skills can have lethal consequences for patients and that the NHS as a whole lags unacceptably behind other safety-critical industries, such as aviation, in this respect.[23] The following are key human factors that are essential to the effective working of the trauma team.

EXHIBIT 2-1

CREW RESOURCE MANAGEMENT KEY PRINCIPLES

- Know the environment.
- Anticipate and plan.
- Call for help early.
- Exercise leadership and followership.
- Distribute the workload.
- Mobilize all available resources.
- Communicate effectively.
- Use all available information.
- Prevent and manage fixation errors.
- Cross (double) check.
- Use cognitive aids.
- Reevaluate repeatedly.
- Use good teamwork.
- Allocate attention wisely.
- Set priorities dynamically.

Data source: Rall M, Gaba D. Human performance and patient safety. In Miller R, ed. *Miller´s Anesthesia.* Philadelphia, PA: Elsevier Churchill Livingstone; 2005: 3021–3072.

Communication

It is essential that the flow of information from the point of wounding to the trauma team in the field hospital is accurate. The initial military communication tool is the "9 liner"[24] evacuation request, which medics on the ground use to request the evacuation of a casualty. Once the casualty has arrived at the Role 3 field hospital, a standardized report is given by the evacuation team detailing the trauma incident. Unless an obvious problem must be immediately addressed (eg, airway compromise), it is important that all receiving team members remain silent and listen during this exchange to maintain their own personal situational awareness.

Other essential lines of communication for the trauma team include the following:

- The trauma surgeon needs to liaison with the team leader about the timing of procedures and movement to the operating room or computed tomography (CT) scanner.
- Radiology personnel are often present in the emergency department to provide immediate digital x-rays or ultrasound scans. They also require communication for CT scans if appropriate.
- Staff providing transfusions need to be updated on resuscitation requirements if additional "shock packs" or other blood products are required.
- Operating room staff must understand the patient's injuries to prepare the operating room to receive the casualty.
- Critical care unit services are often required after surgery.
- Evacuation assets should receive early communication in preparation for transfer to Role 4.

Use of Standard Operating Procedures

SOPs are developed from available evidence and expert opinion and provide guidance to ensure a consistent approach to patient management, which may improve the quality of care.[25] SOPs have been commonplace in the airline industry for many years, covering all phases of flight, with the aim of preventing disaster.[26] Recently the World Health Organization has introduced a surgical checklist[27] to improve patient safety; it has now been implemented in many UK hospitals, where its use is mandatory prior to starting a procedure. For unusual or acute conditions, an SOP can provide important guidance to clinicians.[27] Decisions about the performance of standard emergency

procedures should be made in advance with the benefit of expert opinion[28] and best evidence. During stressful situations memory can be error-prone, resulting in omissions of treatment,[29] and incorrect actions may cause harm with serious consequences.[30]

Situational Awareness

Situational awareness has been defined as "the perception of the elements in the environment within a volume of time and space, the comprehension of their meaning and the projection of their status in the near future."[31] There are three elements to situational awareness[32]:

1. **Gathering information**. This first element consists of collecting information from the surroundings to monitor the state of the work environment and facilitate progress on tasks. Errors can arise if data is not available, if it is misinterpreted, or if individuals display "tunnel vision" or develop fixation errors.
2. **Interpreting information**. In the second element, gathered information is processed to improve understanding of the current situation. This stage is improved when individuals have developed experienced-based mental models from previous deployments or have similar predeployment training. Errors can arise when there is a failure to comprehend the situation due to a lack of or incorrect mental model, or from individual memory failure.
3. **Anticipating future states.** The final element allows the individual to anticipate and plan for future possibilities, refining the mental model.[32]

A loss of situational awareness may arise when there is ambiguity, fixation, confusion, a lack of information, or a failure to maintain critical tasks.[32] Situational awareness is also affected when individuals are fatigued, stressed, or distracted. Strategies to maintain situational awareness have been suggested,[33] including routines for scanning vital signs and instrument functions. In the operating room, strategies for improved awareness involve the use of checklists, training to allocate attention more effectively, learning to multitask, and surgical team briefings to minimize distraction during critical stages of the procedure (eg, induction of anesthesia). Over 10 years of experience in theater has allowed UK and US anesthetists the opportunity to build up mental models and refine techniques in damage control resuscitation. Predeployment training reinforces these tools, ensuring that every member of the team is effectively following the same practices.

Leadership/Followership

Effective military trauma team function depends on effective communication among all members of the team. This objective requires both capable leadership and willingness of team members to display proficient followership skills. Seriously injured patients require early treatment pathway decisions to be made immediately, and these decisions must be accurately communicated to all team members. Everyone must know who the leader is at each stage; the team leader may change as the patient moves from the emergency department to operating room and on to intensive care.

So that the resuscitation area does not become too crowded, it is vital that the team leader is able to exercise a degree of crowd control. Specialists will often stand back until invited by the trauma team leader to offer advice. At times the team leader's job is similar to that of an orchestra conductor, with multiple teams working on a severely injured patient and numerous others supporting the resuscitation.[34] Orders for surgical interventions and additional imaging requirements must also be clearly established within the team. It is vital that the team leader maintains situational awareness and is able to anticipate and effectively respond to physiological changes.

Familiarization With the Environment and Equipment

Predeployment training allows for hands-on opportunities to use unfamiliar equipment prior to stressful situations. High-fidelity simulation is employed to recreate certain aspects of the deployed environment (discussed below).

THE MULTIDISCIPLINARY TRAUMA TEAM

Figure 2-1 shows the make-up of a generic trauma team, and Table 2-1 describes each member's role. This team represents a best practice model. Where there are limited resources, individuals in the team will assume more than one role and specialist resources (eg, surgeon) may move serially from one patient to another depending on the need for specialist assessment and intervention skills. When there are multiple casualties, the emergency medicine consultant will often coordinate the whole department and delegate the team

leader role to another suitably qualified individual.

The process of activating the trauma team is crucial. In the deployed environment, the same team is often on call for prolonged periods. Frequent unnecessary activation calls, particularly in the middle of the night, will leave teams fatigued. The activation criteria, as laid down by CGOs,[6] is described in Exhibit 2-2. Once activated, the trauma team will begin preparing the trauma bay, checking equipment, and organizing drugs.

TRAINING THE TRAUMA TEAM

Using simulation, which allows the delivery of facilitated learning, trainers have the ability to set the training agenda with predefined learning objectives and instant feedback in a safe environment.[35] Simulation is becoming increasing important; the Chief Medical Officer of England and Wales has recently suggested that simulation-based training be fully funded and integrated within training programs for clinicians at all stages.[36] A recent survey of UK military anesthetists has shown general support for simulation in predeployment training.[37] Teaching damage control surgery using a team-oriented approach has been described as an innovative educational method and found to be beneficial.[38]

LIKELY ANESTHESIA TASKS AND CONSIDERATIONS

Scenario 1. Bilateral Above-Knee Amputations

A 22-year-old soldier arrives at the field hospital via helicopter. He has been injured by an improvised explosive device (IED) and has sustained bilateral above-knee amputations with fragmentation injuries to the left hand.

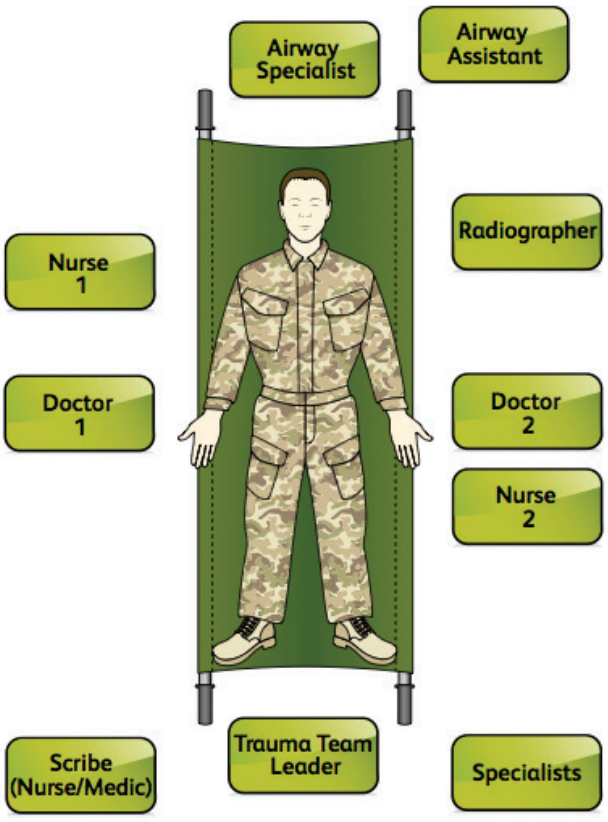

Figure 2-1. Trauma team roles and positions.

Decisions

The notice to the trauma team and the amount of information available will vary. However, upon being alerted to the impending admission, the trauma team must decide on and confirm roles and prepare to receive the patient.

- Roles are assigned.
- The need for surgery is clear but the timing of surgery needs to be determined.
- Should the patient be admitted to the emergency department or move directly into the operating room?
- If the patient is stable after initial resuscitation, is a move to CT scan safe or should damage control surgery be first?

Human Factor Elements

- Assemble trauma team per hospital protocol.
- Balance must be reached between assembling the trauma team and denying members sleep before the next busy workday.
- Careful anticipation and planning ensure that equipment is in the correct location and is functioning.
- Mobilize other key staff:
  - Radiographer to prepare CT scanner.
  - Laboratory staff to prepare for the possibility of a massive transfusion.
- All team members must be aware of their environment:
  - Situation brief by team leader.
  - Overall team composition.
  - Equipment and preparation.

TABLE 2-1

THE ROLES OF THE TRAUMA TEAM

Position	Responsibilities and Skills
Team leader (usually the emergency physician)	• Controls and manages resuscitation. • Team leader (usually the emergency physician) • Prioritizes investigations and treatment. • Makes time-critical decisions. • Has good leadership skills. • Ensures the environment is such that only his or her own voice can be heard. • Clearly communicates and delegates tasks.
Airway specialist (anesthetist)	• Responsible for assessment and management of the airway and ventilation. • Counts the initial respiratory rate. • Administers oxygen. • Performs suction. • Inserts airway adjuncts. • Performs endotracheal intubation (RSI) • Maintains cervical spine immobilization and controls the log roll. • Takes an initial history (AMPLE).
Airway assistant	• Assists in preparing equipment for advanced airway intervention. • Assists with advanced airway intervention, eg, applies cricoid pressure.
Doctor 1	• Undertakes the primary survey: <C>+B to E. • Clearly communicates clinical findings to team leader (recorded by scribe). • Performs procedures depending on skill level and training.
Doctor 2	• Performs procedures depending on skill level and training.
Nurse 1 (emergency department nurse responsible for airway)	• Applies monitoring equipment. • Assists advanced airway intervention (unless ODP is present). • Assists with procedures.
Nurse 2 (emergency department nurse responsible for circulation)	• Undresses patient. • Assists with procedures.
Scribe (emergency department nurse or medic or HCA)	• Collates all information and records decisions on trauma chart • NOTE: All team members are responsible for ensuring their findings and decisions are correctly recorded.
Radiographer	• Takes x-rays as directed by the team leader .
Hospital specialists	• Undertakes secondary survey and advanced procedures (eg, general surgeon to undertake secondary survey of the head and torso; orthopedic surgeon to undertake secondary survey of the limbs, pelvis, and spine; surgeon, emergency physician, or ultrasonographer to undertake FAST).

AMPLE: allergies; medications; past medical history, injuries, illnesses; last oral intake and menstruation; events leading up to the injury and/or illness
<C>+B to E: control of catastrophic hemorrhage, breathing, circulation, disability, exposure
FAST: focused assessment with sonography for trauma
HCA: health care assistant
ODP: operating department practitioner
RSI: rapid sequence induction

    - Standard operating procedures.
- Crosscheck and double-check:
    - Primary and secondary survey per the Battlefield Advanced Trauma Life Support Course.[39]
    - Communicate findings to team leader.
- Cognitive aids:
    - Local SOPs.
    - Clinical Guidelines for Operations.
    - Surgeon General's policy letters.

- Leadership will change if the patient is transferred to the operating theater.

Scenario 2. Possible Hypovolemic Shock

A 24-year-old is involved in a vehicle explosion from an IED. He arrives via the Medical Emergency Response Team and has bilateral above knee amputations with perineal penetration and buttock wounds. Cardiopulmonary resuscitation is in progress for pulseless electrical activity arrest. A decision is made to move the patient directly to the operating theater, where the trauma team is assembled. The most likely diagnosis is hypovolemic shock.

Decisions

- Timing of thoracotomy vs securing intravenous access.
- There are problems with vascular access due to lost limbs and soft tissue damaged by blast or thermal injury.
- Special equipment is needed for vascular access (eg, catheters, ultrasound).

Human Factors Elements

- This scenario is common in the current military theater environment.
- Train with rehearsals.
- Acquire preformed mental models built up from prior experience.
- Continuous practice and refinement of damage control resuscitation has led to much success: individuals are tuned into to their environment, knowing their roles, theater-specific equipment, and communication issues.
- Crowd control requires effective leadership.
- Use cognitive aids.
- Assemble the trauma team early.
- Anesthetic team will have a person responsible for:
  - Leading the anesthetic intervention.
  - Airway management.
  - Vascular access.
- If there is a high index of suspicion that a casualty may require a direct transfer to the operating room, equipment such as the rapid infuser and central line kit will be set up and ready to go in both the emergency room and the operating room.
- Decisions are made quickly but with discussions among the team leader, anesthetic personnel, and surgical teams.
- Anesthetist is aware of the stage of the resuscitation.
- Surgeon is focused on thoracotomy and vascular control.
- Communication between surgeons and anesthetists is vital during damage control surgery because bleeding can be due to a cause amenable to surgery or to derangement of clotting that could be corrected as guided by thromboelastometry.
- Priorities are set dynamically and the situation is constantly reevaluated.
- Additional communication with:
  - Laboratory staff.
  - Intensive care.

Scenario 3. Damage Control Surgery With Multiple Injuries

A multiply injured patient is anesthetized in the operating room for damage control surgery with multiple surgical teams.

Decisions

- Sequence of surgical procedures depending on the priority.
- Need to move patient to CT scanner following damage control surgery.
- The process of achieving vascular control in damaged limbs.
- Where to site vascular access.

Human Factors Elements

- This is a clinical situation that does not frequently occur in routine NHS practice.
- Predeployment training includes:
  - Familiarization with environment.
  - Familiarization with equipment and SOPs.
- SOPs will to encourage teamwork, communication, and effective leadership and followership.
- Leader must:
  - Communicate with the surgical teams over when to operate and when to pause, depending on the patient's physiology and the stage of resuscitation.
  - Coordinate with the anesthetist responsible for vascular access.
  - Coordinate with the anesthetist responsible for airway management.
  - Coordinate with operating room staff about running the rapid infusion device.

  - Maintain situational awareness, which is crucial to success because anticipation and planning for frequent changes in the patient's condition will be necessary.
- Patient's condition and management are constantly reevaluated in case changes need to be made to the original plan.
- Additional communication with:
  - Laboratory regarding additional blood products.
  - Critical care unit to plan timing for critical care evacuation.

Scenario 4. Fluid Replacement With Multiple Injuries

A multiply injured patient is anesthetized in the operating room and receiving a high volume fluid replacement.

Decisions

- Where to site intravenous access (the current practice is a large-bore subclavian central venous pressure line); will be influenced by site of injury.
- Blood and blood products.
- The rate of fluid administration.
- Anesthetic drugs.
- Which clinical parameters are being aimed for.
- Monitoring and management of hyperkalemia, hypocalcemia, and coagulopathy.
- Pain management plan.

Human Factors Elements

- Ongoing experience with damage control resuscitation has allowed the practice and refinement of this process.
- It is important that all member of the team are following the damage control resuscitation flow sheet.
- Use of SOPs improves teamwork and communication.
- Communication with:
  - The team controlling the level one infuser.
  - Laboratory staff.
  - Intensive care.

Further Crew Resource Management (If Patient Goes to Operating Room)

- Effective leadership handover and timing.
- Designated leader in operating room.
- Personnel must understand:
  - The plan.
  - SOPs.
  - Sequence of surgical procedures.
  - Who is in charge (although leadership roles may change).
- Communication with surgical teams about:
  - When to operate.
  - When to pause, depending on the patient's physiology.
  - The stage of the resuscitation.
  - Vascular control in damaged limbs.
  - Whether there is a time limit.
  - Whether further CT imaging is needed.

SUMMARY

This chapter outlines the key human factors that are required by the whole trauma team in dealing with a patient with complex injuries. The human factors have been illustrated with examples, concentrating on communication, decision-making, leadership, and teamwork. It is believed that exemplary human factors are responsible for the success of the trauma team in recent conflicts.

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Chapter 3

MILITARY PREHOSPITAL MEDICINE

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INTRODUCTION

In Afghanistan, the long distances, restriction of movement on the ground, and domination of the airspace by the coalition have made helicopters essential in both moving and treating casualties in the prehospital battlefield arena. The difficulty now faced is identifying which aspects of care have led to improved patient survival and how to apply these lessons in future conflicts.

The military medical evacuation system has significantly evolved in the past 11 years with a change from step-wise movement of casualties through the different echelons of care to the present practice of direct transfer from point of injury to Role 2 or 3 hospitals. All areas of military medical care have improved, and the evacuation chain of survival for the combat casualty is now highly evolved. Between April 2, 2006, and July 30, 2008, 85 patients who were predicted to die from massive injuries sustained in the fighting in Afghanistan survived.[1] The combined medical services of all nations in Afghanistan continue to produce statistically unexpected survivors from trauma. Soldiers are now surviving injuries that would have been uniformly fatal in previous conflicts.

This evolution has led to a complex system of systems within areas of Afghanistan (the most current example) in which all forms of CASEVAC (the movement of casualties aboard nonmedical vehicles or aircraft) and MEDEVAC (the movement of casualties via ground or air by a dedicated_medical evacuation platform) may be employed in the course of providing combat casualty care. A patient may arrive at a Role 2 or 3 medical treatment facility (MTF) aboard just about any platform, having received a wide range of en-route care interventions. The spectrum of care ranges from ground vehicles of opportunity providing no en-route care at all to an armed and armored CH-47 helicopter with a four-person medical emergency response team, led by a doctor experienced in prehospital care and equipped to perform extensive resuscitation en route.

Currently in parts of Afghanistan, at least three different systems of forward aeromedical evacuation may be operating: US Army MEDEVAC helicopters (known since the Vietnam War as "Dustoff"), US Air Force Pararescue helicopters (referred to as "Pedro"), and the United Kingdom (UK) Medical Emergency Response Team (MERT) aboard CH-47 aircraft. This mixture of forward aeromedical evacuation capabilities is in addition to various means of ground evacuation that may be employed as dictated by weather, threat, or other considerations.

These capabilities are employed in individual missions through a system of "intelligent medical direction" or "intelligent tasking," which normally resides in the Combined Joint Operations Center of the regional command and is directed by a patient evacuation coordination center (PECC). PECCs are staffed by experts in medical and aviation operations and often include a person with clinical expertise, usually a nurse. The detailed processes at work in a PECC are beyond the scope of this chapter but serve overall to assure that the goals of "right patient, right platform, right escort, right time, right destination" are all achieved. These five "rights" give casualties the greatest possible chance to survive and medical professionals the greatest possible chance to apply their expertise in caring for them. Given the wide variation among different evacuation platforms, it behooves the medical professional to have some familiarity with their various capabilities to be prepared to receive patients ranging from those who received no en-route care at all to those who were fully resuscitated prior to arrival at the MTF.

It is likely that in the next war, the environment, the threat, and the platforms used to evacuate casualties will be different. The various evacuation capabilities, from ground CASEVAC to MERT or even some capabilities more complex than are presently feasible, will need to evolve to continue providing life-saving care for service members. The lessons learned on (and over) the plains and mountains of Afghanistan may also have application in the civilian arena, particularly in areas where trauma systems are in a state of continuous evolution. The current Joint Theater Trauma System, built in the combined and joint environment of Afghanistan, is achieving unprecedented levels of performance and saving a larger proportion of seriously injured combat casualties than in any previous conflict.

THE EVACUATION CHAIN

The Chain of Survival

The delivery of critical care forward of the hospital must be placed within the wider continuum of care. Of the many available multinational prehospital evacuation assets, the helicopter-based medical care forms are the most widely used within the chain of evacuation from the battlefield to Role 3. Without immediate and effective first aid at the point of injury, none of the most critically wounded would survive to be evacuated.

The first link in the chain of survival requires soldiers (referring here to any service member) to treat themselves or for a buddy to apply first aid.

Self Treatment

All soldiers are taught basic first aid. This training is comprised of triage, basic life support, pressure dressings and tourniquets, placement of chest seals, use of the sit-up and lean forward position for oral cavity and oropharyngeal hemorrhage, and administration of the combat pill pack (analgesics and antibiotics). The initial treatment is likely to be by the casualties themselves before any help arrives as mission priorities during a fire fight continue. The most common life-saving self treatment is applying a tourniquet for extremity hemorrhage.

Buddy-Buddy and Team Medics (UK)

For UK armed forces the next phase of care is provided by other soldiers. The "buddy-buddy" system simply allows the same level of care to be provided by those uninjured or less injured than the casualty. A minimum of one in every four soldiers will be trained to the level of team medic, with a higher level of first aid training and usually carrying additional medical items such as intravenous (IV) cannulae; however, this soldier's primary role is not medical. Training is based on British "CABC" military trauma management: treatment of Catastrophic or massive hemorrhage, including the use of tourniquets; Airway; Breathing; and Circulation. The emphasis at this stage is on control of catastrophic hemorrhage. Communication and organizing the evacuation of the casualty down the chain are concurrent activities. Many patrols also include a combat medical technician, a professional medic with the skills to deliver more advanced techniques if necessary, such as vascular access, pelvic splints, and airway management.

Combat Casualty Care (US)

The equivalent stage in US forces is termed combat casualty care or sometimes care under fire, which also begins at the point of injury. Medical care is administered by either the injured casualty (self-care) or by buddy care via a fellow combatant with no specific medical training, or an individual with basic first aid skills training, termed a combat lifesaver. Specific combat units may have medical technicians with specific entry level or advanced training embedded in their ranks for the purpose of providing more advanced care at the point of injury (eg, combat medic, Special Forces medical technician or sergeant, pararescueman, corpsman).

Higher Levels of Care (Roles 1 through 3)

Rapid evacuation of casualties is a priority because it increases casualty survival and allows units to continue the mission once the wounded are removed. The traditional UK military evacuation of casualties occurs through Role 1, a mobile, lightly equipped unit consisting of a doctor and a number of medics called a regimental aid post (RAP). Each battalion has one RAP, which is typically augmented by another physician and additional medics in certain areas of operations. The United States has Role I MTFs, most commonly termed a battalion aid station (BAS). This unit, comparable to the RAP, conducts triage, resuscitation, and simple life-saving interventions, and facilitates evacuation to a higher level of care. The BAS is staffed by a physician or physician assistant as well as medics, and again like the RAP has few patient hold capabilities and no surgical capabilities. The BAS is typically collocated far forward with a maneuver unit and is able to stabilize casualties for evacuation to higher roles and facilitate return to duty when appropriate.

The next stage in the UK is Role 2, a mobile surgical facility commonly described as a forward surgical team, typically consisting of two surgeons, two anesthetists, an emergency physician, and variable numbers of medics, operating room staff, nurses, and support staff. It commonly has two operating tables, and its main aim is to provide damage control surgery with a limited capacity for equipment and blood products. Rarely does this unit have any patient holding capability, and its ability to provide life- or limb-saving surgery is dependent on efficient evacuation to the next level. Role 3 is a field hospital with variable capacity depending on the predicted need and casualty estimate, normally with at least an emergency department, two operating tables, four critical care beds, and twenty-five other beds with the appropriate staff.

The US Role 2 MTF, in addition to basic primary care capabilities, can provide blood transfusion (typically uncross-matched blood), brief inpatient admission, dental and mental health, and when augmented, damage control surgical services. According to the *Emergency War Surgery Handbook*,[2] Role 2 MTFs with surgical capabilities can conduct life-saving general, thoracic, vascular, orthopedic, and neurosurgical procedures. This surgery is focused on hemorrhage control, control of contamination, and overall stabilization. These augmented units include the US Army forward surgical team, which has one orthopedist, three general surgeons, two nurse anesthetists, and two critical care

Figure 3-1. Boeing CH-47 Chinook helicopter. Reproduced from: http://upload.wikimedia.org/wikipedia/commons/4/41/CH-47F_at_NTC_2008.jpg

Figure 3-2. Sikorsky UH-60M Black Hawk helicopter.

nurses and technicians. Capabilities include the ability to conduct 30 surgical procedures over a 72-hour period and postoperative intensive care bedding for up to eight casualties over a 6-hour period. Other mobile forward surgical teams include the US Air Force mobile field surgical team (a 5-person team capable of 10 damage control procedures over 48-hour period), and the US Navy surgical company (3 operating theaters with a 60-bed capacity and a 72-hour holding period).

Evacuation to Higher Level of Care

In Afghanistan, the classical concept of care delivered in a linear fashion from point of wounding through Roles 1 and 2 before evacuation to Role 3 and beyond no longer necessarily applies. The exception to this is in areas of the country where time and distance factors between units on the ground, Roles 1 and 2 MTFs, and definitive care at Role 3 dictate making stops along the way to stabilize or resuscitate patients prior to onward evacuation. The majority of casualties, however, are retrieved from the battlefield and rapidly evacuated via aircraft to the nearest Role 3 hospital, bypassing smaller medical facilities. Often confounding evacuation methods is the ever-present enemy threat to a valuable evacuation asset sitting on the ground vulnerable to small arms fire. There is a mix of airframes, ranging from a spacious CH-47 (Figure 3-1) to a more space-limited UH-60 (Figure 3-2) platform. (The UH-60 focuses on the "scoop-and-run" concept, whereas the space in the CH-47 allows resuscitation to occur in flight, combining "scoop-and-run" with the "stay-and-play" concept.) Ultimately decisions about whether to begin treatment on the ground or immediately launch and provide care en-route are made on the aircraft by the pilot in command with input from the medical team in the back, soldiers providing protective fire on the ground or in the air, and the pilot's command and control element. For the last 5 years in Afghanistan, very little patient care has been provided by medical evacuation assets on the ground and most care has been performed in flight, with the exception of extreme circumstances such as casualties trapped in vehicles or mine fields.

The majority of casualties will be delivered to the nearest Role 3 facility. However, a small number will benefit from primary evacuation to other facilities with specialist capabilities such as neurosurgery and ophthalmic surgery. Individual aircrews must be aware of available assets to facilitate decisions on the best facility for individual patients and ensure that timelines, especially for neurosurgery, can be met. Also, evacuating local nationals with certain injuries to host nation medical facilities helps prevent military facilities from becoming overwhelmed. Such decisions must be made rapidly and accurately and depend on the wider tactical picture, the number of casualties, the pattern of injury, and the experience of the individuals.

The remainder of this chapter will describe the three major platforms providing helicopter-based medical evacuation support to combatant forces in a theater of operation (currently Afghanistan): (1) the UK's CH-47-based MERT, (2) the US Army's UH-60–based Dustoff, and (3) the US Air Force's UH-60–based Pedro.

THE MEDICAL EMERGENCY RESPONSE TEAM

The MERT concept has evolved since 2006. In previous operational theaters (Northern Ireland, the Balkans, and Iraq), the Immediate Response Team (IRT), consisting of aircrew, the MERT, and the Force Protection

Teams (at minimum) was used, but the high numbers of casualties sustained in southern Afghanistan has driven the development of the IRT's medical component. A standing team consisting of a nurse, a paramedic, and a combat medical technician was, when required, supplemented by a hospital clinician; this team was known as enhanced MERT or MERT(E). Beginning in 2008, doctors were deployed specifically to be part of a MERT. Using doctors experienced in prehospital emergency medicine allowed higher level interventions such as thoracostomy, rapid sequence induction (RSI) of anesthesia, and prehospital blood component transfusion, as well as rapid senior-level decision making. At the same time MERT standard operating procedures (SOPs) were written to standardize the training given to the teams and the care given to patients.

Composition of the Team

There are usually two MERTS deployed to cover the British area of operations in Afghanistan, with a third team on standby in the UK. Currently one team consists of four personnel (Table 3-1). Although combat medical technicians work well within the teams, an effort is underway to deploy more trained paramedics as the fourth practitioner, giving them the opportunity to work under the supervision of a more experienced paramedic during their first deployment.

Predeployment Preparation

All team members have experience in civilian prehospital care. A combination of civilian courses such as Advanced Paediatric Life Support (APLS) and military medical courses such as Battlefield Advanced Trauma Life Support (BATLS), together with specialty courses, provide a platform on which to build integrated team training. These elements are brought together in the MERT course, in which teams are exposed to treating simulated casualties in increasingly challenging environments, with a final assessment on board a flying CH-47 in United Kingdom. Training is further consolidated at the whole-hospital predeployment exercise, in which simulated casualties are transferred to the hospital from a simulated Role 1 or point-of-wounding environment with subsequent resuscitation.

In-Theater Training

Once in the deployed setting, teams receive generic training to allow team members to refresh their skills and receive updates on the current tactical and clinical

TABLE 3-1

COMPOSITION OF THE MEDICAL EMERGENCY RESPONSE TEAM

Clinician	Background	Responsibilities
Flight doctor	Consultant or senior registrar (post-fellowship) in anesthesia or emergency medicine. Experienced in prehospital emergency medicine. HEMS doctor. Should have prehospital care as part of their job plan when not deployed.	Clinical leadership of the team. Senior decision-making. Control of hemorrhage, sedation, anesthesia, intubation, transfusion, packaging, and management of the patient in flight.
Flight nurse	Registered emergency medicine nurse. Should be experienced and clinically current. Civilian prehospital experience desirable but not always possible.	Administration of the team. IV/IO access, monitoring of the patient, blood component transfusion. Assisting during induction of anesthesia. Communications, especially between MERT and aircrew.
Flight paramedic	State registered paramedic. Regular work as a paramedic must be part of their job plan when not deployed.	Handover of the patient from the ground call sign. Loading and unloading of patients. Control of catastrophic hemorrhage. IV/IO access, assisting in blood component transfusion. Patient packaging. Handover to the hospital trauma team.
Fourth practitioner	In recent years this is usually a more junior paramedic, also state registered. Other team members fulfilling this role have included operating department practitioners and combat medical technicians.	Handover of the patient from the ground call sign. Loading and unloading of patients. Control of catastrophic hemorrhage. IV/IO access, assisting in blood component transfusion. Patient packaging. Handover to the hospital trauma team.

HEMS: Helicopter Emergency Medical System
IV/IO: intravenous/intraosseous
MERT: Medical Emergency Response Team

situation, before moving on to the team integration and familiarization phase. An appreciation of the multinational forces and differences in their SOPs is also presented. The location of all medical assets and their capabilities, both military and local national facilities, is briefed to aid in correctly directing patients to facilities with the appropriate capabilities for their injuries.

Combat Casualty Retrieval

The IRT is on permanent standby to move. Response times are necessarily longer at night due to the requirement for extra mission planning, but the team aims to be airborne in a matter of minutes following receipt of a casualty report. In addition to the minimum MERT personnel, other elements such as combat troops, explosive ordnance disposal specialists, and fire fighters are available, depending on mission requirements. When on standby, the teams live together, close to the airframe, to facilitate communication and mutual understanding and to minimize response times.

US MEDICAL EVACUATION ASSETS

In Afghanistan the majority of casualties are carried by US medical evacuation assets. These assets may be ground or air platforms as the terrain and tactical situation dictates. Evacuation by air is the preferred means when possible.

CASEVAC

Because CASEVAC occurs on nonmedical vehicles or aircraft, en-route care beyond self-care or buddy care (tourniquets, IV, or intraosseous [IO] access) may be limited. A key distinction between CASEVAC and MEDEVAC is that only MEDEVAC platforms are identified by the Red Cross and thus enjoy protections under the Geneva Conventions. CASEVAC is rarely used in the current theaters of operations, although it is necessary when no MEDEVAC assets are available but combat aircraft are in proximity to casualties.

MEDEVAC (Dustoff)

Although the term "MEDEVAC" is commonly understood to refer to dedicated US Army air ambulances (known since the Vietnam War as "Dustoff" aircraft), medical evacuation is technically defined as the movement of patients by dedicated air or ground platforms with care provided en route. Medical evacuation may be from the point of injury, a casualty collection point, an ambulance exchange point, or from one treatment facility to another. Ground evacuation platforms differ based on the type of unit; ie, the M113A3 ambulance in armor, mechanized, and cavalry units provides direct medical support to their parent organizations, and light vehicles, M996 or M997 HMMWVs, are used in light infantry units. Throughout Operations Enduring Freedom and Iraqi Freedom, medical evacuation has been primarily conducted by MEDEVAC aircraft on an area basis utilizing HH-60M Blackhawk helicopters due to their speed, the geographical emplacement of forces, the restrictive terrain, and enemy use of improvised explosive devices. MEDEVAC ground and air platforms are identified with a red cross and operate completely unarmed except for the defensive personal weapons assigned to each crew member (tactical commanders may limit the display of the red cross according to operational and tactical circumstances). MEDEVAC platforms may be escorted by combat vehicles or aircraft as the tactical situation requires.

Training

En-route care is provided by a flight medic with a minimum emergency medical technician-basic (EMT-B) certification. Flight medics trained by the US Army School of Aviation Medicine receive advanced training and certifications such as Advanced Cardiac Life Support, International Trauma Life Support, and Pediatric Education for Pre-hospital Professionals or Pediatric Advanced Life Support, as well as courses in critical care management of a patient in a medical evacuation aircraft platform, RSI, helicopter underwater egress, high altitude operations, personal recovery operations, survival, and canine trauma management. The Joint Enroute Care Course, optional for flight medics, covers quality en-route care for a postoperative critical care patient. As a result of a recent initiative by the Army Medical Department, EMT-Paramedic certified flight paramedics are currently being fielded throughout the operational force.

En-Route Care

With a heritage dating to the first helicopter rescue in 1945, Dustoff is the only dedicated aeromedical evacuation platform with its own equipment, personnel, and doctrine within the armed forces. The scope of care flight medics can provide varies by unit and training, and is outlined in the unit's treatment protocols, which are developed by brigade or battalion flight surgeons. EMT-B–trained flight medics

can provide basic resuscitative and treatment capabilities including tourniquet application, infusion of crystalloid and colloid fluids, splinting (including pelvis), pain management, basic airway management (bag-valve mask and laryngeal mask airway), defibrillation, and needle decompression. Due to the frequent transfers of critically injured, mechanically ventilated casualties between Roles 2 to 3 MTF, an emerging tactic, technique, or procedure (TTP) calls for some Dustoff crews to be augmented by an en-route critical care nurse (ECCN) or flight surgeon/aviation physician assistant to provide postoperative en-route critical care.

Capabilities

US Army Dustoff units are assigned 15 aircraft and are placed as far forward as possible. Typically, forward support MEDEVAC teams have three aircraft and are collocated with an aviation task force. Urgent and urgent surgical missions are mandated to be complete in 60 minutes (the "golden hour") from the time the unit receives the mission to the patient's delivery to the appropriate MTF. This is a recent change in doctrine based on a 2009 Secretary of Defense Memo for Record. The memo also spelled out a launch goal of under 15 minutes from time of mission receipt to aircraft launch.

The placement of MEDEVAC aircraft in theater is critical due to the austere and distant locations of some US and coalition forces. Because of the wide dispersal of forces and finite MEDEVAC aircraft available, commanders must accept certain risks to accomplish the overall mission. MEDEVAC aircraft are positioned in conjunction with the population at risk, enemy threat, and mission requirements. The location of MEDEVAC aircraft is closely synchronized with medical assets that have resuscitative surgical capability. MEDEVAC companies are organized within general support aviation battalions in each Combat Aviation Brigade and provide area or direct support as directed. The HH-60 is a two-pilot aircraft staffed with a Medical Service Corps officer and warrant officer conducting flight operations, and one crew chief (15T) and one flight medic (68WF) occupying the cabin area. Patient capacity ranges from two to six litter patients (based on configuration) and up to eleven ambulatory patients. The cabin area of the aircraft is modified for medical treatment to include an internal or external hoist with a forest penetrator and litter system, a medical equipment set, and auxiliary oxygen.

TTPs continue to evolve in an effort to increase favorable patient outcomes and reduce morbidity. ECCNs have been utilized on a limited basis in Operation Enduring Freedom for the evacuation of high acuity patients from Role 2 to Role 3 MTFs to enhance survivability. As of this writing, one class of the Critical Care Flight Paramedic program has graduated, with planning for a total of 950 graduates by 2018. Enhancing en-route care capabilities, graduates from the new course are given 1,100 additional training hours and are capable of administering blood products when authorized by a physician or physician assistant.

US Air Force Pararescue

History and Description of the Platform

The Air Rescue Service was an outgrowth of the need for an organized rescue asset after World War II, when airframe and aircrew losses and crashes in remote and austere environments were extensive. The initial aircrew focused on remote survival and basic medical support after physicians, who were initially involved, were removed. During the Vietnam War the capabilities included rescue of downed aircrew and rescues during active fire fights. This led to the formal introduction of tactical training for the pararescuemen (known as "PJs"). The call sign "Pedro" (used by an air rescue squadron in Laredo, TX, in the 1950s) was adopted in 1967. In the 1990s pararescuemen began obtaining paramedic certification, which remains the current foundation of their medical education, with further specialized supplementation.

US Air Force air rescue is comprised of the HH-60 helicopter (Pedro), fixed-wing HC-130P Hercules (Fever), and the Guardian Angel Weapon System (GAWS), staffed by pararescuemen and combat rescue officers. The primary role of Air Force rescue is personnel recovery, including combat search and rescue and the recovery of isolated personnel (traditionally referring to downed pilots and aircrew in remote or nonpermissive environments). Due to the infrequency of aircraft loss and the Army's need to meet the golden hour requirement, Pedro has augmented tactical evacuation. The pararescue airframe is the US Air Force HH-60G Pave Hawk helicopter (Figure 3-3). The helicopters fly in pairs and have two or three pararescuemen each. Pararescuemen are US Air Force special operating forces trained to the level of EMT-P, with the ability to perform advanced airway management (bag-valve mask, laryngeal mask airway, endotracheal intubation, surgical airways), IO access, tourniquet placement, splinting (including pelvis), pain management, needle decompression, finger and tube thoracostomy, and (since December 2010) transfusion of uncross-matched red blood cells and plasma.

Fever, an intratheater MEDEVAC asset deployed

Figure 3-3. US Air Force HH-60G Pave Hawk helicopter.

to Camp Bastion, Helmand Province, is equipped to move critically injured patients to a Role 3 MTF from an airstrip in proximity to a Role 1 or 2 facility. The medical crew was originally made up of a US Air Force flight surgeon and two pararescuemen, but was recently augmented by an emergency medicine doctor and critical care nurse (ECCN). Capabilities include advanced en-route critical care with ventilator and hemodynamic support. Fever evolved due to the necessity to evacuate casualties from remote locations, often after damage control surgery, to a Role 3 MTF more rapidly than possible with rotary wing aircraft. The airframe can carry multiple litter patients (up to nine), allowing evacuation of multiple casualties from an overwhelmed Role 1 or 2 facility, and does not need en-route refueling in Afghanistan.

The primary role of the GAWS is personnel recovery. The GAWS is composed of combat rescue officers and pararescuemen. To carry out personnel recovery for the Department of Defense, pararescuemen are trained as the most advanced tactical and technical rescue specialists in the world. Pararescuemen are paramedics with supplemental training in tactical medicine and evacuation, advanced care for extended times in remote and austere environments, and in-flight care on fixed or rotary wing aircraft. Other capabilities include precision parachuting, scuba diving and surface marine operations, rescue swimmer, weapons and small unit tactics, communications, small vehicle and water craft use, high altitude and cold weather operations, alternate means of insertion and extraction via rotary wing aircraft, and multiple rescue skills including confined space, high angle, swift water, vehicle extrication, fire suppression, and structural collapse. Because of the need to maintain these capabilities, clinical medical training is limited. However, pararescuemen are able to go into conflicts with active enemy contact and effect rescues of injured personnel that the other platforms cannot.

Pararescue Medicine

Pararescue medicine is a unique conglomerate of EMT-P certification, Tactical Combat Casualty Care (TCCC), Special Operations medicine, wilderness medicine, dive medicine, and en-route care in flight. TCCC care is formally recognized by the National Association of Emergency Medical Technicians. It includes three phases: (1) care under fire, (2) tactical field care, and (3) tactical evacuation. Pararescuemen are the only medical asset to routinely provide care during all phases. TCCC began as an empiric approach to combat injury care in 1997 but was recently validated in a report by Kotwol[3] and now represents evidence-based medicine. TCCC is based on data on the use of tourniquets, gauze impregnated with hemostatic agents, early antibiotics, and surgical airways for severe maxillofacial trauma. This approach has the potential to fundamentally transform civilian prehospital trauma care.

CARE DURING MEDEVAC

Because the MERT includes a highly qualified doctor and the adequate space to carry out highly skilled interventions, the medical team on board are able to bring damage control resuscitation and trauma anesthesia out of the hospital, to the patient during evacuation. This ability to carry out advanced resuscitation has been termed by some US authors as "advanced medical retrieval" (AMR), and they have used this term in comparing MERT to more conventional military retrieval methods. Recent studies by these authors comparing, among other things, mortality among casualties evacuated by these different capabilities indicate that conventional platforms are effective when patients have a low injury severity level.[4–7] However, the studies found evidence that a definable injury severity exists for which evacuation with an AMR capability is statistically associated with improved outcomes. They also discovered that over all injury severity scores, there was a lower than predicted mortality on an AMR platform.[4,7] Interventions carried out aboard AMR platforms tended also to be associated with shorter times between arrival at the emergency department of the receiving MTF and entering the operating

room for surgery,[4] and shorter times to initiation of resuscitation.[6] Discussed below are some of the issues facing combat casualty care aboard platforms used for evacuation.

Concurrent Resuscitation

Aboard larger aircraft with multiple medically trained personnel on the crew, the concept of concurrent resuscitation becomes possible, as described best by MERT team members. As soon as the patient touches down on the deck of the aircraft, treatment begins. Because of injury severity in a significant number of these casualties, there is no place for the "vertical" assessment of the patient through a rigid protocol; instead, these steps must happen simultaneously in a "horizontal" fashion (as though the patient is a racing car and the medical team the pit crew). With training and practice medical teams can apply tourniquets, dressings, and a pelvic binder; initiate monitoring; intubate and ventilate the patient; and administer at least four units of warmed blood components in the space of 6 minutes. This can happen only when all members of the team know their exact roles and work quickly and efficiently together.

Control of Catastrophic External Hemorrhage

Stopping external hemorrhage is a key element in reducing mortality associated with blast injury. In the case of traumatic amputations this is most commonly achieved by the rapid application of a tourniquet to the injured limb or limbs by the patient, other soldiers, or a team medic. Once the patient is on board the airframe, tourniquets are checked and when necessary tightened and additional tourniquets placed. Experience has taught that patients with above-knee amputation will require two combat application tourniquets applied side-by-side to ensure that hemorrhage control is achieved. Stumps are placed in a box splint to protect them and to exploit the analgesic effect of immobilization. Hemostatic agents such as Celox (MedTrade Products Ltd, Crewe, UK) followed by 5 minutes of direct pressure may be required on areas of noncompressible hemorrhage, particularly the junctional areas of the neck, the axillae, and the groin. Because a high frequency of pelvic injuries is associated with traumatic amputations,[8] pelvic ring injury should be suspected and treated prophylactically using a pelvic binder in all lower limb traumatic amputations. Perineal injuries are common, particularly in high amputations, and require aggressive packing with hemostatic agents to successfully control bleeding.

The Log Roll

Use of the log roll assessment technique is controversial. The patient will benefit from minimal movement during this phase of their injury: clots have not yet consolidated and fractures are not stabilized. However, missing a penetrating injury to the back of the head or chest, or missing an area of external hemorrhage, is considered to be more dangerous for the patient than being moved, so a single log roll is mandated. Hemostatic agents must be on hand during the procedure, and particular care must be taken not to dislodge IV and IO cannulae and the endotracheal tube.

Access to the Vascular Space

While the priority for treatment firmly rests with hemorrhage control, in practice, at the same time as the tourniquets are being checked, access to the intravascular space is being gained. Large-bore peripheral IV cannulation is ideal because high flow rates can be achieved; however, it is difficult, time-consuming, and associated with high failure rates due to the environmental constraints of operating on airframes and the extreme hypovolemia seen in MEDEVAC casualties. IO access using the EZ-IO (Vidacare, Shavano Park, TX; see Chapter 39, Basics of Pediatric Trauma Critical Care Management, Figure 39-4) and FAST (Pyng Medical, Richmond, Canada) is therefore routine due to the speed, reliability, and lower failure rates of these devices. The EZ-IO is most commonly placed in the humeral head in the military population due to the high frequency of injuries to the legs and to take advantage of the higher flow rates.[9] EZ-IO can be placed in the tibia or pelvic crest, if possible, but consideration should be given to major venous injury in thoracic and abdominal injury. FAST is placed in the sternum. While it can be used for blood products, it is principally used for the administration of drugs. All blood products must to be warmed prior to administration and are administered via an enFlow (Vital Signs Inc, Totawa, NJ) warming device using a 50 mL-syringe and a 3-way tap. Although IO access can in theory remain in use for up to 24 hours, it will be superseded by the insertion of central venous trauma lines immediately on arrival at the Role 3 hospital.

The Gold Standard (Rapid Sequence Induction)

The MERT is one of the few teams around the world that performs drug-assisted intubation on board a helicopter while in flight. Most civilian Helicopter Emergency Medical System (HEMS) teams argue that

patients should be fully stabilized and packaged before movement by helicopter. However, civilian helicopters are smaller than military combat support helicopters and have limited interior space to perform procedures. The use of large-body helicopters, particularly the CH-47, makes it possible to gain good 360° access to the patient, to carry large amounts of equipment including monitoring devices, and to perform intubation safely. Although there are known advantages to techniques such as prehospital anesthesia, patient outcome following trauma is directly related to the time between injury and surgical intervention. Performing procedures on the aircraft while in flight reconciles both of these approaches.

Severely injured patients will require more than one route of intravascular access in order to administer anesthetic agents concurrently with starting the massive blood transfusion. This may be peripheral intravenous access, intraosseous access, or a combination of the two. Ideally the patient should be preoxygenated before drugs are administered. The patient should be monitored with pulse oximetry, 3/4-lead electrocardiogram, noninvasive blood pressure, and end-tidal CO_2 at a minimum. A set of observations should be recorded before RSI is carried out. The patient should be intubated by the most experienced practitioner (the flight doctor), assisted by a trained assistant (the flight nurse). Endotracheal tube placement must be confirmed by capnometry or (preferably) capnography. Monitoring must be continued during flight and during handover to the hospital trauma team.

Ideally, the entire team will be focused on delivering care involving RSI to one patient, enabling the high standards described above to be met. Frequently, however, multiple patients require RSI on the same mission. In these circumstances the flight doctor must decide whether or not performing RSI on one patient will compromise the management of the others.

Constraints in Flight

There are clear difficulties with performing RSI in the back of a helicopter moving tactically. A formal risk assessment register is maintained in theater, and every effort made to mitigate the threats identified. Some examples are listed in Table 3-2.

TABLE 3-2

RISKS OF RAPID SEQUENCE INDUCTION DURING FLIGHT

Risk	Mitigation
Enemy action on the ground	The MERT is escorted by a Force Protection Team. Time on the ground collecting patients is kept to a bare minimum. All team members must pass generic predeployment fitness and military tests.
Enemy action against the aircraft	Use of armed and armoured military aircraft. Specific risks are addressed by Joint Helicopter Command. Where possible the aircraft flies tactically or at an altitude higher than enemy munitions can reach. Team wears full category 3 personal protective equipment, including ballistic helmet, eyewear, and body armor.
Aircraft noise	Team is issued personal role radios cleared for use in flight, with noise cancelling headsets. Hand signals used as backup.
Aircraft movement	Predeployment training includes training in flight on CH-47 aircraft. Equipment is secured. Good communication with the pilots.
Extremes of temperature	Equipment is ruggedized. Patient warming with Blizzard Blanket* and fluids warmed with enFlow.† Awareness of heat injury risk. Drugs and equipments such as pediatric endotracheal tubes that soften in high temperatures are kept in a cool box.
Darkness	Team is equipped with night vision equipment and blue filtered torches. Limitations of these are understood. All monitoring equipment is visible in darkness (with LEDs or backlit screens). Bags are secured in standard way to facilitate locating equipment.
Multiple taskings	Missions may change in flight. Plenty of backup equipment is stored onboard the aircraft.

*Blizzard Protection Systems Ltd, Bethesda, Gwynedd, UK
†Vital Signs Inc, Totawa, NJ
LED: light-emitting diode

Indications for Rapid Sequence Induction

The decision to perform RSI on a patient must be made quickly by the flight doctor and communicated to the team. Ideally different clinicians and different teams would make similar decisions; however, some variability in decision-making is inevitable. Indications for intubation are similar, but not identical, to those used by civilian HEMS teams (Table 3-3). (Note that "crash" intubation of patients in cardiac arrest is a separate indication whereby intubation is performed in order to assist resuscitation. Drugs may not be required, so it is not considered part of RSI.) Two patterns of injury stand out:

TABLE 3-3

INDICATIONS FOR RAPID SEQUENCE INDUCTION BY MEDICAL EMERGENCY RESPONSE TEAMS

Indication	Discussion
Loss of airway, anticipated loss of the airway	Frank loss of airway or anticipated loss of airway, eg, in airway burns or catastrophic maxillofacial bleeding.
Respiratory failure	Assisting ventilation, eg, with flail chest or multiple penetrating chest injuries. However, converting a patient with chest injuries from negative to positive pressure ventilation is likely to cause further problems. Converting a pneumothorax to a tension pneumothorax must be anticipated.
Hemorrhage	Catastrophic hemorrhage itself is not a direct indication; however, these patients may have a diminished level of consciousness in addition to needing intubation and ventilation to reduce the physiological demands of shock. There are frequently concurrent indications for RSI.
Head injury	Blunt or penetrating head injury with reduced or falling GCS. Maintaining oxygenation, normocapnia, and blood pressure are a priority.
Humanitarian issues	The procedures required to stabilize a severely injured trauma patient such as tourniquets, IO access, and pelvic binding are frequently themselves distressing and painful. Performing RSI allows these to be carried out by the team rapidly without fear of causing the patient more pain. The levels of analgesia required for these patients may well require the airway to be secured.

GCS: Glasgow coma scale
IO: intraosseous
RSI: rapid sequence induction

1. **Traumatic amputee**. When improvised explosive devices detonate underneath or close to dismounted service members and result in double or triple limb amputation, patients are usually in shock, have multiple injuries including pelvic and spinal column disruption, and may be suffering from concomitant head injury. RSI is often necessary because ventilator compromise frequently is associated with massive polytrauma from blast.
2. **Penetrating head trauma**. Penetrating injury to the head from gunshot wound or high velocity shrapnel may well require early intubation. Any reduction in Glasgow coma scale alerts the team to a potential deterioration; the outcome from head injury is improved if falls in oxygenation or blood pressure are avoided. In addition, transfer times may be prolonged if the patient requires neurosurgical intervention. In these cases the patient may be flown directly to the neurosurgeon, who may be at a location further away.

Drugs Used With Intubation and Ventilation

The flight medical officer prepares drugs at the beginning of each shift. Syringes are drawn up in standard fashion, in standard concentrations, and are labelled. This time-saving measure allows the immediate administration of anesthetic agents when intravascular access has been gained on the casualty and reduces the risk of drug administration errors. Unused drugs must be wasted after 24 hours without use due to the risk of bacterial contamination. Standard MERT RSI is as follows:

- Ketamine induction, 1–2 mg/kg.
- Succinylcholine for muscular paralysis, 1.5–2.0 mg/kg.
- Continuation of anesthesia with aliquots of 1–2 mg morphine and 1–2 mg midazolam or ketamine, depending on the patient's physiology.
- Extension of paralysis with nondepolarizing neuromuscular blocking agent. Vecuronium 0.1 mg/kg has the advantage of not requiring refrigeration.

Ketamine is the main choice of induction agent. Ketamine has several applications and may be used for analgesia, procedural sedation, or induction of anesthesia. Many providers consider ketamine the first choice for trauma analgesia because of favorable properties compared to morphine. The advantage of ketamine for trauma induction is its relative cardiovascular stability: blood pressure does not fall on administration, which is considered beneficial in a patient population frequently presenting with hemorrhagic shock. Onset of anesthesia is relatively fast, and ketamine has a wide therapeutic-toxic range. Disadvantages of using ketamine include its chronotropic effect and stimulation of lacrimation and salivation, all of which may make the depth of anesthesia difficult to judge.

Succinylcholine remains the initial paralyzing agent of choice. There is a strong argument that in the airway compromised trauma patient population there is no option to discontinue induction and to wake the patient up in the event of a failed intubation. Therefore, longer acting neuromuscular blocking agents can be considered an appropriate choice in this environment.

Morphine and midazolam are useful to maintain anesthesia in the absence of gaseous anesthetic agents that would be impractical and dangerous in a combat environment. Morphine doubles as an analgesic agent for more minor casualties. Midazolam has a direct hypotensive effect, and its use should be carefully titrated to resuscitation status. However, this combination represents one of the most dangerous combinations on the battlefield and should be used with great care and only when one-on-one caregiver-to-patient care situations exist.

Fentanyl is a useful adjunct. It is used to mitigate the effect of laryngoscopy in patients with head injury and is a very effective analgesic for patients who are awake. Onset of action is fast and it can be administered nasally using a MAD Nasal (LMA, San Diego, CA) mucosal atomization device when needed.

Intubation

Improving the first-attempt intubation success rate is all important (Tables 3-4 and 3-5). To this end the most experienced practitioner (the flight doctor) performs the intubation, assisted by the flight nurse, who is trained in the drugs, equipment, and monitoring required to safely administer anesthesia. The patient is preoxygenated using 15 L/min via Hudson mask (Hudson RCI/Teleflex, Research Triangle Park, NC). Positive pressure ventilation during the preoxygenation phase (using the bag-valve-mask technique) is avoided whenever the patient is making respiratory effort. This avoids gastric insufflation with the consequent risk of vomiting and diaphragmatic splinting. Many patients will already have been administered high flow oxygen before loading onto the airframe.

Confirmation of Endotracheal Tube Position

Visualization of the endotracheal tube passing through the cords is the most obvious confirmation of successful placement. Visualization alone is not enough, however; confirmation of end-tidal CO_2 is mandatory. Several devices are available to provide redundancy (Table 3-6).

The flight nurse assists the flight doctor in applying monitoring equipment and measuring end-tidal CO_2. Once both are satisfied with the placement of the

TABLE 3-4

IMPROVING THE SUCCESS RATE OF INTUBATION

Factor	Best Case
Experience	Flight doctors are already experienced in prehospital anesthesia.
Training	All team members go through role-specific predeployment training. Training continues in theater.
Patient population	Generally fit service members; however, civilian casualties provide increased risk for difficult intubation.
Positioning	The flight doctor has space to move and will generally intubate in the lying or kneeling position.
Equipment	All prehospital intubations are carried out using either a stylet or gum-elastic bougie technique. Bougie intubation requires use of an assistant. Suction is on-hand.
Drugs	Unless cardiac arrest has been diagnosed, success rates are improved by consistent use of drugs to assist intubation.

TABLE 3-5

INTUBATION SUCCESS RATES FOR MEDICAL EMERGENCY RESPONSE TEAMS

	Civilian UK Emergency Department[1]	London HEMS[2]	MERT[3]
Mallampati Grade I or II view	92%	81%	96%
1st attempt	87.7%	87.5%	94.4%
1st or 2nd attempt	Data not available	97.8%	98.8%

HEMS: Helicopter Emergency Medical System
MERT: Medical Emergency Response Team
UK: United Kingdom
(1) Graham CA, Beard D, Oglesby AJ, et al. Rapid sequence intubation in Scottish urban emergency departments. *Emerg Med J.* 2003;20:3–5. (2) 11. Harris T, Lockey D. Success in physician prehospital rapid sequence intubation: what is the effect of base specialty and length of anaesthetic training? *Emerg Med J.* 2011;28:225–229. (3) Kehoe A, Jones A, Marcus S, et al. Current controversies in military pre-hospital critical care. *J R Army Med Corps*. 2011;157(3 Suppl 1):305–309.

TABLE 3-6

METHODS OF MEASURING END-TIDAL CO_2

Method	Description
Colorimetric capnography	Changes color within a few breaths, useful to confirm ETT placement in the initial stage and can then be discarded to remove dead space from the circuit.
EMMA* Emergency Capnometer	Lightweight device connecting in-line with the ETT. Measures CO_2 and displays a useful visible LED readout.
Side-stream or in-line capnography	The monitors used by MERT can display end-tidal CO_2 capnography. They are the gold standard and should be used on all intubated patients. CO_2 waveform provides useful information during resuscitation.

*Masimo, Danderyd, Sweden
ETT: endotracheal tube
LED: light-emitting diode
MERT: Medical Emergency Response Team

Figure 3-4. Thomas Tube Holder in use.
Thomas Tube Holder is a copyright of Laerdal Medical. Photo used with permission from Laerdal Medical. All rights reserved. Thomas Tube Holder is a copyright of Laerdal Medical. Photo used with permission from Laerdal Medical. All rights reserved.

endotracheal tube, the laryngoscope can be withdrawn from the patient's mouth and the tube secured, preferably using a Thomas Tube Holder (Laerdal Medical, Wappingers Falls, NY; Figure 3-4). A cervical collar and head blocks are usually applied, both to secure the cervical spine and to help prevent the endotracheal tube from becoming dislodged.

Failed Intubation Drills

Any indication that the endotracheal tube has been incorrectly placed mandates adherence to failed intubation drills. Extubation and a second attempt to intubate may be possible provided the patient's saturations can be maintained by bag-valve-mask ventilation. Rescue devices available include alternative laryngoscopy aids and supraglottic devices. The final common pathway is to perform a surgical airway. This technique must be practiced as a drill by the team so that when it does occur, the chances of success, and delivering a live patient to hospital, are maximized.

Ventilation

Aboard aircraft ventilation is usually achieved using a transport-style ventilator (Oxylog 1000, Draeger Medical, Hemel Hempstead, UK), which provides a constant minute volume with stability in end-tidal CO_2, a benefit in head-injured patients. The device also frees the doctor to perform other tasks. Some practitioners prefer to maintain a "feel" of the lung dynamics by continuous manual ventilation. A rise in airway pressures may herald the development of a tension pneumothorax.

Thoracostomy

Pneumothoraces are common in blast injury. Converting a patient from self-ventilation (negative-pressure ventilation) to positive-pressure ventilation is a high-risk strategy. A small pneumothorax may become larger or may become a tension pneumothorax. The entire team must be aware of this possibility at the moment of intubation and be ready to perform thoracostomies, bilateral if necessary, to relieve tension pneumothorax. During short transfers thoracostomies alone will be adequate, provided they are re-fingered when necessary. During longer transfers or to maintain the patency of the incision, battlefield chest drains can be inserted. Note that in self-ventilating patients, chest drains must be used.

Resuscitation

MERT has pioneered the administration of blood products in flight since 2008. Crystalloid fluid resuscitation is now rarely used in the context of major hemorrhage; rather, the focus is on replacing blood loss with blood products. This minimizes dilutional

coagulopathy and reduces pathophysiological triggers for the acute coagulopathy of trauma by ensuring adequate perfusion and maintaining tissue oxygen delivery. The triggers for transfusion are:

- absence of a radial pulse,
- tachycardia over 120 bpm,
- anticipation of massive transfusion, or
- pattern of injuries (two or more or more amputations or penetrating torso trauma associated with changes in the vital signs).

Hypotensive Resuscitation

Traditional prehospital guidelines were to maintain a radial pulse or systolic blood pressure of 90 mm Hg until the patient reached the surgeon. The reasoning behind this method was to achieve a balance between maintaining organ perfusion and allowing a clot to form on the injured area. However, traumatic hemorrhage models involving blast have shown that maintaining these conditions for over an hour is detrimental, and casualty survival diminishes significantly with increasing acidosis.[10]

Novel Hybrid Resuscitation

Novel hybrid resuscitation has been shown to improve survival of traumatic blast casualties. It entails a goal of hypotensive resuscitation initially—achieving a radial pulse or a systolic blood pressure of 90 mm Hg—followed by the reestablishment of normotension thereafter. Exceptions to hypotensive resuscitation include head injury and pregnancy. The hypotensive phase of this resuscitation approach should last no more than 1 hour from time of injury. If at any time the patient begins to show signs of metabolic deterioration, the hypotensive resuscitation protocol should be stopped.

Blood Product Administration

Blood products are carefully regulated in keeping with best practices. Blood products are kept in specially designed "Golden Hour" boxes to maintain integrity of temperature control; the boxes may not be opened until the blood products are needed. The transfusion is administered in a 1:1 ratio of O negative packed red cells and AB positive fresh frozen plasma through the enFlow fluid warming device. The "lethal triad" of hypothermia, acidosis, and coagulopathy[10] is actively avoided by anticipating hypothermia and actively warming, minimizing acidosis by reestablishing peripheral perfusion, and correcting coagulopathy and fibrinolysis. Because fibrinolysis is common, tranexamic acid 1g IV/IO is administered during the transfusion. The administration of blood products inevitably results in hyperkalemia and hypocalcemia, so 10 mL of 10% calcium chloride is administered after the fourth unit of blood. Early results from a MERT study with an animal model of ballistic injury have shown that the transfusion of early prehospital blood products minimizes the subsequent deterioration in physiology and in particular the level of acute coagulation of trauma.

THE FUTURE

Because of the success of far forward critical care in Afghanistan in improving survival following combat trauma, it will likely remain a component of medical support for future operations. Although operations currently enjoy air superiority, a relative surplus of support helicopters, and minimal ground-to-air threat, these conditions are unlikely to be the case indefinitely. The use of ground transport is a distinct probability, along with prepositioning of critical care assets both for specific operations and for at-risk populations. The secondary transfer of critically injured service members is another likely scenario, either by air or ground, utilizing a prehospital critical care team. The lack of availability of larger support helicopters may change the doctrine to provide a limited "stay-and-play" capability, stabilizing casualties prior to evacuation on smaller airframes or other forms of transport. Conversely, specialized airframes would enable the use of equipment such as rapid infusion systems that is currently available only in Role 3 facilities.

Interventions available to military prehospital practitioners are likely to evolve further. Early use of fibrinogen replacements, proximal control of the aorta via the chest,[9] and endovascular balloon occlusion of the aortic are all future possibilities. The continuing evolution of resuscitation protocols is likely to see a greater breadth of other drugs and novel technologies employed, such as synthetic oxygen-carrying fluids or blood product storage systems. The management of trauma patients in cardiac arrest is an area that requires further work. The success of post-arrest cooling in the civilian arena and work done on emergency preservation in resuscitation (rapid cooling of the brain) may open up yet further improvements in the rate of survival.

As standard operating procedures develop, the type of patient and situation in which primary transfer to specialist facilities such as neurosurgery is feasible will become better established, resulting in decision protocols for clinicians and more consistent care for casualties.

SUMMARY

Medical evacuation from the battlefield has evolved and improved over the past 8 years. Both UK and US assets have played a significant part in the survival of injured military personnel, and the lessons learned must be carried forward into the next operational arena. The UK facility, with its greater capability for advanced resuscitation, has shown benefits for patient survivability; however, the US assets have a crucial place within the battlefield for patient transfer and evacuation, particularly when the tactical situation dictates speed and maneuverability. Each MEDEVAC asset can learn from the others, and the continued working relationship among them will aid in the ongoing improvement of military prehospital casualty care.

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Chapter 4

CONDUCTING A COMPLEX TRAUMA ANESTHETIC

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''Train hard, fight easy.''
—Attributed to General Aleksandr Vasil'evich Suvorov (1730–1800)

INTRODUCTION

Over the past 11 years of conflict in Iraq and Afghanistan, the severity of injuries has increased, yet combat fatality rates are declining. This is due in part to the improved capability of frontline medical personnel and evacuation assets. In both the civilian and combat casualty care arenas, there has been improved application of damage control techniques, more effective rewarming, and more aggressive correction of coagulopathy.[1]

A unique pattern of injuries has emerged these conflicts termed "dismounted complex blast injury." This injury pattern consists of traumatic lower extremity amputations, penetrating abdominal/pelvic trauma, and urogenital injuries. These patients often arrive in hemorrhagic shock, requiring emergent damage control surgery and resuscitation.[2] These casualties have significant injury severity scores,[3] including injuries requiring a massive transfusion following hypovolemia.The devastating clinical injury scenarios that have characterized the conflicts in Afghanistan and Iraq underline the need for complex anesthesia capabilities on the modern battlefield.

The anesthetic management of military trauma is directed at restoring physiology by reversing or minimizing the "lethal triad" of acidosis, hypothermia, and coagulopathy. Coagulopathy, independent of injury severity score, surgical intervention, or blood product transfusion, is strongly associated with early death following major trauma. Abnormal coagulation, reflected by an increased prothrombin time and partial thromboplastin time, is present in 25% of trauma patients with major injuries upon presentation. Coagulation is an integral part of the inflammatory system, and activation results in systemic inflammatory response syndrome and increased susceptibility to infections and sepsis.[4] Avoiding the lethal triad requires a thorough understanding of damage control philosophy,[5,6] whose clinical features are summarized in Exhibit 4-1.

In the majority of these patients the immediate surgery is limited to definitive control of hemorrhage, wound debridement, and prevention or limitation of contamination.[7] Only in selected cases will definitive restoration of function be undertaken. In such procedures the patient must be physiologically stable and deemed healthy enough to sustain a prolonged intraoperative course. These are conditions better attempted following repatriation from the deployed setting and will not be discussed further here.

DECISION-MAKING

In the early stages of casualty reception, careful time sequencing and communication are critical to success. The anesthetist is an integral member of the emergency department team. Casualties can be classified into those who are physiologically stable and those who are unstable. The primary decision in respect of the unstable group is whether they require immediate surgery. If immediate transfer to the operating room is required, current practice in the NATO hospital facility in Afghanistan is for the emergency department team to escort the patient there. "Control" is handed over to the anesthesia/surgical team only when a definitive airway and intravenous access have been established and an initial surgical plan agreed upon. Those casualties who do not require immediate surgery will have their injuries more fully characterized with a computerized tomography scan.

CLINICAL MANAGEMENT

Airway management is the first critical step in caring for the severely injured combat casualty. This may present unique challenges for the anesthesiologist. Vascular access will often be performed concurrently, a procedure that has popularized the use of large-bore central venous access. Initially, intraosseous access may be required, especially in the prehospital phase. Early attention to ventilation strategies to promote oxygenation while limiting potential lung damage requires the early involvement of established intensive care principles, especially in respect of blast casualties. Appropriate blood product resuscitation in a targeted manner, preferably guided by thromboelastographic testing technology at Role 3, and a thorough understanding of the pathophysiology of coagulopathy, is necessary to ensure optimum clinical outcomes while minimizing the risks of transfusion.

As resuscitation progresses, interactions among the surgical, anesthetic, and nursing teams become increasingly complex. Maintaining situational aware-

EXHIBIT 4-1

CLINICAL FEATURES OF DAMAGE CONTROL PHILOSOPHY

- Anesthesia and surgery are contemporary; surgery is part of resuscitation.
- Physiological control is directed at restoration of tissue oxygenation.
- Management of the "lethal triad" includes the use of massive transfusion protocols.
- Surgical episodes may be abbreviated depending upon the patient's physiological status.

EXHIBIT 4-2

THE STAGES OF A COMPLEX MILITARY ANESTHETIC

1. Secure or confirm a definitive airway.
2. Establish appropriate intravenous access.
3. Obtain laboratory investigations.
4. Control ventilation, respecting the possibility of lung injury.
5. Restore tissue oxygen delivery by reversal of hypovolemic shock.
6. Treat coagulopathy.
7. Ensure a full secondary survey is completed.
8. Maintain good CRM principles throughout.
9. Initiate appropriate postoperative care including attention to pain management.

CRM: Crew Resource Management

ness is paramount to ensure good Crew Resource Management (CRM) practice throughout. In particular, the secondary survey including imaging identifies all significant injuries to ensure long-term morbidity is minimized. Postoperative care starts in the operating room and includes the early involvement of intensive care, acute pain management, and aeromedical evacuation teams. The key clinical steps for anesthesia are summarized in Exhibit 4-2.

TRAINING FOR WAR

The workload imposed by the severely injured military patient is intense and demanding of logistics both within and outside the operating theater. The United Kingdom (UK) Defence Medical Services specifically trains and exercises their operational and CRM capabilities prior to deployment.[8] The critically injured casualty will often require the attention of two anesthetists while a team of as many as ten surgeons operate. Anesthetist and surgeon must be in constant dialogue so each is aware of the evolving clinical plan

EXHIBIT 4-3

THE DEPLOYED MILITARY ANESTHESIA SYSTEM

- Appropriately trained and experienced anesthetists, and allied anesthesia support staff.
- Equipment "fit for purpose," including near-patient testing such as thromboelastometry, blood gas analysis, and ultrasonography for regional anesthesia.
- Large and small team training, eg, CRM and the horizontal team approach.
- Integrated laboratory support particularly for transfusion and blood products, including the facility for a donor panel to collect fresh whole blood and platelets.
- Coordinated intensive care to manage staged damage control procedures and for stabilization prior to aeromedical evacuation.
- An acute pain service.
- Facilities for data collection and audit of all anesthetic activity.
- Academic support to promote evidence-based initiatives for current and future conflicts.

CRM: Crew Resource Management

and the patient's temporal physiology. Clinical teams and hospital management must communicate effectively to ensure that overall situational awareness is maintained. There may be further incoming casualties as well as a need to consider the onward movement of casualties already admitted.

Military trauma teams are consultant lead on a 24-hour basis (in contrast, only 3% of UK's National Health Service hospitals offer this level of care); however, clinical care is delivered by a team of all ranks and specialties, and constant communication and situational awareness is emphasized. Exhibit 4-3 lists the essential elements of the deployed military anesthesia system. To improve the system for successful deployed clinical teams, there must be an active ongoing collection of data and audit. The results are processed by academic clinicians and fed back into the system to improve current protocols and inform future generations.

CONCLUSION

The technical details of the components of damage control are discussed in depth by a series of authors in the chapters following this introduction. As this chapter's title implies, a successful outcome relies upon orchestration of the parts. In comparison to many civilian trauma care systems,[9] this is a requirement that historically the military manages extremely well.

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Chapter 5

VASCULAR ACCESS AND INFUSION DEVICES FOR COMBAT ANESTHESIA

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INTRODUCTION

Establishing vascular access is of paramount importance for the successful resuscitation of the trauma patient. It is essential for administering intravenous (IV) fluids; it is the optimal route for administering anesthetic and other drugs; and it may be a useful adjunct for measuring physiological parameters both during primary resuscitation and subsequently in the postoperative period.

It is likely that some form of access will have been established prior to the first interaction with the anesthesia provider. Indeed, in civilian practice prehospital providers successfully establish IV access in approximately 90% of cases.[1] However, the anesthesia provider must be familiar with the entire spectrum of IV techniques in order to manage patients with either absent or suboptimal vascular access. Any IV access lines in situ on the patient's arrival must be checked thoroughly for function and adequate flow, and a plan made for the establishment of further access if required (Exhibit 5-1).

The patient's physiological status will dictate the urgency of the procedure and may direct the clinician toward one particular technique over another. Establishing or upgrading vascular access may be concurrent with other aspects of resuscitation, for example, airway control or surgical hemostasis. IV access should be a priority task assigned to a specified individual or individuals.[2,3]

The most suitable site and technique used will depend on several factors, including pattern of injury, the patient's current physiological condition, and the ability to physically access the proposed insertion site.

There must be a patent circulatory system proximal to the site of insertion. In the case of abdominal and thoracic trauma, IV access must be obtained in a tributary of the superior vena cava (SVC) because veins below the injury may not be in continuity with the central circulation as a result of inferior vena cava (IVC) injury. Insertion of IV devices in injured limbs should be avoided because there is no guarantee that infused fluid and drugs will reach the central circulation. Furthermore, extravasation of fluid may result in a compartment syndrome, thus exacerbating a preexisting injury.

CATHETER AND CANNULA SIZES

Currently two systems are in common use to describe the size of cannulae that may be used to secure IV access:

1. Smaller diameter cannulae tend to be expressed by a number based on either the US Birmingham wire gauge or the British Standard wire gauge. In this system as the diameter of the device increases the gauge number (G or Ga) decreases. The relationship between the gauge of the cannula and its actual measured diameter does not follow a linear relationship but rather is standardized by convention.
2. The diameter of larger cannulae tends to be expressed by reference to the French gauge (Fr or F). In this notation 1 Fr equals one third of a millimeter; therefore, the diameter in millimeters can be calculated by dividing the gauge size by 3. It follows that as the number increases, so does the diameter.[4]

PHYSICS OF FLOW

Traditional teaching is that flow in an IV cannula is governed by the Hagen-Poiseuille law, which relates flow (Q) to the pressure gradient (ΔP), viscosity of infusate (ρ), and the length (l) and radius (r) of the catheter. In this relationship the primary determinant of flow is the radius, such that doubling the radius increases flow 16-fold.

PERCUTANEOUS CENTRAL VENOUS ACCESS

It may be difficult to establish early peripheral IV access in the critically hypovolemic patient with a collapsed circulation. In these cases early recourse to central access should be made. The ideal for managing massive hemorrhage in adults has been described as 8 Fr central access.[3] Large-bore central venous lines allow rapid infusion of resuscitation fluid. Therefore, once established, direct access to the central venous system provides the most effective route for resuscitating the profoundly hypovolemic patient.[5,6] Central access also allows the delivery of potent vasoactive drugs and irritating or hypertonic solutions, as well as routine blood sampling.

Central venous access has been utilized extensively since the 1950s, when Swedish radiologist Sven Ivar Seldinger described the use of a flexible metal leader

EXHIBIT 5-1

VASCULAR ACCESS TASKS TO BE COMPLETED ON ARRIVAL OF PATIENT

- Identify vascular access devices in situ.
- For each device assess and record:
  - o Site
  - o Flow characteristics
  - o Security
  - o Current infusions
- Plan for further vascular access as required:
  - o Peripheral
  - o Central
- Discuss with operating surgeons whether cutdowns or direct atrial cannulation are required.
- Plan for arterial access if appropriate.

to exchange a needle for a catheter during arteriography.[7] However, early central venous catheters were long, small in diameter, and with a high resistance to flow, which proved ineffective in the volume resuscitation required in major trauma. Interest in their use in trauma increased during the 1980s, when it was recognized that percutaneous pulmonary artery catheter introducers could be used to deliver rapid volume replacement.[8,9]

Initial worries about the safety of central access in the trauma setting have been unfounded. Central access for the initial resuscitation of trauma patients has been demonstrated to be safe and quick to establish. Complications can and do occur with any route chosen; however, it is unclear if complication rates are increased in the trauma population.[10,11] It is recognized that complication rates are inversely related to the experience of the operator.[10,12] Therefore, central vascular access should be assigned to the most experienced individual. The central venous circulation is commonly accessed via the subclavian (SCV), internal jugular (IJV), and femoral veins.

Subclavian Vein

SCV access with a large-bore catheter has been described as the "gold standard" for fluid resuscitation in the military setting.[13] Venous access via the SCV is a useful technique in major trauma and cardiopulmonary resuscitation. It has a relatively constant anatomic position behind the clavicle, a large diameter of 12 to 25 mm, and has been shown to remain open even in critical hypovolemia.[14] As a tributary of the superior vena cava, the SCV is regarded by some as the method of choice in major abdominal trauma.[13] The area of insertion may remain available for line insertion during emergency airway management, cervical spine immobilization and resuscitative surgery.[15]

Patient Positioning

Optimal positioning of the patient may be compromised by the clinical situation. Use of a moderate Trendelenberg position may be useful.[16] With regard to the infraclavicular approach, anatomical studies have shown that arching the shoulders or turning the head may reduce the diameter of the vessel; however, these positions may be useful to reduce the incidence of arterial puncture. Caudal traction on the arm may facilitate needle entry to the vein.[17]

Techniques

An infraclavicular approach to the SCV was first described by Aubaniac in 1952.[18] The most often used technique describes the point of needle puncture as occurring just caudal to the junction of the medial and middle thirds of the clavicle. The needle is directed under the clavicle toward the sternal notch. To reduce the risk of pneumothorax, the needle should not be allowed to angulate below the coronal plane.[19]

Access to the SCV via a supraclavicular approach was described by Yoffa in 1965.[20,21] In this approach the needle is inserted 1 cm cephalad and medial to the midpoint of the clavicle. The needle should be directed to bisect the angle between the sternocleidomastoid muscle (SCM) and the clavicle, and be angulated 10° to 20° anterior to the coronal plane. This approach has been shown to be safe, with complication rates comparable to other methods of central access.[22,23]

Another typical method is a modified Seldinger technique. If a J-tip metal leader wire is used, the tip should be directed in a caudal direction to reduce the incidence of passing the wire and subsequent catheter into the IJV.[24]

The use of tandem large-bore subclavian catheters has also been described. In this technique the two guide wires are inserted into the chosen vessel before the catheters are inserted.[25]

Internal Jugular Vein

Catheterization of the IJV is a popular choice for central venous access. It is readily achieved with the operator standing at the patient's head; therefore, it may be the most readily accessible site once surgery has commenced. This technique has been readily

adapted to ultrasound guidance. However, it also has several disadvantages. It may be difficult to achieve in patients with cervical spine immobilization as well as with concurrent advanced airway management. If IJV cannulation is undertaken during the management of a patient with major chest injury, as with SCV cannulation, it is advisable to perform the procedure ipsilateral to the injury.

Patient Positioning

The patient is positioned supine with the head rotated 30° to 40° degrees away from the side of puncture. Most commonly the right side is the site of first choice, which is usually more convenient for right-handed operators. The route from the right IJV to the SVC also follows a more linear course, making passage of the catheter more straightforward. Anatomical considerations may make complications more common with a left-sided approach.

A roll may be placed anywhere under the chest to facilitate extension of the neck. Use of a Trendelenberg position may increase engorgement of the neck veins and facilitate cannulation, although these effects must be moderated in the case of acute neurological injury.

Techniques

At least 13 approaches to the IJV have been described. They can broadly be divided into anterior, central, and posterior approaches, depending on their relationship to the SCM.

The anterior approach has a point of needle insertion at the midpoint of the anterior border of the SCM and aimed towards the junction of the middle and medial thirds of the clavicle. The angle of insertion should be 30° to 45°. It is important in this approach to retract the carotid artery medially to avoid inadvertent carotid puncture.

The most commonly used central approach has a site of puncture at the intersection of the clavicular and sternal heads of the SCM; this should be at the level of the cricoid cartilage. The needle is directed towards the ipsilateral nipple at an angle of 45° to the skin. Entry is made to the vein where it lies lateral to the pulsation of the carotid artery.

The posterior approach to the IJV has a needle puncture site at the lateral border of the SCM at the level of the thyroid cartilage or the superior border of the external jugular vein that crosses the muscle belly at this point. The needle is directed toward the contralateral nipple. At this point the IJV lies anterior to the carotid artery.

Femoral Vein

Cannulation of the inferior vena cava via the femoral vein is a well-documented route for fluid resuscitation in trauma. A technique for accessing the vein was first described in 1949.[26] Femoral cannulation is possible in trauma patients with neck immobilization and may be easily established in patients undergoing cardiopulmonary resuscitation or advanced airway management. This route may be unsuitable, however, in cases of severe lower limb injury and abdominal trauma when the continuity of the femoral vessels and the inferior cava may be questionable.[27]

Patient Positioning

Most commonly the patient is positioned supine with the legs slightly abducted. The chance of successful cannulation may be improved by externally rotating the leg.[28]

Techniques

When cannulated by a landmark technique, the femoral vein is located medial to the pulsation of the femoral artery as it passes from under the inguinal ligament. The point of needle insertion should be such that the needle does not pass superior to the inguinal ligament; typically insertion is made a few centimeters distal to the junction of the middle and medial third of the ligament.[29] The performance of a Valsalva maneuver can double the cross sectional area of the vein and may make location of the vessel easier.[30]

Ultrasound-Guided Central Venous Access

The use of ultrasound in central venous access has gained great popularity in recent years in anesthesia practice throughout the United States. In 2001 the Agency for Healthcare Research and Quality recommended the use of ultrasound for the placement of central venous catheters as one of eleven practices to improve patient care.[31] Many studies have favored the use of ultrasound-guided techniques in comparison to more traditional landmark techniques. A metaanalysis by Hind et al[32] revealed that in 18 trials comparing two-dimensional ultrasound-guided techniques with landmark methods, ultrasound-guided cannulation of the jugular vein in adults was associated with a significantly lower failure rate both overall and on the first attempt. The same study found limited evidence to support ultrasound guidance in placing femoral and subclavian catheters in adults. Additionally, Doppler-guided cannulation of the IJV was more successful than the landmark method.[32]

Extrapolation of such findings to trauma must be guarded given the fluid nature of the trauma experience and the time required to perform an ultrasound-guided technique. Additionally, the often limited space in the deployed field environment argues against ultrasound-guided techniques, especially when coupled with the goals of damage control surgery. Currently, trauma central venous access is conducted rapidly, often simultaneous with damage control surgery, and is done to facilitate the massive transfusion process. The traditional landmark technique for accessing the subclavian vein followed by the IJV is the current practice in combat trauma anesthesiology. Despite this practice, there may be a role for ultrasound-guided access of the internal jugular vein if theater CSHs continue to receive two-dimensional ultrasound capabilities.

Complications of Central Venous Access

Pneumothorax is the most common complication of central venous access. Several anatomical considerations related to this complication should be used to help guide the operator. The dome of the pleura is higher in the left hemithorax than the right, and both may rise higher during positive pressure ventilation. Care should therefore be taken to minimize tidal volumes during SCV puncture. It is unclear whether this complication is more common with IJV or SCV catheter insertion.[33] Pneumothoraces may develop quickly or many hours after line insertion[34] and will most likely require the insertion of a chest drain. Some authors suggest it is prudent to insert IJV and SCV lines ipsilateral to preexisting chest injuries.[13,15]

Vascular injuries may occur with all access routes. Arterial puncture is more common with IJV (the carotid artery is at risk) than SCV cannulation. Carotid puncture is usually well tolerated; however, it is recognized that the SCV artery is difficult to compress behind the clavicle, so an injury may be more likely to be clinically significant. A hemothorax may develop after arterial puncture in these areas. Pressure to control a developing carotid hematoma has been associated with cerebrovascular accidents and death. The most severe complications of femoral vein access arise from inadvertent femoral or iliac artery puncture. Above the inguinal ligament, arterial puncture may result in a hemoperitoneum or retroperitoneal hemorrhage (such a complication may be more likely in severe hypovolemia in the absence of a palpable femoral pulse).[27] If either complication is followed by coagulopathy, the result can be severe. Other vascular injuries may involve the aorta and any of the great vessels; injuries to the walls of the cardiac chambers. Overall, femoral access carries the highest complication rate.[35] Other complications of central venous access include line malposition, air embolism, cardiac dysrhythmias, chylothorax, and peripheral plexus or nerve damage. With all routes, the strongest predictor of complications is the number of insertion attempts.[36]

INTRAOSSEOUS ACCESS

Access to the circulation via the bone marrow was first described in the 1920s by Drinker and Doan and the technique gained widespread support during the Second World War. Subsequently, with the development of more reliable and robust IV cannulae, it fell out of favor. However, the technique came back into use in the 1980s, especially in the pediatric population. Intraosseous (IO) vascular access is increasingly recognized as a valuable tool in the initial resuscitation of combat-injured patients.[13,37–39]

Several commercial kits are available to achieve IO access quickly and safely. These can be grouped into manual needles, impact-driven needles, and power-driven needles. It is essential that operators be familiar with the particular devices they may encounter in their practice.

Potential sites for IO access, dictated by the device to be used, include sternum, proximal and distal tibia, humeral head, and iliac crest. Other sites that have been described include the medial clavicle and calcaneum[40]; however, these are used much less frequently. Correct placement may be indicated by the aspiration of bone marrow and the ability to infuse fluid without subcutaneous extravasation. Extra care should be taken with sternal insertion because of the potential for rare complications including mediastinal injury and pneumothorax.

IO access has been shown to be significantly quicker to establish than central venous access.[41] However, it is recognized that maximum infusion rates are not comparable and that fluids must be infused under pressure. Most currently available devices use a 15-G cannula and provide flow rates comparable to a 20-G peripheral cannula.[41]

It is possible to infuse all commonly used anesthetic drugs via the IO route, and bioequivalence with the direct IV route has been demonstrated.[42,43] In addition to drugs, blood, crystalloids, and artificial colloid-containing solutions may be infused. Blood samples drawn from an IO needle demonstrate hemoglobin, electrolyte, and blood gas analysis results comparable to peripheral venous blood.[44,45]

Complications of IO access are rare (the overall rate is less than 1%[43]) and can be mitigated by familiarity with equipment. Patients commonly experience pain on infusion, which can be relieved by local anesthetic administered into the medullary cavity.[46] Fluid extravasation may occur with an improperly sited cannula, which can cause a compartment syndrome.[47] Osteomyelitis has been reported, with an incidence of approximately 0.6%, usually associated with prolonged infusions.[43] Iatrogenic fractures have been reported in children. A rare reported complication is retention of the cannula, necessitating surgical removal.[48] Long-term follow-up has shown that bone deformity due to needle insertion is not a problem.[49]

PERIPHERAL VENOUS CUTDOWN

Peripheral venous cutdown had been a popular route for fluid resuscitation in critically ill patients with shock since the first reported use in 1940.[50] However, the technique declined in popularity with the increased use and familiarity of the Seldinger technique for percutaneous access. Although setting up and starting IV resuscitation via venous cutdown techniques has been shown to take significantly longer to infuse fluids with by percutaneous femoral vein cannulation,[51] a cutdown remains a viable option in cases where percutaneous access proves impossible.

Popular sites for cutdown to be performed are the great saphenous vein, in the groin and distally at the ankle, and the basilic vein proximal to the elbow. At these sites it is usually possible to insert an 8.5-Fr catheter. If IV tubing is used, insertion is made easier by shaping the end of the tubing with a 45° bevel.[52] Exposure and cannulation of the great saphenous vein at the groin is the preferred technique. At the groin, the classic technique involves a transverse skin incision through superficial subcutaneous tissue in the proximal thigh.

A technique for rapid access to the femoral vein has been described utilizing a skin incision from a point inferior and lateral to the pubic tubercle, directed toward the medial epicondyle of the femur. Manual distraction of the subcutaneous tissue allows visualization of the vein at the base of the incision.[53] In all cases the vein is then identified, and a venotomy is performed and cannulated using either a modified Seldinger technique or classic technique without a guidewire. The line is then usually secured with sutures.

A contraindication for the technique is trauma to the ipsilateral extremity. Infection at the intended site and anatomical variance are relative contraindications.

The most common complications include cutaneous nerve damage and damage to vascular structures. If vascular structures are disrupted during attempted insertion, the site should be packed and explored in the operating room. Rates of infection rise significantly with duration of infusion, so lines inserted using cutdown techniques should be removed as soon as practical.[52]

DIRECT ATRIAL CANNULATION

Performing an emergency resuscitative thoracotomy may afford a further option for vascular access. Direct infusion of fluid to the heart is possible.[54] A technique has been described utilizing a 16-Fr or larger Foley catheter passed into the right atrial appendage via an atriotomy incision.[55] However, a number of complications may result from this procedure. First, air may be entrained into the cardiac chambers. The small amount of air that may enter at the time of the initial incision is considered to be of no consequence; however, care should be taken to ensure a good seal between the inflated balloon of the catheter and the atriotomy. This seal will also help to limit the leakage of blood from the heart.

A second complication is cardiac distension, which may occur if the technique is utilized in conjunction with proximal aortic occlusion. Manual and visual examination of the contracting heart should be used to guide fluid resuscitation along with other hemodynamic parameters.

ARTERIAL ACCESS

Once the anesthesia team is satisfied with the adequacy of the venous vascular access, access to the arterial system for rapid blood gas measurement and beat-to-beat blood pressure measurement can be conducted. Although venous blood gases may be used in extremis and utilized for base deficit determination for trauma,[56] using arterial blood for analysis is ideal. Arterial blood gas determination of base deficit is the best indicator for determining the adequacy of resuscitation for a combat trauma patient who is receiving a massive blood transfusion. Additionally, beat-to-beat blood pressure measurements are ideal for large-volume trauma resuscitations. The mere presence of systolic pressure variation can provide

real-time information about intravascular volume status during resuscitation.

Again, due to the massive nature of injuries and diffuse injury pattern caused by IEDs, considerable yet rapid consideration must be given to the site for arterial cannulation. A review of the literature published between 1978 and 2001 on the most common arterial cannulation sites (radial, femoral, and axillary) revealed that major complications occurred at a rate of less than 1%, with similar rates for radial, femoral, and axillary arteries.[57] The location of the arterial site during combat anesthesia operations is often dictated more by the location of injury than an individual anesthesia provider's particular preference. Femoral arterial cannulation may be necessary if the soldier has injury to the bilateral upper extremities. Traumatic amputation of the upper extremity is a devastating injury, yet not uncommon because of the high velocity of IED projectiles. In these cases placement of a femoral line by a trauma surgeon would facilitate arterial blood gas analysis as well as rapid preparation for damage control surgery.

The femoral arterial route may be less desirable with intraabdominal or groin-penetrating injuries; in these cases a more traditional route may be preferred. Most anesthesia providers have a high comfort level with the radial arterial site given its use in routine practice. Despite this familiarity, it is sometimes difficult, if not impossible, to obtain the radial artery pulse with traditional palpation techniques and cannulate the radial artery. Often severely traumatized combat patients present with profound tachycardia, hypotension, and peripheral vasoconstriction, which makes palpation of a radial arterial difficult, particularly with systolic blood pressures below 60 mm Hg. Hypothermia and hypovolemic shock may result in peripheral vasoconstriction. This also makes radial arterial access and monitoring difficult. Although the use of ultrasound to obtain a radial arterial line is possible, the equipment is often impractical in the cramped and chaotic field environment.

An attractive alternative to the traditional radial artery site is the cannulation of the axillary artery. Adler was the first to describe the use of the axillary artery for intravascular monitoring in 1973.[58] Obviously this line is precluded in the presence of an upper extremity injury, but it is an invaluable technique for the severely traumatized combat soldier. The axillary vessel has a relatively larger caliber compared to the radial artery, so it is less likely to be affected by vasoconstriction. The more central location of the axillary is also advantageous for more accurate central pressure measurements. Additionally, the axillary arterial line is very durable and is in an ideal position for patient transport to higher levels of care.

The axillary artery is easily accessed with the aid of ultrasound[59]; again, however, the use of ultrasound for vascular access may be limited in the field environment. If ultrasound is unavailable, the axillary artery can easily be accessed from the head of the bed by placing the patient's hand behind the head and flexing the upper extremity at the elbow. By doing this the anesthesiologist can place the axillary line at the same time as the surgeons are performing damage control surgery.

The brachial artery should be cannulated with care during trauma. The brachial artery is an end-artery, and the presence of a vascular access line may result in ischemia of the forearm and hand on the side of arterial cannulation. The axillary artery is not an end-artery and when appropriately managed does not pose the same risk of ischemia as the brachial artery catheter.

EXHIBIT 5-2

POST-ANESTHESIA CARE OF VASCULAR ACCESS DEVICES

- For each device assess:
  - o Site
  - o Flow characteristics
  - o Security
- Reassess circumstances of insertion of each device.
- Plan for removal of devices no longer required.

BEYOND THE FIRST 24 HOURS

IV access lines should be removed as soon as they are no longer needed. Complications relating to duration of insertion, particularly infection, may affect subsequent recovery. Infective complications may be more common than in lines placed electively because they may have been placed in less than ideal circumstances; indeed, some may remain from the prehospital phase. Thorough handover to the next role of care must include discussion of current vascular access devices (Exhibit 5-2).

SUMMARY

In summary, vascular access in combat trauma anesthesia is done in the context of damage control surgery and often with the requirement for massive transfusion. It is critical for the anesthesia team to work in concert. The anesthesia team must attack the lethal triad of acidosis, hypothermia, and coagulopathy by performing targeted intravascular line placement with appropriate escalation to central venous access. This must be done simultaneously with damage control surgery and never delay the combat trauma patient from reaching the operating room. Intravascular access should augment the overarching goal of damage control surgery to stop the bleeding, and at times should be delayed to allow a more hypotensive strategy, especially when vascular injury is suspected. Obtaining good vascular access is a priority task in the early management of the injured. It will facilitate subsequent therapeutic maneuvers and is essential for the optimal resuscitation of the critically injured patient. A sound knowledge of the options available to access the circulation and familiarity with the commonly used equipment will allow anesthesia providers to quickly optimize this aspect of patient management.

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Chapter 6

MANAGING THE AIRWAY

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INTRODUCTION

Since 2000, the incidence of combat face and neck injury has increased in relation to total battle injury compared to the 20th century.[1] Penetrating injuries are the most common cause of combat injuries to the face and neck,[1] resulting in an increased incidence of airway compromise.[2] Penetrating airway injury is unusual in the civilian context, where the bulk of airway injury is blunt trauma due to motor vehicle collisions.[3] Military clinicians rarely manage these types of injuries except when deployed, highlighting the importance of appropriate training.

Penetrating injuries to the face and neck can result from either gunshot wounds or explosive events. Explosive events have become the most common cause in recent years (81%), with gunshot wounds accounting for only 19% of battle injuries to these anatomical areas.[1] Penetrating injuries can result in severe disruption of both soft tissue and bone,[4] and if the airway is disrupted, surgical emphysema may occur, leading to air being trapped under the skin. The airway is relatively superficial throughout the face and neck and, with the exception of the mandible, has no bony protection. Airway embarrassment may result from relatively innocuous wounds because fragments need to travel less than 15 mm through skin to damage the airway, especially in the anterior neck. The airway can also be compromised by blood, secretions, and foreign bodies. The multiple potential approaches to airway management in casualties with penetrating injuries, as well as advantages and disadvantages of each technique, airway devices, team considerations, and suggested guidelines will be discussed in this chapter.

EVIDENCE FOR CURRENT PRACTICE

The anesthetic management of penetrating neck injuries is poorly reported in the literature,[5,6] and manuscripts generally concentrate on surgical management[7] or related case reports.[8] The publications on this type of injury reveal a lack consensus among the anesthetic community,[9] with great variability described in their management.[7] A literature review of papers published on the subject between 1995 and 2010[8] identified 51 relevant papers. Only three of these papers involved military patients, and all were case reports.

Facial Injury

Awake fiberoptic intubation (AFOI),[10–13] rapid-sequence induction (RSI),[14,15] and surgical airways[15,16] have all been described in the airway management of patients with extensive facial injuries. Even in the presence of extensive facial injury, oropharyngeal intubation is generally considered the anesthetic modality of choice in both civilian[10,11] and military clinical series.[8] Large defects should be directly intubated and smaller defects visualized through fiberoptic intubation. Indications for a surgical airway include intraoral hemorrhage and extensive disruption of the mandible or maxilla.

Penetrating Neck Injury

Neck injuries are currently found in 11% of battle injuries in United Kingdom (UK) forces, compared to 2% to 5% in US forces.[16] In the neck, both the trachea and laryngopharynx are superficial and are commonly injured.[17–20] Various techniques have been described in the literature to manage penetrating airway damage, including orotracheal intubation,[18,21–23] flexible bronchoscopy,[20] use of a light wand following failure of direct laryngoscopy,[17] RSI,[10–18] and AFOI.[10–18] Surgical techniques described include both surgical cricothyroidotomy[24] and tracheostomy.[23]

Determining where the airway is injured is the first step in managing penetrating neck injury. This determination is best approached on a zonal basis.[21,25] Zone I of the neck represents the area between the clavicles and the cricoid cartilage, zone II the area between the cricoid cartilage and the angle of the mandible, and zone III the area between the angle of the mandible and the base of the skull. Injuries to the anterior and lateral aspects of the neck compromise the airway more often than those in the posterior region in civilian trauma[21] and probably military cases as well: a military study found that anterior wounds accounted for 79% of fragment wounds to the neck.[22] Once the zones involved have been identified, the clinician should then consider the presence of injury to the airway's lumen (with associated blood and debris), injury within the airway wall, or injury outside the wall (eg, expanding hematoma or surgical emphysema). Guidelines for managing injuries in each zone are listed in Exhibit 6-1.

Optimal intubation conditions may be difficult to achieve, and injuries may compromise positive pressure ventilation with bag-valve-mask devices.[7] Not all patients will be in extremis, however, and time may be available to consider additional investigations to characterize the injury. Computed tomography (CT) angiography is the first-line investigation in stable patients with penetrating neck injuries[24] to identify sites

EXHIBIT 6-1

SUGGESTED GUIDELINES FOR MANAGEMENT OF PENETRATING AIRWAY INJURY

Zone I injury
- Direct intubation through a large defect
- Surgical cricothyroidotomy in an emergency or tracheostomy in the semi-elective setting
- Thoracotomy in complete tracheal transaction

Zone II injury
- CT scan to exclude distal airway injury (provided there is no immediate impending obstruction of the airway)
- Oral intubation by RSI for injuries proximal to the larynx
- Fiberoptic intubation for injuries distal to the larynx
- Surgical airway for injuries distal to the larynx

Zone III injury
- Oral intubation by RSI for small defects
- Surgical airway for gross disruption

For any large airway defect: direct intubation through the defect

When a distal airway injury has not been excluded: primary surgical airway may be the most appropriate plan.[1]

CT: commuted tomography
RSI: rapid-sequence induction
1. Nelson LA. Airway trauma. *Int Anesthesiol Clin 2007;*45:99–118.

of potential bleeding that may be suitable for surgical or endovascular interventions. Such an investigation will also demonstrate the location, nature, and extent of any airway injury.

Penetrating Neck Injury With Associated Vascular Injury

Despite the high prevalence of airway damage to the neck, the most common cause of death from combat neck injury is secondary to exsanguination from the carotid arteries or jugular veins.[26] Vascular damage should be suspected in any airway injury to the neck due to the close anatomical proximity of major blood vessels to the upper airway. Vascular damage can result in bleeding into the airway itself, or it can cause a rapidly expanding hematoma, resulting in progressive airway obstruction.[27] RSI should be considered the airway modality of choice in these cases.

AIRWAY DEVICES

New technology in electronics and materials has led to an increase in the availability of new airway devices. It must be borne in mind, however, that many new products have not undergone rigorous testing, particularly in the trauma setting. Although many different devices are now on the market, it is advisable for military providers to practice using the equipment in advance (a crisis situation is not a good time to experiment with unfamiliar devices). Some of the newer devices, with their advantages and disadvantages, are listed in Table 6-1.

ANESTHETIC CONSIDERATIONS

Airway Bleeding, Facial Distortion, and Patient Positioning

Blood and debris may be soiling the airway. Conscious casualties who are maintaining their airway satisfactorily do not require immediate airway intervention apart from a jaw thrust. Such patients should be allowed to adopt the most comfortable position. Lateral, sitting, and prone positions have all been described in case reports. The importance of allowing

TABLE 6-1

NOVEL AIRWAY DEVICES

Device	Advantages	Disadvantages
Video laryngoscopy • GlideScope (Verathon, Bothwell, WA) • McGrath (LMA North America, San Diego, CA) • C-Mac (Karl Storz, Tuttlingen, Germany) • Airway Scope AWS-S100 (Pentax, Tokyo, Japan) • Airtraq (Prodol Meditec, Getxgo, Spain)	A recent field trial of Airtraq for use in a military prehospital setting was favorable.[1]	Not intuitive New skill must be learned
Supraglottic airways • LMA ProSeal (LMA PacMed, Burnley, Victoria, Australia) • LMA Supreme (LMA PacMed) • i-gel (Intersurgical Ltd, Wokingham, Berkshire, England)	These three devices have been shown to be easy to place with a 97%-98% first time placement possible. They have been shown to be capable of rescuing ventilation when facemask and tracheal intubation have failed.	None identified
Flexible fiberoptic laryngoscopes	The replacement of external light sources with battery light sources makes fibrescopes truly portable. The use of chip camera technology offers good quality images and recording facilities.	Decontamination and cleaning of the traditional fiberoptic laryngoscope in the deployed field hospital is difficult (but these problems are negated by a disposable fiberscope).

1. Dawes RJ. Military difficult airway equipment evaluation. *JR Army Med Corps.* 2010;156:60.

these patients to choose their own positions must be reinforced during patient handover. Oropharyngeal tubes are a useful interim measure and should be used in preference to nasopharyngeal tube in head, face, and neck injuries because it is impossible to exclude base-of-skull fractures in the acute setting. Comminuted mandibular fractures may result in loss of tongue support, resulting in the tongue moving backwards and obstructing the airway. This may be temporarily resolved by placing a single suture through the tongue allowing the tongue to be pulled towards the chin.[28] Conscious patients with this form of injury often want to sit upright, and clinicians should be wary of laying these patients supine.

Anesthetic Approaches to Penetrating Airway Injury

The principle clinical features mandating early tracheal intubation are acute or worsening respiratory distress, an airway compromised by blood and secretions, extensive surgical emphysema, tracheal deviation by hematoma, or a decreasing level of consciousness.[29] Although anesthetists routinely perform endotracheal intubation, this procedure should be approached with great caution in patients with a penetrating airway injury.[30]

Direct Laryngoscopy

It is important for anesthetists to be aware that despite the appearance of an intact laryngeal inlet, a tracheal tear may present underneath. In such a case, if an endotracheal tube is placed under direct laryngoscopic vision, the tip of the tube could pass through the defect. This problem may go unrecognized and risks airway obstruction, pneumomediastinum, and the creation of a false passage.[30] Direct laryngoscopy is in effect a blind technique that may completely disrupt the larynx. The incidence of complications is unknown, but they are potentially lethal and difficult to reverse even with an emergency surgical airway (especially if gross surgical emphysema has been created).[21] Direct laryngoscopy under topical anesthesia (an "awake look") has been recommended,[7] but this technique will not reveal any injuries distal to the vocal cords.

Rapid Sequence Induction

Despite the common use of RSI to secure the airway, the technique is controversial. Some authors hold that RSI should be the default method of airway control,[31] and evidence suggests it is safe[32] and has a high success rate[33–35]; however, other researchers argue against RSI in certain cases.[36,37] It is not recommended in cases of near or total airway transection, where paralysis will abolish the supportive muscle tone, which may be all that is holding the airway together.[7,38] For these reasons, some authors advocate maintaining spontaneous ventilation at all costs.[30] Current UK anesthetic practice includes the use of cricoid pressure[39] during an RSI, but such pressure may distort the airway, change the anesthetist's view, and result in a more difficult airway.[30,40] Cricoid pressure is not used in other countries.

Blind Nasal Intubation

The consensus of opinion is that blind intubation methods including nasotracheal intubation should not be used in patients with penetrating neck injury because further injury or complete airway obstruction may be induced.[41] A single paper reviewing a case series of patients successfully managed with blind nasotracheal intubation has challenged this advice.[42] This technique is rarely taught in UK hospitals, and if it is not part of their regular practice, clinicians should not use it.

Fiberoptic Intubation

AFOI is the gold standard for safely securing the airway in a casualty with traumatic airway injury. This technique allows the lumen of the airway to be identified by direct vision throughout the intubating process, so the anesthetist can be confident about siting the endotracheal tube distal to any visualized tear. However, AFOI depends on availability of a fiberscope, the cooperation of the patient,[30,43] and the skills of the operator. Another confounding factor is that foreign bodies or blood hinder the use of the fiberscope,[30] although in skilled hands it has proved very effective.[10–13, 15, 44] Sterilizing the fiberscope can also cause difficulties with AFOI in the field hospital; disposable versions have recently been developed but are yet to be evaluated in this setting.

SURGICAL CONSIDERATIONS

A surgical airway is generally considered the first choice intervention for penetrating laryngeal injuries[30,37] because placing an endotracheal tube under direct vision reduces the potential for misplacement. Cricothyroidotomy is the surgical modality of choice in the emergency setting,[38] with conversion to tracheostomy performed semi-electively. Tracheostomy should be performed at least one tracheal ring below the injury to avoid complications.[15] If a difficult intubation is suspected, it is advisable to prepare the patient's neck, and the surgeon should be ready to perform a surgical airway.[30] The anesthetist should be mindful that it might be difficult for the surgeon to rapidly create a surgical airway, particularly if there is overlying hematoma or other gross anatomical disruption.

TEAM CONSIDERATIONS

Because of the issues discussed above, the team dealing with airway injuries must consider the likely fragment or projectile trajectory and potential airway effects. Whether the anesthetist or the surgeon performs the surgical airway will be determined by the skills and experience of each team member. Human factors[45] (or nontechnical skills such as leadership, teamwork, communication, and situational awareness) play an important role in ensuring that individuals in a clinical team perform to the highest standard.[46] The authors believe that the principles of Stanford School of Medicine's Anesthesia Crisis Resource Management (ACRM) training[47] are crucial to ensuring the best possible outcome when faced with a patient with severe blast or ballistic injuries. Swift, coordinated decision-making by all members of the team is essential.

SUGGESTED TECHNIQUES AND GUIDELINES IN THE DEPLOYED SETTING

UK Defence Medical Services anesthetists spend the majority of their clinical practice working with civilian patients in the National Health Service and generally deploy on military operations every 6 to 18 months. The deployed environment has a much different case mix to that experienced in the civilian setting. Standard operating procedures have been developed for management of the difficult airway by the American Society of Anesthesiologists,[48] and for the unanticipated difficult airway by the Dif-

ficult Airway Society.[49] Both of these protocols were designed to deal with a civilian patient population in the setting of a general hospital, and do not reflect the circumstances currently encountered in the deployed military environment. Management of "anticipated difficult airway" has recently been evaluated to some extent in a civilian setting[50]; however, the unusual nature of penetrating airway injury necessitates its own standard operating procedure for use in the deployed field hospital. Key points are listed in Exhibit 6-2 and potential pitfalls are recorded in Exhibit 6-3. (See Exhibit 6-1 for suggested guidelines for the airway management of blast or ballistic injury.) These lists are provided to help anesthetists improve their nontechnical or human factors skills in the clinical environment.

SUMMARY

Because of the multiple potential approaches to airway management of casualties with penetrating injuries, as well as the low incidence of these injuries in the civilian context, it is important to develop guidelines that allow planning and anticipation of these cases prior to deployment. Use of an algorithm, however, should not be substituted for common sense. The newly developed technologies describe here can aid in airway management, but the anesthetist must be aware of their limitations.

EXHIBIT 6-2

KEY POINTS FOR THE MANAGEMENT OF PENETRATING AIRWAY INJURIES

- Monitor patient with full AAGBI standard monitoring[1] (especially $ETCO_2$)
- Preoxygenation
- Airway optimization
  - o Allow conscious patient to adopt most comfortable position
  - o Use jaw thrust in unconscious patients
- Consider the urgency that a secure airway is required
  - o Not all patients will be in extremis; there may be time to consider additional investigations to characterize the injury. CT is considered the first-line investigation in stable patients with penetrating neck injuries.[2]
  - o However dire the situation, take a few seconds to think before acting
- Consider the site of injury
  - o Blood and debris may be soiling the airway
  - o May require clearing prior to securing the airway
- Consider the availability of suction
  - o Two devices are preferable
  - o Have a bougie readily available
- When securing the airway consider:
  - o Chin lift
  - o Jaw thrust
  - o Head tilt
  - o Basic airway adjuncts
  - o Positioning head up
  - o Using a smaller endotracheal tube
  - o Using a hollow bougie to allow continual insufflation
- If C-spine immobilization is present, remove and nominate one person to maintain manual-in-line stabilization

AAGBI: Association of Anaesthetists of Great Britain and Ireland
CT: commuted tomography
$ETCO_2$: end-tidal carbon dioxide

1. Association of Anaesthetists of Great Britain and Ireland. Recommendations for Standards of Monitoring During Anaesthesia and Recovery. 4th ed. London, England: Association of Anaesthetists of Great Britain and Ireland; 2007. Available at: http://www.aagbi.org/publications/guidelines/docs/standardsof monitoring07.pdf. Accessed on: January 9, 2012.
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EXHIBIT 6-3

POTENTIAL PITFALLS OF AIRWAY MANAGEMENT

Ventilation: Positive pressure ventilation risks enlarging tears and causing surgical emphysema.
- Try to preserve spontaneous ventilation prior to intubation.
- Use bag-valve-mask ventilation as a last resort.
- Beware of using a supraglottic airway device in injuries distal to cords.
- Avoid transtracheal jet ventilation.

Intubation: Blind placement of the tube risks causing the tip to pass through the defect and lie outside the airway; this prevented only by fiberoptic intubation or a surgical airway.

Intubation: Endotracheal intubation should be approached with caution.
- Avoid oral intubation when the injury is distal to the vocal cords.
- Avoid blind nasal intubation.
- Fiberoptic intubation is likely to be difficult or impossible when there is bleeding into the airway.

Surgical Airway: potentially extremely difficult in the presence of subcutaneous emphysema or expanding hematoma. Direct laryngoscopy is also likely to be difficult.

Drugs: Avoid muscle relaxants with near or complete airway transaction. Muscle tone may be important for airway integrity.

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Chapter 7

DAMAGE CONTROL RESUSCITATION

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INTRODUCTION

Damage control resuscitation (DCR) is defined as a systematic approach to major trauma, combining a series of clinical techniques from point of wounding to definitive treatment to minimize blood loss, maximize tissue oxygenation, and optimize outcome. The three major components of DCR are surgery to control bleeding, massive transfusion, and hemostatic resuscitation, instigated simultaneously (Figure 7-1).[1,2]

The term "damage control" originated in the United Kingdom (UK) Royal Navy as early as the 17th century and relates to doing whatever is required to bring a damaged ship home to port. DCR was introduced into the UK Defence Medical Services (DMS) in 2005 with the publication of *Battlefield Advanced Trauma Life Support*[3] (BATLS) and later enhanced with hemostatic resuscitation and massive transfusion guidelines.[4] Further work in US military vascular trauma cases highlighted the value of implementing this important concept.[5] DCR represents a major step in the evolution of military trauma care. Since its inception, there has been a significant improvement in the number of unexpected survivors and a marked reduction in mortality from massive transfusion, despite increasing injury severity.[6]

THE EVOLUTION OF MILITARY TRAUMA CARE

Trauma care dates back to ancient Egypt, Greece, and Rome, inextricably linked to the wars these empires were built upon. The Edwin Smith Papyrus from the 17th century BCE details the clinical treatment of 48 cases of war wounds in ancient Egypt. Homer's *Iliad* records 147 types of wound with an overall mortality rate of 77.6% during the Trojan War. The Romans probably created the first trauma center hospitals, called "valetudinaria," during the 1st and 2nd centuries CE. Eleven such centers existed in Roman Britain.[7]

Trauma care did not advance greatly until the 14th century when, Guy de Chauliac (often termed the "father of surgery") practiced the use of inhalational anesthesia, antisepsis, trephination, and thoracic surgery. From 1797 to 1812 Dominique Larrey acted as Napoleon's surgeon general and developed what was at the time a revolution in military trauma care: he introduced field hospitals located close to the front line and "flying ambulances" to quickly transport the wounded to the operating theatre, thereby reducing mortality in the perioperative period. Larrey understood the need for predeployment training for his ambulance teams (consisting of eight surgeons) and exercised them daily until their operations and application of bandages showed "the greatest degree of emulation and that the strictest discipline were prevalent among all the surgeons."[8]

Conflict continues to drive advances in military medicine. The sustained casualty rates since 2003 of UK military personnel in Operation Telic (Iraq)

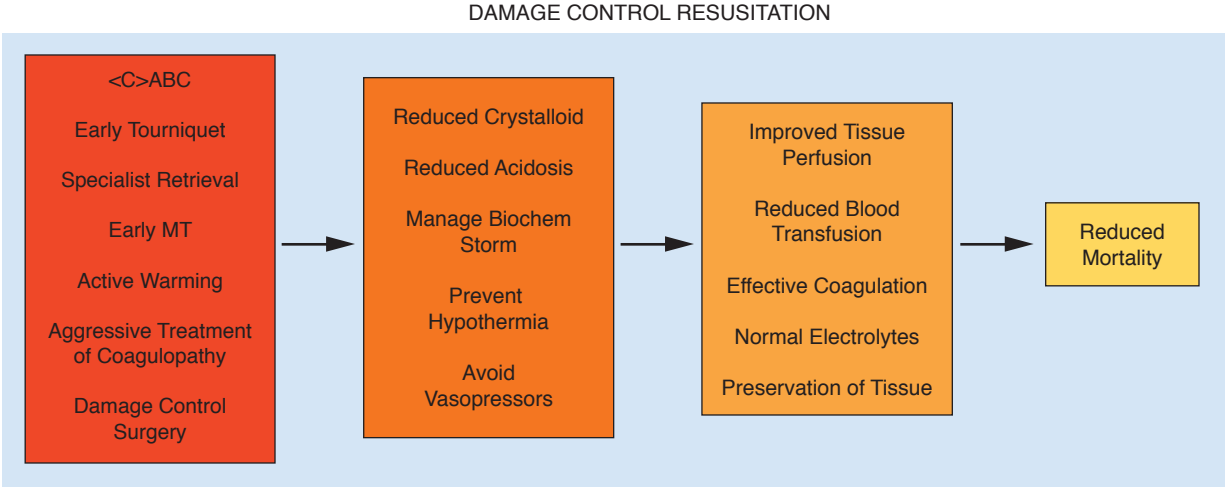

Figure 7-1. Damage control resuscitation. Surgery to control bleeding, massive transfusion, and hemostatic resuscitation are encompassed within the the first (red) box. The positive sequelae from appropriate treatment are shown in subsequent boxes. <C>ABC: catastrophic hemorrhage, airway, breathing, and circulation; MT: massive transfusion

and Operation Herrick (Afghanistan) have allowed greater understanding of the pathophysiology of major trauma and stimulated the development of new paradigms of care and structured practice guidelines. The new practices, which evolved into DCR, are focused on a common team approach to ensure rapid restoration of physiology in preference to definitive surgical treatment.[1,9] To deliver effective DCR, this approach must be rehearsed in predeployment training so that each provider becomes familiar with new and often unfamiliar team dynamics. DCR principles include a horizontal trauma team in which all members (surgeons, anesthesiologists, nurses, and others) are encouraged to contribute to the trauma management discussion in order to solve problems and improve care. To this end, DCR training should train those who will be working together in future deployments. This training has been widely termed "crew resource management"; it aims to ensure all team members are familiar with the environment, equipment, and roles in future challenging DCR situations. All UK deploying clinicians attend a 5-day predeployment clinical exercise practicing challenging medical scenarios in an exact copy of a Role 3 hospital.

During DCR it is possible that individuals, especially clinicians performing critical procedures, may become overly focused on immediate tasks and lose overall situation awareness (termed "reduced bandwidth"). Therefore, a designated leader of the resuscitation team must ensure effective communication among all key members. During the initial stages of resuscitation, the primary goal is restoring physiology, while surgery is limited to controlling hemorrhage and minimizing wound contamination (see Damage Control Surgery, below). It is incumbent on the resuscitation leader to ensure this aim is achieved. This may require a pause in surgery to focus on additional dressings, packs, clamps, or direct pressure to reduce bleeding while volume is restored by the anesthesia team. Once volume has been restored, surgeons should do the minimum surgery needed to save the patient's life. Further debridement and definitive surgery can occur at a later stage in the evacuation process, hours or days after the initial resuscitation.

PRINCIPLES OF DAMAGE CONTROL RESUSCITATION

Pathophysiology

Major trauma is a generic term covering a wide range of injuries and injury mechanisms. The physical injury itself differs according to its etiology, for example blunt, penetrating, or blast, as well as the individual patient. Many factors, including presence of hemorrhage, head injury, coagulopathy, and hypothermia, as well as the care given, influence the initial physiologic insult sustained in the initial trauma.

DCR includes the accepted concepts of the "lethal triad" of hypothermia, acidosis, and coagulopathy; however, recent major advances in the understanding of coagulopathy have occurred.

"Trauma-induced coagulopathy" is a term encompassing the coagulopathy related to the traumatic insult and includes acidosis, hypothermia, platelet consumption, blood loss, and dilution. More recently a further subgroup of trauma-induced coagulopathy, termed "acute trauma coagulopathy" (ATC), has been described.[2] This is a primary pathological event whose cause remains uncertain, but increasing evidence points toward a mechanism that includes tissue hypoperfusion, hyperfibrinolysis, activation of protein C, and up-regulation of thrombomodulin. The driver of ATC appears to be related to poor tissue oxygen and results in activation of the vascular endothelium. The endothelium is a poorly understood structure that is implicated not only in ATC but also in the development of systemic inflammatory response syndrome, which can ultimately lead to multiorgan dysfunction and increased mortality.[10] This process occurs independently of crystalloid administration, acidosis, and hypothermia.

DCR aims to restore tissue oxygen delivery in order to reverse the pathological processes driven by the hypoxic endothelium and restore normal physiology. This approach is coupled with treatment and prevention of hypothermia, acidosis, and coagulopathy. The military approach is to target these pathologies as early and as aggressively as is practical from the point of wounding, along the evacuation chain, and into the Role 3 hospital. At the Role 3 hospital, the three major components of DCR, surgery to control bleeding, massive transfusion, and hemostatic resuscitation, are instigated simultaneously.[1,2]

Point of Wounding

Hemorrhage remains the leading cause of death in military trauma.[11] Early treatment or temporary control at the point of wounding is essential for survival.[11] Much effort has gone into improving the care given at point of wounding.[11] This includes training in the use of the <C>ABC (catastrophic hemorrhage, airway, breathing, and circulation) paradigm, which is the core of the BATLS system and incorporates the use of the hemostatic agents and the timely application of tourniquets.

Specialist Retrieval Teams

Care by appropriately trained prehospital doctors (with critical care and airway skills) has been shown to significantly improve survival from major trauma.[12,13] An integral part of the British military trauma system is the medical emergency response team (enhanced), or MERT(E), which consists of a prehospital-trained attending anesthesiologist or emergency physician, two paramedics, and an emergency department flight nurse. This specialist team is able to initiate DCR in flight by gaining rapid intravenous or intraosseous access and administering warm blood products with other specific therapies such as tranexamic acid. The ability to perform advanced airway maneuvers, diverting casualties to the appropriate medical facility, and senior decision-making have contributed to the success of this type of prehospital care. However, this model of prehospital care is very different from what is performed in the civilian setting or military medical systems of other nations The MERT(E) model has been validated since its introduction and robust evidence of its effectiveness has been produced to guide other services.[13–15]

Damage Control Surgery

Damage control surgery, the surgical component of DCR, is focused on control of major anatomical bleeding, removal of dead tissue, and gross decontamination.[16]

Permissive Hypotension

Hypotensive resuscitation is a standard of practice in hemorrhaging patients without traumatic brain injury.[17] Numerous animal models of uncontrolled hemorrhagic shock have demonstrated improved outcomes when a lower than normal mean arterial pressure of 60 to 70 mm Hg is used as the target for fluid administration during active hemorrhage.[18] Two large human trials have demonstrated the safety of this approach (relative to the conventional target of greater than 100 mm Hg), suggesting various benefits including shorter duration of hemorrhage and reduced mortality.[19,20]

Animal models incorporating blast injury and hemorrhage, however, have demonstrated a high mortality if hypotensive resuscitation is used in blast injury patients. Follow-up studies using a prehospital blood pressure profile (termed "novel hybrid resuscitation") utilized a blood pressure of 90 mm Hg (palpable radial pulse) for 60 minutes, followed by volume boluses to a target blood pressure of 110 mm Hg. The study concluded that more animals survived overall, and for longer, than those animals allowed 120 minutes of permissive hypotension. Using the results of this study's parameters with battlefield casualties may permit a longer survival timeline, allowing live casualties to reach a facility where DCR can be instigated.

Fluids

Crystalloid has been the mainstay of resuscitation since the Vietnam War, when it was first popularized.[21] In 1978 it was adapted by the American College of Surgeon's Advanced Trauma Life Support Group, who advocated using two large-bore cannulas and 2,000 mL of lactated Ringer solution for the hypotensive casualty.[22] With the introduction of the DCR protocol, however, the overall use of crystalloid has decreased dramatically.[23] This has helped reduce the major adverse effects of unchecked crystalloid administration: acute lung injury and acute abdominal compartment syndrome (diagnosed since the Vietnam War), together with acute renal failure, multiorgan dysfunction, and anastomotic leaks (termed the "vicious salt water cycle").[21]

Reperfusion injury is marked in hemorrhaging trauma casualties.[24] There is evidence that crystalloid activates white cells, thereby stimulating systemic inflammatory response syndrome. This is probably potentiated by the "d" stereoisomer of lactate (present in Hartmann solution), which the body fails to metabolize.[25] Over-resuscitation with crystalloid may lead to uncontrolled hemorrhage due to dilution of clotting factors, causing a hypocoagulable state with the sequelae of reduced organ perfusion, and abdominal compartment syndrome (Figure 7-2).[26] These complications predispose casualties to multiple organ failure and increased mortality, compared with moderate resuscitation.[21–26]

Hemostatic Resuscitation

Hemostatic resuscitation is defined as the rapid proactive treatment of coagulopathy associated with major trauma[27] and aims to gain physiological control of bleeding through the use of massive hemorrhage protocols with high ratios of packed red blood cells : fresh frozen plasma : and platelets (PRBC:FFP:PLT), as well as medical adjuncts guided by laboratory and point-of-care testing. This has been shown to improve outcomes.[17] Early in resuscitation, the patient is managed empirically using massive hemorrhage protocols with the emphasis on restoring lost blood volume, treating shock, and improving tissue oxygenation to avoid further development of ATC. Once bleeding has stopped and shock is reversed, a more goal-directed

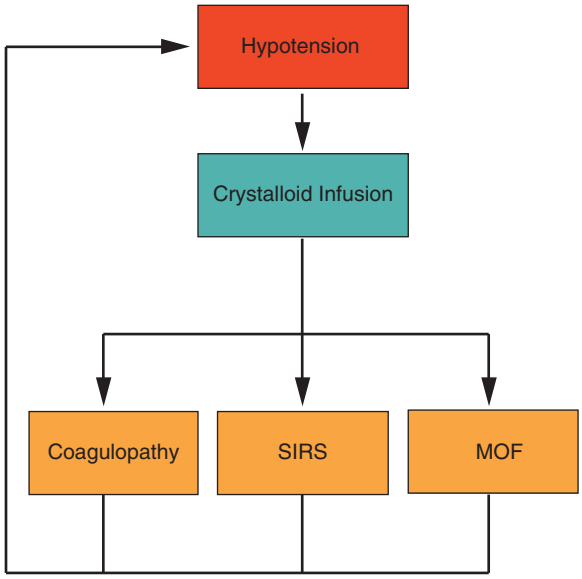

Figure 7-2. Perils of over-administration of crystalloid, which may lead to coagulopathy, systemic inflammatory response syndrome, and multiple organ failure.
MOF: multiple organ failure
SIRS: systemic inflammatory response syndrome

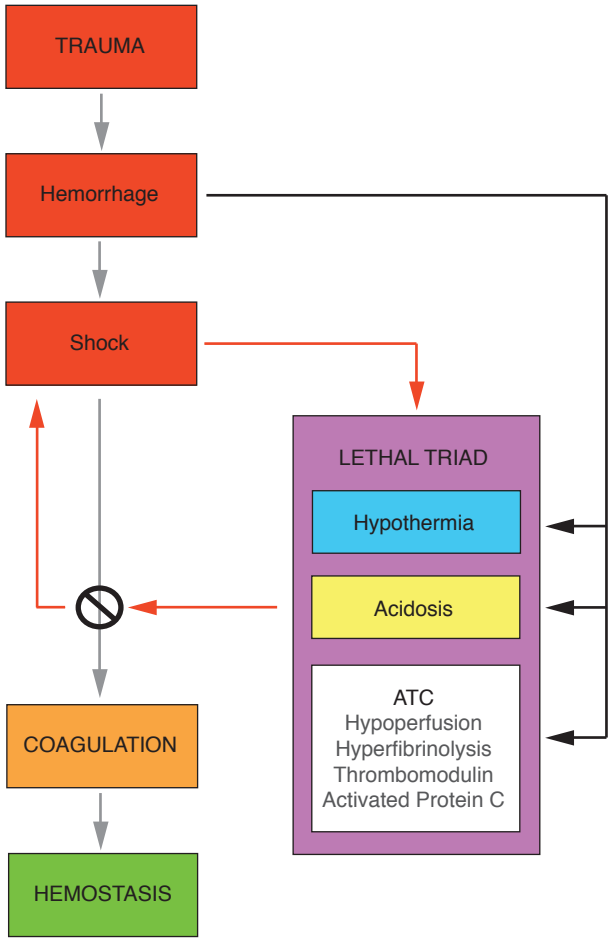

Figure 7-3. Hemostatic resuscitation.
ATC: acute trauma coagulopathy

approach to managing coagulopathy is advocated; its aims are to prevent dilution, replace factors lost due to consumption and bleeding, and treat deficiencies caused by ATC through the administration of effective whole blood resuscitation using blood product components (Figure 7-3). This is done with the use of point-of-care coagulation testing such as ROTEM (TEM International GmbH, Munich, Germany) or TEG (Haemonetics Corp, Braintree, MA) and standard laboratory blood counts. To achieve effective whole blood replacement, PRBC:FFP:PLT ratios of approaching 1:1:1 have been advocated. Fresh whole blood is still available to the deployed clinician.

Point-of-Care Testing

Point-of-care testing is becoming increasing important and recognized as crucial to the treatment of the patient during DCR. It includes arterial blood gas analysis and coagulation monitoring using thromboelastometry (ROTEM or TEG). Either ROTEM or TEG can determine failure in each part of the clotting cascade from clot initiation to propagation, amplification, and stabilization. Recent clinical use of ROTEM with experimental use of platelet function analysis (multiplate) has implied that platelet function is affected early in trauma, so some new guidelines now incorporate the early, empiric use of platelets rather than using the traditional trigger of a platelet count below 100.[28] Point-of-care testing gives results to clinicians in a clinically relevant time, allowing them to concentrate on delivering blood and blood products to the patient, and reduces the logistical delay and burden on laboratories. Ultimately it allows for more individualized patient treatment.

MANAGING THE PHYSIOLOGY

The evolution of DCR philosophy, coupled with increased training and experience in current conflicts, has resulted in very effective and aggressive administration of large amounts of blood products according to massive hemorrhage protocols. Massive transfusion rapidly treats underlying hemorrhagic shock but, unless carefully monitored, has potentially lethal sequelae. Base deficit, calcium, and potassium levels

are all available in point-of-care blood gas analysis and should be performed at least every 30 minutes in the early stages of resuscitation.

Acidosis

Virtually all coagulation stages are inhibited by acidosis. Platelets alter shape at a pH below 7.4, and Ca^{2+} binding sites are pH-dependent,[29] but the main process inhibited is thrombin generation.[30] Unsurprisingly, trauma nonsurvivors were more likely to have a lower pH than survivors.[26] Martini et al demonstrated that thrombin generation was inhibited by a pH of 7.1 by as much as 50% with an additional 35% reduction in fibrinogen. Platelet count was also reduced by 50%.[30]

In addition to pH, base deficit is a sensitive indicator of hypoperfusion and correlates with mortality.[26] At levels below -12.5, it has been demonstrated to directly inhibit coagulation.[30–32] Base deficit has also been used to predict transfusion requirements.[33] A recent review concluded that a notable impairment of hemostasis arises at a pH of 7.1 and below, with similar effects observed at base deficit of -12.5 or less.[34] Thus, when there is severe hemorrhage and acidemia, buffering toward physiologic pH values is advantageous, especially when massive transfusions of older PRBCs displaying exhausted red blood cell buffer systems are used.[30]

Calcium

JR Green was the first to show that "calcium is instrumental in bringing about coagulation when added to plasma which shows little or no tendency to clot, and that coagulation in its absence is almost or quite prevented"(1887).[31] Hypocalcemia is common in critically ill trauma patients and is associated with increased mortality.[32,33] It has since been shown that calcium is required for several reactions in the coagulation cascade and in platelet activation.[32–34] Citrate (a chelating agent) is added to blood products (FFP in particular) to prevent clotting during storage. It follows that a patient receiving a massive transfusion will be hypocalcemic and coagulopathic regardless of other measures taken to improve coagulation. It is recommended that ionized calcium levels be maintained above 1.0 mmol/L for effective coagulation to occur.[35]

Potassium

Hyperkalemia is common after PRBC transfusion, and often severe.[36] It appears to be more common after transfusion under pressure and with older units of PRBCs, most likely due to increased cell lysis. It is therefore essential to closely monitor potassium levels throughout the resuscitation and maintain a concentration within the normal range. If hyperkalemia is detected, the myocardium should be protected by administering calcium and starting an insulin/dextrose infusion immediately to regain control.

Hypothermia

The causes of hypothermia in the trauma patient are reduced heat production and increased heat loss. Hypovolemic shock results in an inadequate oxygen delivery to the tissues, which impairs cellular respiration and results in a decreased heat production. Hypotension and hypovolemia inhibit shivering, preventing this normal response to hypothermia. Increased heat loss occurs through environmental exposure and, during the resuscitation phase, is primarily caused by administering cold fluids. Heat loss is proportional to the volume given and the temperature difference between the patient and the fluid.[37] The energy needed by the body to warm 2,000 mL of fluid infused at 25° C within 1 hour exceeds the energy that can be delivered by conventional warming methods in the same time.[38]

Hypothermia has effects on all body systems, including reduced cardiac output and impaired respiratory and endocrine function. During DCR, its most significant effect is on coagulation, producing a reduction in platelet function and number, inhibition of the coagulation cascade, and increased fibrinolytic activity.[39] A significant effect on platelet function is observed even in mild hypothermia (34 °C) through an inhibition of thromboxane B_2 and reduced expression of surface molecules, leading to poor aggregation. At the same temperature the reduction in platelet numbers is due to sequestration in the liver and spleen.[40]

Coagulation cascade enzyme reactions are strongly suppressed by hypothermia. In one study, a temperature of 34° C was the critical point at which enzyme activity in trauma patients slowed significantly. At temperatures below 33° C, hypothermia produces a coagulopathy equivalent to 50% of activity at normothermia, despite the presence of normal clotting factor levels.[41] It is important to note that this effect on coagulation occurs even in isolated areas of the body, and superficial cooling of a limb with preserved core temperature results in a significantly prolonged bleeding time. It should also be noted that tests of coagulation are performed at 37 °C, so a purely hypothermia-induced coagulopathy will not be demonstrated by laboratory tests.[26]

Avoiding and correcting hypothermia is critical in preventing or correcting coagulopathy in a patient receiving massive transfusion. The resuscitation and

operating rooms must be warmed. All fluids administered must pass through a warmer. Hot air convection should be used above the patient, and electric mattresses under the patient have been proven useful when multiple body cavities are being operated on simultaneously. The patient should be insulated as much as possible, which can be difficult when large surgical exposure is required.

MEDICAL ADJUNCTS

Antifibrinolytics

The 2010 CRASH-2 trial showed that administering tranexamic acid to adult trauma patients with, or at risk of, significant hemorrhage within 8 hours of injury reduced all-cause mortality with no apparent increase in vascular occlusive events.[42] As a consequence of this trial, tranexamic acid has been incorporated into trauma treatment protocols worldwide. A further analysis of the CRASH-2 study demonstrated a 32% increased survival if the tranexamic acid is given within 3 hours to bleeding trauma patients, but beyond this time it is less effective and could be harmful. The trial protocol involved administering 1 g as soon as possible, followed by another 1 g administered over the subsequent 8 hours. This procedure rarely takes place in military medicine because the first dose is often administered near the point of wounding (eg, during the MERT(E) stage), and then further blood products and adjuncts are given when further laboratory or thromboelastometry results are known.

Recombinant Activated Factor VII

Factor VII is a crucial component of coagulation, binding to tissue factor (a lipoprotein present in endothelial cells) exposed by injury, generating activated factor X and subsequently thrombin. Recombinant activated factor VII (rFVIIa) is licensed for use in patients with hemophilia and inhibitory antibodies. Its enhancement of hemostasis directly at the site of injury has stimulated research into possible uses in trauma.[43,44] Two parallel, multi-center, randomized controlled trials have shown a statistically significant reduction in blood transfusion requirements in blunt (but not penetrating) trauma patients treated with rFVIIa.[45] Although the trial and statistics have been the subject of criticism, these studies remain some of the best evidence available. The effectiveness of rFVIIa (not limited to trauma) has also been assessed by a Cochrane review, which concluded that rFVIIa as a hemostatic adjunct in trauma remains unproven.[46]

The benefit of using rFVIIa must be balanced against its thrombogenic potential.[47] Although not licensed for the treatment of traumatic hemorrhage, rFVIIa use continues. Given its substantial cost, further research is warranted. Previously, it had been DMS policy to give rFVIIa to any salvageable patient with continuing hemorrhage that has failed surgical and nonsurgical treatments. However, with the advent of DCR, patients are now less coagulopathic, acidotic, and hypothermic, and consequently the use of rFVIIa has declined considerably. During mature operations such as Afghanistan the use of rFVIIa is now limited; however, in less well located and supported hospitals, there may be potential for the use of rFVIIa to help reduce blood usage and extend resuscitation timelines.

END POINTS OF RESUSCITATION

DCR is a concept aimed at restoring physiology, and end points are critical in determining when aggressive protocols should cease and more measured approaches begun. Current end points are summarized in Figure 7-4. Critical to resuscitation is returning blood pressure to normal values when central circulation is filled. However, the patient still requires further resuscitation to ensure that peripheral circulation is also filled. To achieve this, targeted resuscitation must continue after blood pressure returns to normal. Many approaches to targeted resuscitation may be used, but most military proponents now administer a high-dose opioid anesthetic similar to cardiac anesthesia. This procedure causes a degree of vasodilatation allowing resuscitation of the peripheral compartment. It is fair to say that end points of resuscitation remain uncertain; however, markers of tissue perfusion are likely to provide the most information about whole-body tissue oxygenation.

Future strategies are focusing on the treatment of coagulopathy with statins, with optimum ratios of FFP : PRBC : platelets, and earlier use of blood products, such as in the prehospital phase. Ongoing military research involves the use of prehospital recombinant erythropoietin and better use of blood products in the prehospital phase by using freeze dried plasma (Lyoplas; DRK-Blutspendedients West gGmbH, Hagen, Germany) and synthetic hemoglobin. Recent work with dogs cooled to below 18°C to ascertain whether surgery at this temperature would "suspend" further

tissue damage and allow trauma surgery to be undertaken with lower risk has moved into human trials. Also, work has recently begun at the UPMC Presbyterian Hospital in Pittsburgh, Pennsylvania, with deep hypothermia for trauma victims, termed emergency preservation and resuscitation. Lead researcher Dr Sam Tisherman and his team of surgeons are hoping to replicate animal research in this area. Probably the greatest advances in care will occur with earlier and more targeted treatment in the prehospital phase, particularly where long transport times are prevalent. Prehospital systems in Europe are already trialling the use of extracorporeal membrane oxygenation, and the first prehospital resuscitative endovascular balloon of the aorta (REBOA) has already been used successfully in London. Early use of synthetic blood products, better patient warming, intelligent tasking of doctor-led prehospital teams, and shortened on-scene times, together with early computed tomography scanning, will allow time to surgery to be reduced in those patients who require it. These efforts hold promise to improve outcomes and reduce morbidity.

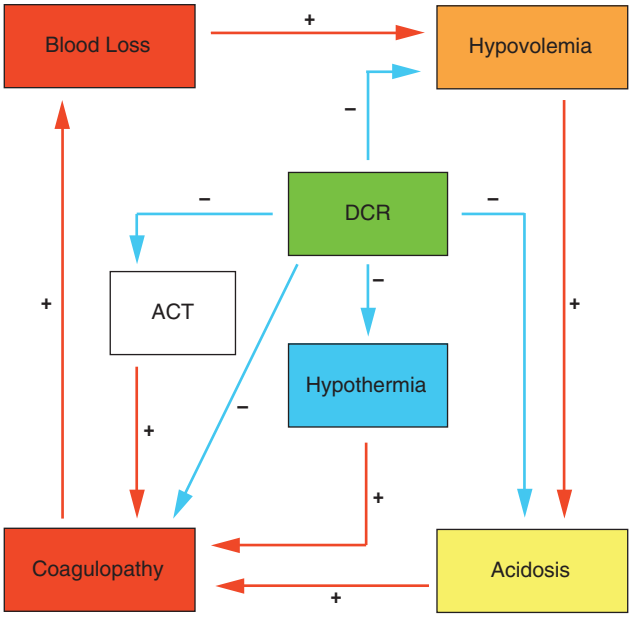

Figure 7-4. End points of damage control resuscitation.

SUMMARY

DCR is a complex process that aims to restore physiology in order to save life. If practiced effectively by well-rehearsed, experienced teams, life can be saved, physiology rapidly restored, and surgical options increased. It is a technique that requires flexibility, a thorough understanding of the pitfalls of massive transfusion, and attention to detail.

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Chapter 8

MASSIVE TRANSFUSION IN THE FIELD

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INTRODUCTION

"The advantage of the direct transfusion of human blood in cases of severe haemorrhage which we encounter in the emergencies of military surgery cannot to our minds, be overestimated."[1]

The above comment, from a paper presented by Lieutenant Colonel A Primrose of the Canadian Army Medical Corps in 1916, remains as pertinent today as during those earlier days of military transfusion medicine.[1] Hemorrhage remains the most common cause of death in combat trauma and, more importantly, is the most frequent cause of preventable death.[2] On today's battlefield, advances in body armor, field resuscitation, and casualty evacuation have resulted in more potentially salvageable patients with massive trauma arriving alive at Role 2 and 3 care facilities. Such casualties, who in earlier conflicts likely would have died at or near the point of wounding, now often present to the combat support hospital (CSH) emergency department in the last few seconds of that "momentary pause in the act of death" that is severe shock.[3] Massive transfusion is just one component in the multifaceted approach to managing the modern combat polytrauma patient.

The standard approach to casualties with major trauma is now termed damage control resuscitation (see Chapter 7, Damage Control Resuscitation), the aim of which is to "minimize blood loss, maximize tissue oxygenation and optimize outcome."[4] It encompasses rapid control of compressible hemorrhage in the field, swift retrieval with ongoing resuscitation during transport to the medical facility, focused airway management and oxygen therapy, hemostatic resuscitation, permissive hypotension, damage control surgery, and critical care. This chapter will focus on the interplay between hemostatic resuscitation, permissive hypotension, and massive transfusion. Hemostatic resuscitation is the proactive management of hemorrhage aimed at offsetting the effects of acute trauma coagulopathy before it is compounded by dilution of clotting factors and the development of acidosis and hypothermia. In the United Kingdom (UK) military, hemostatic resuscitation is initiated as early after wounding as possible by the Medical Emergency Response Team–Enhanced (MERT-E) and continued until surgical and microvascular hemorrhage is controlled.

In the more severely injured this approach involves the massive transfusion of blood products in, as near as possible, predetermined ratios that have been shown to improve outcome. For this approach to succeed, adequate quantities of blood products must be available and a pre-agreed massive transfusion protocol (MTP) must be in place to avoid delays in blood product administration and the use of unnecessary and potentially harmful crystalloids or colloids. The use of such MTPs has also been shown to improve outcomes.[5]

A major change in trauma patient transfusion medicine during the current conflicts is the administration of blood products in increased ratios of plasma and platelets to packed red blood cells (PRBCs). A retrospective study of combat casualties in Iraq demonstrated that survival is significantly improved with these increased ratios.[6] As a result the UK military aims to transfuse severely injured casualties in a ratio of 1:1 PRBC to fresh frozen plasma (FFP), with platelet support as indicated, while the US policy is to use 1:1:1 PRBC to FFP to platelets.[7,8]

Massive transfusion has been variously defined, but most definitions include the transfusion of 10 units of blood, or 1 to 1.5 times the patient blood volume, in 24 hours. Other criteria, more meaningful in acute major hemorrhage, include 4 units in 1 hour, 50% blood volume in 3 hours, or a rate of loss of over 150 mL/min.

INITIATION OF MASSIVE TRANSFUSION PROTOCOLS

A key factor in the effectiveness of the MTP is the timely and appropriate initiation of the protocol. Frequent inappropriate initiation of the MTP will likely result in diminished blood product supplies (particularly plasma) and greatly contribute to medical personnel fatigue. A number of authors have developed criteria for instituting an MTP. These tools use various combinations of mechanism of injury, vital signs, and laboratory results. Schreiber et al[9] found that an international normalized ratio over 1.5, hemoglobin under 11g/dL and penetrating trauma mechanism independently predicted the need for MTP. McLaughlin et al[10] used admission heart rate over 105 beats/min, systolic blood pressure under 110 mm Hg, pH under 7.25, and hematocrit under 32%. These criteria demonstrated a positive predictive value of 66%. Revealingly, McLaughlin noted that some of the more severely injured requiring massive transfusion did not necessarily demonstrate these laboratory criteria on admission because they were diverted directly to the operating room. Larson et al,[11] who used a similar model, pointed out in their conclusions that the decision to activate the MTP is quite subjective, relying on experienced clinicians to assess the severity of injuries. It may well be that these models will prove their utility in the hands of the less experienced, under the stress of dealing with multiple

casualties and in the less clear cut cases.

In these models, waiting for laboratory results can result in decision delay. A scoring system described by Cotton et al,[12] referred to as the Assessment of Blood Consumption (ABC) score, eliminates these factors by using penetrating mechanism of injury, arrival systolic blood pressure less than 90 mm Hg, arrival pulse rate over 120 beats per minute, and a positive focused assessment with sonography for trauma (FAST) exam. One point is allocated for each of the four markers and a total score of 2 or more was shown to predict the requirement for MTP with 84% to 87% accuracy.

In combat, most severe injuries are penetrating in nature (primarily the result of blast but also gunshot wounds) so that if the ABC scoring system is applicable, the casualty would only have to demonstrate the falling blood pressure or tachycardia to be triaged to the MTP. This is in fact what happens currently in the prehospital phase in British combat operations: the MERT-E clinician identifies the mechanism of injury, checks for the presence of shock, and initiates the transfusion of blood and plasma in flight while at the same time passing a pre-agreed codeword on to the field hospital alerting the emergency department to the requirement for massive transfusion. With nonpenetrating injuries, the FAST examination is performed immediately on arrival at the CSH. It should be noted that the ABC score was developed in civilian centers and needs to be validated in a military cohort.

In the civilian hospital, a high degree of specificity in predicting the need to initiate the MTP is ideal to avoid wasting blood products. During busy combat operations at the CSH, such specificity is less of a concern because prepared but unused blood products will most likely be required for other casualties before they need to be discarded. In these circumstances a high degree of sensitivity in prediction is of greater value in avoiding delays.

Other areas of concern include the over-transfusion of blood products (failing to recognize when hemorrhage is controlled and resuscitation efforts can be scaled back) and the recognized complications generally associated with blood transfusion. To avoid over-transfusion, it is essential to carefully monitor the patients' vital signs and laboratory indices. This monitoring includes recognizing a developing or resolving coagulopathy using clinical observation, standard coagulation tests, and, increasingly, thromboelastography (see below). Equally, close observation of the patient who has undergone large volume transfusion is required in the CSH intensive care unit, during repatriation flights, and in the Role 4 hospital intensive care unit, in anticipation of possible complications associated with massive transfusion.

COMPLICATIONS OF MASSIVE TRANSFUSION

Although massive transfusion can be life-saving, it is important to consider the possible complications that, left untreated, could be detrimental to the patient. Problems associated with massive transfusion cover a wide spectrum and include infections, immunologic changes, metabolic derangements, coagulopathies, and physiologic abnormalities. The majority of these complications can occur with transfusions of any magnitude; with some patients there is a significantly increased risk during large volume transfusion. The specific complications related to massive transfusion include hypocalcemia, hyperkalemia, acidosis, hypothermia, and dilutional coagulopathy.[13] Considering the concomitant pathophysiology surrounding the initial traumatic injury, these complications can significantly worsen the clinical picture; therefore, it is imperative for the clinician to be vigilant with respect to these complications and understand how to prevent and treat them.

Hypocalcemia

Hypocalcemia is commonly seen in massive transfusion due to the anticoagulant citrate used in blood products, which binds to ionized calcium. Plasma and platelets have the highest citrate level; therefore, these products have a higher risk. Since citrate is usually rapidly metabolized by the liver, the associated hypokalemia during standard transfusion is transient; however, when large volumes of blood products are administered against a background of impaired hepatic function due to hypothermia and hypoperfusion, the effect may be dramatic.[13] For example, a healthy adult liver can metabolize 3 grams of citrate every 5 minutes, and one unit of PRBCs usually has about 3 grams of citrate. Therefore, if transfusion rates exceed one unit every 5 minutes, which is common in massive transfusion, citrate levels will increase and hypocalcemia will result.[14] As the citrate level increases, signs of citrate toxicity and severe hypocalcemia can develop, including tetany, prolonged QT interval, decreased myocardial contractility, hypotension, narrowed pulse pressure, elevated end-diastolic left ventricular pressure, and elevated central venous pressure.[15] Hypocalcemia can also predispose to hyperkalemia-related arrhythmias as well as pulseless electrical activity arrest and ventricular fibrillation.[13,14] In addition, hypocalcemia has been implicated as a contributing factor in

the coagulopathy associated with massive transfusion. With this in mind, it is important to frequently monitor ionized calcium blood levels and treat low levels with intravenous calcium chloride or calcium gluconate to maintain levels within the normal range. Also, when possible, slowly infusing citrate-containing blood products can decrease the degree of citrate toxicity and hypocalcemia.[13]

Hypomagnesemia

Hypomagnesemia, which occurs with massive transfusion, is thought to result from the transfusion of large volumes of magnesium-poor fluids as well as the binding of magnesium to citrate.[14] This effect of citrate explains why hypocalcemia, hypomagnesemia, and citrate toxicity are often seen concurrently. Low levels of magnesium can lead to QT prolongation and ventricular arrhythmias, and may contribute to the coagulopathy associated with massive transfusion.[16] For these reasons, it may be important to monitor magnesium levels during trauma resuscitation and administer intravenous magnesium when indicated.

Hyperkalemia

Hyperkalemia is commonly seen during massive transfusion. Extracellular potassium increases as red blood cells (RBCs) are stored, which is attributed in part to inactivation of RBC membrane adenosine triphosphatase pumps.[14] The average extracellular potassium level in blood after 7 days of storage is 12 mmol/L, increasing to 32 mmol/L after 21 days.[17] After a unit has been transfused, the extracellular potassium is taken into RBCs as adenosine triphosphatase pump activity is restored. The increase in plasma potassium levels is therefore typically transient and without physiologic effect. However, during massive transfusion, large volumes of RBCs are typically administered through central venous catheters, so that a large extracellular potassium bolus may reach the right heart prior to intracellular uptake or dilution in the total blood volume. It is this delivery of extracellular potassium to the right heart that results in ventricular arrhythmias and cardiac arrest.[18] As mentioned above, simultaneous hypocalcemia can further predispose patients to potentially dangerous dysrhythmias. Methods to reduce the risk of hyperkalemia include using fresh blood (less than 14 days old), transfusing blood products through lines further away from the right atrium, and using washed RBCs.[13,14] In addition, correcting acidemia will prevent the extracellular shift of potassium. It is important to frequently check plasma potassium levels to detect hyperkalemia and treat elevated levels appropriately with standard therapies such as insulin with dextrose, β-2 (adrenergic) agonists (when blood pressure allows), bicarbonate, and intravenous calcium.

In addition to the effect on potassium level and pH, the storage of RBCs alters two other properties that change the ability of transfused cells to deliver oxygen to tissues. RBC deformability, which allows RBCs to navigate microvasculature, decreases with storage. In addition, 2,3-diphosphoglycerate decreases in stored RBCs, therefore effectively increasing hemoglobin's affinity for oxygen and decreasing the unloading of oxygen at tissues.[13] During massive transfusion in trauma, it is important to realize that older units of RBCs will not deliver oxygen to hypoperfused tissues as well as newer ones.

Although severely traumatized patients may present with an endogenous coagulopathy due to the nature of their injury and hypoperfusion, now referred to as acute traumatic coagulopathy, resuscitative efforts and the combination of acidosis, hypothermia, and coagulopathy (often referred to as the "bloody vicious cycle") may also contribute to the overall trauma-induced coagulopathy.[14] It is therefore essential that each of these components be carefully monitored and any problems promptly treated.

Acidosis

During trauma-related hemorrhage, acidosis mainly results from hypoperfused tissues, producing lactate. However, massive transfusion can worsen this acidemic state because stored RBCs are acidic due to the citrate phosphate dextrose adenine (anticoagulant) solution in which they are suspended, as well as the accumulation of the products of continuing cellular metabolism.[14] Stored RBCs have a pH of 7.16 at the time of collection and progressively become more acidic, with a pH of 6.87 at 21 days and 6.73 at 35 days. Once transfused, the acid present in blood products is immediately metabolized by the liver, but this typically rapid process may be impaired and overwhelmed during massive transfusion in the face of hemorrhagic shock. The injudicious administration of large volumes of nonbuffered crystalloid solutions may also lead to worsened acidemia, because the hydrogen ion is dissociated from water due to high levels of chloride relative to sodium. The physiologic consequences of acidemia include dysrhythmias, decreased cardiac contractility, hypotension, and decreased response to catecholamines. In addition, acidosis has been found to independently lead to coagulopathy because an acidic environment in the blood leads to decreased enzymatic activity of clotting factors, reduced thrombin genera-

tion, and impaired platelet aggregation.[13] For example, it has been shown that at a pH of 7, the activity of factor VIIa, VIIa/tissue factor complex, and factor Xa/Va complex decreases by 90%.[19] Acidosis attributable to massive transfusion can be lessened by using fresher blood and treated with alkalinizing solutions, such as sodium bicarbonate or tromethamine although the use of these agents is contentious in terms of both efficacy and necessity. Tromethamine is currently licensed and widely used in the United States but not in the United Kingdom.

Hypothermia

Hypothermia, defined as core body temperature below 36°C, is commonly seen in trauma patients and is associated with an increased risk of uncontrolled bleeding and mortality. Patients can become hypothermic from the environmental conditions at the time of injury, during evacuation, and during exposure for examination and surgery. Also contributing to hypothermia is the impaired thermoregulation related to shock and anesthesia.[13] In addition, infusing large volumes of inadequately warmed intravenous fluid and blood products will contribute to the dangerous hypothermia that is too often seen in trauma. Blood products are normally stored between 1°C and 6°C, and for this reason it is imperative that fluid warmers be used during transfusion.[14] Because serious physiologic effects of hypothermia include impaired oxygen delivery by hemoglobin, decreased cardiac output, increased risk of cardiac dysrhythmias, and increased cardiac toxicity from electrolyte derangements, maintaining normothermia is essential. In addition, hypothermia contributes to coagulopathy, affecting both platelet function and the coagulation cascade. Platelet dysfunction occurs as hypothermia leads to reduced thromboxane A_2 production, impaired platelet adhesion and aggregation, and decreased generation of thrombin on platelets.[13] The coagulation cascade is affected as reduced temperatures impair the activity of coagulation enzymes, resulting in a 10% reduction in coagulation factor activity for each 1°C reduction in temperature.[19] These platelet and coagulation derangements are resolved as the temperature returns to 37°C, emphasizing the importance of being vigilant with respect to patient temperature during massive transfusion.[20]

Dilutional Coagulopathy

In addition to the detrimental effects on coagulation of acidosis, hypothermia, and the consumption of clotting factors, massive transfusion of blood products can itself contribute to coagulopathy. This dilutional component of coagulopathy is the result of replacing lost blood with large volumes of stored RBCs and fluids that do not contain clotting factors or platelets. Dilutional thrombocytopenia associated with massive transfusion was seen in both the Korean and Vietnam conflicts because stored whole blood, which does not have functional platelets, was used extensively. In addition to low platelets, labile clotting factors such as V and VIII deteriorate with blood storage times.[13,21]

Today, fractionated component transfusions are most commonly used and involve the transfusion of PRBCs, FFP, and platelets. Although this practice has been proven to result in less dilution when compared to transfusing PRBCs alone or stored whole blood, the risk of thrombocytopenia, hemodilution, and a resulting coagulopathy still exists. The reason for the continued risk of coagulopathy with blood component therapy is that when whole blood is used to make these three components, RBCs, platelets, and factors are diluted through processing and the addition of preservatives.[13] The resulting 660 mL achieved when recombining one unit of each component results in a net deficit, with the reconstituted "whole" blood having a hematocrit of 29%, a mean platelet count of 85,000/μL, and a mean coagulation factor activity of 62%.[14] During the current conflicts interest has resurged in the use of fresh whole blood (FWB) for resuscitating combat casualties; 6,000 units of FWB were administered by the US military to casualties in Iraq and Afghanistan, and many practitioners would argue for its use in cases of refractory coagulopathy.

In addition to issues with blood component therapy, infusing crystalloid or colloid into the bleeding patient results in the further dilution of cells and clotting factors, contributing to coagulopathy. Colloids such as hydroxyethyl starch have been shown to impair von Willebrand factor activity in plasma.[22] As a result it is now standard in military practice to limit crystalloid or colloid infusion as much as possible.

Because conventional coagulation tests were not designed for monitoring transfusions and are time-consuming relative to the rapidly changing situations in casualty resuscitation, the use of real time thromboelastometry (ROTEM; TEM UK Ltd, Hartlepool, UK) may help to assess the patient's current coagulation state and guide further transfusions. The potential value of ROTEM is under evaluation.

Immunologic Complications

Immunologic complications of massive transfusion include acute hemolytic reactions, which are very rare when units of "trauma blood" (uncross-matched

type O) are used. However, acute hemolytic reactions can be observed after patients have received large amounts of type O whole blood and then receive type-specific blood. This is due to transfused isoagglutinins from whole blood reacting against type A or B antigens found in type-specific blood.[23] Microchimerism (the harboring of small numbers of cells that originated in a genetically different individual) is another immunologic-related complication and involves the persistence of allogeneic cells for years posttransfusion. It can be seen in up to 10% of trauma patients receiving transfusions, but its clinical significance remains unknown. Another immune-related process is immunomodulation. Although its exact mechanism of action remains unclear, immunomodulation has been associated with an increased risk of bacterial infection (especially with older PRBC units), acute lung injury or acute respiratory distress syndrome, systemic inflammatory response syndrome, and multiple organ failure.[13,24]

The leading cause of transfusion-related death, with a reported mortality rate of about 25%, is transfusion-related acute lung injury (TRALI).[19,25] TRALI typically occurs within 6 hours after transfusion, but can occur up to 24 hours later. The type of blood product transfused determines the risk of TRALI, which has been found to be 1 case per 5,000 units of PRBCs, 1 per 2,000 units of FFP, and 1 per 400 units of platelets.[14] Clinically, it is indistinguishable from acute respiratory distress syndrome and involves acute onset of noncardiogenic pulmonary edema; severe hypoxia; and bilateral, fluffy infiltrates on chest radiograph. It is thought to be an immune-mediated process, wherein donor antibodies activate recipient leukocytes, causing pulmonary injury through microvascular occlusion, endothelial damage, and capillary leakage.[25] Treatment is supportive critical care including mechanical ventilation, fluids, and inotropes as needed.[25] TRALI should be distinguished from transfusion-associated circulatory overload (although the distinction is sometimes unclear), which is hydrostatic pulmonary edema occurring in approximately 1% of transfusions.[14,19]

PRINCIPLE CONSIDERATIONS IN MASSIVE TRANSFUSIONS DURING MILITARY OPERATIONS

Given the risks outlined above, the overarching goal in managing massive transfusions for combat casualties must be to prevent the exacerbation of coagulopathy while avoiding unnecessary use of blood products. To this end the UK and US military have both published documents laying out the principles involved in damage control resuscitation in general and massive transfusion in particular. These are the *UK Armed Forces Surgeon General's Policy Letter on the Management of Massive Hemorrhage On Operations* (dated 27 February 2009)[7] and the *US Joint Theater Trauma System Clinical Practice Guideline on Damage Control Resuscitation At Level IIb/III Treatment Facilities* (dated 10 August 2011).[8]

UK Operational Massive Transfusion Protocol

The 2009 UK policy letter describes the massive transfusion protocol adopted for UK military operations as an "aggressive massive transfusion protocol based on a 1:1 ratio of red cell concentrate (RCC) to FFP with platelet component support when needed" (Exhibit 8-1). It differs from the US equivalent in that platelets and cryoprecipitate are administered only on an as required basis. The policy outlines the following approach to massive transfusion:

1. Avoid hypothermia by using fluid warmers and rapid infusion devices.
2. Maintain hematocrit at 35%.
3. Use FFP and RCC in a 1:1 ratio as soon as practicable.
4. Use cryoprecipitate early to maintain the level of fibrinogen above 1.0 g/L.
5. Initiate early intervention with platelet support to maintain the platelet count above 100×10^9/L using UK-derived (or more local source if appropriate) platelet components, or platelets donated using field apheresis, both in preference to whole blood from the emergency donor panel (a group of preidentified and screened blood donor volunteers readily available to the field hospital).
6. Frequently take a full blood count and conduct coagulation studies to confirm successful application of the MTP.
7. Frequently measure potassium and calcium levels to identify the presence of hyperkalemia or hypocalcemia, followed by appropriate therapy as needed.
8. Use appropriate intervention with recombinant factor VIIa in accordance with current military guidelines.
9. Regularly assess the base deficit (along with hypothermia and coagulopathy) to monitor the lethal triad associated with massive trauma.

US Military Massive Transfusion Protocol

Not surprisingly, the US military has focused an enormous amount of time and research on developing

MTPs during the wars in Iraq and Afghanistan. Much of this research was done at the US Army's Institute of Surgical Research (USAISR) at Fort Sam Houston, Texas, which has the mission of "providing requirements-driven combat casualty care medical solutions and products for injured soldiers." Among much of the valuable literature published by the USAISR are the Central Command/Joint Theater Trauma System Clinical Practice Guidelines (http://www.usaisr.amedd.army.mil/clinical_practice_guidelines.html). These generally evidence-based guidelines represent the US military's current thinking on a host of medical issues, including massive transfusion. The Clinical Practice Guideline covering damage control resuscitation and massive tranfusions[8] includes as an appendix the example of an MTP for a CSH (Exhibit 8-2).

FIBRINOLYTICS AND RECOMBINANT FACTOR VIIA

The use of antifibrinolytics such as tranexamic acid should also be considered to counter the hyperfibrinolysis that can be a feature of as acute traumatic coagulopathy. A randomized controlled trial of tranexamic acid, called CRASH-2, revealed a significant decrease in all-cause mortality and death due to bleeding in bleeding trauma patients treated with the drug; the effect was most apparent in patients administered the drug less than 3 hours after injury. As a result the investigators recommended that tranexamic acid be administered as soon as possible after injury[26]; the drug is available for use in the prehospital phase of resuscitation in Afghanistan by UK MERT-E clinicians.

When coagulopathy persists despite appropriate blood product therapy, the use of recombinant factor VIIa has been considered to initiate a thrombin burst at the sites of injury. The safety of this off-label use of factor VIIa approach has, however, been questioned.[27] A study of 328 massively transfused trauma patients revealed a significantly improved 24-hour survival but no benefit in late survival to discharge.[28] In the authors' opinion, the use of factor VIIa has decreased as the use of higher ratios of FFP to PRBC has become standard, although there may be a place for factor VIIa on military operations where there is limited transfusion capability. The initial dose is 10 μg/kg IV, which may be repeated after 15 to 20 minutes.

MILITARY USE OF FRESH WHOLE BLOOD

FWB has been used in the trauma setting since World War I, serving as an ideal resuscitation fluid, intuitively appealing in that it "replaces what is bled." However, with the development of fractionation of whole blood into PRBCs, platelets, FFP, cryoprecipitate, and various concentrated coagulation factors, component therapy (CT) supplanted the use of whole blood in the operating room. The use of CT over FWB in the trauma setting therefore developed in part as an untested extension of CT's widespread use in the elective surgical world, and to preserve valuable resources. Trauma resuscitation strategies using CT were extrapolated from studies of euvolemic patients undergoing elective surgeries, resulting in unproven blood product recipes that relied heavily on crystalloid fluids and front-loaded PRBCs, preserving more precious blood products such as FFP and platelets until blood samples, often drawn during a hectic resuscitation, demonstrated their need.

Blood fractionation capabilities are not always in place in an austere combat theater of operation at the time of arrival of troops and casualties, as was the case in the wars in Iraq and Afghanistan, where trauma resuscitation early in the conflict included the use of both CT and FWB. At the time many surgeons and anesthesiologists using FWB were impressed with its efficacy and convenience. Favorable editorials and research supporting the use of FWB for trauma ensued, such as a retrospective analysis by Spinella et al, which looked at 354 US combat casualties comparing two groups: (1) those who received warm FWB, RBCs, and plasma, but not apheresis platelets, and (2) the CT group, who received RBCs, plasma, and apheresis platelets, but not warm FWB. This study found improved 24-hour (96% vs 86%, $P = 0.018$) and 30-day survival (95% vs 82%, $P = 0.002$) among the FWB group.[29]

"Walking blood banks" (ie, using FWB taken directly from volunteer donor soldiers) became commonplace in US CSHs in Iraq and Afghanistan, and by the summer of 2007 over 6,000 units of FWB were transfused to combat casualties. The procedures for walking blood banks differ depending on the level of care and within military medical facilities themselves. In general, however, donor pools consist of local service members within the hospital or at nearby military units. Military medical facilities that anticipate large transfusion requirements often develop lists of prescreened potential donors based on blood type. When the walking blood bank is activated, donors are gathered, rescreened for recent changes to their medical history, tested for anemia using a copper sulfate test, and then cross-matched to the recipient's blood. Blood is then collected into 400- to 500-mL bags containing citrate phosphate dextrose adenine and imme-

EXHIBIT 8-1

UNITED KINGDOM OPERATIONAL MASSIVE TRANSFUSION PROTOCOL

Step 1. Primary clinical assessment of a casualty at risk of requiring massive transfusion support

If patient has:

- severe injury (eg, bilateral proximal amputations or truncal bleeding and one proximal amputation) OR clinically obvious massive trauma or hemorrhage,
- PLUS either temperature < 96°F or 35°C or systolic blood pressure < 90 mmHg or abnormal mental status,

then proceed to Step 2. (Secondary laboratory assessment criteria are INR > 1.5, base deficit of > 6 and Hb < 11 g/dL. These results support the requirement for massive transfusion but are not required to activate the protocol.)

↓

Step 2. Activate massive transfusion protocol

↓

Step 3. Action

Clinicians:

- Demand issue of a "shock pack."
- Send samples for FBC, PT, APTT, fibrinogen, cross match, U&E, calcium, blood gases, and base deficit testing.
- Actively avoid hypothermia by passing all replacement fluids to be given through a blood warmer or rapid infusion device.
- Monitor the FBC, coagulation, blood gases, U&E, calcium (and lactate) closely once the patient is out of the "shock phase." In the meantime, the calcium and potassium levels should be aggressively managed.

Laboratory (upon receipt of shock pack request):

- Issue four units compatible RCC[1] (group O Rh D-negative unless lab testing for casualty was previously undertaken) and four units of FFP (group AB).[2]
- Ensure sufficient numbers of staff are in the laboratory to provide a rapid response to the developing situation.[3]
- Defrost and store at 4°C six more units of FFP.
- Prepare to issue a further six units of RCC (either group-specific or fully cross matched depending on the time scale available).

↓

Step 4. Continuing requirement for massive transfusion

Laboratory issues:

- six units of RCC (preferably group-specific or fully cross matched, depending on time frame), and
- six units of FFP (group selected).

Laboratory prepares to issue:

- cryoprecipitate if required,[4]
- platelets (to maintain platelet count above 100 x 10^9/L),[5] and
- six units of FFP (unless the EDP has been used to supply whole blood).

↓

Step 5: Requirement for massive transfusion support continues

Laboratory issues:

- six units of group selected RCC,[6]
- six units of FFP.
- platelets (dosage is dependant on FBC results; each adult equivalent dose of platelets can be expected to increase the platelet count by 30 to 40 x 10^9/L), and
- cryoprecipitate (dosage is dependent on fibrinogen results or clinical assessment).

Consider use of rVIIa.

Laboratory actively manages blood stocks and requests urgent resupply if appropriate.

↓

Step 6. Requirement for massive transfusion support continues[7]

Repeat Step 5.

Consider giving one unit FFP, one pool of cryoprecipitate, one unit of platelets and rVIIa ("Bastion glue"), or using of cross-matched fresh whole blood derived from the EDP.

---

1. The first 10 units of RCC issued must, as soon as possible, be retrospectively cross-matched.
2. It may be appropriate, during high-tempo operations or following notification of the imminent arrival of a severely injured casualty, for the laboratory to anticipate the need for a massive transfusion and defrost four units of FFP (and hold them for up to 5 days at 4^0 C). This approach will result in more waste but support the aggressive treatment required.
3. The laboratory may well need to suspend nonurgent testing during a massive transfusion situation.
4. The dosage of cryoprecipitate given should, when possible, be modified depending upon the results of fibrinogen tests to avoid unnecessary donor exposure.
5. In severe trauma one unit of platelets may be required for every 2.5 units of red cells given.
6. There is no requirement to fully cross match RCC after the first 10 units have been transfused in a massive transfusion situation UNLESS the patient has a clinically significant antibody. All patients should be converted to Rh D-positive units at this stage (to conserve Rh D-negative stock) UNLESS they are female of child-bearing age. If the patient has a clinically significant antibody, it may be necessary to deliberately select incompatible units during the mid-phase of a massive transfusion in order to preserve the compatible blood for use once hemostatic control has been achieved.
7. There is no clear threshold beyond which blood usage is futile. There is, however, a need to ensure that blood stocks are not exhausted in a futile effort to save a life. Close liaison, detailed attention to stock management, and effective communication is essential.

APTT: activated partial thromboplastin time
EDP: emergency donor panel
FBC: full blood count
FFP: fresh frozen plasma
Hb: hemoglobin
INR: international normalized ratio
PT: prothrombin time
rVIIa: recombinant factor VIIA
RCC: red cell concentrate
U&E: ureas and electrolytes

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diately transfused to the patient. In ideal conditions, a military medical facility with a well-rehearsed walking blood bank program can have warm FWB transfused within 20 to 30 minutes of activation.[30]

The risk of infection using walking blood banks is thought to be minimized by the reliance on service members, who are screened for HIV every 2 years (and often retested prior to deployment), must be vaccinated for hepatitis B, and undergo routine screening for illicit drug use. In an effort to further diminish the infectious risk, some military medical facilities in Iraq and Afghanistan with high-volume FWB requirements sent samples from potential donor pools back to the US for formal screening for transfusion-associated diseases before collecting blood.[31]

Despite these efforts, the risk of transfusion-acquired infection is inherently higher with FWB compared to the more rigorously screened CT blood products, and for this reason the Food and Drug Administration does not approve the use of FWB in the United States. In 2011, a study undertaken by the US Armed Services Blood Program Office (and other institutions) looked at blood samples from 761 service members taken before and after they received emergency transfusions of FWB in Afghanistan and Iraq. The study determined that one service member acquired a hepatitis C infection from a transfusion and four service members had hepatitis C infections prior to their injuries and subsequent transfusions. Because these four service members were themselves potential donors, the finding suggests that the service-member donor pool was not as safe as first thought.[32]

The USAISR annually reviews and modifies its Clinical Practice Guidelines for the use of FWB (originally released in 2006). The current guidelines describe the advantages of FWB as providing blood product in a favorable 1:1:1 ratio, its availability in austere conditions, and that it has no loss of clotting factor, platelet activity, or RBC "storage lesion" compared to CT. Among the principal disadvantages of FWB are that it must be ABO-type specific because it contains both RBCs and plasma, which creates a greater op-

EXHIBIT 8-2

EXAMPLE OF A MASSIVE TRANSFUSION PROCEDURE AT A US CENTRAL COMMAND ROLE 3 FACILITY

The following flexible massive transfusion (MT) procedure can be used in the emergency department (ED), operating room (OR), or intensive care unit (ICU). It may be initiated or terminated by the site-specific provider as dictated by the patient's needs in each specific venue. It consists of batches or packs, as defined below, which vary in composition but should approximate a 1:1:1:1 ratio of packed red blood cells (PRBC), fresh frozen plasma (FFP), platelets, and cryoprecipitate.

Pack One: Four units of PRBC and four units of FFP. Additionally, consider using six packages of platelets, one 10-unit bag of cryoprecipitate, and possibly factor VII (obtained from the pharmacy). Use emergency release blood. Strongly consider the early use of tranexamic acid: infuse 1 g of tranexamic acid in 100 mL of 0.9% normal saline over 10 minutes intravenously (IV) in a separate IV line from any containing blood and blood products. (More rapid injection has been reported to cause hypotension.) Hextend (Hospira, Lake Forest, IL) should be avoided as a carrier fluid. Infuse a second 1-g dose intravenously over 8 hours infused with 0.9% NS carrier.

Pack Two: Four units of PRBC and four units of FFP.

Pack Three: Four units of PRBC, four units of FFP, six packages of platelets, one 10-unit bag of cryoprecipitate, and consider factor VII (obtained from the pharmacy).

Pack Four: Four units of PRBC and four units of FFP.

Pack Five: Four units of PRBC, four units of FFP, six packages of platelets, and one 10-unit bag of cryoprecipitate. At this time, providers should reassess the progress of the resuscitation, hemostasis, and the need to continue the MT procedure.

Packs Six, Seven, Eight and **Nine** are identical to **Packs Four** and **Five**.

Emergency release: four units of uncross-matched PRBC (two units of O+ and two units O-) and four units of AB or A FFP. (A FFP is not a universal donor product, but its use in MT patients when supplies of AB FFP are limited or absent may improve survival and help preserve resources, with a low risk to the patient. The decision to use A FFP or to switch from AB FFP to A FFP in the same patient should made by the medical and surgical staff in concert with laboratory staff. Once the patient's type has been identified, type-specific plasma should be given as soon as possible.)

Pack: A single group of type-specific, cross-matched PRBC and FFP (four units of each), which later in the procedure may also include cryoprecipitate, platelets, and/or factor VII.

portunity for clerical errors. Moreover, the collection of FWB usually creates diminished exercise tolerance in donors, who may be members of the wounded soldier's own unit (and thus may still be needed in ongoing combat). For these reasons and the known increased risk of transfusion-acquired infections, the Joint Theater Trauma System Clinical Practice Guideline recommends that FWB be reserved for severe casualties expected to need massive transfusions (10 or more units within 24 hours) with clinically significant shock or coagulopathy, when other blood products are not available, are not effective, or cannot be delivered rapidly enough to resuscitate an actively bleeding patient.[8] Meanwhile, until the appropriate place for FWB in massive transfusion has been established, UK military policy is to confine its use to situations where full CT is not yet available in theater or when coagulopathy persists despite targeted CT.

SUMMARY

Hemorrhage remains the most common cause of death in combat, and in many cases these deaths are preventable with clinical vigilance and proactive care. Massive transfusion of blood products is a key part of the damage control resuscitation paradigm conceived to manage these severely injured casualties from point of wounding to critical care (described in Chapter 7, Damage Control Resuscitation). The earlier the appropriate transfusion of blood products is initiated, the better the chance of survival; however, prediction tools are not yet adequately sensitive or specific, so when to initiate MTPs remains a clinical decision, particularly during the prehospital phase.

Lieutenant Colonel Primrose noted during World War I that the first result of blood transfusion in the treatment of hemorrhage is that "*it increases the power of coagulation of the blood*."[1] This lesson has been relearned in recent conflicts so that now the purpose of transfusion

is not merely the replacement of volume and oxygen-carrying capacity, but also the active prevention or treatment of coagulopathy. The complications of transfusion described above are those most likely to be of concern during or soon after initial resuscitation, requiring attentive monitoring and treatment in the field hospital.

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Chapter 9

PERIOPERATIVE AND INTER-OPERATIVE CRITICAL CARE

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INTRODUCTION

Decision-making and treatment for physiological correction following major combat-related injury can have effects beyond the initial surgical and anesthetic interventions. It is vital that the entire clinical team (including the intensive care physician) is involved as early as possible in the treatment pathway. The treatment of these patients can be protracted (commonly referred to as the prolonged-care phase), and the initial injury may become of secondary importance to the effects of systemic inflammatory response syndrome (SIRS), acute lung injury (ALI), nosocomial infection, and intercurrent multiorgan dysfunction syndrome (MODS). Patients with multiple injuries often require lengthy periods of mechanical ventilation. The surgical approach to the most injured patients has changed in recent years with the adoption of damage control surgery and resuscitation.[1,2] The triad of hypothermia, acidosis, and coagulopathy, as well as complications such as abdominal compartment syndrome, require surveillance and management prior to and after admission to the intensive care unit. Recent research and clinical work have clearly shown that the early phase of trauma is characterized by a marked inflammatory response. Modifying this response in the initial treatment and early intensive care phase has the potential to mitigate its progression and impact on end organs.[3,4]

Trauma patients requiring intensive care interventions generally fall into two groups: (1) those immediately requiring organ support upon admission to the hospital, and (2) those experiencing the later complications of trauma, such as SIRS, sepsis, ALI, or MODS. The first group includes patients with thoracic injuries, major head injuries, or circulatory shock, as well as those who require an extended recovery period following resuscitative surgery. For service personnel, extended care and rehabilitation are usually delivered back in their home nations. Local civilians may pose particular challenges because their medical requirements may exceed the capacity of facilities within the theater of operations to manage their care in the medium to long term (see Chapter 42, Ethical Challenges of Deployed Military Critical Care).[5–7]

INITIAL MANAGEMENT

Treatment of trauma patients begins with the standard <C>ABC (catastrophic hemorrhage, airway, breathing, circulation) assessment by the trauma team. Intensive care specialists should be represented within the trauma team; this is often in the role of an anesthesiologist, whose responsibility goes beyond assessment of the airway (Exhibits 9-1 and 9-2, and Figure 9-1). Knowledge of the injury mechanism allows potential injuries to be identified or ruled out; specifically, the team must know whether the injury was blunt or penetrating and if it involved blast and penetrating fragments. The team should be aware that although most cases of shock in trauma will be hypovolemic, shock may have other causes (eg, cardiogenic from a myocardial injury, sepsis if the presentation is delayed, tension pneumothorax); the injury mechanism can assist with this diagnosis.

The concept of damage control resuscitation is now well established and over the last decade has become the accepted method of managing unstable trauma patients. Damage control surgery is a component of damage control resuscitation, and resuscitation occurs both before and after the surgical intervention. Major hemorrhage from amputated limbs or internal hemorrhage requires immediate surgical intervention to gain proximal vascular control. Hemorrhaging patients require endotracheal intubation concurrently with insertion of large-bore venous access and hemostatic resuscitation. The patient may be resuscitated in the emergency department first or taken immediately to the operating room.[8]

IDEAL ASSESSMENT REGIME

History

Attempted verbal communication gives a good indication of whether the patient has a patent airway (is able to speak clearly), has sufficient respiratory drive (is able to speak in full sentences), has sufficient blood pressure to provide adequate cerebral perfusion, and can facilitate determination of the Glasgow coma score and level of pain. Asking the patient to cough gives valuable quick information about respiratory capacity. An AMPLE history (Exhibit 9-3) can be taken in less than 30 seconds.

Airway

Airway can be assessed within the first few seconds. A patent airway without requirement for airway adjuncts or jaw thrust, in a conscious patient, can be considered to be stable. A rapid sequence induction of anesthesia (RSI) should be considered urgently in

EXHIBIT 9-1

IMMEDIATE REQUIREMENTS AND DECISION POINTS IN TREATING THE SEVERELY INJURED PATIENT

1. Consider the injury mechanisms and potential structures at risk in blunt and penetrating trauma.
2. Initiate damage control resuscitation and massive transfusion protocol.
3. Administer appropriate and timely airway and ventilatory interventions.
4. Accurately assess volume status, particularly in patients in compensated shock.
5. Achieve appropriate endpoints of preoperative and intraoperative resuscitation.
6. Use advanced monitoring to aid resuscitation.
7. Assess requirement for intensive care and communicate with nurse in charge.

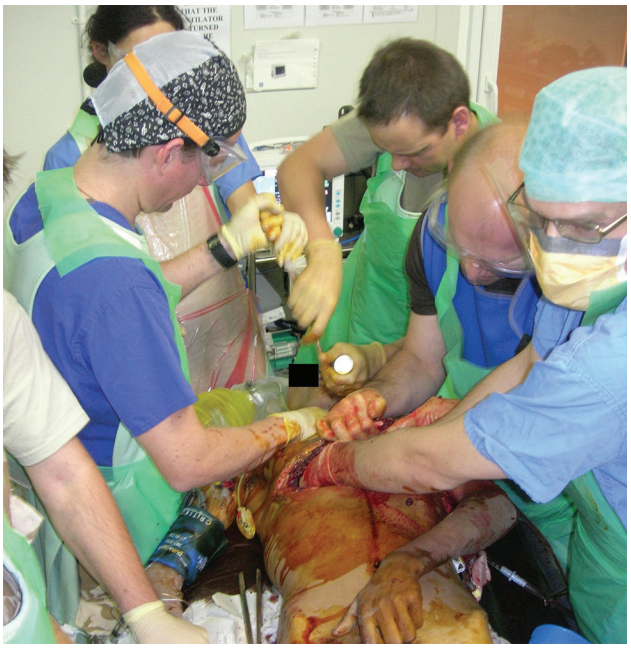

Figure 9-1. The trauma team in action: simultaneous resuscitation and damage control surgery.

patients with conditions listed in Exhibit 9-4. The last two indications listed are subject to debate because of concerns about physiological decompensation. RSI requires an appropriate patient assessment and appropriate drug dosages for the patient's condition, as described below. There is usually no good reason to excessively delay the provision of anesthesia and airway support to severely injured patients. Most trauma patients will have a hard collar in place for C-spine protection. This collar should be removed for intubation, with manual in-line stabilization maintained throughout the RSI. The need for C-spine control implies that all trauma intubations should be considered more challenging than elective intubations, and a bougie should be routinely used. The importance of trained assistants in trauma airway management cannot be overstated.

Although no induction agent is ideal, most can be used appropriately if titrated according to the hemodynamic status of the patient. Most anesthetic agents are direct vasodilators and negative inotropes. Even

EXHIBIT 9-2

EARLY INTENSIVE CARE REQUIREMENTS FOR THE SEVERELY INJURED PATIENT

1. Ensure safe transfer to the CT scanner, the operating theater, or the intensive care unit.
2. Clear the cervical spine in a sedated and ventilated trauma patient.
3. Determine timing of fracture fixation and further surgery.
4. Achieve medium and longer-term resuscitation endpoints.
5. Follow aseptic strategies for mitigation of central venous catheter bloodstream infections.
6. Instigate enteral nutrition.
7. Perform an early rehabilitation assessment.

CT: computed tomography

EXHIBIT 9-3

THE "AMPLE" HISTORY

Allergies

Medications

Past medical history

Last food or drink

Everything else: history of incident including mechanism, time, etc

EXHIBIT 9-4

CONDITIONS REQUIRING RAPID SEQUENCE INDUCTION OF ANESTHESIA

1. Actual or impending loss of airway.
2. Reduced consciousness level; patient is unable to maintain own airway.
3. Head injury, especially if patient is combative or has a low GCS.
4. Injuries or decreased level of consciousness causing ventilatory failure.
5. Likelihood of immediate surgical intervention being required on arrival at surgical facility due to injury pattern.
6. Humanitarian reasons (distress and pain relief).

GCS: Glasgow coma score

agents, recommended for their cardiovascular stability, such as etomidate and ketamine, have indirect negative effects mediated through reduction of circulating catecholamine levels in patients who are in pain or otherwise physiologically stressed.[9] Induction doses should be reduced by up to 50% to 90% in hemodynamically compromised patients, and maintenance doses should be started at low levels and titrated upward only as tolerated. An induction with propofol reduced to as little as one tenth of normal induction doses has been shown to produce equivalent reductions in cerebral electrical activity when administered to an animal in shock, even after adjustment for a reduced blood circulating volume.[10]

Although hemorrhage control should not be delayed to pursue resuscitation, bolus administration of fluid at the time of anesthetic induction may help to prevent catastrophic vascular collapse. In the hemorrhaging patient, this bolus therapy should be carefully monitored and balanced against the administration of anesthetics to preserve a state of controlled hypotension.[11] Practice varies depending on the provider's training and background.

Clearance of the Cervical Spine

Cervical spine injury occurs in 5% to 10% of blunt polytrauma patients. Despite several published clinical guidelines, treatment remains controversial. The application of these guidelines to the obtunded trauma patient is limited. The presence of a severe head injury increases the relative risk of a cervical spine injury by as much as 8.5 times, and the risk of focal neurological deficit by 58 times.[12] When the patient is unlikely to be fully evaluated within 24 hours, complications associated with prolonged immobilization shifts the risk–benefit analysis: rather than waiting for an opportunity to do a full clinical evaluation, providers should opt for a nonclinical clearance, given that the vast majority (90% –95%) will not have a cervical injury. A protocol using computed tomography (CT) scanning for blunt trauma patients who were obtunded has shown the risk of missing a cervical spine injury to be 0.04%.[13] In any of the following circumstances casualties should undergo CT imaging of the cervical spine as the primary imaging modality:

- Glasgow coma score below 13 on initial assessment;
- the patient is intubated;
- plain film series is technically inadequate (for example, desired view unavailable), suspicious, or definitely abnormal;
- continued clinical suspicion of injury despite a normal x-ray; or
- the patient is being scanned for multiregion trauma.

Many combat trauma patients have one or more of these risk factors after complex blast injuries, so the early involvement of a radiologist is essential.[12–14]

Ventilation

Pulmonary dysfunction in trauma patients is multifactorial and may result from direct contusion of the lung tissue, lung injury by fractured ribs, loss of chest wall function, fat embolism to the lung from long bone fractures, aspiration of blood or gastric contents, the activation of SIRS, shock, reperfusion, or transfusion therapy.[15,16] Ventilatory failure can have a profound effect on physiology.

The lungs are especially susceptible to injury from blast shock waves because of the contrast between tissue density and the gas inside. A specific syndrome of lung damage can result from blast shock waves: "blast lung" is a progressive condition characterized by the development of pulmonary inflammation and edema following initial intrapulmonary hemorrhage. These conditions can cause a decrease in pulmonary gas transfer, initially leading to hypoxia and hypercarbia. The resulting lung damage is exacerbated by the other mechanisms of traumatic lung injury described.[17–19]

Pneumothoraces are frequent in trauma patients and should be actively excluded in the initial examination. They may expand to produce tension physiology:

impaired ventilation due to decreased air entry and hypotension from vena caval compression. Mediastinal shift is common in the absence of tension physiology and cannot guarantee the diagnosis of tension pneumothorax.[20] Small pneumothoraces (visible on chest CT but not chest radiograph) may not require treatment.[21] However, pneumothoraces behave very differently in spontaneously versus mechanically ventilated patients. Rapid development of tension physiology is much more likely in the ventilated patient, so vigilance should be maintained. If in doubt, immediate decompression of the thoracic cavity either with a simple thoracostomy or chest drain is required.

It is important to reduce the additional iatrogenic effects of mechanical ventilation, which can cause barotrauma, volutrauma, and atelecttrauma[22–24] and potentiates circulating inflammatory markers (biotrauma).[25] Physiological tidal volumes and limiting the inspiratory plateau have been widely accepted in patients with ALI.[26,27] Because all trauma patients are at risk of developing ALI, strategies to protect the lung should be applied immediately following intubation, aiming for tidal volumes of 6 to 8 mL/kg and plateau airway pressures below 30 cm H_2O. This reduction in alveolar volume may result in alveolar derecruitment if insufficient positive end-expiratory pressure (PEEP) is applied to prevent alveolar collapse. The use of high PEEP to compensate for this de-recruitment may be associated with excessive lung parenchyma stress and strain, which may have the most impact on a severely injured lung after traumatic injury. However, stepwise titration of PEEP to higher levels has been shown to have positive effects in trauma patients.[28,29] A minimum PEEP of 5 cm H_2O should be applied and adjusted upward to ensure adequate oxygenation, with awareness that increasing the PEEP may precipitate hypotension in hypovolemic patients. In patients with head injuries, there may be a need to control P_{CO_2}, causing potential conflicts with the ventilation strategy employed to protect the lungs.[30–33]

In most cases conventional lung-protective ventilation has provided adequate respiratory support of blast casualties. A small number of these patients have required high frequency oscillation ventilation, but only after the first 24 hours of management. This type of ventilation is currently unavailable in the operational theater. [34]

Pump-less interventional lung assist (iLA) can be used in patients with acute respiratory distress syndrome (ARDS) to improve extracorporeal gas exchange by means of a membrane integrated into a passive arteriovenous shunt. Effective carbon dioxide removal through iLA has been demonstrated in early studies, but only a moderate improvement in oxygenation, with no survival benefit, in life-threatening hypoxemia and hypercapnia was shown.[35] More recently iLA has been used for extracorporeal carbon dioxide removal to enable lung-protective ventilation in patients with ARDS. In life-threatening gas exchange limitation, iLA has been used to facilitate lung-protective ventilation by enabling low tidal volume and reduced inspiratory plateau pressure.[36] Insertion of the arteriovenous iLA is not without risk, including critical limb ischemia, and high bilateral amputees may have difficult groin access. In the deployed intensive care setting it may be appropriate to consider iLA early to aid lung-protective ventilation and transfer to Role 4 in some multiply injured patients. The procedure has been used successfully in US soldiers being transferred from Iraq to higher echelons of medical care.[37]

Circulation

The decompensated, bleeding patient requiring massive transfusion should be immediately resuscitated as described in the massive transfusion chapter. The subsequent management of these patients becomes more challenging. End points in resuscitation (below) should be used to guide continuing volume resuscitation and thromboelastography (as described in other chapters), as well as the use of clotting product to correct the coagulopathy of trauma that will inevitably be present. Both the injury process and treatment can have a profound effect on the patient's physiology.

Recognition of a hemodynamically compensated trauma patient who is not obviously exsanguinating can be difficult; the use of pulse, respiratory rate, and blood pressure is neither sensitive nor specific for hemorrhagic shock.[38,39] Blood pressure is determined by the ratio between the functional capacity of the vascular system and the volume of fluid that fills it, along with the pumping power of the heart. Young adults may lose 30% of their circulating volume with little change in their vital signs, and up to 40% of the normal circulating volume can be lost before the limits of compensation are reached and catastrophic vascular collapse occurs. Again, knowledge of the injury mechanism is important.

Vascular Volume Status

The traditional approach to measuring intravascular fluid volume is changing. Routine clinical use of "gold standard" methods, such as central venous pressure (CVP) monitoring and pulmonary artery catheter monitoring, are declining. CVP correlates poorly with total blood volume, and does not always reliably predict fluid responsiveness, so its use in guid-

ing fluid management should be limited.[40,41] Like CVP, pulmonary artery occlusion pressure may fail to reflect changes in preload and may not always be suitable for predicting the response to further fluid administration. However, the effect of volume therapy can be detected in combination with other measures derived from the pulmonary artery catheter, such as cardiac output and mixed venous oxygen saturation. Decreased venous saturation is an indirect indicator of poor tissue perfusion and the need for resuscitation. Newer technologies are available to guide fluid resuscitation with a more dynamic approach, which works better than using historical static parameters. Determining where patients lie on their individual Starling's curve during the resuscitation process may be more important than the type of fluid being administered.[42]

Arterial pressure waveform systems function on the relationship between pulse pressure and stroke volume. Systolic pressure variation, the difference between maximum and minimum systolic pressure during one mechanical breath, has been shown to predict fluid responsiveness to volume loading. Concepts such as pulse pressure variation and stroke volume variation in ventilated patients have been extensively reviewed in the literature and found to be reliable predictors of volume responsiveness.[43] Arterial-based systems in clinical use today include the PiCCO (Phillips; Andover, MA); PulseCO (LiDCO Ltd; Lake Villa, IL); and the FloTrac/Vigileo (Edwards Lifesciences; Irvine, CA). These systems are all minimally invasive.[43]

Stroke volume variation and pulse pressure variation are more reliable indicators of volume responsiveness than CVP, artery occlusion pressure, left ventricular end-diastolic volume index, and global end-diastolic volume index. However, stroke volume variation and pulse pressure variation do have limitations in clinical use. They can be affected by alterations in ventilator settings, chest wall compliance, and dysrythmias, as well as by pharmacologically induced changes in ventricular and aortic compliance.[44]

Esophageal Doppler has been shown to be a clinically useful alternative to thermo-dilution in determination of cardiac output,[45–48] but the process shares a common problem of noninvasive monitors in that the interpretive algorithms have been developed and much of the clinical validation studies performed in relatively healthy and normal patients. However, in multiple trauma patients with at least 2 liters of blood loss, optimization of intravascular volume using esophageal Doppler was associated with decreased blood lactate levels, a lower incidence of infectious complications, and reduced duration of time in intensive care.[48]

Ultrasound assessment of the inferior vena cava to evaluate intravascular volume during resuscitation in trauma patients might be useful in the diagnosis of hypovolemia and is more sensitive than blood pressure alone.[49] A focused transthoracic echocardiographic assessment to evaluate cardiac function and volume status in trauma and critical care patients correlates well with data obtained from a pulmonary artery catheter.[50]

One of the most recent developments in technology is the development of tissue hemoglobin oxygen saturation (StO_2) monitoring. StO_2 has been shown to accurately correlate with peripheral oxygen delivery and to be predictive for those patients who are likely to decompensate and die early from exsanguinating hemorrhage. StO_2 employs near-infrared spectroscopy to permit continuous, noninvasive measurement of StO_2 in muscle. StO_2 is a parameter of tissue perfusion and oxygenation and performs similarly to base deficit in predicting the development of MODS or death after severe torso trauma.[49] It can be used to monitor resuscitation status and guide therapeutic end points in severely injured trauma patients.[50,51]

The splanchnic bed is very sensitive to hypoperfusion and is thought to play a major role in postinjury multiorgan failure.[52] Tissue oxygen tension measurements of the deltoid muscle reflect liver oxygen tension and may serve as a surrogate marker of splanchnic perfusion.[53] As such, these measurements may be used as an index of adequate resuscitation and a predictor of infection and multiorgan failure.[54]

Sublingual and buccal capnometry may be useful in identifying patients in a state of occult circulatory failure and in predicting survival in the trauma patient.[55,56] Although these techniques appear promising, large clinical studies are required to confirm their usefulness.

Correction of Base Deficit and Lactate

Lactate and base deficit have been used as parameters of tissue hypoperfusion and predictors of outcome in hemorrhagic shock. Elevated lactate levels on hospital admission and delayed normalization are associated with increased mortality.[57,58] The lactate level and base deficit should be used to identify patients who need more fluid resuscitation.[59]

The inability to normalize lactate is a predictor of death after trauma, but means to measure lactate may not be immediately available in every facility. It has commonly been thought that, in a normal acid-base environment, lactate would correlate with the anion gap and the base excess of an arterial blood gas. Neither anion gap nor base excess can be used to predict lactate levels; therefore, lactate must be directly measured. The lack of correlation between anion gap and base excess or lactate suggests the presence of unmeasured anions, an impairment in acid-base regulation after injury and resuscitation, or both.[60]

END POINTS IN RESUSCITATION

The goals of resuscitation include restoring circulatory volume via fluid resuscitation, restoring microcirculation, preventing clot disruption (thereby preventing re-bleeding), and maintaining adequate perfusion pressure to the brain and other vital organs. An adjunct to these goals is modification of the inflammatory process.

It is important to correct acidosis by resuscitating the patient to appropriate end points. Current monitoring technology now allows the physician to more rapidly identify the relationship between oxygen delivery and consumption (Exhibit 9-5). End points allow uniformity in gauging the adequacy of resuscitation: preventing under- and over-resuscitation and serving as a basis to compare outcome measures in resuscitation trials. Recent technological advances in patient monitoring allow a wider scope of clinical data to be obtained via less invasive means.

The expedient detection and correction of tissue hypoperfusion associated with compensated shock may limit organ dysfunction, reduce complications, and improve patient outcome. It seems logical that the earlier tissue hypoperfusion is detected and corrected, the greater the likelihood that outcome (ie, lactate and base deficit) will be improved to maximize oxygen delivery and correct tissue dysoxia. The use of StO_2 monitoring is undergoing evaluation in an operational setting. It is difficult to understand why esophageal Doppler technology in particular has not been evaluated in the same way. In the absence of specific cardiac output monitoring in current deployed operations, a combination of transthoracic echo assessment of both the heart and the inferior vena cava caliber on admission,[61] followed by CVP, pulse pressure observations, and regular measurements of base excess and lactate should be used to guide resuscitation.

HYPOTENSIVE RESUSCITATION

Uncertainty exists as to whether blood pressure and heart rate should be restored to normal before definitive hemorrhage control has been established. Work in 1994 showed better outcomes from patients with penetrating torso trauma if fluid resuscitation was delayed until surgical control was achieved.[62] The study patients had reached the hospital quickly, in less than 75 minutes (which happens with some but not all military casualties), and it is unclear whether the finding is applicable in cases of blunt trauma.[63]

Evidence from investigations into combined hemorrhage and blast injury showed that hypotensive resuscitation exacerbated a profound acidosis, possibly due to a compromise in tissue oxygen delivery, which is not compatible with survival after primary blast exposure.[64] Other studies have looked at intraoperative hypotensive resuscitation, initially in both blunt and penetrating trauma patients, then subsequently in only penetrating trauma patients. The researchers found that patients in the lower mean arterial pressure (LMAP) group, who were resuscitated to a mean arterial pressure (MAP) of 50 mmHg, received significantly less blood products and total intravenous fluids than those in the control high MAP (HMAP) group, who were resuscitated to a target MAP of 65 mmHg. The LMAP group also had a significantly lower mortality in the early postoperative period, and a significantly lower international normalized ratio in the postoperative period, indicating less severe coagulopathy. The study has limitations, however, and the results encompass only patients with penetrating injuries, but its findings support hypotensive resuscitation, particularly in penetrating injuries.[65]

As with ventilation, a dilemma exists in treatment of bleeding patients with traumatic brain injury.[66,67] Under-resuscitation, hypotension, and decreased cerebral perfusion can result in devastating secondary brain injury. In contrast, over-resuscitation in the face of ongoing hemorrhage increases blood pressure, reverses vasoconstriction, dislodges early thrombus, and causes dilution coagulopathy. These effects promote further bleeding and accelerate hypothermia, acidosis, and coagulopathy.[63] Fluid overload causes or exacerbates tissue edema, manifested by worsening ALI and brain edema in patients with head injury. Uncontrolled resuscitation is an independent predictor of secondary abdominal compartment syndrome, which is associated with MODS and a poor outcome.[68–70] Even after normalization of blood pressure and heart

EXHIBIT 9-5

IDEAL GOALS OF RESUSCITATION

1. Systolic blood pressure greater than 100 mm Hg.
2. Hematocrit greater than 30%.
3. Urine output at least 1 mL/kg/hour.
4. Base deficit on the arterial blood gases less than -3.
5. Cardiac index at least 4.5 L/min/m2.

rate, up to 85% of severely injured patients still have evidence of inadequate tissue oxygenation (ongoing metabolic acidosis), ie, they are still in compensated shock.[59]

ADJUNCTS AND FLUIDS IN TRAUMA RESUSCITATION

Fluids

Patients resuscitated with crystalloids require a larger amount of fluid to achieve the same end points of resuscitation as compared to plasma expanders, which results in more edema formation.[71] Colloids are generally considered to be a more effective volume expander than crystalloids. However, in the case of an impaired endothelial barrier and increased capillary permeability (as in trauma patients), this volume expansion effect is grossly reduced and is potentially counterproductive.[72,73] Several adverse effects (renal failure, bleeding complications, and anaphylaxis) have been reported with the use of artificial colloids. Colloids are not superior to crystalloids in treating hypovolemia in critically ill patients and show no survival benefit.[74,75] As a result, the use of crystalloids is currently recommended in trauma resuscitation.[76]

Hypertonic saline solutions are effective and well tolerated in the treatment of hypovolemic shock. Potential benefits are reduced requirements for fluids as well as less edema formation and immune modulation, thereby decreasing the risk of ARDS and MODS. Hypertonic saline is effective in reducing intracranial pressure in traumatic brain injury patients, but its use in trauma resuscitation is not associated with improved survival.[77]

Inotropic and Vasoactive Agents

Comparing the early use of vasopressors with the aggressive early use of crystalloid resuscitation in severely injured patients has revealed that early use of vasopressors almost doubles mortality.[78] Aggressive crystalloid resuscitation was independently associated with a survival benefit in the younger population (age < 55 years). The study concluded that early hemodynamic support in the trauma patient should rely primarily on aggressive fluid administration, and vasopressors should not be used in the early resuscitation period.[78] Beneficial effects of both arginine vasopressin and phenylephrine, as compared with crystalloid alone, have been found with traumatic brain injury, pulmonary contusion, and hemorrhagic shock. The Vasopressin in Refractory Traumatic Hemorrhagic Shock (VITRIS) study[79] is an international randomized controlled trial, recently initiated to assess the effects of arginine vasopressin in traumatic hemorrhagic shock patients who don't respond to standard shock treatment in the prehospital setting. Vasopressin may have three different beneficial effects: increasing blood pressure in refractory shock, shifting blood from a subdiaphragmatic bleeding site towards the heart and brain, and decreasing fluid resuscitation requirements.[79]

FRACTURE FIXATION

The optimal timing of fracture fixation in multiply injured patients is still widely debated. However, there is no doubt that early fixation of fractures reduces inflammation at the site of injury and decreases pain and opiate requirements. Evidence also shows that this approach reduces overall pulmonary complications and promotes early mobilization. Larger studies tend to indicate that the early stabilization of femoral fractures with definitive intramedullary nailing appears to be the treatment of choice, even for patients with combined head and chest injuries.[80–82] Practically, in the field hospital, external fixation will be the norm due to damage control resuscitation and infection control principles. It is vital that the timing of definitive fixation is discussed as part of the onward movement of the patient.

ADMISSION TO THE INTENSIVE CARE UNIT

When the patient first arrives in the intensive care unit, physiological correction and further evaluation of injuries must continue. The patient needs to be fully evaluated as quickly as possible so that all injuries and concurrent medical conditions are recognized.[83] The more severely injured patients, particularly those with traumatic brain injury, are at the greatest risk for occult lesions. A common pitfall is to focus only on the immediately life-threatening wounds, while inadvertently ignoring less obvious but potentially debilitating injuries. Repeated limb compartment checks and continued presence of distal pulses must be recorded in all patients with limb injuries. Clinical vigilance (and rising serum creatinine kinase levels) at this stage can prevent limb loss later. A thorough examination of the eyes

and ears is also indicated (an often overlooked part of the examination).

Infection Care Bundles

In addition to mechanical and thrombotic complications of central venous catheter (CVC) insertion, which can be life threatening, CVC-related blood stream infections are a significant source of morbidity and mortality. CVC insertion guidelines should be adhered to in the intensive care unit at all times, and should be the gold standard in the trauma resuscitation room as well. In sick patients where central venous access is a a time-critical intervention, strict adherence to an aseptic technique is not always possible, in which case any deviations from local guidelines should be documented. Some units advocate changing the CVC line within 24 hours of admission to intensive care. Clear documentation is the key to avoiding potential contamination from catheters left in place too long.

In addition to particular attention to CVC care, all staff involved in the care of critically ill trauma patients must pay close attention to simple precautions to prevent the spread of infections. Hand-washing and use of alcohol gel after contact with any patient or bed space, and again before the next patient contact, must be rigidly enforced. Gloves and aprons should be worn for any direct patient contact. Ward rounds should be limited to essential team members only around the patient bed space. Senior staff (medical and nursing) have a significant leadership role in gaining compliance with these simple measures.[84–86]

Early Enteral Nutrition

Clinical practice guidelines have been published that recommend initiating enteral nutrition (EN) in the trauma patient "as early as feasible."[87,88] Laboratory studies have established the physiological benefits attributable to the provision of EN in trauma patients within 24 hours of injury. Early EN (within 24 h) is associated with a significant reduction in mortality Trials have reported significant reductions in infectious complications in patients receiving early EN, and one reported a trend toward a decrease in the severity of MODS in patients receiving early EN.[89] The provision of early EN in the critically ill trauma patient is standard practice to preserve gut barrier function and maintain gut-associated lymphoid tissue mass and function.[85,86]

By maintaining the host defense functions of the intestine, translocation of bacteria from the gut into the bloodstream and consequent systemic infectious complications are reduced.[90] The provision of early EN also down-regulates the systemic immune response to bacterial translocation, which reduces overall oxidative stresses and moderates the expression of SIRS[89] and subsequent progression to MODS. The appropriate provision of EN may also decrease aspiration-related complications such as pneumonia.[90,91]

SUMMARY

Field anesthesia and intensive care are inextricably linked.[92] However, the progress of severely injured patients through the medical chain has a tendency to be compartmentalized. Recent experience has shown that austerity is no barrier to high standards of care and successful outcomes.[93] The role of the intensive care team in general and the physician in particular should extend beyond the boundaries of the intensive care unit. The expectation should be that care in the field will strive to follow recognized good practices established at home, including simple preventive strategies such as sepsis bundles and other quality improvement measures. Rather than being associated with particular equipment and technology, high-quality intensive care should imply the vigilant attention of a skilled multidisciplinary team. Operations in Iraq and Afghanistan have allowed development of military trauma systems resulting in an increasing number of multiply injured patents surviving.[94–97]

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Chapter 10

HEAD AND NECK TRAUMA

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INTRODUCTION

Injury to the head and neck has always been a major cause of morbidity in military casualties. The principles for dealing with these injuries were defined in the work of Major Harvey Cushing during World War II. Close attention to the principles of resuscitation combined with advances in surgical and intensive care have markedly improved the outcome from neurological combat injury.

CERVICAL SPINE INJURIES

Epidemiology and Injury Patterns

Cervical spine injuries (CSIs), which include bony and ligamentous injuries to the spine and spinal cord injuries with or without a detected fracture, are a common complication of acute traumatic events. CSI sometimes occurs with other major injuries, but other major injuries nearly always accompany CSI. The estimates of cervical spine trauma or spinal cord injury in civilian trauma patients has ranged from less than 0.41%[1] to 4.3%[2] to 6%,[3] and the incidence of CSI varies with mechanism of injury. Rhee et al found a three-fold higher risk of a CSI after gunshot wounds compared to blunt trauma, and a 3.5-fold lower risk of a CSI among stab-wound victims compared to blunt-trauma victims.[1]

Victims of direct injuries to the neck carry a higher risk for a CSI. One study found a 12.1% rate of CSI after a cervical gunshot wound, and a 1.5% rate of CSI after a cervical stab wound.[4] An earlier study found a similar rate for CSI among patients with penetrating neck trauma.[5] Data from combat-wounded service members in Iraq and Afghanistan suggests CSI is common with penetrating neck injuries sustained in combat. Just over 22% of 90 patients with penetrating neck injuries seen at a British combat hospital were found to have CSI.[6] A US study found a 30% rate of CSI after patients sustained penetrating neck wounds. This evidence suggests combat-related penetrating neck trauma may be complex and yield a significant incidence of CSI.

Certain injury patterns, such as facial injuries, are associated with CSI. Among all facial injuries, there was a 6.7% incidence of CSI in a retrospective study,[7] and the incidence appears to increase with greater facial injury severity.[8] Among patients with head injuries, there was a 7% rate of CSI.[7] Patients with known CSI are also at high risk for concomitant injuries. CSI patients had a 13.5% incidence of facial injuries and a 40% incidence of head or brain injuries[7]; however, CSI should be considered in all trauma patients. Similarly, patients with known CSI should be considered high risk for coexisting head and facial injuries.

The most common site of a cervical spine fracture is C2 (24% of fractures), and the most common region for a cervical spine fracture is the lower cervical region (39% at C6 or C7).[9] The vertebral body is the most common location of a fracture. Injury to the spinal cord can result from fractures or ligamentous disruption. Axial-load injuries are seen in diving accidents among the civilian population, but may also occur in the combat environment; a C1 fracture (Jefferson burst fracture) can result from an axial load and may cause neurologic damage directly or as a result of damage to the vertebral arterial system. Flexion injuries can lead to anterior cord syndrome, which may result in quadriplegia and the loss of pain and temperature sensation. Extension injuries can also occur from rapid deceleration injuries. Neurologic assessment should focus on motor and sensory function to determine a deficit and the level of injury.

Airway Management

For patients with known or possible CSI, provide a patent and secure airway and avoid the secondary insult that can result from hypoxia or hypoperfusion; minimize or prevent further neurological insult that can result from mechanical forces. Airway support may be needed before CSI can be assessed or ruled out. If a patient must be intubated prior to a full cervical spine evaluation, providers should proceed using manual inline stabilization (MILS) and rapid-sequence induction if successful intubation is anticipated. If intubation attempts will be predictably unsuccessful based on standard airway evaluation criteria, other options should be employed. MILS is preferable to traction for minimizing cervical motion during intubation[10]; it is also an effective technique for minimizing cervical spine motion while attempting intubation compared to the use of a soft or rigid collar.[11] The best method to immobilize an adult's cervical spine is with the use of a rigid cervical collar, sand bags, and restraining tape holding the patient's head to a spine board.[12] Unfortunately, the use of maximal immobilization techniques makes intubation more difficult. Full immobilization leads to a worsened grade of view of the vocal cords with direct laryngoscopy. One study found a 64% incidence of a grade III or worse view, compared to a 22% grade III or worse view with MILS.[13] The use of a

rigid cervical collar also decreases mouth opening.[14] The best-practice recommendation is to use MILS and a rigid cervical collar with the front portion removed to allow for greater mouth opening.

No significant difference in cervical spine motion has been found when comparing the various blades used for direct laryngoscopy in cadaveric models or in live patients without cervical pathology.[15,16] Several comparisons of video laryngoscopes to direct laryngoscopy tools have shown some degree of superiority of the video laryngoscopes. Use of the Bullard laryngoscope (Circon ACMI, Stamford CT) decreases cervical spine motion and improves vocal-cord visualization compared to direct laryngoscopy[17,18] and, although the Bullard laryngoscope was associated with a longer time to successful intubation, the mean time was less than 30 seconds.[19] Similar results were found for the Glidescope (Verathon Inc, Bothell, WA) when compared to direct laryngoscopy.[20,21]

Awake fiberoptic intubation for patients with a possible or known CSI is common. In cadavers with induced cervical instability, tracheal intubation using a flexible fiberoptic bronchoscope caused the least cervical spinal canal distortion of all techniques used.[22] The disadvantage of intubating a patient's trachea using a flexible fiberoptic bronchoscope prior to inducing anesthesia is that it can be time- and resource-consuming and it requires patient cooperation and operator experience. There is no living human data supporting the theory that an awake technique with a flexible bronchoscope will lead to lower rates of secondary CSI, but it is standard practice when time and patient condition allow.[23]

In emergent intubations, awake intubation with a flexible fiberoptic bronchoscope may not be feasible. In this situation, providers must determine if the patient can be successfully intubated, then MILS should be employed with an apneic technique for laryngoscopy and intubation. When ventilation or intubation difficulty is anticipated, awake fiberoptic bronchoscopy or awake tracheostomy can be performed. In one study, tracheostomy in a cadaveric model of cervical instability led to a small amount of canal distortion of unknown clinical significance.[24]

The impact of cricoid pressure on cervical spine motion has been evaluated. No significant change in spinal canal size or shape was found after cricoid pressure was applied in cadaveric models of cervical instability.[25] Other airway maneuvers, such as jaw thrust and chin lift, may displace a patient's cervical spine in a similar magnitude as direct laryngoscopy.[26] A laryngeal mask airway decreases the motion in the cervical spine relative to direct laryngoscopy in living patients and cadavers with cervical pathology,[27] but the insertion and inflation of an laryngeal mask airway was found to increase pressure in the cervical spinal canal in a cadaveric model.[27] The significance of increased pressure in the spinal canal is unknown but should be considered when weighing the benefits of a laryngeal mask airway (ie, decreased neck motion and an improved ability to ventilate or intubate a patient when conventional methods for either are inadequate) in a patient with a possible CSI.

Tracheal intubation with a nasotracheal tube using a flexible fiberoptic bronchoscope reduces cervical spine motion relative to other forms of airway manipulation,[22] but inserting a tube through the nares is concerning because it could pass out of the nasopharynx or potentially worsen a facial fracture in a patient with facial and head injuries. There is some evidence that this phenomenon is not clinically significant and nasal intubation may be safe in patients with head and facial trauma.[28] Despite its safety, nasal intubation is time consuming and can lead to epistaxis, which can make airway management far more challenging and lead to an emergent direct laryngoscopy with resulting cervical motion or an aspiration event.

Radiologic Assessment

If a patient is stable and there is not an obvious CSI, yet injury mechanism provides reason to suspect one, CSI should be evaluated. There are two major sets of criteria used to determine whether further radiological assessment is necessary or an injury can be ruled out without further studies. The National Emergency X-Radiography Utilization Study (NEXUS) criteria have been proposed as a way to determine the need for radiologic studies to evaluate for a CSI. Patients who meet NEXUS criteria for low risk of CSI should not have further radiological studies.[29] Low-risk criteria are as follows:

- No midline cervical tenderness
- No focal neurological deficit
- Normal alertness
- No intoxication
- No painful distracting injury (in which pain from the injury distracts from pain associated with a coexisting cervical spine injury).

The second set of criteria is the Canadian C-Spine Rule (CCR; Exhibit 10-1). Using the CCR, if the patient does not meet high-risk criteria but does meet one or more low-risk criteria and can turn his or her head 45 degrees left and right, further studies are not needed. If the patient has limited cervical motion, high-risk criteria, or no low-risk criteria, he or she must be

EXHIBIT 10-1

CANADIAN C-SPINE RULE HIGH- AND LOW-RISK CRITERIA

High-risk criteria:
- age greater than 64
- dangerous mechanism of injury
- paresthesias in the extremities

Low-risk criteria:
- simple rear-end motor vehicle accident
- sitting position in the emergency department
- patient was ambulatory at any time after the injury
- delayed onset of neck pain
- no midline cervical tenderness

evaluated radiologically.[30] The CCR was more sensitive than the NEXUS criteria (99.4% versus 90.7%, $P < 0.001$) and more specific (45.1% versus 36.8%, $P < 0.001$) in one comparison, and CCR usage would lead to a lower rate of radiological studies indicated (55.9% versus 66.6%, $P < 0.001$).[31] The NEXUS criteria were also found to be insufficiently powerful when the incidence of CSI was higher. The NEXUS criteria was found to have a sensitivity of 82.8%, a specificity of 45.7%, and a negative predictive value of 97.6% in a single-site study with a higher percentage of CSIs sustained than in the NEXUS trial (6.02% versus 2.4% in the NEXUS trial) and using computed tomography (CT) scans rather than radiography to detect cervical fractures.[3] Including high-risk injury (as in the CCR) improved the sensitivity of the NEXUS criteria. The CCR appears to be a superior tool for evaluating the need for a radiological study, but the NEXUS criteria are nearly as good in the low-risk population.

The best radiological study for detecting CSI has also been evaluated. Radiographs of the cervical spine were traditionally performed, including lateral, anterior-posterior, open mouth odontoid, and swimmer's views. In a metaanalysis of several comparisons between plain films and CT scans, plain films had a sensitivity of 52% while CT scans had a sensitivity of 98% for detecting a cervical spine fracture in blunt-trauma victims.[32] Nearly all fractures missed on CT scan were determined to be clinically insignificant. CT scans were also more time efficient because radiographs frequently provided poor visualization and had to be repeated.[33] If it is available and the patient is stable, a cervical CT scan is preferable in both efficacy and efficiency to plain films.

In cases of negative CT scans, ligamentous injury or occult spinal cord injuries are still possible. There is considerable controversy about how to proceed with patients who have normal cervical spine CT scans but unreliable examinations because of depressed level of consciousness. After the initial resuscitation and stabilization, patients with a residually depressed level of consciousness should be examined using magnetic resonance imaging as soon as feasible. Multiple studies have shown low rates of injuries missed on CT scan evaluation, but these studies have disagreed on the clinical significance; some studies have classified the few cases of CSI missed on CT scan as clinically insignificant,[34,35] while others have found significant rates of instability requiring intervention.[36,37] In facilities with magnetic resonance imaging capability, obtunded patients are evaluated after stabilization with magnetic resonance imaging before the cervical collar is removed. In forward settings, case-by-case decisions of when to discontinue CSI precautions must be made by weighing the harm of immobilization against the low likelihood of a significant CSI after a normal CT scan in a patient without a highly suggestive injury pattern.

Steroids

Steroids have been used to treat blunt spinal-cord injuries. Preclinical animal data has shown steroids provide a post-induced-injury benefit, but human studies have been ambiguous on neurological outcomes and may suggest greater overall harm.[38] Systemic glucocorticoid treatment may be considered when caring for combat-injured patients, but it is not recommended. Patients with complex polytraumatic combat injuries will likely suffer greater overall morbidity or mortality if given steroids. The decision to administer steroids should be made by the trauma team on a case-by-case basis.

HEAD TRAUMA

Assessment and Monitoring

Traumatic brain injury (TBI) is common among polytraumatized war casualties. Over 200,000 US service members suffered TBI from 2001 to 2011.[39] The most commonly used assessments of brain injury are the Glasgow coma scale (Table 10-1) and CT imaging. When available, CT imaging is indicated in obtunded patients or when TBI is suspected. Imaging can yield signs of intracranial pathology, such as bleeding, or

TABLE 10-1

GLASGOW COMA SCALE

	Eye Opening	Verbal Response	Motor Response
1	None	None	None
2	To painful stimuli	Incomprehensible sounds	Decerebrate
3	To verbal command	Confabulation	Decorticate
4	Spontaneously	Confused, disoriented	Generalized pain response
5		Oriented and appropriate	Localizes painful stimuli
6			Follows commands

signs of intracranial hypertension, such as midline shift or diminished ventricular volume. In one study, patients with suspected TBI and abnormal CT scan had a greater than 50% chance of developing intracranial hypertension.[40]

Monitoring intracranial hypertension can be difficult in obtunded or anesthetized patients. The Brain Trauma Foundation recommends placing an intracranial pressure (ICP) monitor in salvageable patients with Glasgow coma scale scores less than 8 and CT scans revealing hematoma, contusion, swelling, herniation, or compressed basal cisterns.[41] An intracranial monitor should be considered in TBI patients anesthetized or sedated for long periods of time to monitor ICP for severe elevations. There are many ICP monitors available, but an extraventricular drain is the most commonly used in the forward-deployed setting.

Management

Cerebral oxygen delivery depends on arterial oxygen content and cerebral blood flow (CBF). The first challenge in optimizing cerebral oxygen delivery is avoiding hypoxia. Arterial hemoglobin oxygen saturation levels below 90% have been linked to worsened outcomes.[42] Hypoxia results from five major causes:

1. low inspired oxygen content (eg, resulting from fire, or carbon monoxide inhalation);
2. hypoventilation (potentially due to drugs or a brainstem injury);
3. perfusion deficits in the lung (eg, hypotension, embolic events);
4. ventilation deficits in the lung (eg, pneumothorax); or
5. poor diffusion into pulmonary capillaries (resulting from pulmonary edema, acute respiratory distress syndrome, transfusion-related acute lung injury, etc).

The second portion of cerebral oxygen delivery is via CBF. The most common clinically used measure of CBF in patients with head injuries is cerebral perfusion pressure (CPP). CPP can become critically decreased even with supranormal cardiac output when ICP is elevated. CPP can be calculated using the following formula:

$$\text{CPP} = \text{mean arterial blood pressure} - \text{central venous pressure or ICP}$$

Patients who experience brain hypoperfusion (measured as a single systolic blood pressure reading of less than 90 mm Hg) have worse outcomes than those who do not.[42] Previously, authors had advocated maintaining CPP greater than 70 mm Hg based on this finding, but the cause-and-effect relationship was unclear. Recent recommendations have changed based on evidence of worsened overall mortality from systemic complications. Currently there is no clear recommended target CPP, and the Brain Trauma Foundation states maintaining a CPP between 50 and 70 mm Hg is an option for care, but it does not meet criteria for a recommendation.[43] Maintaining a goal CPP of greater than 70 mm Hg should be avoided (Brain Trauma Foundation class II recommendation)[43] based on the potential for overall harm to the patient. There is currently no evidence to determine the benefits versus the harm of delayed resuscitation on brain-injured patients.

In addition to concern for mean arterial blood presure, providers need to consider central venous pressure and ICP; increases in either can decrease cerebral perfusion. Central venous pressure can be increased by using positive end-expiratory pressure, or relatively increased in the brain if the patient is in the Trendelenburg position or if the normal venous outflow pathways are obstructed. The need for positive end-expiratory pressure for better oxygenation must be balanced against the resulting change in central venous pressure and CPP. There are no studies of the impact of jugular venous system cannulation on direct measurements of cerebral venous outflow, though one case series reported no adverse events related to the placement of an internal jugular cannula in patients with intracranial hypertension.[44] There is significant collateral brain drainage in healthy patients via the

vertebral venous system.[45] Patients who are positioned with their heads in a manner that does not compress the contralateral jugular or vertebral venous drainage systems will not likely suffer damage from a unilateral jugular cannula; however, the likelihood of possible concomitant CSI means most patients with severe head injuries and intracranial hypertension will likely require cervical collars, and a subclavian vein cannulation may be more easily accomplished acutely. If subclavian vein cannulation is contraindicated, an internal jugular vein cannulation is likely safe. The addition of positive end-expiratory pressure or the impact of positioning on venous drainage and the resulting change in CPP has been well documented and described, but each patient is different and may tolerate differing levels of each.[46,47]

An elevation in ICP can also decrease CPP and lead to a worsened neurological injury.[48] The degree of ICP elevation that leads to harm is debatable, but the Brain Trauma Foundation recommends that ICP values of 20 to 25 mm Hg should be treated to avoid harm.[49] This recommendation should be taken in context of the patient's clinical examination and CT scan results, if available. If the patient is anesthetized or obtunded and no CT scanner is available, ICPs above 20 mm Hg should be treated. ICP is determined by the volume of brain, blood, and cerebrospinal fluid present in the closed cranial vault.

ICP can be decreased in several ways, targeting all three components. Blood volume in the brain can be altered to decrease ICP, and improved venous drainage from reverse Trendelenberg positioning can decrease the volume of venous blood in the intracranial space. Arterial blood flow can also be reduced through hyperventilation, though hyperventilation is mostly detrimental in cases of head injury and should be reserved for the most refractory cases of acute intracranial hypertension.[50] Hyperventilation will rapidly decrease ICP, but after several hours the brain will equilibrate to the lowered arterial partial pressure of carbon dioxide and the reduction in cerebral arterial blood flow will be decreased. After equilibration, an abrupt normalization of arterial partial pressure of carbon dioxide will lead to increased cerebral arterial blood flow and worsened ICP. Hyperventilation should be used sparingly and reversed slowly.

Brain parenchyma volume can also be decreased to reduce ICP. Mannitol can be used to reduce ICP be removing cytosolic fluid from brain tissue; it is believed the fluid loss occurs mostly from healthier parts of the brain because edema in an injured brain results from chemical pathways that may not be responsive to osmolar gradients. Mannitol may impart a survival advantage, but this effect has limited documentation[51] and further analysis is necessary. The Brain Trauma Foundation recommends the use of 0.25 to 1 g/kg of mannitol for intracranial hypertension management (class II evidence). The clinical effects of mannitol can be seen in 15 to 30 minutes and may last up to 6 hours. The potential side effects of mannitol use are renal failure and hemodynamic compromise from fluid shifts and diuresis. Providers should restrict the use of mannitol to patients with signs of intracranial hypertension or elevated measured ICPs.[52]

Other strategies for reducing ICP include removing cerebrospinal fluid or improving compliance with a decompressive craniectomy. Cerebrospinal fluid can be withdrawn using a ventriculostomy, but the benefit can be short-lived due to continuous production.[53] Decompressive craniectomy has been used as an effective treatment for intracranial hypertension.[54] The benefit of early craniectomy, as opposed to craniectomy as a treatment for refractory intracranial hypertension, is still controversial.

Decreasing Cerebral Oxygen Consumption

The cerebral metabolic rate of oxygen ($CMRO_2$) can be reduced pharmacologically or through the use of hypothermia. Hypothermia has been shown to reduce oxygen consumption in experimental models, but clinical results are mixed. There is insufficient evidence to recommend induced hypothermia after a traumatic brain injury.[55] Patients who are unintentionally hypothermic are at higher risk of morbidity (odds ratio 2.9; 95% CI, 1.3–6.7), although this may indicate worse injury or greater need for resuscitation rather than a primary effect of hypothermia.[56] Hypothermic patients suffer from myocardial depression and a coagulopathy. Additionally, maintaining mild hypothermia is challenging through the evacuation chain.

A drug-induced "coma" or electroencephalographic burst suppression can decrease ICP and $CMRO_2$.There is no evidence of a survival benefit for treating intracranial hypertension with a barbiturate coma,[57] nor is prophylactic barbiturate coma beneficial in patients at risk for intracranial hypertension.[58] A pharmacologic coma should be used as a last-line medical therapy for refractory intracranial hypertension. Propofol has also been used to effectively induce burst suppression, but it carries the risk of propofol infusion syndrome (PIS) with prolonged use. The signs of PIS are lactic acidosis, rhabdomyolysis, and cardiac collapse. PIS appears to be related to dose rate and duration of drug therapy, occurring with doses of 4 mg/kg/h or greater after several hours.[59]

Other Anesthetics and Intracranial Hypertension

Volatile anesthetic agents cause a dose-dependent decrease in $CMRO_2$. Isoflurane produces the greatest decrease (up to 50% reduction in $CMRO_2$), while halothane produces the least effect (less than 25% reduction). Volatile anesthetics also have direct cerebral vasodilatory effects, which can increase CBF and ICP in cases where intracranial compliance is abnormal. These CBF and ICP increases can be inhibited with hyperventilation. Patients with intracranial hypertension undergoing prolonged anesthetics with high doses of volatile agents would likely benefit from ICP monitoring because of the mixed effect the agents have on $CMRO_2$ and CBF. Lower doses generally reduce ICP through reduced $CMRO_2$ but higher doses, approaching 1 MAC (minimum alveolar concentration percentage at 1 atmosphere), will lead to an elevation of ICP through increased CBF.

Intravenous anesthetic administration leads to the preservation of cerebral autoregulation and responsiveness of the vasculature to carbon dioxide. Barbiturates and propofol decrease CMR and ICP even at higher doses. Commonly used opioids have minimal intrinsic effect on ICP, but can increase ICP by depressing respiratory drive and the resulting hypercarbia. Regional anesthetics may be appropriate in patients with head injuries who are undergoing surgical procedures or for pain control.

Resuscitation and Intracranial Hypertension

Many authors have advocated small-volume resuscitation and the use of hypertonic saline in civilian patients with head trauma; however, complex polytraumatic injuries usually require large-volume resuscitations. The goal of resuscitation should be restoration of adequate systemic perfusion and oxygen delivery. Although over resuscitation should be avoided, it should not be used as a justification for under resuscitating because many patients with complex polytraumatic injuries have lost greater than one blood volume in the first 24 hours after injury. The need to limit cerebral edema must be carefully balanced on a case-by-case basis against the need for further volume expansion to combat shock. Hyponatremia should be avoided, which is relatively easy in large-volume resuscitations because of the amount of blood that is given with 0.9% normal saline. Excessive hypernatremia appears to be a more significant problem than avoiding hyponatremia. In many cases, severe-trauma patients with refractory acidosis required treatment with tris(hydroxymethyl) aminomethane[60] rather than sodium bicarbonate because of the severe hypernatremia that resulted from resuscitation (Exhibit 10-2).

EXHIBIT 10-2

TREATING INTRACRANIAL PRESSURE

Optimize oxygenation

- Increase oxygen delivery by treating hypoxia and hypotension.
- Decrease oxygen demand by inducing hypothermia or coma.

Decrease ICP

- Decrease CSF volume with ventriculostomy; decrease production and increase absorption.
- Decrease intracranial blood volume by hyperventilation or by placing the patient in the reverse Trendelenburg position.
- Decrease brain parenchymal volume with mannitol or by inducing hypernatremia.
- Improve intracranial compliance with decompressive craniectomy.

CSF: cerebrospinal fluid
ICP: intracranial pressure

Seizures

Seizures occur in a significant number of patients who sustain head trauma. Posttraumatic seizures can be categorized as early (occurring in the first 7 days after injury) or late. Antiepileptic drugs given shortly after injury decrease early seizure activity but not late seizures. In a study by Temkin et al, there was no survival difference between groups treated with antiepileptic drugs and untreated groups.[61] The decision to administer prophylactic antiepileptic drugs should be made on a case-by-case basis, but all patients who develop PTS should be treated. Risk factors for PTS include the following:

- younger age;
- penetrating head wound;
- depressed skull fracture;
- subdural hematoma, intracerebral hematoma, epidural hematoma, or cortical contusion; and
- Glasgow coma scale score less than 10.

SUMMARY

Severe neurological injury can result in a devastating outcome. Optimum survival requires close medical support from the point of wounding and throughout the continuum of care. The anesthetic input can be best defined as detailed attention to each aspect of resuscitation and above all else avoiding morbidity from secondary cerebral insult.

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Chapter 11

THORACIC INJURY

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INTRODUCTION

Thoracic injuries remain a common presentation to medical facilities during current military operations. These cases are predominantly ballistic penetrating trauma, but also include blunt trauma from tertiary blast injury and road traffic collisions, as well as behind-armor blunt trauma (BABT).

This chapter discusses the incidence and pathophysiology of chest injury with particular emphasis on the anesthetic and surgical considerations from point of wounding through the emergency room to the operating room.

BACKGROUND

Penetrating wounds to the thorax frequently occur during military conflict. These injuries are often fatal before the casualty reaches medical care. This high probability of death has changed little from World War I, when the mortality from all penetrating thoracic injuries was 74%, through to Vietnam, when a single assault rifle gunshot wound to the chest resulted in an 80% mortality rate.[1] Thoracic wounds remain common during the conflict in Afghanistan, where approximately 13% of ballistic injuries require a thoracic intervention (chest drain, tissue debridement, or thoracotomy),[2] and thoracic injuries contribute to 30% of combat deaths.[3]

PATHOPHYSIOLOGY AND SPECIFIC INJURIES

The thorax is a semirigid structure that affords protection to the lungs, heart, and great vessels. Injury to these structures therefore typically requires a significant magnitude of force delivered by either penetrating or blunt injury. Penetrating injuries (Figure 11-1) frequently necessitate thoracotomy during initial resuscitation and will often require a surgical intervention, whereas blunt injuries (Figure 11-2) tend to be managed conservatively or with the placement of an intercostal drain.

Chest injury leads to hypovolemia secondary to major organ or vessel injury, or to hypoxia as a result of disruption to the mechanics of ventilation. A combination of hypoxia and reduced cardiac output can occur as a result of cardiac tamponade or tension pneumothorax (see Figures 12-1 and 12-2).

Intrathoracic Airway Injuries

Tracheobronchial injuries are found in 0.8% of blunt thoracic trauma victims presenting for emergency surgery.[4] The vast majority of these injuries are found within 2.5 cm of the carina[5] and are associated with a high mortality because of the difficulties in maintaining adequate ventilation and oxygenation, as well as delayed diagnosis.[5,6]

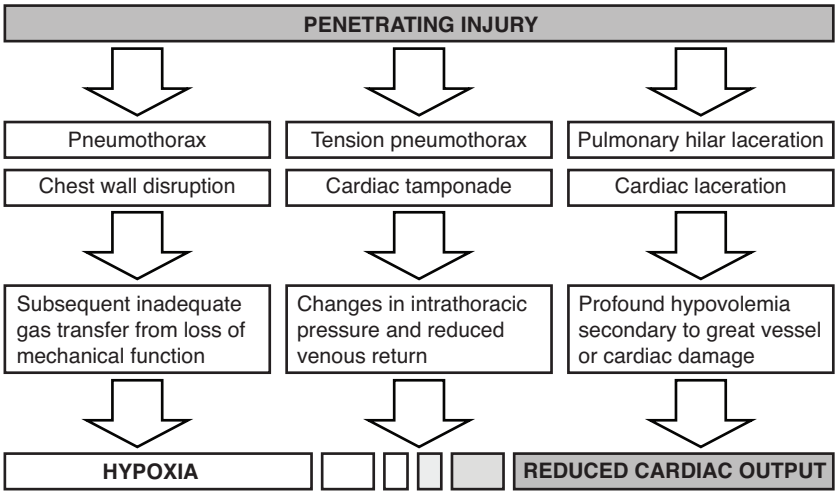

Figure 11-1. Effects of penetrating thoracic injury.
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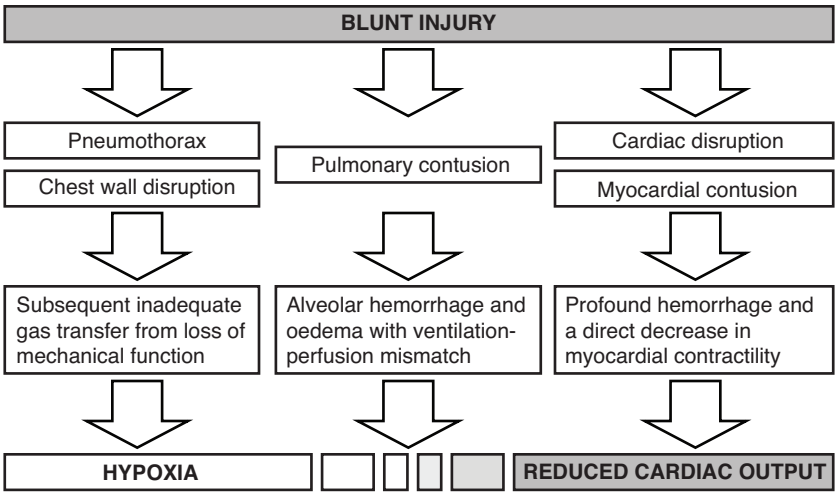

Figure 11-2. Effects of blunt thoracic injury.
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The management of intrathoracic airway injuries should ideally involve a thoracic surgeon at an early stage because operative repair will usually be required.[6] However, most trauma surgeons are not cardiothoracic specialists; therefore, adequate risk assessment must be done before embarking any operative procedure. In the deployed environment, conservative management frequently becomes the best course of action in carefully selected stable patients. There is evidence to support this view: in a study of 20 patients with tracheobronchial injuries who were managed conservatively, four died. The authors concluded that surgery should be performed in cases of associated esophageal injuries, progressive subcutaneous or mediastinal emphysema, severe dyspnea requiring intubation, difficulty with mechanical ventilation, pneumothorax with air leak through chest drains, or mediastinitis. The remaining cases are likely to do well with conservative medical management.[7]

The aim of initial management should be to improve ventilation and reduce air leak. This can be achieved by placing a cuffed airway device distal to the site of injury typically with the use of a fiberoptic bronchoscope, although this instrument may not be available in the field setting.

Cardiac Injury

Cardiac injury can occur secondary to blunt or penetrating trauma. Blunt cardiac injuries can present as a spectrum ranging from isolated electrocardiogram (ECG) abnormalities to myocardial rupture, with the right ventricle and interventricular septum the most frequently involved.[8] Cardiogenic shock may ensue as a result of arrhythmias, structural damage, or impaired ventricular contractility.

ECG abnormalities that may indicate cardiac injury include ST segment changes and arrhythmias. These patients should have continuous ECG monitoring. If hemodynamic instability is manifest, a transthoracic or transesophageal echocardiogram should be performed. Measuring troponin levels is probably of little benefit in management of blunt cardiac injury.[9] In the advent of cardiogenic shock, consideration should be given to placing an intraaortic balloon pump to off-load the left ventricle (although this procedure is unlikely to be possible in the deployed field hospital).[10]

BABT is a nonpenetrating injury resulting from the deformation of body armor after ballistic impact. It has been shown in animal models that apnea occurring after BABT is a vagally mediated reflex that results in severe hypoxia. Supportive ventilation should begin immediately in BABT casualties who are unconscious and apneic.[11]

Penetrating cardiac injuries are typically fatal. Only 6% of patients with penetrating anterior chest wounds causing cardiac injury survive to reach hospital care.[12] Presentation frequently includes the signs of cardiac tamponade, which are classically the Beck triad of hypotension, muffled heart sounds, and distended neck veins. Typically a globular heart is seen on chest radiograph, although in practice this is a subtle sign (Figure 11-3). Cardiac tamponade has also been reported with low-energy ballistic wounds.[13] The current practice of performing a rapid focused abdominal scan for trauma should include examination of the pericar-

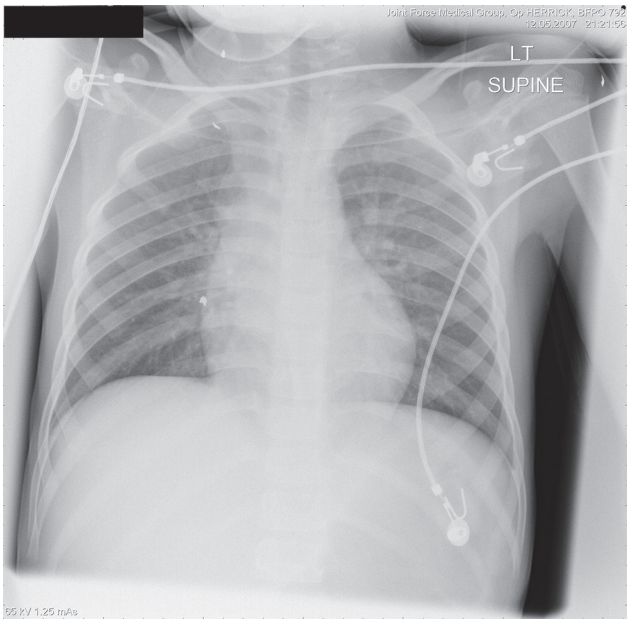

Figure 11-3. Chest x-ray showing cardiac tamponade.

dium via the substernal window. Management is by needle pericardiocentesis or thoracotomy; the latter is preferable and necessary for definitive treatment. Patients in extremis are candidates for emergency resuscitative thoracotomy (defined as thoracotomy in casualties with vital signs absent for less than 10 to 15 minutes).[14,15] In the civilian setting most chest wounds are caused by low-energy mechanisms (stab wounds or low-energy ballistics). In these circumstances, thoracotomy performed at the roadside on patients with a penetrating chest injury observed to lose their cardiac output has a survival rate of about 7%.[16] Military trauma is different and tends to produce complex injuries that require complex surgery; for this reason, thoracotomy should not be undertaken forward of a hospital with a surgical facility and blood products available to handle such cases.[2]

Aortic Injuries

Blunt aortic dissection is the second most common cause of death (after head injury) in blunt trauma.[17] However, diagnosis can be difficult and a high index of suspicion should be maintained. Computerized tomography and transesophageal echocardiogram are invaluable in establishing the diagnosis. Early decision-making and prompt thoracotomy with proximal control of the aorta is essential. Resuscitation with permissive hypotension (approximately 90 mm Hg) should be instituted until control is established, with correction of coagulopathy, acidosis, and hypothermia. It is essential to maintain good blood pressure control perioperatively. Glyceryl trinitrate and beta blockers should be used to keep the systolic pressure less than 140 mm Hg to minimize further aortic dissection.

Increasingly, endovascular stent grafting is being used rather than the conventional open aortic repair.[15] Neither of these options are likely to be available in forward surgical locations, however.

Lung Injury

The pathophysiology of lung injury, whether blunt or penetrating, can be seen as a 2-fold process with direct injury to the lung parenchyma followed by a systemic inflammatory response causing alveolar-capillary changes. This leads to acute lung injury (ALI) or acute respiratory distress syndrome (ARDS). ALI is defined by the following features: acute onset, bilateral infiltrates on chest radiograph, pulmonary artery occlusion pressure less than 18 mm Hg, Pa_{O_2} to Fi_{O_2} ratio less than 40 kPa. ARDS is defined by the same criteria except the Pa_{O_2} to Fi_{O_2} ratio is less than 27 kPa. These conditions represent a spectrum of increasing severity of lung dysfunction. Excessive bronchial secretions predispose the patient to lobar collapse and a further decrease in lung compliance, with pneumonia ensuing in approximately 50% of cases. The end result is a significant ventilation/perfusion mismatch with the additional effect of decreased oxygen delivery to vital organs.[18]

Blast lung results in an extreme form of pulmonary contusion. Shock waves from the blast cause both intraalveolar and intrabronchial hemorrhages with a sudden increase in lung weight.[19,20] The hemorrhage decreases lung compliance and leads to severe impairment of alveolar gas exchange and a rapidly worsening ventilation/perfusion mismatch. Typically these casualties present with dyspnea, cough, hemoptysis, and chest pain following blast exposure. The onset of hypoxia can occur as quickly as 1 to 2 hours after the blast exposure, in contrast to the 24 to 48 hours that has traditionally been described in this type of injury.[21] Theoretically, the administration of recombinant factor VIIa (rFVIIa) may rapidly control the pulmonary hemorrhage associated with blast lung. Prompt control of lung hemorrhage may improve the ALI/ARDS picture and prevent the need for mechanical ventilation. In an Israeli case series, full recovery in soldiers with life-threatening blast lung treated with rFVIIa was reported.[22] This indication for rFVIIa remains controversial and off-license. More studies are required to prove its efficacy in this situation.

Management of traumatic lung injury is supportive.

It should aim to minimize the systemic inflammatory response syndrome and its progression to ALI/ARDS. This is achieved by using hemodynamic monitoring to avoid excessive fluid overload or profound hypovolemia. The treatments indicated may include the use mechanical ventilation with lung-protective strategies (as discussed later in this chapter), crystalloids (in restricted volumes), colloids, diuretics, and inotropes.

OPERATIVE INTERVENTION

Although the majority of trauma cases with thoracic involvement can be managed without thoracotomy, a significant proportion do require thoracotomy, particularly with penetrating trauma. Following the principles of damage control resuscitation may further lower the threshold for operative intervention to control hemorrhage.

For those unfamiliar with thoracic surgery, the prospect of performing a thoracotomy may be daunting. The most common concerns are about the indications and timing of such intervention. Resuscitative thoracotomies—those performed for a patient in cardiac arrest—have been the subject of much debate and study (albeit retrospectively). It is generally accepted that resuscitative thoracotomies are most likely to be successful in the context of cardiac tamponade, when the patient has lost vital signs during the resuscitative process, and the period of cardiopulmonary resuscitation was limited. Those who sustained blunt trauma or had no vital signs for some time are less likely to survive. In certain polytrauma victims with a blunt thoracic injury in cardiac arrest, it is much more likely that cardiac arrest is secondary to hypovolemia from the nonthoracic injury, for example, a traumatic limb amputation. In these cases a thoracotomy would in all likelihood delay fluid resuscitation and hence worsen outcome.

It must be borne in mind that there is a limit to what can be achieved rapidly through a left anterior thoracotomy during resuscitation. Relief of tamponade or undiagnosed tension pneumothorax may lead to the restoration of a cardiac output; however, identifying and controlling a source of hemorrhage can be challenging, and cross-clamping the aorta may not be as straightforward as expected. Better exposure is almost always required, typically through conversion to a bilateral anterior thoracotomy (clamshell) incision, enabling a better assessment of the injuries and actions required to address them. In a series of traumatic cardiac arrest cases from the conflict in Afghanistan, all of the survivors (4 survivors out of 52 cardiac arrest patients) had a thoracotomy, although only one had an intrathoracic injury.[23] Whether or not thoracotomy, in itself, is beneficial in these cases is unknown.

Once the chest is opened, rapid assessment of cardiac filling and function can be made, the aorta cross-clamped, transfusion lines inserted under direct vision, and internal cardiac massage started. Deciding whether or not to perform an early thoracotomy in a patient in extremis is challenging and will, to a large extent, depend on the personalities involved. In the military setting, prehospital thoracotomy is unlikely to be a life-saving procedure due to the complex injury pattern associated with high-energy ballistic trauma and should arguably be avoided.[2] That said, prehospital thoracotomy has been found not to be an independent predictor of mortality (albeit in the civilian setting), leading to the conclusion that this intervention applied early, to a well-selected group of patients, may be of benefit.[24]

The more common scenario, and arguably more challenging in terms of decision-making, is the patient who does not require an immediate resuscitative thoracotomy, but has thoracic trauma that is not being adequately managed with simple maneuvers such as chest drain placement and volume resuscitation. The danger for patients such as this is to delay thoracotomy and persist with fluid resuscitation without controlling hemorrhage (and often air leak), which may allow the acidotic, hypothermic, and coagulopathic triad that damage control resuscitation seeks to prevent. The challenge is in identifying which patients are likely to need a thoracotomy and making the decision to proceed. Using absolute volumes of chest drainage as a trigger point for thoracotomy may be tempting, but this approach ignores the effects of air leaks and trauma elsewhere in the body. Instead, the logical and well-defined Advanced Trauma Life Support physiological principles should be used to guide the decision-making process, by identifying "transient" or "non-responders"' to fluid resuscitation: patients who have either partial or no improvement in circulatory status in response to fluid resuscitation.[25] Current resuscitative strategies have moved away from initial resuscitation with crystalloids in favor of early use of blood products, but distinguishing those responding to volume replacement from those with ongoing massive hemorrhage is pivotal in the decision-making process. Other indications for early thoracotomy include massive air leaks affecting the ability to ventilate or oxygenate, and more obvious conditions such as foreign body transfixion.

Once the decision to perform a thoracotomy has been made, the next dilemma is the surgical approach

to be taken. The classical posterolateral thoracotomy used for elective pulmonary surgery is arguably the least useful incision in the trauma setting. This technique relies on effective one-lung ventilation, and access to other parts of the chest cavity is limited. Establishing one-lung ventilation represents a challenge in a field hospital environment for a variety of reasons including lack of equipment or expertise, the need to rapidly establish a secure airway, and the lack of bronchoscopes to optimally position a double-lumen tube.

The two incisions most frequently employed for access to the thoracic cavity in trauma are therefore the bilateral anterolateral thoracotomy (the clamshell incision) and the median sternotomy, as shown in Figure 11-4. Both provide good access to the pericardial cavity without creating any of the additional anesthetic or physiological stresses associated with one-lung ventilation. The median sternotomy provides excellent access to the great vessels and, in the case of junctional trauma, is easily extended superiorly into the root of the neck or inferiorly into a laparotomy incision. Disadvantages include the need for a sternal saw or a Gigli saw to perform the incision; the former is not always readily available and the latter is slower and can be harder to keep in the midline. The other disadvantage of this approach is the relatively limited access to the lungs and pleural cavities, which may be particularly important in penetrating trauma associated with projectile injuries.

The clamshell incision is easily performed with scalpel and scissors. The sternum can usually be divided across the midline to join the anterior thoracotomies with bone shears or a Gigli saw. These thoracotomies should be performed in the fifth intercostal space, because making the incision too low can restrict access, particularly to the great vessels. The advantages of the incision are immediately apparent on retracting the thoracotomies: excellent exposure of both pleural cavities and the pericardial cavity. It is also the logical method of opening the chest following a resuscitative left anterior thoracotomy by simply extending the incision across the midline. It is important that bleeding from the transected internal thoracic arteries should be addressed early in the procedure, preferably when identified. A transected vessel overlooked at this stage may not be apparent until the casualty is resuscitated to more normal physiological values, leading to significant delayed hemorrhage.

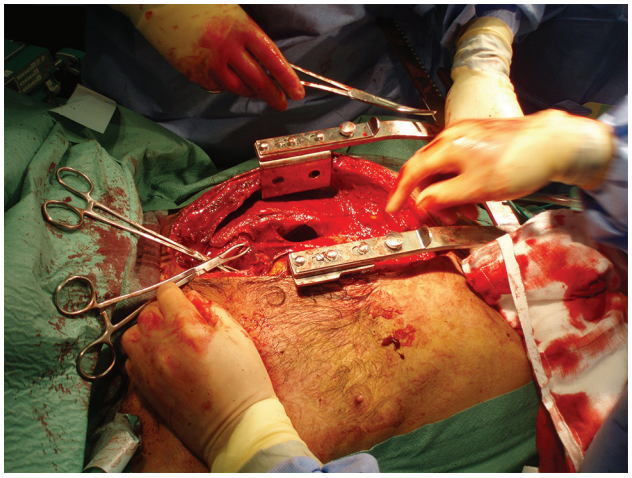

Figure 11-4. Median sternotomy.

Once the thorax is opened, the typical next step is to open the pericardial cavity. This should initially be done longitudinally, then making an inverted-T incision, avoiding the phrenic nerves, particularly on the left where is it most vulnerable to iatrogenic damage. Subsequent surgical maneuvers depend on the pathology found, but the main principles of any trauma surgery apply: first control hemorrhage. Bleeding from the heart or great vessels is initially controlled with digital pressure, possibly with the subsequent application of side-biting clamps to enable direct suture repair. Massive pulmonary hemorrhage or air leak can be controlled by temporarily clamping the lung at the hilum. A pneumonectomy is occasionally necessary but is associated with a high mortality of around 75%.[26,27] The high incidence of mortality is related to exsanguination, right heart failure, and pulmonary edema in the remaining lung. Oversewing pulmonary lesions, or performing a tractotomy with standard stapling devices for deeper lacerations, is often adequate to achieve permanent control; wedge resections may also be required. Inevitably thoracotomies performed for blunt or blast trauma will be more challenging, typically due to the magnitude of soft tissue and chest wall injury, and there are no easy ways to deal with these.

Junctional trauma is a well-recognized issue; traumatic injuries do not respect anatomical boundaries and chest injuries are not infrequently associated with abdominal, neck, and spine injuries. As discussed, a median sternotomy is easily extended into a laparotomy, or vice versa, and the clamshell incision can also be combined with a midline extension. Extending the operative field from the chest into the abdomen is something that most trauma surgeons should be comfortable with, but the root of the neck can prove more challenging. Access to the subclavian vessels, particularly on the left, can be difficult, and often necessitates a trap-door incision with division or removal of part of the clavicle. Definitively dealing with great vessel trauma in this challenging region requires good

exposure, which initially can seem daunting.

A range of other related injuries and structures are not addressed here, thoracic duct and esophageal injuries for example. The need to understand the anatomy of the thoracic cavity and neighboring areas cannot be over emphasized.

PRINCIPLES OF ANESTHESIA

The patient should be initially assessed and managed in accordance with Battlefield Advanced Trauma Life Support guidelines in the deployed military setting. Broadly speaking, the principles of thoracic trauma anesthesia involve the restoration of circulating volume, maintenance of adequate oxygenation, and correction of hypothermia and coagulopathy (ie, the goals of damage control resuscitation).

It is highly likely that a definitive airway will need to be secured at an early stage. The usual practice to achieve a definitive airway is a rapid sequence intubation of the trachea, with cricoid pressure applied and the cervical spine controlled with manual in-line stabilization. However, this practice can increase the likelihood of difficult intubations. Difficult intubation should be dealt with in accordance with local guidelines. Any unexplained hypotension and difficulty in ventilation should arouse suspicion of a tension pneumothorax. A retrospective analysis of 978 penetrating chest trauma casualties during the Vietnam War found radiographic evidence of tension pneumothorax in 198 of the cases. The researchers concluded that tension pneumothorax was the cause of death in 3% to 4% of fatally wounded combat casualties.[28]

Anesthesia for an emergency thoracotomy may include one-lung ventilation. In the shocked patient, ketamine is considered by some to be the induction agent of choice because it preserves blood pressure and cardiac output.[29] Alternative strategies involve a significantly reduced dose of other induction agents or opioid-based anesthesia. Hypotension immediately postinduction should be anticipated and treated with further intravenous fluid resuscitation (usually blood products) or sympathomimetic drugs in a euvolemic patient. Anesthesia is usually maintained with a low-dose volatile agent and nondepolarizing muscle relaxation. Using nitrous oxide should generally be avoided because it has a propensity to increase the size of gas-filled cavities including air emboli and pneumothoraces. Monitoring should include invasive arterial blood pressure measurement as well as placement of a central venous catheter.

PRACTICAL CONDUCT OF ANESTHESIA

The common theme in all cardiothoracic procedures is the close communication required between surgeon and anesthetist. This is true through the decision to operate, surgical approach, lung isolation, and problem solving. Only by paying close attention to the progress of the surgery and understanding the implication of surgical maneuvers can the anesthetist interpret changes in airway pressure, blood pressure, or heart rhythm correctly.

The choice of surgical approach can obliviate the need for lung isolation, and a clamshell or median sternotomy are optimal approaches for intrathoracic procedures for this reason. Lung isolation with a double-lumen endobronchial tube (DLEBT), standard for elective thoracic work, allows the surgeon to operate on a nonventilating lung. Alternatives to the DLEBT in providing one-lung ventilation include bronchial blockers and the Univent (Fuji Systems Corp, Tokyo, Japan) tube.[30] Fiberoptic scopes are used to facilitate and confirm placement. Some or all of this equipment may not be available in a deployed field hospital. The DLEBT can be difficult to place by those not regularly undertaking routine thoracic anesthesia, and the tubes are commonly associated with malposition and a high complication rate, which can be compounded in the rapid sequence intubation situation.[31]

Several simple strategies are available to a nonthoracic anesthetist providing anesthetic for emergency intrathoracic surgery to facilitate surgery on the lung or chest cavity. Most emergency thoracotomies carried out as part of damage control resuscitation can be managed with a single-lumen tube and two-lung ventilation. Typically a single-lumen tube passed beyond the carina will rest in the right main bronchus (which is in a straighter line from the larynx than the left main bronchus). A single-lumen tube can readily be used to isolate the left lung and preferentially ventilate the right. Simply advancing (an uncut) tube should provide lung isolation with this technique. Single-lumen tube design will then almost inevitably occlude the take-off of the right upper-lobe bronchus, which may worsen hypoxemia. Rotation of the patient's head to the right on insertion can sometimes allow the tube to pass into the left main bronchus and allow ventilation of the left lung.

Lung movement can be minimized by periods of relative hypoventilation or even disconnection from ventilation for brief periods to allow a specific surgi-

cal maneuver to be performed. Combined with gentle surgical retraction of the lungs, this technique will allow most emergency procedures to be undertaken.

Hypoxemia is common during single-lung ventilation and may also be "permissive" due to hypoventilation during particular surgical maneuvers such as lung retraction. Inspired oxygen percentage can be increased and acceptable arterial oxygenation monitored with arterial blood gases. Lung injury from contusion or blast lung is common in military trauma, and every effort should be made to keep tidal volumes and ventilator pressures low intraoperatively. Airway pressures can be affected by mechanical retraction of the airway, as can hypotension.

Hypotension is frequently caused by surgical retraction around the mediastinum or great vessels. Periods of hypotension should first be addressed by ensuring there is no mechanical cause. Surgical stimulation around the mediastinum is a potent cause of arrhythmias, which are usually self-limiting once the surgical stimulus is withdrawn. Turning a patient with a massive hemothorax to the lateral position can provoke profound hypotension both through redistribution of blood volume and also from the contained blood's pressure on the mediastinum. For this reason it is recommended that the supine position be used wherever possible (with incision via clamshell or median sternotomy).

Fluid management for major lung resections is challenging, as reflected in the high mortality associated with traumatic pneumonectomy.[26,27] It seems reasonable to adopt a more cautious approach to transfusion than in non-chest-related trauma. Resuscitation should aim to correct initial acidosis and hypotension with blood and blood products, which will maintain a normal plasma oncotic pressure. Over-transfusion and increasing right ventricular load should be avoided at all costs with major lung resection.

VENTILATION STRATEGIES

The incidence of ventilator–associated lung injury has changed the way that mechanical ventilation is delivered. The Acute Respiratory Distress Syndrome Network study[32] used tidal volumes of 6 and 12 mL/kg to ventilate patients with ARDS. A significant absolute reduction in mortality was achieved using the lower tidal volumes and maintaining inspiratory plateau pressures less than 30 cm H_2O. However, these protective ventilation strategies frequently result in hypercapnia and respiratory acidosis.[32] When conventional methods of mechanical ventilation fail in patients with an asymmetric lung injury, intensivists may resort to methods such as independent lung ventilation to maintain oxygenation.

One-lung independent ventilation (OL-ILV) is a technique that allows ventilation of one lung while the other main bronchus is artificially blocked to isolate fluid (ie, blood) or secretions, avoiding contamination of normal lung alveoli and improving gas exchange. OL-ILV can be facilitated by deliberate left or right main bronchus intubation with a normal endotracheal tube, the use of a DLEBT, or placement of a bronchial blocker.

Two-lung independent lung ventilation (TL-ILV) allows different ventilatory parameters or ventilatory modes to be applied to each lung. Separate ventilator circuits are used for each lung. TL-ILV can be applied synchronously or asynchronously. Synchronous TL-ILV maintains the same respiratory rate for both lungs but the flow rates, tidal volumes, and positive end-expiratory pressure are set separately. Asynchronous TL-ILV must use two separate ventilators to deliver different modes as well as different ventilator settings.

PRINCIPLES OF ANALGESIA

The ongoing management of thoracic casualties hinges on providing excellent analgesia, which allows weaning from mechanical ventilation and hence restoration of normal respiratory mechanics. Using local anesthetic techniques avoids the respiratory depressant side effects of opiates. However, regional techniques to provide adequate analgesia for significant thoracic injuries are relatively complex. Thoracic paravertebral blocks are attractive because they have no unwanted effects on the uninjured side, are safe to perform and relatively easy to learn, and provide analgesia comparable to that of epidural analgesia.[33] Paravertebral blocks can also be placed under direct vision by the surgeon during thoracotomy.[34] Thoracic epidurals, however, are likely to be the technique of choice for most anesthetists. These blocks provide bilateral analgesia but also cause muscle weakness; however, improvements in respiratory mechanics resulting from excellent analgesia more than offset this side effect.[35]

CONCLUSION

The majority of combat thoracic injuries can be managed with simple interventions, such as placement of an intercostal drain or soft tissue debridement. It is the minority of cases, when thoracotomy is indicated, that pose the greatest challenge for the military anesthetist and surgeon.

The essential principles for dealing with these injuries are:

1. Recognize life-threatening problems and intervene accordingly.
2. Understand the prevailing pathophysiology.
3. Adopt a multidisciplinary approach to provide care from point of wounding though resuscitation and surgery to intensive care.

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Chapter 12

EXTREMITY, JUNCTIONAL, AND PELVIC TRAUMA

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INTRODUCTION

Recent conflicts have resulted in a high proportion of limb injuries, accounting for 50% to 70% of all injuries treated during Operation Iraqi Freedom.[1] This is reflected in the steady increase in the incidence of traumatic limb amputations reported in military casualties.[2] Evidence from previous conflicts including Vietnam and Iraq also suggests that exsanguination from these injuries accounts for the greatest proportion of preventable deaths on the battlefield.[3] As a result of these experiences, significant advances in hemorrhage control have been developed, implemented, and tested during recent conflicts. In addition, experiences in Afghanistan have revealed an increasing burden of severe extremity, junctional, and pelvic trauma,[2] which may represent an increased prevalence and evolution in the design of improvised explosive devices (IEDs)[4] against both military and civilian targets.

IEDs are defined by the US Department of Defense as "devices placed or fabricated in an improvised manner incorporating destructive, lethal, noxious, pyrotechnic or incendiary chemicals, designed to destroy, disfigure, distract or harass."[5] These weapons have been responsible for a significant proportion of deaths in Afghanistan. IED survivors may sustain injuries ranging from relatively minor wounds, with little or no physiological compromise, to multiple traumatic limb amputations with possible pelvic involvement. IEDs contain strong explosives with little primary fragmentation material, thereby giving rise to an injury pattern caused by the primary blast wave with secondary fragmentation. Current combat body armor provides defense to the thorax and abdomen but lacks protection for the limbs and perineum; therefore, highly contaminated junctional trauma with perineal wounds and pelvic fractures are common.[6,7]

Recent improvements in the design of military helmets and combat body armor, in addition to the relatively high mortality of torso injuries, have also contributed to this changing injury pattern.[6] Innovations in military medical care, such as the increased use of tourniquets and novel hemostatic treatments, are considered to have improved outcomes in military patients with trauma. Military trauma doctrine now places increasing emphasis on the early identification and treatment of catastrophic hemorrhage,[8] so military anesthetists must have a thorough knowledge of the challenges posed by these injuries.

CLASSIFICATION OF EXTREMITY, JUNCTIONAL, AND PELVIC TRAUMA

Extremity Injuries

Limb injury and traumatic amputation is common in the current conflicts. In the lower limb, amputation typically occurs through the upper third of the tibia,[9] although more recent experience suggests an increasing burden of more proximal amputations and junctional injuries. These injuries are associated with other extensive bony fractures, significant soft tissue disruption, and contamination due to separation of fascial planes with embedding of environmental debris. They may occur in isolation, bilaterally, distal, or more proximal, and as a result may present the anesthetist with a range from mild to severe physiological insult and challenges in securing peripheral intraosseous and intravenous access.

Junctional Injuries

Junctional zone trauma, by definition, is an injury occurring at the junction of anatomically distinct zones (Table 12-1). These injuries may be defined as damage to tissues that span the root of an extremity and adjacent body cavity. Such regions include the lower abdomen, groin, axillae, and proximal extremities.[10] The injuries occur as a result of a transfer of energies from the passage of missiles, energized fragments, or blasts. Junctional areas are traversed by major blood vessels and, when injured, may not be suitable for tourniquet application. Consequently, junctional injuries are frequently associated with profound bleeding that may be difficult to control. Trauma to these areas

TABLE 12-1

CLASSIFICATION OF JUNCTIONAL TRAUMA

Type of Injury	Characteristics	Implications
Type 1	Wound encroaches on a junctional zone but surgical control can be gained without entering adjacent body cavity.	Proximal control requires surgical incision extending across the joint flexure.
Type 2	Wound encroaches on a junctional zone requiring surgical access to gain hemorrhage control.	Body cavity may need to be entered, eg, laparotomy/thoracotomy. Potential for occult blood loss.

may therefore need early and aggressive resuscitation and surgical hemorrhage control; occasionally, limb salvage may have to give way to preservation of life.

Pelvic Injuries

Pelvic injury is associated with civilian mortality rates of 18% to 40%, and deaths within the first 24 hours are often due to blood loss.[4] Pelvic injury mechanisms involve strong energies and frequently include damage to the pelvic vasculature and organs. The resultant cardiovascular compromise presents a complex challenge to the trauma team.

Pelvic injuries may be categorized according to the forces applied to the pelvis and the degree of ligamentous and bony disruption. Classifying the injury allows the trauma team to predict associated injuries, pathophysiology, and likely transfusion requirements.[11] The Young and Burgess system divides injuries into three types:

1. **Anterior posterior compression**. Pubic diastasis disruption with or without sacroiliac disruption. Pelvis is potentially unstable, with damage to pelvic vasculature and bleeding likely.
2. **Lateral compression.** Anterior ring injury with or without sacral compression fracture, posterior ilium, or sacroiliac involvement. Pelvis is usually mechanically stable; therefore, any hemodynamic instability is due to other causes such as intraabdominal pathology.
3. **Vertical shear**. Vertical displacement of hemipelvis. Potentially unstable pelvis associated with intrapelvic, intraabdominal, or mediastinal injury.

Anterior, posterior, and lateral compression have further subtypes I, II, and III with increasing severity. It is also possible to have a combination of the types following a complex mechanism of injury.

PREHOSPITAL CARE

Forward projection of military anesthetic care to aid resuscitation and evacuation of casualties back to field hospitals has gained particular prominence in recent years, in line with civilian practice[12]; examples of such models include the UK-led Medical Emergency Response Team (Enhanced) in Afghanistan.[13] The degree of medical intervention in the prehospital phase depends on the type and size of platform used for evacuation (eg, helicopter vs land ambulance) as well as the composition and skill mix of the medical team involved.

Initial care is provided by the casualties themselves, followed by other soldiers ("buddy-buddy" system) and combat medical technicians or corpsmen when available. The main aim of care in the field is early control of bleeding in accordance with military trauma doctrine; this begins with the control of catastrophic hemorrhage, prior to assessment and treatment of the airway, breathing, and circulation (the <C>ABC paradigm).[8] Bleeding from extremities is controlled via application of the combat application tourniquet (CAT), supplemented with direct pressure to the wound and indirect pressure to proximal vessels (eg, femoral artery) where necessary.

Recent conflicts have seen a sharp increase in the use of CATs. A high proportion of casualties with extremity trauma will likely present with at least one tourniquet in place. US data from recent conflicts suggests that CATs were already applied in over 18% of battlefield admissions,[14,15] with significant vascular injury seen in approximately 6.6% of these cases.[16] Providers must be aware that CATs may have been applied as part of "care under fire" protocols, and therefore may not be clinically indicated. However, mortality from limb injury exsanguinations in US troops decreased from 9% during the Vietnam conflict to 2% in more recent conflicts in Iraq and Afghanistan, and consensus remains that a well-applied tourniquet can prevent death from catastrophic hemorrhage. The Israeli experience shows a 0% mortality from similar injuries when tourniquets are applied correctly. To prevent unnecessary limb ischemia, it is recommended that when the tactical situation allows, the tourniquet should be loosened after the bleeding is controlled and where appropriate substituted for a pressure dressing and elevation.[8]

Where junctional trauma has created an insufficient proximal stump for tourniquet application, or when the tourniquet fails, direct pressure with topical hemostatic agents will be required. Modern hemostatic products fall broadly into two categories. The first category of products are the chitosan-based preparations (eg, HemCon [HemCon Medical Technologies Inc, Portland, OR]; Chitoflex [HemCon Medical Technologies Inc]; Celox [Medtrade Products Ltd, Crewe, UK]). Chitosan is a nontoxic derivative of the naturally occurring carbohydrate chitin (found in the cell wall of fungi and crustaceans) and has mucoadhesive properties that accelerate hemostasis in the presence of bleeding. The second are zeolite-based preparations (eg, QuikClot [Z-Medica, Wallingford, CT]). Zeolite is a mineral-based material that promotes hemostasis by concentrating clotting factors in an exothermic reaction

initiated by exposure to water (and therefore blood). All these products are widely available to field medics on the modern battlefield and are likely to have been applied to casualties with severe limb bleeding.

The presence of junctional trauma makes adjoining body cavity and bony injury likely, so thoracic, abdominal, and pelvic injury should be assumed. Thorough examination of the body cavity adjoining the injured limbs should be undertaken to exclude pathology such as clinical signs of thoracic and abdominal disruption. All lower limb junctional trauma should be assumed to involve pelvic fractures until proven otherwise. Suspected pelvic fractures should be stabilized with empiric application of a pelvic binder (or a sheets and sand bag improvisation) and correction of lower extremity external rotation by taping the knees and ankles. The chances of exsanguination and hypovolemia must be mitigated preemptively per current military trauma doctrine.

Major limb trauma is associated with severe pain, and efforts to mitigate pain in the prehospital phase are essential.[17] Splinting fractures to reduce pain and bleeding requires the administration of effective analgesia. Numerous drugs are used in the prehospital environment; the agent chosen will depend on physician familiarity and drug availability. In all cases, the aim is to alleviate pain while minimizing serious adverse effects such as oversedation (loss of verbal contact), respiratory depression, hypotension, nausea, and vomiting.

ROLE 3

When severe extremity, junctional, and pelvic injuries are reported, an appropriately staffed trauma team should be mobilized. Most casualties should receive standard trauma care in the emergency department; however, unstable casualties (eg, in traumatic cardiac arrest with cardiopulmonary resuscitation in progress, or with limb or torso trauma and signs of critical hypovolemia) and those with junctional injuries with incompressible hemorrhage may bypass the emergency department and be taken straight to the operating room (OR) to allow damage control resuscitation and surgery to commence simultaneously.[18] The requirement for large bore intravenous (IV) access is paramount and should be immediately established when the casualty arrives at Role 3 if not already in place. The laboratory liaison should ensure suitable supplies of O Rh-negative, and AB-negative plasma should be requested and used until group-specific agents are available.

All patients will receive a focused assessment with sonography in trauma (FAST) scan, and the majority of blast injury patients will undergo multislice computed tomography (CT) scanning as part of their initial management, to help guide surgery. The decision to scan must balance the risks of delaying the surgical control of significant bleeding with gaining diagnostic information that may allow more appropriate surgery. The use of truncal angiography is gaining in popularity, with the arterial phase often available to the mid lower limb. Approximately 20% of whole body CT scans are extended to include peripheral lower limb angiography. Such preoperative knowledge of arterial damage and collateral vessel "run off" (when accompanied with tourniquet release) greatly assists in assessing hemorrhage sites, determining limb viability, and surgical decision-making (personal communication from Surgeon Commander R Miles, RN, Defence Consultant Advisor, Radiology; Plymouth, UK, August 2011).

Extremity Injuries

Injured limbs associated with potentially catastrophic hemorrhage will have had tourniquets placed in the prehospital phase, and the requirement for these should be reassessed at Role 3. Loosening tourniquets should only be done with the immediate availability of dressings, other hemostatic products, and surgical expertise. If a tourniquet is still required, it should be replaced with a padded pneumatic surgical tourniquet, once the patient is anesthetized. This tourniquet allows more measured application of compression, and the wider dimensions are considered less likely to cause further damage to soft tissues and nerves.

Conscious, physiologically stable patients with extremity trauma should be examined and the extent of injury documented. Digital photographs should be taken of all wounds when possible and with appropriate consent, to aid subsequent reconstructive procedures. The neurovascular integrity of affected limbs should be assessed (with the help of Doppler ultrasound if necessary) and documented to identify any subsequent worsening of the injury. More extensive examination, irrigation, and instrumentation of the wound should be avoided at this stage to prevent further contamination and bleeding. Injured limbs should be carefully redressed with iodine-soaked gauze and secured with a crepe bandage.

Splints applied in the prehospital phase should be reviewed by a surgeon to confirm correct position-

ing when plain radiographs are available. It is likely that improvised splints will be substituted for a more conventional splint (eg, a Thomas splint) by the attending surgeon if the patient is not proceeding to the OR imminently. Appropriate analgesia should be administered to allow the splinting.

Junctional Injuries

The surgeons must consider the approach needed to achieve proximal control, taking into account whether a type 1 or type 2 junctional injury exists. Diagnostic imaging with FAST scanning will aid the surgical decision as to the need for thoracotomy or laparotomy. In the face of physiological instability, a positive FAST scan will encourage urgent surgery for proximal hemorrhage control. If the patient is stable, the investigation of choice to assist in surgical planning is high resolution CT angiography.

Pelvic Injuries

Life-threatening pelvic hemorrhage remains a source of preventable death in the trauma population and can occur with all pelvic fracture patterns. The three major bleeding sources are fractured cancellous bone and venous or arterial laceration. Priorities are the early identification of fractures and associated bleeding, with early mechanical stabilization and aggressive volume resuscitation. A multidisciplinary approach with coordination of early surgery is key. Pelvic radiographs will guide the surgeon to achieve restoration of the pelvic ring. FAST scanning in the emergency department will assist in diagnosis of abdominal and pelvic blood and inform further treatment.[19] The presence of intraabdominal blood will trigger urgent laparotomy and pelvic fixation, whereas a negative scan will trigger further resuscitation and pelvic fixation if cardiovascular stability is not achieved.

OPERATIVE INTERVENTION

In the OR, procedures may be conducted under regional anesthesia, general anesthesia, or a combination of the two. In all cases, the aim is to maintain adequate tissue perfusion, oxygenation, homeostasis, hemostasis, and analgesia.

Generic Considerations

Positioning

It is important that the often numerous surgical teams have adequate access to the injury sites to effectively perform the surgery. The abdomen and chest may need to be prepared and opened to achieve proximal vascular control. Care should therefore be taken to ensure that vascular access sites are accessible and secured. Additionally, it is important to ensure that all limbs, whether injured or otherwise, are adequately padded and joints placed in neutral positions to prevent further neurovascular injury. Placing the patient in the cruciform position addresses these issues while providing good surgical access.

Infection Control

Blast and ballistic wounds are at high risk of bacterial infection due to extensive contamination from environmental debris. The majority of organisms causing life-threatening infections (eg, *Clostridium perfringens* and *Streptococcus pyogenes*) are sensitive to relatively narrow spectrum antibiotics such as benzylpenicillin. Extensive use of broader spectrum agents has been linked to the increased incidence of multidrug resistant species such as *Acinetobacter* species.[20,21] Where the tactical situation allows, parenteral antibiotics should be administered during the prehospital phase (eg, benzylpenicillin 1.2 g IV or intramuscular), although the evidence base for this is not extensive.[22] In the OR, a suitable choice would be co-amoxiclav 1.2 g IV (clindamycin 600 mg IV if the patient is allergic to penicillin), cefuroxime 1.5 g IV in the presence of fractures, adding metronidazole 500 mg IV with compound fractures and severe soft tissue injury.[18] Those without immunity to tetanus should receive relevant prophylaxis.[23]

Communication

The anesthesiologist has a key role in aiding the situational awareness of the surgeon, who is likely to be focused on the surgical field. The anesthesiologist may assist in the coordination of numerous simultaneously operating surgical teams often operating on different anatomical regions of the casualty. It is important to maintain effective communication and convey vital information, including duration of tourniquet inflation, clamping of major vessels, and significant changes in physiological parameters. It may be necessary in the face of significant blood loss for the anesthesiologist to call for a "hemostatic pause," when surgery is halted while blood products and medication are administered to correct coagulopathy and control bleeding.

Regional Anesthesia

Regional procedures can be effectively performed in the emergency department but are usually reserved for the OR. Peripheral nerve blocks may provide excellent anesthesia and analgesia, for both intraoperative and postoperative pain control, avoiding the systemic side effects of many parenteral analgesics and potentially reducing the incidence of postoperative neuropathic and phantom limb pain. Nerve blocks may take the form of single-shot injections of local anesthetic or infusions via peripheral nerve catheters,[24] which are increasingly being inserted under direct vision using ultrasound. Such procedures are ideally suited for trauma patients, who may require numerous trips to the OR for staged surgery. Following the correction of coagulopathy, epidural catheters may be considered a more appropriate option in the case of bilateral lower limb injuries.

Tourniquet Use

It is important to avoid the serious and largely dynamic complications associated with tourniquet application and reperfusion (summarized in Table 12-2). Cuff inflation results in rapid increases in systemic vascular resistance, and central blood volume increases following exsanguination of the affected limb. A tachycardia develops as tourniquet pain evolves after approximately 45 minutes, and together these effects lead to increases in cardiac output and mean arterial blood pressure that may completely conceal an underlying hypovolemia. Careful volume replacement must be instituted preemptively to avoid precipitous hypotension when the cuff is deflated at the end of the procedure. Cuff pain may be severe and often persists for several hours following cuff release as reperfusion occurs. It is characteristically difficult to relieve with systemic analgesics, although because the underlying mechanism is thought to involve *N*-methyl-D-aspartate receptors, ketamine may be more effective.[25] Regional anesthetic techniques should be considered by the anesthesiologist to provide adequate analgesia in the perioperative period.

Peripheral nerve injury is uncommon but may be devastating. Rather than ischemic time, it appears that compressive shearing forces across the nerve are likely to be the most important mechanism influencing nerve injury.[15,26] This problem can potentially be minimized by using wide, contoured cuffs for the minimal necessary time. Excessive cuff pressure should be avoided by targeting the limb occlusion pressure, which is the minimum cuff pressure required to interrupt distal flow (demonstrated by loss of distal pulses, infrared oxygen saturation, or Doppler measurements). After limb occlusion pressure has been determined it is common practice to add cuff pressure as a safety margin to account for surgical changes that may require increased pressure.

Cuff release leads to a predictable reperfusion phenomena characterized by vasodilation, lactic acidosis, hypercarbia, hyperkalemia, hypothermia, and pain.

TABLE 12-2

COMPLICATIONS ASSOCIATED WITH TOURNIQUET USE

System	Responses	Mechanisms	Actions
Respiratory	↑MV, ↑$Paco_2$, hypoxia thromboemboli	Pain, reperfusion, pulmonary embolism	Control ventilation, increase inspired O2 fraction as required. Consider imaging and anticoagulation, depending on clinical context.
Cardiovascular	↑SVR, ↑MAP, ↑CO, ↑HR	Occlusion of vessels, ↑central blood volume, catecholamine release	Vasodilators if severe but beware of underlying hypovolemia
Neurological	↑ICP, peripheral nerve damage, pain	↑Cerebral blood volume, nerve compression, ischemia	Ventilate to normocapnia, target cuff pressure (LOP),* minimize cuff time, consider regional anesthesia
Metabolic	↓pH, ↑lactate, ↑K+	Reperfusion of ischemic tissue	Anticipate and treat fluid resuscitation

*to stop the flow of arterial blood into the limb distal to the cuff
↑: increased
↓: decreased
CO: cardiac output; HR: heart rate; ICP: intracranial pressure; LOP: limb occlusion pressure; MAP: mean arterial pressure; MV: minute ventilation; SVR: systemic vascular resistance

This metabolic storm is often only transient (typically 10–15 min) but may be pronounced. In severe cases, myoglobinuria and rhabdomyolysis may combine to precipitate acute renal impairment.

Surgical Considerations

General

Knowledge of surgical approaches to battlefield trauma allows the anesthetist to optimize the condition of the surgical field and preempt any predictable changes in physiology caused by surgery. A number of surgical principles are common to the management of all traumatic injuries (Table 12-3). There may be times when the casualty is so sick that surgery is limited to hemorrhage control only, with the priority being further resuscitation, restoration of physiology, and transfer onward for further debridement at Role 4.

Extremity Injuries

Orthopedic. Stabilization of fractures reduces pain, infection, and further soft tissue damage as well as facilitating bone healing. Strategies for fracture stabilization include:

- Nonoperative
  - Plaster. Suitable for simple or closed fractures. Simple and effective.
  - Traction. Effective and used widely in military and civilian casualties. Its simplicity makes it a particularly attractive option in civilian patients who can be discharged to civilian hospitals with such devices in situ.
- Operative
  - External fixation. Allows more effective elimination of movement at the fracture site, transportation of casualties with open fractures, and access to soft tissue wounds to permit wound care and revascularisation procedures. However, there are some concerns about external fixation; one study suggested that complications requiring revision or removal occurred in 86.7% of cases.[27]
  - Internal fixation. Inappropriate for the acute management of war injuries because of the unacceptably high risk of infection associated with contaminated wounds.

Vascular. Goals of damage control vascular surgery on extremities are controlling exsanguinating hemorrhage, rapidly restoring blood flow to an ischemic limb, and preventing compartment syndrome.[28] It may involve clamping a proximal vessel, followed by simple ligation of damaged arteries and veins; limb salvage may be a secondary priority. If the patient is stable and the expertise of the surgeon allows, simple vascular shunts or grafts may be employed to salvage limbs. Coagulopathy often prevents the use of systemic heparin, although with increasingly successful treatment by aggressive targeted administration of blood products, it may still be necessary for surgeons to infuse dilute heparin (1 unit/mL) proximal and distal to the injury. This procedure is supported by anecdotal accounts from Iraq and Afghanistan, when shunts inserted on

TABLE 12-3

GENERAL PRINCIPLES OF SURGICAL MANAGEMENT OF BATTLEFIELD WOUNDS

Surgical Principles	Rationale
Hemorrhage control	External then potentially internal proximal control
Damage control surgery	An operative strategy that sacrifices the completeness of the immediate surgical repair in order to address the physiological consequences of the combined trauma (double hit) of injury and surgery.[1] Integrated and combined with damage control resuscitation
Debridement	Removal of all foreign material and nonviable tissue to leave a bed of healthy tissue on which subsequent reconstruction can be performed. Usually performed with a tourniquet
Wound excision	Excision of no more skin than that sufficient to leave healthy wound edges. Skin must be retained for later reconstruction procedures
Wound extension	Extension of the wound will be necessary to allow adequate examination of the zone of injury, which will extend proximally and distally
Removal of nonviable tissue	Necessary to prevent the establishment of infection. Nonviable tissue is characterized by dark coloration, mushy consistency, lack of capillary bleeding, and lack of muscle contractility when touched with a diathermy probe or crushed with forceps.
Closure	Wounds NOT treated with primary closure

1. Hawley JS, Murray CK, Jorgensen JH. Colistin heteroresistance in acinetobacter and its association with previous colistin therapy. *Antimicrob Agents Chemother.* 2008;52(1):351–352.

the battlefield reportedly clotted during evacuation back to rearward echelons of care.[29]

Many casualties with vascular injuries will be at risk of developing compartment syndrome, and fasciotomies may be performed. In the civilian extremity trauma population, surgical fasciotomy is performed in less than 1% (upper limb injuries) to 5% (lower limb injuries) of cases, while recent studies have suggested that 16% of casualties evacuated from Iraq and Afghanistan underwent fasciotomies.[30] Early clinical diagnosis is key (excessive pain, exacerbated by compression and passive extension, a palpably tense compartment, impaired neurology, and loss of distal pulses are very late signs) and has been shown to be as reliable as invasive compartment pressure monitoring.[31] A low threshold for fasciotomy therefore stems from a combination of factors including a significant mechanism of injury, the potential for prolonged warm ischemic times, and the use of regional anesthesia techniques to the injured limbs. Another important factor is the prolonged evacuation times to Role 4 in an environment where the clinical monitoring of the limbs is difficult and compartment pressure transducer monitoring is not routinely available. One study of military casualties suggested that 35% of fasciotomies performed were done so without a diagnosis of compartment syndrome.[32]

Amputation. Decision to amputate is not always straightforward, and civilian limb salvage scores are not useful. Relative indications to amputate a limb may include severe bone and/or soft tissue loss, extensive arterial injuries, prolonged warm ischemic times, and lack of appropriate surgical experience. Relative indications to salvage a limb may include upper limb injuries, bilateral limb injuries, and injuries in children.[33] If damage is extensive and/or there is questionable circulation to the limb, amputation should be undertaken. Amputations are made at the lowest possible level, with wounds left open (fashioning of flaps should be initially delayed).[34]

Debridement. Debridement of lower extremity wounds should be performed through inspection of each muscle with transection of necrotic areas. Contamination should be identified, with careful inspection of intermuscular planes for necrosis. Proximal vascular control should be maintained until debridement is completed to allow a clearly visible surgical field.

Junctional Injuries

Type 1 injuries are often unilateral, do not require body cavity exploration, and are associated with less physiological disturbance. Hemorrhage may require an extraperitoneal approach with control at the external iliac level. Type 2 injuries are those in which the peritoneal or thoracic cavities must be entered to gain proximal control, commonly seen in bilateral injuries when immediate laparotomy and control at the level of the distal aorta or iliac system is conducted.[35] This procedure has the dual effect of limiting blood loss from both the amputation site and from pelvic and perineal injuries. Laparotomy also allows direct visual inspection of abdominal viscera, which may also have suffered as a result of the primary blast wave. Anesthesiologists should be prepared for the surgical invasion of any body cavity and the physiological insults that occur as a result.

The surgical priorities in junctional injuries are continued direct pressure at the wound site, compressing the wound against the underlying axial

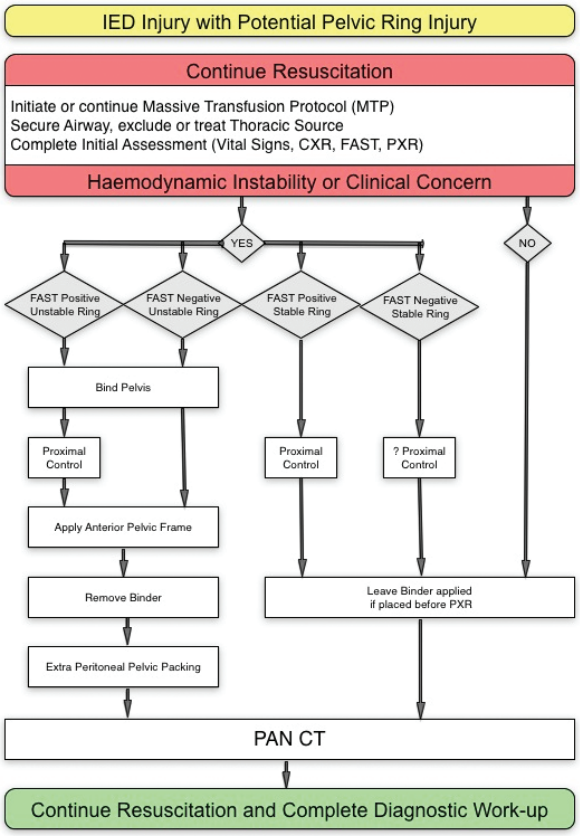

Figure 12-1. A suggested management strategy for pelvic fractures.
CXR: chest x-ray
FAST: focused assessment with sonography in trauma
IED: improvised explosive device
Figure: Courtesy of Lieutenant Colonel SA Adams, Royal Army Medical Corps, Consultant Orthopaedic Surgeon, 16 CS Medical Regiment.

skeleton while preparing for the OR. Once in the OR, manual pressure will be replaced by a sponge stick, held in place until both proximal and distal control have been achieved.

Lower limb junctional injuries can be controlled with femoral or iliac proximal control depending on the site of vessel injury. Whether control is achieved above or below the inguinal ligament will directly affect anesthesia as a result of potential surgical entry into a neighbouring body cavity. Upper limb junctional injuries are less common, but no less challenging to achieve proximal control. Right upper limb injuries may require median sternotomy for proximal aortic arch control compared with thoracotomy for left upper limb injuries; each imposes different challenges to the anesthesiologist. More distal vessel injuries may be accessed by periclavicular incisions and affected vessels ligated with reliance on collateral flow.

Pelvic Injuries

The aim of surgery for pelvic fracture is the restoration of skeletal stability and volume,[12] although further examination in the OR is essential. Pelvic fracture is often associated with damage to the pelvic veins, the pelvic viscera, and the iliac arteries and their divisions. Over 70% of hemorrhage associated with blunt pelvic trauma is venous and may be controlled with maneuvers that reduce the pelvic volume and stabilize the pelvis. The other nearly 30% is arterial and often requires surgical packing or embolization.[4] Figure 12-1 shows a suggested management strategy for pelvic fractures.

Anteriorly, the urethra and bladder are often threatened, and posteriorly, the lumbosacral and coccygeal nerve plexuses, as a result of their proximity to bone. It is injury to these structures that results in a high morbidity and mortality.

External fixation of the pelvis is often necessary to restore stability using an anterior external fixation frame or a posterior C-clamp. If arterial bleeding continues, ligation or tamponade via catheter balloons may be necessary. Embolization of damaged pelvic vasculature is unlikely to be readily available, but extraperitoneal packing of the pelvic vasculature is a common alternative. This is a simple and potentially lifesaving procedure. It is performed via a midline incision from the umbilicus to the symphysis pubis. Tissues are dissected down to the peritoneum, where clots may be removed and swabs inserted.[36] When a laparotomy is indicated in the presence of pelvic fractures, it is performed with the pelvic binder in place, followed by pelvic ring stabilization using iliac crest pins. The binder can then be removed and if necessary the extraperitoneal pelvis packed.[19]

Open pelvic fractures and perineal disruption are associated with extremely high mortality although the number of survivors is increasing. These injuries are managed as above but with extensive debridement and antibiotics. Perineal injuries may require selective fecal diversion with a divided sigmoid colostomy. Damage to the bladder, rectum, and small and large bowel and gross soiling of the peritoneum should be anticipated. Vascularized testicular remnants are preserved, scrotal injuries debrided, and urethral injuries managed by urethral or suprapubic catheterization.[37]

POSTOPERATIVE CARE

The course of the postoperative phase depends largely on the severity and distribution of the casualty's injuries. The physiologically unstable, severely or multiply injured casualty is likely to be transferred sedated to the intensive care unit for further care and stabilization. Particular care should be taken to observe for physiological derangements caused by reperfusion of ischemic limbs following limb salvage procedures. Perfusion of limbs must also be closely monitored and optimized by aggressively treating hypothermia and hypovolemia and avoiding vasopressor drugs. Clinical diagnosis of compartment syndrome must be suspected in all injured limbs and surgeons immediately informed of any concerns.

It is important to liaise closely with intensive care staff when surgeons identify a need to return a patient to the OR for further procedures. Extremity injuries are likely to need repeat debridements and irrigation at intervals of 24 to 48 hours in the initial stages. Casualties returning to the surgical ward either from the OR or intensive care may require ongoing anesthesia input. When peripheral nerve or epidural catheters are indicated, they should be inserted prior to waking and extubation to allow successful weaning from the ventilator. These catheters may be used for anesthesia for any subsequent surgical procedures, contributing greatly to multimodal analgesia and reducing the need for opioid medications. When using catheters is not possible, a multimodal approach should be taken, and the use of patient-controlled analgesia should be considered.

Casualties with extremity, junctional, and pelvic trauma will require multiple surgical procedures and prolonged rehabilitation in other facilities either within the operational theater or in a Role 4 hospital overseas. This can involve the transportation of critically ill ca-

sualties with complex injuries over many thousands of miles, frequently coordinated by intensive care and anesthesiology personnel (see Chapter 38, Air Transport of the Critical Care Patient).

SUMMARY

Extensive trauma involving the extremities and junctional and pelvic regions accounts for an increasing burden of trauma on the modern battlefield. It is associated with significant mortality and presents unique challenges to the medical team and anesthesiologists. Initially, treatment will focus on the control of potentially life-threatening hemorrhage; in junctional and pelvic trauma, hemorrhage control is difficult and requires early, clear, and concise discussion among the surgical team. Considerations about how to manage treatment must be made, in the context of the casualty's physiological condition. The anesthesiologist is optimally placed to advise on the patient's physiological context and inform decision-making.

Operative management of these casualties primarily involves aggressive management of hypovolemia and associated coagulopathy. Communication with the surgical team is paramount to optimize surgical access, maximize limb perfusion, and react to changes in surgical strategy that may occur as wounds are explored. A hemostatic pause should be discussed whenever ongoing bleeding and worsening coagulopathy become apparent. Postoperative anesthetic management focuses largely on pain relief. A body of evidence suggests an increasing role for regional anesthetic techniques in these casualties.

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ATTACHMENT: LIMB INJURY REVASCULARIZATION AND MANAGING REPERFUSION WHEN THE TOURNIQUET COMES OFF

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Introduction

The perioperative management of patients after vascular reperfusion may present a challenge to the anesthesia provider. Presently several methods of vascular occlusion are available to achieve a bloodless field during surgery or control hemorrhage in traumatic injury. With combat injury and trauma, these methods, specifically tourniquets, are often applied preoperatively. The use of vascular occlusion devices produces mechanical, metabolic, and physiologic changes that may be difficult to manage during occlusion and at reperfusion, when the tourniquet is released. While the entire medical team must be aware of these changes, anesthesiologists must specifically understand the type of vascular exclusion employed and the changes the patient will experience perioperatively to avoid the morbidity associated with these devices.

Battlefield and Preoperative Hemorrhage Control

Hemorrhage is a major cause of morbidity and mortality in trauma, and hemorrhage from limb injuries has been recognized as the most important cause of avoidable battlefield death.[1] Important advances in hemorrhage control have been developed, implemented, and tested in the current wartime environment. The US military has mandated medical training for all deployed military personnel focusing on hemorrhage control. Military medics follow specific algorithms to control blood loss and as a result have helped decrease mortality due to exsanguination from major limb trauma.[2]

The simple application of direct pressure is the primary step in hemorrhage control, and its application is of utmost importance prior to surgical treatment. The failure of direct pressure to stop bleeding usually signifies severe vascular injury or multiple vascular injuries likely requiring surgical intervention. In cases of severe arterial bleeding or other major vascular injury, other methods are utilized to control bleeding. These include pressure dressing, proximal arterial compression, hemostatic agents, tourniquets, and vascular clamps. Compression of proximal arteries including the axillary, brachial, and femoral arteries, can be applied to stop distal arterial bleeding in traumatic limb injuries. Hemostatic agents currently in use include HemCon (HemCon Medical Technologies Inc, Portland, OR) and QuickClot (Z-Medica Corp, Wallingford, CT), which activate the coagulation cascade, forming vascular plugs at sites of hemorrhage. These products are available in powder or dressing form.

When other methods of hemorrhage control have failed to control severe limb bleeding, tourniquets may be applied. These severe injuries often require multiple tourniquets to prevent battlefield exsanguination. In many combat situations tourniquets may be the best option to control bleeding; they require minimal observation, allowing other casualties to be cared for.[3]

These methods of hemorrhage control are frequently employed in the preoperative setting by lay persons, local emergency services, and medical professionals. The anesthesiologist must be aware of the technique used in the field due to the significant impact it has on anesthetic management.

Tourniquets

The history of tourniquets dates back centuries, but it wasn't until the latter half of the 20th century that the modern pneumatic tourniquet, with pressure control, a timer, and wider cuffs, was invented. These pneumatic cuffs are frequently applied during orthopedic surgery and on trauma patients in the operating room. Intraoperative pneumatic tourniquets, usually applied by technicians, registered nurses, or physicians have a long history and proven track record for safety.

To control hemorrhage on the battlefield, soldiers are currently issued a nonpneumatic tourniquet system, the combat application tourniquet (CAT; Figure Attachment 12-1). Both pneumatic tourniquets and nonpneumatic

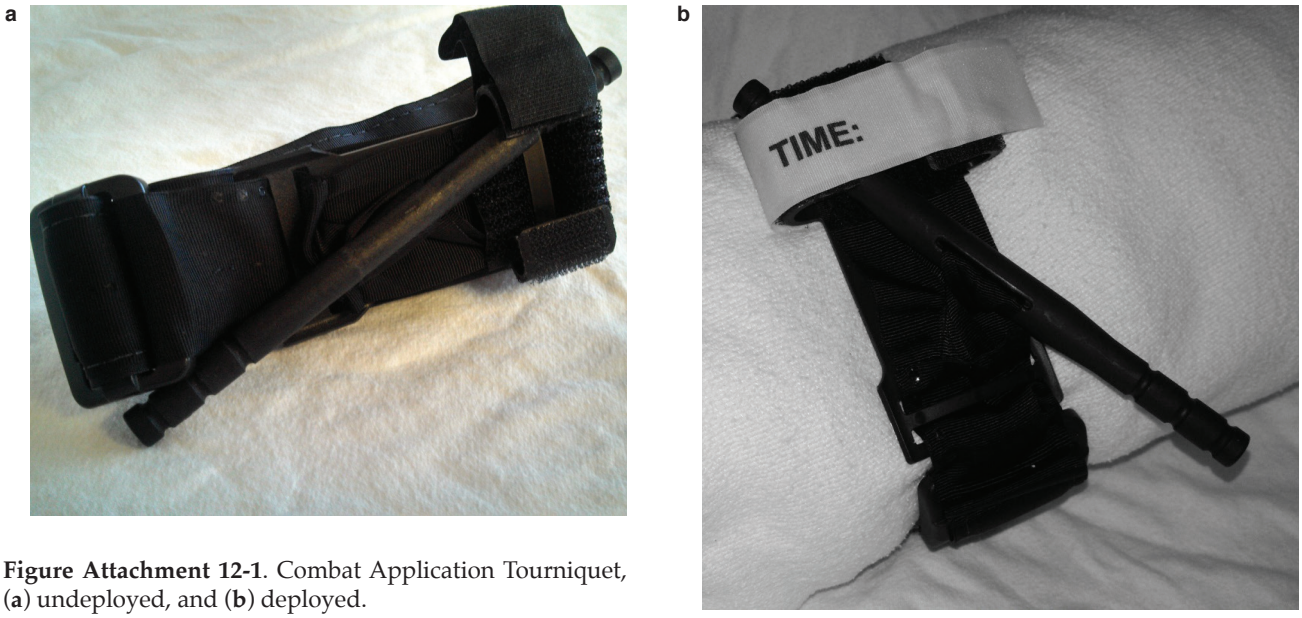

Figure Attachment 12-1. Combat Application Tourniquet, (**a**) undeployed, and (**b**) deployed.

tourniquets, similar to the CAT, are in use by local emergency services.[4] Military tourniquets are frequently applied by a range of people from lay persons to medically trained professionals. Despite training in nonpneumatic tourniquet use, controversy exists on their safety and appropriate use in the prehospital setting.[5]

Limb injuries make up a high percentage of battlefield trauma; some studies suggest that up to 61% of casualties in the current armed conflicts have some form of limb trauma, a majority of these occurring in the lower extremities.[6] The high incidence of extremity trauma and the issuance of CATs have increased the use of tourniquets to over 18% of battlefield admissions.[6,7] Significant vascular injury is seen in approximately 6.6% of these admission.[8]

Most clinicians' knowledge of tourniquets is limited and does not extend beyond the mechanism of action and awareness that they should not be inflated for prolonged periods of time.[9] The anesthesia provider, however, is well aware of tourniquet mechanism and time limits, but also (more importantly) deals with the physiologic result of their use.

Physiologic Effects of Tourniquet Use

Several complex physiologic disturbances occur with tourniquet use, as shown in studies on systemic and local changes during and after tourniquet use.[10–15] These changes are dynamic, usually transient, and drastically different depending on the phase of tourniquet use. These changes can at times have a significant impact on anesthetic management. While most of the changes are well tolerated in the healthy patient undergoing elective surgery, some patients with underlying systemic disease or polytrauma may not tolerate the physiologic insult associated with tourniquets. It is important for the surgical team and especially the anesthesiologist to understand these changes, anticipate complications, and develop practices to minimize compilations associated with the use of tourniquets. The anesthesiologist must remain vigilant throughout the perioperative period to prevent serious complications.

Cardiovascular Effects

Initial tourniquet inflation causes immediate and delayed changes in hemodynamic parameters. In nontraumatic surgery, the limb is exsanguinated by the use of gravity or an Esmarch band. This exsanguination initially increases intravascular volume and decreases the vascular bed.[11] The immediate increase in central blood volume and decrease in vascular bed increases the mean arterial pressure (MAP), believed to be secondary to increased systemic vascular resistance (SVR). As tourniquet time increases, MAP and SVR continue to increase, likely from increased endogenous catecholamines caused by the tourniquet pressure, limb ischemia,

or tourniquet pain. Initially there may not be a heart rate response to inflation; however, heart rate increases as tourniquet time increases. Additionally, with increased duration of tourniquet use, the elevation in MAP and SVR increase stroke volume and contributes to the elevated cardiac output seen during tourniquet inflation (Figure Attachment 12-2).[10]

The dynamic nature of tourniquet use is most evident at deflation. Hemodynamics begin to change instantly, observed as a significant decrease in MAP and SVR. The cause of this is multifactorial and related to vasodilatory effects of metabolic mediators, pain relief, and redistribution of blood flow. Heart rate decreases initially but rapidly returns to baseline.[10] Careful monitoring of the patient is essential at this stage of deflation because of the risk of a sudden release of large venous emboli (although this complication is rare).[4]

Published data on physiologic changes associated with traumatic amputation is limited; however, it is well observed that the hemodynamic changes occurring with tourniquet application in a nontraumatic patient may be less predictable in trauma patients. Tourniquet use in trauma is usually in response to uncontrollable bleeding and vascular injury. The patient may present with severe hemodynamic instability and other physiologic changes that do not produce the predictable hemodynamic changes of elective tourniquet use. These patients often have severe multilimb injuries involving massive blood loss and multilimb amputations. The tourniquet role changes from a bloodless field strategy to a life-saving strategy.

The physiologic and metabolic changes more consistent with a trauma patient include coagulopathy, hypothermia, and acidosis. The role of an anesthesia provider is to direct resuscitation while surgical hemorrhage control is achieved. At times patients will undergo amputation of multiple limbs as a means of controlling hemorrhage, which may lead to a substantial decrease in the vascular bed, followed by increased SVR and the potential for overresuscitation. Vigilance on the anesthesiologist's part in fluid management is important when a large decrease in the vascular bed has occurred.[10]

Respiratory Effects

Tourniquet deflation produces a significant and rapid rise in $PaCO_2$ and partial pressure of end tidal carbon dioxide ($PetCO_2$), peaking at 2 to 3 minutes postdeflation and returning to baseline within 10 minutes. Minute ventilation increases as $PetCO_2$ increases, with a peak minute ventilation approximately 2 to 3 minutes postdeflation and returning to baseline within 5 minutes. Spontaneously ventilated patients demonstrate a rapid compensation by increased minute ventilation and fast return to baseline $PetCO_2$ regardless of the anesthetic type. However, controlled ventilation produces prolonged periods of elevated end tidal carbon dioxide ($ETCO_2$) unless minute ventilation is increased to compensate for the influx of hypercarbic blood from the ischemic limb (Figure Attachment 12-3).[12,13]

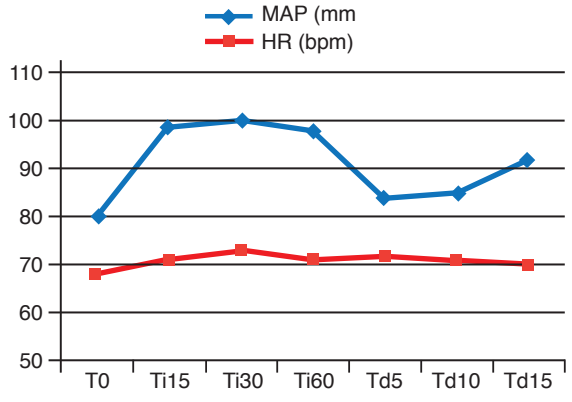

Figure Attachment 12-2. Heart rate (HR) and mean arterial pressure (MAP) changes following inflation and deflation of a tourniquet. Ti: time at inflation; Td: time at deflation; number: the minutes follow each event.

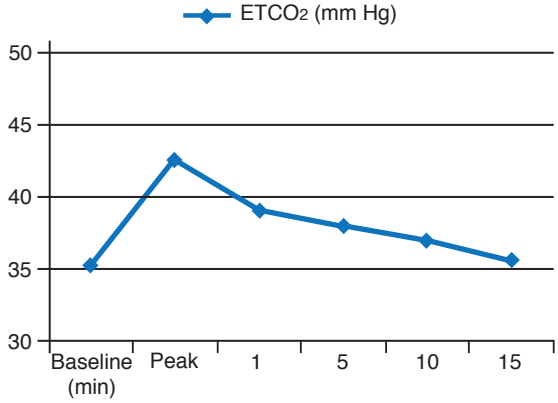

Figure Attachment 12-3. Change in end-tidal carbon dioxide ($ETCO_2$) after release of tourniquet.

Neurologic Effects

$PaCO_2$ is an important regulator of cerebral vascular tone and, as mentioned earlier, is elevated above baseline after tourniquet release. At tourniquet release there is also an immediate rise in $ETCO_2$, which may last several minutes depending on the specific ventilatory status. The elevation in $ETCO_2$ is associated with an increase in cerebral flow velocity and increased cerebral blood volume. Hirst's study demonstrated that mean middle cerebral artery blood flow velocity increased 58% from baseline flow rates within 2 minutes of tourniquet deflation (Figure Attachment 12-4).[14] The resulting increase in intracranial pressure (ICP), in combination with an immediate decrease in MAP, can reduce cerebral perfusion pressure to dangerously low levels. The transient increase in cerebral blood flow and ICP is usually tolerated in healthy patients, but may lead to secondary brain injury in patients at risk for cerebral ischemia,[14] including polytrauma patients with underlying traumatic brain injury and elevated ICP.

Although the use of tourniquets in the medical profession is common, it is important to remember that tourniquet use is not benign and is associated with long-term complications and sequelae. Nerve injury is an uncommon injury that may be either minor or devastating. Research on the cause of tourniquet-related nerve injury has shown that neural ischemia related to prolonged tourniquet time is not the key determinant of neural injury, but that compressive shearing forces across the nerve play a major role in nerve injury.[4,6] Nerves are most vulnerable at the edges of the tourniquet, where pressure differences across the tissue are greatest. These forces stretch the nodes of Ranvier, leading to partial or complete rupture of the stretched myelin and resultant nerve palsy.[4] Several factors have been found to increase the pressure gradient (and thus decrease the risk of injury), including the use of noncontoured cuffs, narrower cuffs, larger limbs, higher pressure, nonpneumatic cuffs, and increased tourniquet times.[4]

Controversy exists regarding the use of military nonpneumatic tourniquets. Some authors believe this type of tourniquet use has led to preventable nerve injury and unnecessary limb amputation,[5,6] while other studies consistently show that the appropriate use of combat tourniquets is associated with improved mortality and minor morbidity.[6] In fact, US mortality from limb injury exsanguinations decreased from 9% during the Vietnam conflict to 2% in the current Middle East conflicts. Israeli literature shows a 0% mortality from similar injuries when tourniquets are applied correctly.[4] These and other studies report that nerve palsies and limb shortening are infrequent morbidities usually associated with misuse of tourniquets.[4,6,16]

Tourniquet Pain

The term "tourniquet pain" refers to the observation of increased MAP and heart rate with prolonged tourniquet use. The elevation in systemic blood pressure is often seen after prolonged tourniquet use of over 45 minutes. Tourniquet pain is the most common complication of tourniquet use seen during surgery and is described as a dull, aching pain that increases as tourniquet time increases. It is often difficult to treat, frequently requiring vasodilators to manage blood pressure,[15] may not be fully relieved with narcotics, and can persist for several hours after surgery.[17] After deflation of the tourniquet, a different pain sensation is noted, associated with reperfusion of the limb. This sensation is described as being equal to or greater than the intensity of the discomfort caused by the tourniquet immediately before deflation.[18]

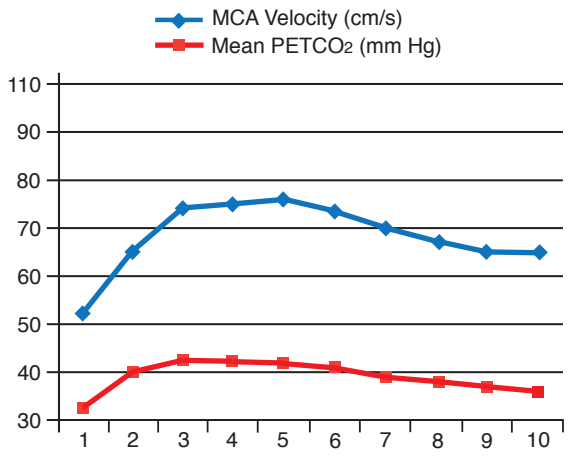

Figure Attachment 12-4. Mean cerebral artery (MCA) velocity and partial pressure of end tidal carbon dioxide ($PetCO_2$) changes over time in minutes

The underlying pathophysiology of tourniquet pain is complex and multifactorial, involving mechanical compression, cutaneous neural pathways, and limb ischemia.[14] The noxious stimulus associated with tourniquet use is thought to be mediated by unmyelinated C fibers rather than myelinated delta fibers. Compression of the neural tissue such as myelinated delta fibers

decreases conduction through the neural fibers, and the neural conduction via these myelinated fibers is blocked, allowing the slower unmyelinated neural tissue, the C fibers, to transmit the dull pain sensation.[15] The C fiber transmission activates *N*-methyl D-aspartate receptors, leading to increased blood pressure.[19] In addition to the neural pathway, other processes that may play a role in tourniquet pain are local tissue ischemia and reperfusion of ischemic tissue after deflation, leading to continued pain despite tourniquet release.

Metabolic Changes

Many of the physiologic changes associated with tourniquet inflation and deflation are due to metabolic changes that occur at the cellular level. As stated above, neural injury in the ischemic limb appears to be related to compressive force. Muscular tissue, however, appears to be more sensitive to the duration of ischemia.[4] After inflation of the tourniquet, anaerobic cellular metabolism predominates, with accumulation of metabolic metabolites including lactate, increased CO_2, and potassium.[19] After release of the tourniquet, the accumulated metabolites are released into the general circulation, and blood levels of lactate increase, $PaCO_2$ increases, serum bicarbonate decreases, and potassium decreases. The pH decrease can last several minutes; in fact it, appears that many of these metabolic changes last several minutes. Some studies show increased lactate and decreased pH lasting longer than 10 minutes.[19]

In addition, core temperature decreases with tourniquet use.[19,20] In cases of extremely long tourniquet times or multiple limb tourniquets, tissue necrosis may occur and cellular components may be released into the circulation, resulting in myoglobinemia and rhabdomyolysis.[18] These metabolic changes are usually well tolerated during elective surgery in the healthy patient. Patients with significant comorbidities may not tolerate such a systemic insult and require slower release of tourniquets. Trauma patients also may not tolerate immediate release of tourniquets; the release can make management of hypothermia, acidosis, hypotension, and ICP more challenging.

The return of toxic metabolites to the circulation results in systemic metabolic dysfunction, referred to as ''myonephropathic metabolic syndrome'' and characterized by metabolic acidosis, hyperkalemia, myoglobulinemia, myoglobinuria, and renal failure. Paradoxically, tourniquet deflation is associated with thrombolytic activity and anoxia, promoting activation of the antithrombin III and protein C pathways, which may be implicated in posttourniquet bleeding.[4]

Safety

In an effort to decrease tourniquet-related injury in the operating room and on the battlefield, guidelines and practices have been proposed by several authors and some organizations. Measurement of limb occlusion pressure before surgery might lead to the use of a lower tourniquet cuff pressure during surgery and thereby reduce the risk of postoperative pain and complications,[20] and limb occlusion pressure (LOP) has consistently shown to be lower than traditional tourniquet use.[18] LOP is defined as the minimum pressure required, at a specific time, by a specific tourniquet applied to a specific limb at a specific location, to stop the flow of arterial blood into the limb distal to the cuff. LOP can be determined simply by increasing the tourniquet until distal flow is interrupted, which may be determined by palpation of distal pulses, use of infrared oxygen saturation, or Doppler. Many modern automated tourniquets are equipped with LOP technology. After LOP has been determined, it is common practice to increase cuff pressure by 20 to 50 mm Hg as a safety margin to account for fluctuating blood pressure as a result of painful and noxious surgical stimuli.

There is an inverse relationship between LOP and the ratio of cuff width to limb circumference. A narrower cuff requires higher LOP and increases the pressure gradient across the underlying nerves, as well as the potential for nerve injury. The use of wider or contoured cuffs results in lower pressure gradients and theoretically a decreased risk of nerve injury.[4]

Military combat tourniquets are nonpneumatic, narrow, applied by nonmedical professionals, and can be applied incorrectly, all factors that increase the risk of morbidity. However, the survival benefit of using tourniquets in severe limb trauma cannot be understated. To decrease the risks of injury, all deployed soldiers in the United States, United Kingdom, and several other countries receive training on the appropriate use of tourniquets, including indications, application, and evaluation. Tourniquet use is tracked and training is tailored according to trends in use and morbidity. Once a combat tourniquet is applied, it is important to monitor the injury for continued bleeding; check whether the tourniquet has loosened or moved, especially if placed over clothes; and continue to reevaluate the indication for the tourniquet.[6,7]

Tourniquet inflation time is another area of uncertainty. It is widely taught that a tourniquet should not be left inflated for longer than 2 hours; however, there are situations when the tourniquet must exceed 2 hours of ischemia in order to complete the surgical procedure. In general, nerves are more susceptible to mechanical pressure, and it is the muscle and other soft tissues that are at increased risk from prolonged tissue ischemia. Many authors suggest that a tourniquet time of 1.5 to 2 hours is an acceptable time for tissue ischemia; however, animal studies suggest that even 1 hour of tissue ischemia can produce muscle weakness and tissue injury lasting up to 7 days.[15] It is generally acceptable to allow a period of reperfusion if the tourniquet is needed for a prolonged period of time, exceeding 2 hours. The optimal duration of reperfusion remains uncertain, but some researchers suggest 10 minutes of reperfusion for every hour of ischemia.[19] In summary, the use of LOP, wider or contoured cuffs, and shorter ischemia intervals or periods of reperfusion may decrease the incidence of neurologic or muscular injury related to tourniquet use.

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Chapter 13

CRITICAL CARE AND ANESTHETIC CARE OF MILITARY BURN CASUALTIES AT ROLE 3 FACILITIES

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INTRODUCTION

In the written history of wars and conflicts, burn injuries have been described for over 5,000 years. In the austere environment, military personnel are at risk of sustaining a variety of burn injuries, including flash injuries, flame burns, contact burns, scalds, chemical burns, electrical burns, and radiation burns. These injuries can result from both combat and noncombat (eg, waste burning, ammunition handling, gasoline) causes. The majority of combat burn injuries result from the detonation of explosive devices[1]; these account for 63% of burn injuries among military personnel. In addition, a military combatant may sustain several types of burn injuries in a single incident. Specific types of burns, risks, morbidity, and mortality are beyond the scope of this discussion.

In Operation Iraqi Freedom and Operation Enduring Freedom, one of the challenges faced by military anesthesiologists was the treatment of the severely burned soldier. Current burn care capitalizes on the recent advancements in evacuation times from point of injury through Landstuhl Regional Medical Center in Germany, to the US Army Institute of Surgical Research (USAISR) Burn Center at Brooke Army Medical Center, Fort Sam Houston, Texas. The USAISR Burn Center is the Department of Defense's only military facility treating severely burned soldiers. Between January 2001 and December 2010, 691 military burn casualties were admitted to USAISR (Jackson BA. Data from US Army Institute of Research Burn Program Manager, Fort Sam Houston, Texas; December 2010). Of these, 216 casualties (31%) had a total burned surface area (TBSA) of over 20%, compared to only 13% of civilian patients treated nationally with that extent of TBSA.[2] In addition to their burn injuries, military burn casualties are often subjected to polytrauma. To date, more than 50% of burn patients admitted to the USAISR had at least one other significant injury, most commonly a fractured extremity.[3]

In the forward deployed environment, burn casualties may initially be treated with the surgical and medical management necessary to save life, limb, and eyesight in the same way as other trauma patients; however, burn casualties have perioperative anesthesia concerns distinct from other surgical populations. These require consideration and planning by the anesthesiologist and surgical team to obtain optimal outcomes. This chapter is written to serve as a guide to anesthesiologists who may have limited experience with burn casualties, and will discuss the practical issues that may be encountered in treating these patients.

ACUTE THERMAL INJURY

The first 24 to 48 hours after a major burn is commonly referred to as the resuscitation phase. The patient will often be treated with massive intravenous (IV) fluids to maintain intravascular volume and urine output. There are several formulas that may guide this therapy (Table 13-1), but the amount is usually titrated to maintain urine output between 0.5 and 1 mL/kg/h.[4,5] Over-resuscitation must be avoided because it leads to increased edema and related complications.[6] During this phase the patient may require escharotomy or fasciotomy to preserve blood flow to extremities or to allow ventilation. Less commonly, a laparotomy is required to treat abdominal hypertension. Blood transfusion is typically not required during these early procedures, because these patients have very low blood loss and their measured hematocrit is commonly normal to high, assuming they have not lost blood from a nonburn injury. Nonburn injuries may also mandate other operative procedures during this phase, such as definitive control of exsanguinating bleeding, neurosurgical management of head injuries, and surgical stabilization of severe orthopedic or musculoskeletal trauma. In most cases, when performing procedures during the resuscitation phase, it is helpful to keep in mind the goal of maintaining organ perfusion as demonstrated by urine output, rather than by volume loading to intravascular euvolemia.

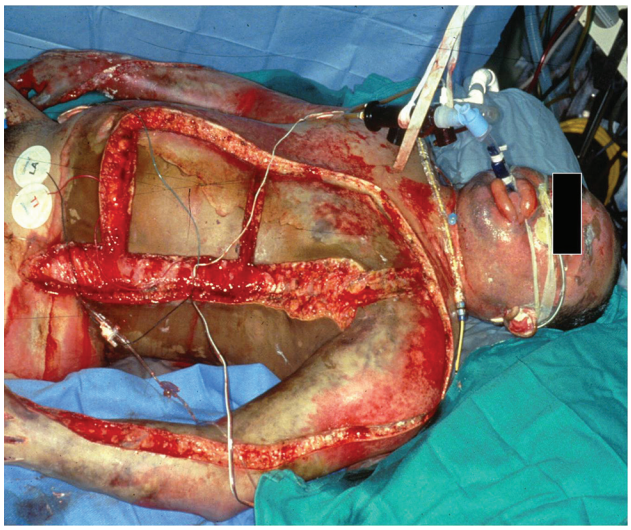

Figure 13-1. Patient with difficult airway and escharotomies performed to facilitate ventilation as well as prevent compartment syndrome of extremities and abdomen.

TABLE 13-1

VARIOUS INTRAVENOUS FLUID RESUSCITATION FORMULAS FOR THE ADULT BURN PATIENT

Formula	First 24 Hours Post-Burn	Second 24 Hours Post-Burn
Burn Budget	LR: 1,000 mL–4,000 mL 0.5 NS: 1,200 mL Colloid: 7.5% of body weight Glucose in water: 1,500–5,000 mL	LR: 1,000 mL–4,000 mL 0.5 NS: none Colloid: 2.5% of body weight Glucose in water: 1,500–5,000 mL
Monafo Hypertonic	250 mEq Na, 150 mEq lactate, 100 mEq Cl with volume adjusted to maintain UOP of 30 mL/h	0.5 NS with volume adjusted by urine output
Brooke	LR: 1.5 mL/kg/% TBSA Colloid: 0.5 mL/kg/% TBSA D5W: 2,000 mL	LR: 0.5 mL/kg/% TBSA Colloid: 0.25 mL/kg/% TBSA D5W: 2,000 mL
Modified Brooke	LR: 2 mL/kg/% TBSA, with half the volume given during the first 8 h and half over the next 16 h	Colloid: 0.3–0.5 mL/kg/%TBSA D5W: to maintain UOP
Parkland	LR: 4 mL/kg/% TBSA Colloid: 0.5 mL/kg/% TBSA D5W: 2,000 mL	Colloid: 20%–60% of calculated plasma volume D5W: To maintain UOP of 0.5 mL/kg/h
Modified Parkland	LR: 4 mL/kg/% TBSA Colloid: 0.5 mL/kg/% TBSA D5W: 2,000 mL	5% albumin at a rate of 0.3 mL/kg/% TBSA 16 per h
Evans	NS: 1 mL/kg/% TBSA Colloid: 1 mL/kg/% TBSA D5W: 2,000 mL	NS: 0.5 mL/kg/% TBSA Colloid: 0.5 mL/kg/% TBSA D5W: 2,000 mL

LR: lactated Ringer solution; TBSA: total body surface area; D5W: 5% dextrose in water; NS: normal saline; UOP: urine output
Data sources: (1) Monafo WW. Initial management of burns. *N Engl J Med*. 1996;335:1581–1586. (2) Warden GD. Burn shock resuscitation. *World J Surg*. 1992;16:16–23.

Anesthesiologists may be called on during this period to intubate burn casualties as ventilatory or respiratory failure is commonly encountered during the resuscitation phase. For patients with burns as their sole injury, those with burns of less than 40% TBSA rarely require intubation, and those with burns of greater than 60% TBSA almost always require intubation. Casualties with inhalation injury may require intubation regardless of the size of their burns. Of note, military burn casualties often present with difficult airways (Figure 13-1) and have a higher percentage of inhalational injury (10.3% with inhalational injury, compared to 5.7% of patients from the 2006 National Burn Repository report).[7] Generally, intubation should be done earlier rather than later in these patients, but it is usually an urgent rather than an emergent procedure. It is paramount to utilize allotted time to optimize and prepare for the procedure. A hastily and poorly planned intubation may bear disastrous consequences.

The decision of whether or not to intubate a given patient in the acute setting often requires considerable judgment. To the astute anesthesiologist, subtle clues such as a subjective change in the patient's voice may tip the decision. The decision having been made, the conduct of acute-phase intubations generally does not require any unusual considerations beyond those for any other trauma patient. If the decision is delayed until the patient is in respiratory distress, the time remaining before hemodynamic compromise may be abridged. Larger endotracheal tubes are much preferred due to the likely need for bronchoscopy and effective respiratory care, including frequent suctioning to clear bronchial clots and mucus plugs. In an emergency situation, a clinician may decide to initially secure the airway with a smaller tube if airway edema makes placement of a larger tube difficult. The smaller tube may then be changed under controlled circumstances, even in the operating room if necessary.

NONSURGICAL BURN CARE

Nonsurgical care of burn wounds is continuously evolving and remains dynamic as new technologies and pharmaceuticals emerge. Antibiotic creams and solutions are often used on partial or full thickness burns. Silver sulfadiazine (Silvadene [King Pharmaceuticals Inc, Bristol, TN]) and mafenide acetate (Sulfamylon [Mylan Inc, Canonsburg, PA]) are the most commonly used agents. Silvadene is considered less painful to apply but does not penetrate intact burn eschar. Silvadene may also cause signifi-

cant leukopenia, typically in the first few days of use.[8] Sulfamylon penetrates burn eschar but can be painful to apply. Sulfamylon is a carbonic anhydrase inhibitor.[9] There are conflicting studies on how often this produces a significant metabolic acidosis.[9,10] In casualties with large burns or renal failure, a hyperchloremic metabolic acidosis is occasionally seen that may be attributable to Sulfamylon and may not resolve until the drug is withdrawn. Silver nitrate is also effective but not frequently used due to its staining ability and lack of superiority over other agents. Other dressings and agents that may have anesthetic considerations are in continuous development.

EXCISION AND GRAFTING

Full-thickness burns, unless miniscule burns, must be treated with excision and grafting. Partial-thickness burns may require excision and grafting or may be treated nonsurgically depending on depth, size, and location. Excision of burned skin and placing skin grafts is the mainstay of burn surgery. There is some controversy over the exact timing of this surgery, but most centers conduct the first operation within a few days of the patient being burned. Over the years, the definition of early excision has changed somewhat, with "early" becoming progressively earlier. Previously, burns were left to slough off weeks after injury, and any excision before then was considered early. Currently, an excision within 48 hours of injury would be considered early. Some surgeons will operate as soon as possible, while others prefer to wait 48 hours to allow the patient to undergo complete initial resuscitation.[11,12] There is no clearly optimal timing for all patients.[12,13] Some surgeons will restrict the scope of the operation to 20% TBSA to minimize blood loss, surgical time, and length of exposure to anesthetic agents. Other surgeons prefer to remove as much of the burn as possible, if not all, on the first procedure. This variability in treatment necessitates situational awareness, communication with the surgical team, and a flexible anesthetic plan.

Excision may be either tangential, in which the burn is shaved off until unburned tissue is reached, or fascial, in which all skin and underlying fat is removed down to fascia, usually by using an electrocautery device. Tangential excision generally produces a better functional and cosmetic result. Fascial excision may be faster and usually involves less blood loss. Whichever method is chosen, these procedures can be unexpectedly bloody, with blood loss varying from 123 mL to 387 mL for each 1% of TBSA excised.[14–16] The larger amount of blood loss arises during operations when multiple surgeons and physician assistants simultaneously excise the majority of burned skin during the initial surgical procedure (Figure 13-2). Also, greater degrees of blood loss are typically seen with older burns, infected burns, and extremity burns.[16] Less blood loss is associated with fascial excisions, fresh burns, and more centrally located burns. The use of tourniquets and fibrin glue may reduce blood loss substantially.[15]

Harvesting of the skin graft may also produce considerable blood loss, especially if the scalp is harvested. Infiltration of epinephrine solution in the area to be harvested can reduce or eliminate this blood loss.[17] Pitkin solution is lactated Ringer solution with 1 to 2 mg of epinephrine per liter. Other combinations of vasoconstrictors in crystalloid solutions are likely equally effective. Postoperatively, most patients report that the site of graft harvest is much more painful than the site of the excised burn. This should be considered if regional anesthesia is part of the plan.

OPERATING ROOM SET-UP

In the deployed and austere environment, supplies and resources for the anesthesiologist may be limited, and improvising is often necessary to ensure good anesthetic outcomes. Taking this into consideration, the following recommendations are best suited for higher echelons of care at military treatment facilities and the definitive care at established burn centers.

All standard anesthesia equipment should be present and machine checks done as for a routine case. The room should be heated to 90°F or as close to that as possible. Commonly, the entire patient's body must be exposed, thus limiting the usefulness of warming blankets. The inability of damaged skin to mitigate heat loss often makes maintaining normothermia an anesthetic challenge (Figure 13-3). As previously discussed, burn injured patients frequently present with difficult airways; it is often necessary to have alternative means of securing the airway beyond direct laryngoscopy. An anesthesiologist may be best served by having emergent airway adjuvants immediately available, as well as video-assisted or fiberoptic devices if possible.

A rapid infusion system capable of warming and infusing blood at 200 mL per minute should be available. Blood should be cross-matched, and two to six units, depending on the circumstances, should be immediately available. For large excisions, 10 to 20

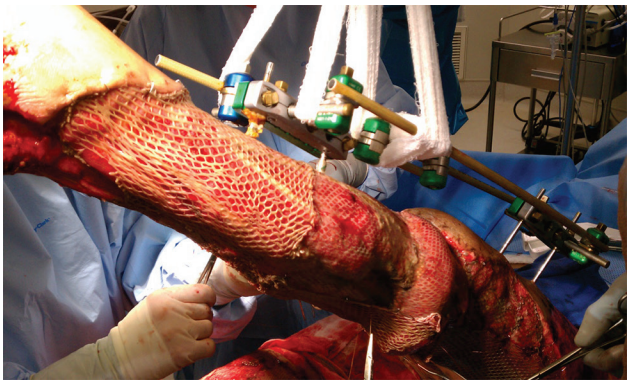

Figure 13-2. This figure illustrates the appearance and placement of a partial thickness skin graft on extensive soft tissue injury of a left lower extremity. Note the external fixating device used to stabilize multiple concurrent fractures.

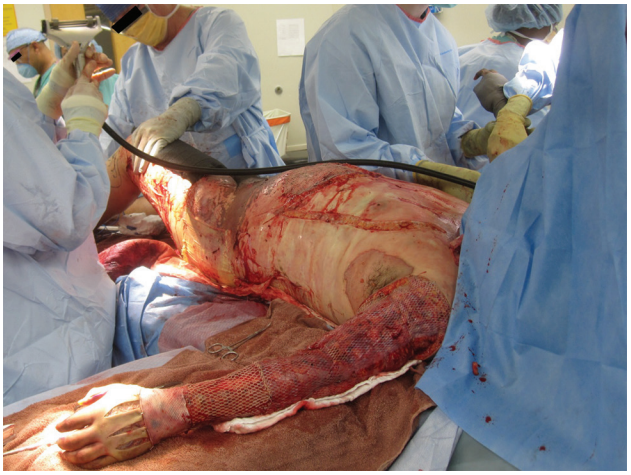

Figure 13-3. Excision and grafting of a severely burned patient. Note the typical amount of exposed body surface area. Extensive surface exposure and damaged skin can make maintenance of normothermia a challenge. In this photo partial thickness autografts are being harvested from the patient's left thigh for grafting onto his left arm. Also note the instrumentation used to harvest skin (top left), and apparent blood loss.

units of packed red cells may be needed over several hours. Platelets and plasma may also be required for larger excisions, even if the patient starts with normal coagulation. Additional IV or central line supplies should be in the room.

PATIENT EVALUATION

Some burn casualties will be extremely ill when scheduled for surgery. In addition to any preexisting disease, burn casualties often have concurrent coagulopathy, hemodynamic variability, and limited oxygenation/ventilation, and they may also be septic from wound infections. It is paramount to realize the patient may not improve until the burn is removed. For example, abdominal compartment syndrome or chest eschar that inhibits patient ventilation can be immediately resolved by surgical intervention. The question to ask is whether there is a problem that can clearly be improved with nonsurgical treatment. If the answer is no, needed surgery should not be delayed.

In addition to the standard preoperative evaluation, several areas dictate extra attention to detail when accessing burn casualties:

- **Airway**. Mask ventilation may be difficult for patients with burn cream on their faces. A standard hand towel may be used to give the hands additional traction on the face, and two-handed masking may be required. Bandages may also be present on the face, making a mask seal quite difficult (Figure 13-4). Patients in the early stages of care may have a tracheostomy. When a patient is no longer ventilator dependent but may require more surgical procedures, surgeons may wish to remove the tracheostomy tube. The anesthe-

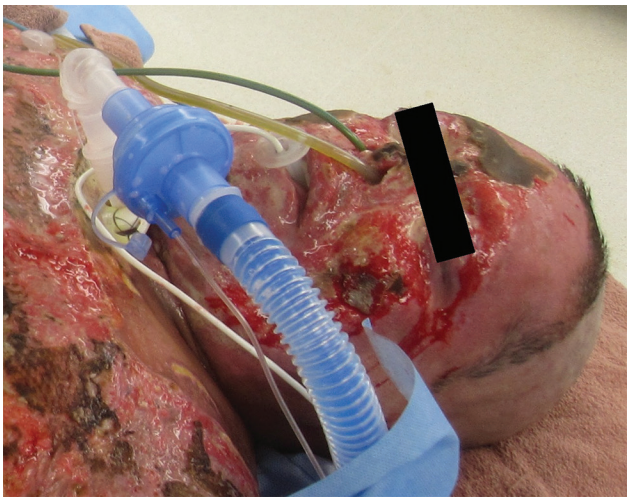

Figure 13-4. With this patient's injuries (without tracheostomy), mask ventilation, securing an endotracheal tube, and providing eye protection may be challenging. Note that the oral airway is being utilized to monitor Spo_2 (oxygen saturation) from the patient's hard palate because of this patient's extensive extremity burns.

siologist should be involved in this decision. Patients with longer times since their injury may develop scarring that limits mouth opening or neck extension. This should be evident from routine examination and may indicate a plan that does not rely on a laryngoscope. For patients who will be intubated in the operating room, some means of securing the position of the endotracheal tube other than adhesive tape should be used. Cloth ties are commonly used, and suturing to a tooth is another viable option. Nasal intubation with cloth ties around the nasal septum is also quite effective.

- **Pulmonary**. The fraction of inspired oxygen (F_{IO_2}), ventilator pressures, and arterial blood gas are very useful in preoperatively evaluating pulmonary issues. Burn patients routinely have higher than normal minute ventilation.[9] High F_{IO_2} and ventilator pressures combined with poor arterial blood gas signal possible difficulty in the operating room. Patients with inhalation injuries frequently produce plugs or clots that can obstruct an endotracheal tube. This is particularly concerning in a patient who will be placed in a prone position. The combination of marginal lung performance, clot production, and prone position can result in a rapidly fatal loss of airway. The anesthesiologist should have a plan for dealing with a plugged prone tube beforehand and communicate that plan to the rest of the operating room crew. Very ill patients may need to remain on an intensive care unit (ICU) ventilator rather than using the anesthesia machine. In this case, the anesthetic plan will require an IV anesthetic. Patients with high ventilator pressures and high inspired oxygen levels may desaturate very rapidly when disconnected from their ventilator, even briefly. This should be considered when planning patient transportation, and alternative plans to maintain positive end expiratory pressure should be utilized, such as clamping the endotracheal tube or placing a positive end expiratory pressure valve during mask-bag ventilation.
- **Circulation**. Patients who survive their initial injury and shock have essentially passed a stress test. These patients should not require further cardiac evaluation except in unusual circumstances. A large burn frequently results in a rise in troponin, even in patients who do not have cardiac disease.[18] Burn patients are typically hyperdynamic and may remain so for weeks or months after their injury. Heart rates in adults of 110 to 120 beats per minute are typical.[18] If a patient is hypotensive early in resuscitation, preload is frequently inadequate; later in the course, afterload is more commonly the cause, but either or both may be present. The patient will frequently require blood transfusion. Knowing the hematocrit at the start of the procedure will help guide the timing and volume of blood products. Critically ill patients with multiorgan dysfunction may require platelets or plasma, as will patients undergoing large excisions. Most patients are treated with some form of thromboprophylaxis. This will be a consideration if nerve blocks or neuraxial anesthesia is planned.
- **Neurologic**. The primary neurologic issues are pain control and sedation. Patients may be receiving substantial doses of narcotic or sedative drugs and remain surprisingly awake.[19] Verifying the patient's level of consciousness and drug doses can help with planning the amounts needed in the operating room.
- **Vascular access**. A well-running 18-gauge IV is sufficient for most cases, as is a well-running central line. A second IV usually provides more than enough access. An introducer is needed only for the largest cases. Some planning is required for smaller lines. Fluid therapy or blood transfusion may need to be initiated earlier if multiple large-bore catheters are not available. An arterial line can be very useful for larger excisions, for both monitoring and lab draws. An arterial line may also be indicated if the surgical plan leaves no suitable site for a noninvasive blood pressure cuff. Placement of all lines must be made with the surgical plan in mind. Placing a line in or near an area to be excised or harvested should be done only if there are no better alternatives. Catheters are prepped into the surgical field when necessary. Even peripheral IV catheters are routinely secured with sutures when fundamentals such as burn creams or prep solutions can render tape useless.
- **Nutrition**. Nutrition is very important to burn healing, and the acute burn casualty generally will have an increased metabolic rate. Thus nonoral nutrition times should be minimized whenever possible. For patients with feeding tubes beyond the gastric pylorus, feeds may be continued until transport to the operating room. There is some controversy about the

need to stop feeds even then. The reasoning for stopping feeds in the operating room is that the patients frequently are treated with vasopressors during and after surgery; it may be detrimental to have feeds continuing at the same time. Patients who are able to eat should generally be allowed to do so until 6 to 8 hours prior to their surgical time.

- **Drips and drugs**. Patients may be on many different infusions, especially in the ICU. Generally, any infusion that is not absolutely necessary is stopped prior to transport to the operating room. This minimizes the equipment needed to transport the patient and reduces the chance of errors, which is especially important in regard to concentrated electrolytes. Preoperative antibiotics are frequently delayed or overlooked. It is worthwhile to verify that any antibiotics ordered have been given.

INTRAOPERATIVE ANESTHETIC MANAGEMENT

Once the patient has been transported to the operating room, the next step is induction of anesthesia. Consideration should be given to induction on the patient's bed if movement is especially painful; however, this is secondary to optimizing a safe and controlled method of securing the patient's airway. Monitor placement and induction are generally routine, with a few exceptions. Monitor placement may be limited by injuries and dressings. Standard electrocardiogram lead placement is rarely essential, and leads may be placed where space can be found. Ordinary leads may not stick to burn patients but may be stapled in place after induction. Noninvasive blood pressure cuffs work surprisingly well over most dressings, but arterial lines are sometimes needed.[20] Creativity may be needed in placing a pulse oximeter. Other than fingers and toes, the ears, nose, lips, forehead, and hard palate (via an oral airway) are some of the sites that may be successful (see Figure 13-4). Exhaled carbon dioxide monitoring is essential in burn patients. It is a reliable indicator of adequate ventilation, as well as a rough guide to cardiac output, and it is the monitor least likely to fail in these patients. Temperature monitoring is almost always used. Inability to maintain a temperature of 36°C warrants maximum effort to warm the patient. A Foley catheter should be used for most cases. Movement of patients to or from the operating table is a high risk period for the inadvertent removal of line or tubes. Several people may be involved in this movement, and one of them should be identified to ensure lines and tubes are in position for movement.

Induction of anesthesia usually involves muscle relaxant drugs. Succinylcholine is widely recognized as being contraindicated in burn patients. Succinylcholine is safe for the first 24 hours after a burn, but beyond that period and for up to a year after healing it may cause a dramatic and fatal hyperkalemia.[21] Nondepolarizing muscle relaxants are regularly used in burn patients, with the understanding that they will require larger doses and will not last as long as in patients without burns.[22] The exception to that rule is mivacurium, which lasts as long or longer in burned patients as unburned patients.[23] Some patients develop a hyperreflexia that is commonly elicited when lifting a leg. This can produce jerking that only stops with muscle relaxation. Aside from that situation, muscle relaxants are generally not required beyond intubation.

Burn patients may be tolerant to opiates, especially if they have been given large doses for several days, but they generally respond normally to the usual induction agents. The burn patient also tends to have an attenuated response to catecholamines. They may require doses of phenylephrine (or other pressors) considerably higher than do unburned patients to achieve the expected hemodynamic response.

Patients who do not require an ICU ventilator may be given inhalational anesthesia. Potent inhalation agents supplemented with opiates work well for most patients. If an IV anesthetic is chosen, propofol supplemented by opiates works well. For patients with a large blood loss, the propofol infusion may have to be decreased to very low rates, even in well resuscitated patients.[24] Ketamine has been a traditional choice in burn patients and works well either as the main agent or as a supplement to other IV agents.[25] Emergence delirium is seldom an issue in critically ill ICU patients, who are generally maintained on sedatives for days after their surgery. Despite its reputation, long term psychological effects have not been documented with ketamine.[26]

Once the patient is prepped, the operation may begin. Blood loss during the excision portion may be dramatic, with 1 to 2 liters lost in a short period of time. Large excisions may cause the loss of 5 or more liters over the course of a few hours (see Figure 13-4). When to transfuse is a decision that must be based on individual circumstances. Healthy young adults can easily tolerate hematocrits of 20 or even less. Once the hematocrit drops below 18, the patients typically become hypotensive and poorly responsive to pressors. Older, less fit patients may be less tolerant of anemia. As a general rule, if the patient is hypotensive,

it is rarely wrong to initiate red blood cell transfusion while investigating the cause, especially if the patient is not responding well to fluid and phenylephrine. The choice between crystalloid and colloid will vary. At many burn centers, the principal IV fluid is Plasma-Lyte (Baxter, Deerfield, IL). Plasma-Lyte is not as acidic as saline and is compatible with blood products. The quality of IV access and speed of blood loss will also influence the timing of transfusion. Some patients will require multiple units of packed red blood cells to be transfused in a short period of time. This may lead to decreased ionized calcium in the patient's blood and the need for IV replacement.[23,24] With larger excisions, other blood products such as plasma and platelets are commonly required. The need for non-red cell blood products varies considerably from case to case and is usually driven by laboratory values, clinically observed bleeding, and the judgment of the staff involved. Recombinant activated Factor VIIa and antifibrinolytics have been used occasionally in cases involving large blood loss, but at present no data proves it is helpful.

For larger excisions or unstable patients, obtaining frequent blood gas measurements may be helpful. The base deficit and hematocrit will guide the choice and amount of fluids used. Venous blood gasses may be used for this purpose if an arterial line is not in place. "Arterialized" venous blood, as it is sometimes called, can be drawn from the distal extremities such as the back of the hand for a more accurate venous estimation of an arterial blood gas. Urine output or lack of it may also guide volume replacement. It is not uncommon to require vasopressors to maintain adequate blood pressure after surgery has begun. This may be due to the release of bacteria or other factors during excision of the wound. Vasopressin, norepinephrine, and phenylephrine are commonly used with effectiveness that varies from patient to patient, but it is essential to keep in mind that the severely burned patient may be catecholamine depleted and thus require larger than anticipated doses of vasopressor agents. Care must be taken to ensure that anemia or lack of preload is not the cause of hypotension before relying on vasopressors to maintain blood pressure.

If blood loss gets ahead of the resuscitation during surgery, it may be necessary to ask the surgeon to stop so the anesthesiologist can continue resuscitation. The basic life support rule of holding pressure to stop bleeding can be very effective. Epinephrine-soaked lap pads are employed to assist in hemostasis. Patients tend not to show a significant response to the epinephrine.[15] Patients may also receive several milligrams of subcutaneous epinephrine from Pitkin solution without change of heart rate or blood pressure. Halothane has a long history of use in burn patients, but may not be desirable when epinephrine is used to assist with hemostasis, due to the risk of ventricular arrhythmias. At least one study, however, has shown halothane to be safe in this situation.[27] In cases of large burns, the patient may receive several liters of Pitkin solution mobilized over the following 24 hours, along with IV crystalloids. This volume must be considered when making decisions such as whether to extubate a patient after surgery.

After skin grafts are placed, they may be covered with a negative pressure dressing or conventional gauze dressings. At this point, protecting the graft from shearing is very important. A smooth, pain-free wakeup will help prevent patient thrashing that may shear the grafts. Narcotic should be titrated to respiratory rate. As noted earlier, patients may require large doses.

POSTOPERATIVE CARE

ICU patients are returned to the ICU and the care of the surgeon or ICU team. With few exceptions, patients should be monitored during this transport. It may also be reasonable to transport them with additional sedating and vasoactive medications, depending on the patient's postoperative stability. Once in the ICU, the anesthesia team should ensure that the patient is rapidly connected to the ICU monitors. If it was necessary to start any vasopressors during the case or if the patient has been unstable, the anesthesiologist should ensure that the physician responsible for the patient in the ICU is aware of these issues and present to manage them.

Ward patients may be taken to the recovery room and treated in the standard fashion. Patients with negative pressure dressings in place should be reconnected to suction without delay. Pain control usually requires treatment with opiates. Morphine, hydromorphone, and fentanyl titrated to effect work well. Meperidine is not used due to the potential for toxic metabolites to accumulate.[28] Methadone has also been used successfully, sometimes when other opiates have failed.[29]

PROCEDURES OUTSIDE THE OPERATING ROOM

Burn patients often require wound care that may be quite painful. Surgeons will commonly attempt to treat these patients with a combination of benzodiazepines and opiates. Some patients cannot tolerate

their care without more profound acute pain control. Wound care procedures are often done in a shower room. Sometimes this may involve removing large adherent dressings or debriding wounds. Aggressive wound care can help some patients avoid the operating room. Numerous regimens are acceptable for this sort of pain control. Choosing a plan that is familiar and comfortable for the anesthesiologist is probably more important than any particular drug selection. Propofol infusions can be very effective for these procedures. Infusion rates of 50 to 200 μg/kg/min are well tolerated. Patients frequently require jaw lift to maintain spontaneous ventilation, especially after a bolus dose, but apnea is very rare if opiates have not been added. If propofol alone is inadequate, small doses of ketamine can be added, typically 10 to 20 mg at a time, up to 1 mg/kg. These cases are routinely performed without need for positive pressure ventilation or supplemental oxygen. Pulse oximetry is sufficient monitoring for most patients in this situation. Exhaled carbon dioxide monitoring can also be very reassuring if it is available. Additional monitors may be considered based on individual patient issues.

ELECTRICAL INJURIES

Electrical injuries, while similar to other burns, have a few distinctions. There may be extensive underlying tissue destruction beyond the obvious contact point, especially with high voltage injury.[30] Muscle destruction is common with electrical injury; monitoring potassium, creatine kinase levels and serum urea nitrogen/creatinine is mandatory for electrical injuries. Patients with electrical injuries are usually monitored in an ICU for 24 hours, even with small burns, to observe for cardiac arrhythmias. That said, malignant cardiac arrhythmias are rare.[31,32] Patients with electrical injury commonly have a superimposed thermal burn injury if the electrical current has ignited their clothing.

NONTHERMAL SKIN DISEASES

Any injury or disease that causes a significant loss of skin may be suitable for treatment in a burn unit. One of the most common disorders is toxic epidermal necrolysis syndrome (TENS). TENS has a reported 30% to 50% mortality rate.[33–35] The disease causes a partial thickness skin injury that may also involve the mucosal membranes. TENS does not usually require skin grafting, but the anesthesiologist may be involved for airway issues. An important consideration is that mucous membranes may slough and cause bleeding with manipulation. Direct laryngoscopy in a TENS patient may result in bleeding sufficient to obscure the view of the airway. The first attempt at laryngoscopy may provide the only good view, and fiberoptic bronchoscopy may be difficult or impossible afterward. This should be considered when planning to secure the airway. TENS patients may also produce plugs that can acutely obstruct an endotracheal tube.

Other skin disorders, such as pemphigus vulgaris, may be treated in a burn unit from time to time, but rarely pose an issue not already considered in the care of burn patients.

CONCLUSION

Burn patients often return to the operating room multiple times over the course of weeks to years. This provides an opportunity for continuity that anesthesiologists seldom get with other patients, as well as the satisfaction of seeing patients progress from critically ill to recovered and functional. Providing anesthetic services to the burn casualty requires knowledge of, and preparation for, several specific issues. The severely burned military casualty is one of the most challenging patient populations to be cared for by perioperative providers and anesthesiology teams alike. In the face of difficult airways, poor IV access, hypermetabolic states with significant challenges in supporting adequate caloric intake, hemodynamic instability associated with burn shock and cardiovascular compromise often requiring multiple vasoactive infusions, and frequent surgical interventions and painful rehabilitation, provision of care to this select population requires knowledge of and preparation for the many specific issues discussed in this chapter. With proper planning and close coordination with the burn care team, these patients can be cared for effectively, safely, and compassionately.

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Chapter 14

IMAGING

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INTRODUCTION

The aim of trauma imaging is to identify suspected or unsuspected life-threatening injuries or hemorrhage. This can be achieved with a combination of traditional x-rays, ultrasound, and computed tomography (CT) scanning, as well as the more recently developed magnetic resonance imaging (MRI). Environmental factors that are typical in a military medical environment, such as noise, heat, vibration, and limited space, or patient factors, such as reduced levels of consciousness, often complicate trauma diagnoses. Therefore the trauma team focuses on rapid physical examination closely followed by prompt imaging in order to identify those conditions requiring urgent treatment. The anesthetist's understanding of appropriate radiological modalities for assessment of the patient and how these techniques guide supportive therapy is paramount.

INITIAL TRAUMA ASSESSMENT

Standardized guidelines drive initial patient assessment.[1] Injuries are searched for in a rapid, systematic, and logical way to identify immediate life-threatening conditions before conducting a more detailed secondary survey. Assessment and treatment of the patient often take place concurrently, allowing rapid recognition of a hemodynamically unstable patient who requires urgent surgery. In such cases, no further time should be spent in the resuscitation bay, and the patient should be transferred into the operating theater for surgery. Any immediate life-saving surgical intervention should not be delayed by imaging.

Basic Radiography

All military trauma patients will get a portable chest and pelvic radiograph in the first 5 minutes utilizing digital x-rays that allow immediate review by the clinicians. These plain x-rays provide essential information on possible life-threatening conditions. Chest x-rays are valuable in the diagnosis of a pneumothorax, hemothorax, rib fractures, or a widened mediastinum. Pelvic x-rays can identify pelvic and hip fractures, although the images provide limited information on fracture stability. Pelvic fractures most often occur from a variety of mechanisms that involve high-energy blunt force trauma and have a high morbidity and significant mortality.

Focused Assessment With Sonography for Trauma

Modern ultrasound technology provides a rapid, portable, and reliable method to screen patients with abdominal trauma for the presence of hemoperitoneum. These techniques evolved into a new ultrasound-guided examination method termed "focused assessment with sonography for trauma" (FAST). FAST has been defined by a consensus conference as an expeditious, focused interrogation of the pericardial and peritoneal space looking for free fluid as a marker of injury.[2] All major trauma patients (as defined by an injury severity score [ISS] of greater than 15 or a significant injury to two or more ISS body regions) receive a FAST performed by a consultant radiologist within the first 5 minutes of arrival in the resuscitation room.

FAST is essentially a method of identifying intraperitoneal fluid with ultrasound by scanning several areas: the hepatorenal space, the splenorenal space, and the pelvis. If fluid is detected, it implies a hemoperitoneum requiring urgent surgery, thus negating the need for another confirmatory diagnostic test in an unstable patient. Therefore, FAST can reduce the time from initial assessment to operative care.[3]

FAST can also be used to examine the heart in vivo, which is especially useful in determining the presence of cardiac activity in cardiac arrest, and in the diagnosis of hemopericardium and potential cardiac tamponade.[4] The same ultrasound probe can be used to create a view of the lung bases and diaphragm, once again looking for fluid. Fluid at the lung bases is highly suggestive of a hemothorax, which provides a diagnosis requiring prompt treatment. As knowledge, skill, and technological advances in ultrasound have progressed, the scan can now be used to detect pneumothoraces; this additional component of the scan has been termed "extended FAST" (EFAST).[5]

The FAST scan has superseded the diagnostic peritoneal lavage (DPL) in assessing the presence of hemoperitoneum, and DPL is now rarely used. The combination of plain radiographs and the FAST scan will help direct the insertion of chest drains, pelvic splinting, or emergent thoracotomy/laparotomy, as appropriate. However, if the scan or radiographs are negative, further investigation may be warranted.

Computed Tomography

Computed tomography (CT) scans have been available for several decades, and since the late 1990s whole-body CT has increasingly been used as part of the initial trauma resuscitation.[6] The process has evolved

since then, mostly due to advances in CT technology. Now, due to their speed and accuracy in diagnosis, CT scans have not only become an important part of the primary trauma survey, but have also been shown to increase the probability of survival in patients with polytrauma.[7] A whole-body scan from vertex to thighs takes less than 10 minutes, and multidetector row spiral CT (MDCT) allows for scanning large volumes in a single breath-hold. CT scanning of trauma patients provides a high yield of unexpected injuries, in up to 38% of patients in some studies.[8]

Routine and liberal use of CT scans is not without problems. There is always a danger in exposure to ionizing radiation and its associated increase risk of cancer.[9] A second potential issue is cost, although any financial analysis should take into account the costs of missed or delayed diagnosis if CT is not used. Lastly, for the abdominal component of the CT scan, intravenous contrast is required, which raises the possibility of contrast-induced nephropathy and the development of acute renal failure. However, using a CT scanner has become easier in theater due to advances in logistic transportation and readily available technical support.

ANESTHETIC MANAGEMENT FOR IMAGING

The use of CT in a trauma patient requires the anesthetist to prepare for the patient's movement from the resuscitation bay to the scanner. Patients should be hemodynamically stable, have a clear airway or be intubated, be adequately ventilating, and have standard monitors in place. The anesthetic issues related to performing a CT scan are summarized as follows:

- Airway
  - Possibly requires intubation and a rapid sequence induction due to full stomach and a potential difficult airway (unstable spine)
- Monitoring
  - Possibly requires invasive monitoring
  - Careful observation is required for cardio respiratory stability
- Transfer equipment
  - Ventilator and sufficient oxygen cylinder capacity
  - Syringe drivers and drug therapies
  - Suction and emergency drugs
- Transfer
  - Patient is transferred from bed or trolley to scanner
  - Awareness of any spinal injuries is required
- CT scan room
  - Limited access to patient
  - Reduced space
  - Reduced staffing
  - Proximity of resuscitation equipment
  - Patient anxiety
- On-going medical care
  - Continued resuscitation with drug therapies
  - Warmed intravenous fluids

IMAGING BY BODY REGION

In the postresuscitation and postoperative phase of trauma care, imaging remains a useful source of information to track patient progress and guide clinical decision-making. Imaging is a routine part of more formal secondary surveys, and should be used prior to any surgery if the patient remains stable. A variety of modalities are available in the current Role 3 combat support hospital, from the basics of plain films and fluoroscopy to ultrasound and CT. Previously available only in Role 4 facilities, MRI has recently become available at Role 3.[10] MRI can identify a number of injuries not readily visible on CT scan; however, because battlefield trauma frequently has a ballistic component, its use may be limited until further in the timeline, once all metallic risk has been assessed. Nonmainstream radiological techniques have possible applications, which will be discussed after a review of current techniques.

Head Injury

CT is the mainstay of imaging for craniocerebral trauma. Any suspected head injury requires CT assessment to identify hemorrhage, mass affect, and edema; CT scans can also be used to accurately and quickly insert minimally invasive intraventricular drains.[11] MDCT, if available, can transform CT scans from a cross sectional view to full 3-dimensional views, allowing greater yield for maxillofacial pathology.

Imaging is critical to both the diagnosis and management of traumatic brain injury (TBI). For diagnosis of TBI in the acute setting, noncontrast CT is the modality of choice because it quickly and accurately identifies intracranial hemorrhage that warrants neurosurgical evacuation. For the management of TBI patients, noncontrast CT readily identifies the progression of hemorrhage and signs of secondary

injury relevant to neurocritical care, such as cerebral swelling, herniation, and hydrocephalus.

Historically, skull plain films have had a place in mild head trauma, identifying fractures, air-fluid levels, and foreign objects. However, intracerebral injury can occur without any of the listed pathology. Therefore, plain films are used less frequently if the mechanism of injury suggests possible craniocerebral pathology, and a full CT scan should be performed if available. The deteriorating patient on the ward, after initial assessment, should also receive a CT because the decline in neurological state would be highly suggestive of an intracerebral cause.

Thoracic Injuries

The plain chest radiograph is a simple and common modality in the diagnosis of thoracic injuries and can provide good information about the thoracic organs and surrounding structures and tissues. On admission to the intensive care unit, the patient routinely receives a chest radiograph, allowing identification of the correct positioning of lines, chest drains, and endotracheal tube. A chest radiograph gives good diagnostic information about the lung fields and can reveal some pathognomic signs for other cardiac complications, such as tamponade or pericardial effusion.

Pneumothorax and hemothorax are readily identified on a plain film, if of sufficient size, but a poor quality film, complicated by supine positioning of the patient, alters the dependent areas and can lead to diagnostic uncertainty, requiring CT for clarification. Lateral supine films can increase yield for recognition of intrapleural fluid and pneumothoraces, but with the availability of CT these are rarely performed. Ultrasound by a skilled operator can be used to identify a pneumothorax, pleural fluid depth, and degree of loculation, as well as to guide drain placement. Differential diagnosis between fluid and blood is difficult with ultrasound and if necessary a CT scan can be used to distinguish between the two.

Lung soft tissue injuries, such as blast lung or pulmonary contusion, are often imperceptible on initial assessment and may only be suggested by a decline in respiratory gas exchange. Plain film changes can often take several hours to develop, whereas CT findings are seen much earlier. It is well accepted that the initial radiological signs of pulmonary contusions often fail to show the extent of the lesion. CT pulmonary angiography also has its place in the diagnosis of pulmonary embolism; however, careful consideration should be given to the impact of contrast medium on the patient and to whether such an investigation would change current management.

Cardiac Injury

Hemodynamic instability in the post–acute-phase trauma patient could point to cardiac compromise if other causes such as hypovolemia and tension pneumothorax have been excluded. Plain chest films are unhelpful, except in excluding these other causes, but echocardiography, either transthoracic or transesophageal is far more useful, allowing diagnosis of pneumopericardium, pericardial effusion and tamponade, together with their effects on cardiac function. Echocardiogram-guided pericardiocentesis also improves safety and success rate. CT scans will show gross anatomy, along with evidence of a pericardial effusion, pneumopericardium, or pneumomediastium, but will not allow assessment of cardiac performance.

Vascular Injury

Blunt aortic injury is considered the second most common cause of death after head injury in blunt trauma patients. If an aortic injury does not cause death immediately, the patient may present with severe hemodynamic instability or even be totally asymptomatic. Accurate and rapid diagnosis is vital. Plain chest films can reveal some signs, such as widened mediastinum, depression of left mainstem bronchus, or lateral displacement of the trachea. However, in one study, nearly half of patients identified with aortic rupture by CT had a normal mediastinum on plain film, and only 12% of patients with a widened mediastinum had an aortic injury.[12] An aortogram has been the gold standard for imaging in suspected thoracic trauma, but it is invasive and time consuming, and does not detect small intimal injuries that are seen on CT or transesophageal echocardiography. One study has shown CT to have a sensitivity of 99%, as compared to 92% in angiography for blunt aortic injury.[13] Angiography has largely been replaced by CT and is now reserved for difficult to diagnose cases.

A newer evolution in using CT to assess peripheral vascular damage in limb injuries is a carefully timed bolus of contrast followed by rapid acquisition of data (10 seconds or less) over long vascular territories.[14] This can be done relatively safely by releasing the tourniquet for this short period of time.

Intraabdominal and Pelvic Injury

CT is the mainstay of radiological assessment for trauma-related intraabdominal and pelvic injury. Damage to any viscus as well as evidence of ischemia can be identified, guiding decisions on further surgical intervention.

Musculoskeletal Injury

Imaging for musculoskeletal injuries is very much the remit of orthopedic surgeons, who require radiography for diagnosis and guiding management. Initial diagnosis of injuries will be with the full body CT and plain film on first trauma assessment. Any further directed imaging such as plain films would be on the advice of the orthopedic team.

ULTRASOUND REGIONAL ANESTHESIA AND VENOUS ACCESS

Ultrasound is routinely used in the combat hospital for regional techniques including nerve blockade and the placement of nerve block catheters. Difficult vascular access can also be aided by ultrasound images, and should be used when possible for gaining central venous access in accordance with guidance from the National Institute of Clinical Excellence.[15].

Interventional Radiology

Because of the patterns of injury in the current conflict, interventional radiology such as embolization of bleeding from pelvic trauma is not used. Interventional radiology does have some application in the civilian world, but will not be discussed here. However, inferior vena cava filters (IVCFs) have been used in combat trauma patients, and have been subject to lengthy discussion. Recent developments in damage control resuscitation, using massive transfusion protocols and early use of recombinant factor VII, render major trauma patients at risk of prothrombotic complications when pharmacological thromboprophylaxis is not appropriate. Use of IVCFs decreases the risk of complications such as pulmonary embolism without the problems associated with pharmacological prophylaxis. However, the lack of prospective randomized trials in this area has created a void in evidence-based recommendations. Retrievable IVCFs, which can be removed at a later date when the risks of venous thromboembolism have decreased,[16] may offer the best risk-benefit ratio for traumatized patients (although they are likely to be used only at Role 4).

FUTURE DIRECTIONS IN IMAGING

An increasing body of published literature is documenting further developments in FAST, including its use in assessing cardiac function as a transthoracic echocardiogram, viewing the inferior vena cava diameter to assess volume status and degree of hypovolemia, and viewing the optic nerve sheath diameter, which can act as an indicator of raised intracranial pressure.

Using data from CT scanning for research could provide further insight into patterns and mechanisms of blast injury, in particular the altered metabolism and hemodynamics in TBI.

New portable technologies for the battlefield may include transcranial Doppler to monitor intracerebral hemodynamics. This technology is readily available and is used in many intensive care units, including military hospitals, to monitor patients for vasospasm. Portable devices for monitoring pupillometry are similarly employed in intensive care units and may prove useful in the battlefield. Similarly, near-infrared optical imaging devices are under development that could measure oxygen extraction ratios with 5- to 15-mm resolution and up to 10 mm deep under the skull. A key issue raised by these emerging technologies is balancing research needs, which might guide new therapies and help prevent injury to soldiers, with the existing burdens of gear weight and the need for medical personnel in the field to focus on delivering life-saving care and moving personnel out of harm's way as quickly as possible.[17]

SUMMARY

An essential part of the initial trauma assessment, imaging comprises plain radiographs, FAST scans, and CT scans. Further imaging may be required after the initial trauma assessment, usually in the modality of a plain film or CT. Ultrasound has many uses apart from FAST, including facilitating vascular access and the provision of regional anesthesia.

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Chapter 15

MANAGEMENT OF STABLE CASUALTIES

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INTRODUCTION

"While it is evident that the general principles of anesthesia are not affected by the circumstances of war, it is equally evident that it is our duty assiduously to seek those means in anesthesia which are especially suited to the exigencies of battle; and I hope to show that although men of the fighting services are of necessity exceptionally fit before an engagement, they may frequently be most urgently in need of the best attention known to anesthesia after the conflict."[1] Wesley Bourne's observations at the Annual Meeting of the Massachusetts Medical Society, May 22, 1941, remain pertinent to the conduct of anesthesia in current combat conditions.

It is self-evident that management of combat-related trauma remains the fundamental clinical activity of a military medical treatment facility. The overall quality of care of the severely injured patient in combat circumstances, who is by definition unstable, has always attracted considerable attention. In the last decade, significant advances in care have resulted in unexpected rates of survival.[2] Similarly, the anesthetic management of the stable casualty will of necessity require attention not merely to technique but also to external factors, including battle tempo and logistics, in order to ensure successful continuum of care.[3]

THE STABLE CASUALTY

Labelling a patient's condition as "stable" may be a potentially risky decision, especially for combat casualties presenting to a military medical treatment facility (MTF). The American Hospital Association advises that the term "stable" not be used, either as a condition or in combination with other conditions, because such statements are often inherently contradictory and misleading.[4] In the context of combat trauma-orientated MTFs, stability is largely denoted by physiological variables. While this approach remains useful, it must also include consideration of the mechanism of injury and the extent of energy transfer. These considerations will help the perioperative medical team maintain appropriate levels of vigilance with a stable patient. The concept of the "metastable" patient (one whose current stability is judged to have the potential to change rapidly) is useful in this regard, particularly in relation to preserving surgical situational awareness and in guiding anesthesia strategy for these patients.[5]

For the purposes of this text, the term "stable" refers to those patients whose condition is not expected to deteriorate in the next 24 hours. Surgery on stable patients will mostly be performed according to scheduled rather than emergency operating lists, and such patients may be:

- casualties requiring surgery for injuries that are not time-critical,
- casualties needing follow-up procedures for wound and injury care, or
- patients with disease non-battle-injury (DNBI) problems (eg, appendicitis).[6]

Consequently, stable casualties range from the minor DNBI patient to the complex polytrauma patient on whom damage control surgery has already been performed. Various observers including the authors have noted the rapidity with which apparently stable patients can deteriorate, either due to the consequences of surgery or because of injuries that have evolved or gone unrecognized.[7–9] Indeed, this situation necessitates that high and specific levels of clinical vigilance be maintained, which can normally be accomplished in a more comprehensive Role 3 MTF or combat support hospital, which would be better resourced than a forward surgical facility.[3] Therefore, anesthesia for stable military patients is likely to be undertaken in a field hospital, either Role 2 (enhanced) or Role 3 because these facilities are expected to possess the necessary resources for maintaining such clinical oversight.[10] Nonemergent surgery is rarely performed further forward than this. The contents of this chapter will also be relevant to the anesthesiologist working in a parent nation's domestic Role 4 hospital, who may be required to anesthetize battlefield casualties within days of their injury.

THE POPULATION AT RISK

The deployed military population is composed of predominantly young, fit, and prescreened individuals. Every effort is made to evacuate the seriously injured military patient to a suitable Role 4 (domestic) hospital as soon as possible, both for clinical benefit and to enhance operational agility in dealing with further casualties at the forward facility. Such evacuation reduces workload, supply consumption, and bed occupancy in the field hospital. Consequently, most of these patients will receive only emergency or damage

control surgery at the field hospital, although weeks of follow-up and reconstructive procedures may await them at Role 4.

The field hospital, deployed on operations other than war such as disaster relief and peacekeeping operations, as well as during combat, can expect a significant number of locally born patients.[11] These may be civilians (both adults and children),[12] military allies, or detainees. An analysis was performed of the surgical workload of a NATO field hospital deployed to Kandahar, Afghanistan, over 5 months in 2006. Of 259 patients treated, 118 were Afghan soldiers or police, 60 were local civilians, and 10 were detainees.[13] Such patients can have poorly managed or untreated chronic disease, which will present an extra challenge to the anesthesiologist. There may also be entitled civilian contractors with undeclared chronic health problems that would have precluded their employment had they been divulged. Conditions such as hypertension, ischemic heart disease, diabetes mellitus, and malignancy have all been seen in this population during recent operations in Iraq and Afghanistan.[6] Civilian patients, in particular, form a significant proportion of the scheduled operating workload, since they may remain under the care of the field hospital for weeks while waiting for transfer to a suitable facility. During this time they can require multiple returns to the operating room for wound debridement and dressing changes.

SPECIAL CONSIDERATIONS IN THE STABLE CASUALTY

It is the stated intent of UK Role 3 MTFs to deliver healthcare at least to the standard of that provided at Role 4 civilian hospitals in the National Health Service in the United Kingdom. In the nonemergent patient, military anesthesiologists must consider the possible requirement to modify their approach to one much more in keeping with a civilian hospital setting. Patients should be fasting, and a well-documented anesthetic history and examination should be performed, which may uncover chronic health issues such as those mentioned above. However, unlike in elective civilian practice, the military anesthesiologist must be prepared to proceed with a medical history that may be fragmentary and inaccurate (especially for local national patients). Therefore, perioperative vigilance is vital to deal with unanticipated problems.

Informed consent for the proposed anesthetic technique should be obtained, following good-practice guidelines[14]:

- fully disclose serious or frequently occurring risks;
- discuss potential benefits and alternatives;
- avoid providing new information immediately prior to anesthesia induction, when possible;
- ensure the patient is able to understand, retain, and use the information provided;
- use a trained interpreter to facilitate communication, if necessary; and
- record the discussion in the clinical notes.

Two particular patient groups require further consideration: pediatric patients and detainees. Experience during recent operations indicates that all expeditionary MTFs will need to be able to manage pediatric patients with trauma, medical conditions, or both.[15] For pediatric patients, parental involvement in treatment decisions and their presence at induction of anesthesia is highly desirable, just as would occur in a Western civilian hospital. For detainees, there may well be security requirements. Security should be in keeping with the expeditionary force's ethical guidelines, and detainees should be provided the same information and treatment choices as any other patient.

COMMON OPERATIONS IN STABLE CASUALTIES

Nonemergent surgery forms a significant proportion of the operative workload of a deployed field hospital. In the previously mentioned analysis at a NATO field hospital in Afghanistan, of the 393 quantifiable procedures performed over a 5-month period, 166 were wound debridements. Dressing changes for trauma or burns were the second most common operations performed by general surgeons (15% of all their procedures). Other nonemergent procedures included drain and packing removal and skin grafting. DNBI patients encompassed 8% of the general surgical workload, including seven appendectomies. Seventeen patients returned to the operating room for further surgery (including one patient who required six separate operations).[13]

By their nature, the injuries of war often require multiple operations, particularly in those patients who have not been repatriated. Wounds are often highly contaminated at presentation and may require repeated dressing changes and debridement. Delayed primary closure of such wounds, if possible, is performed between day 4 and day 6, during the

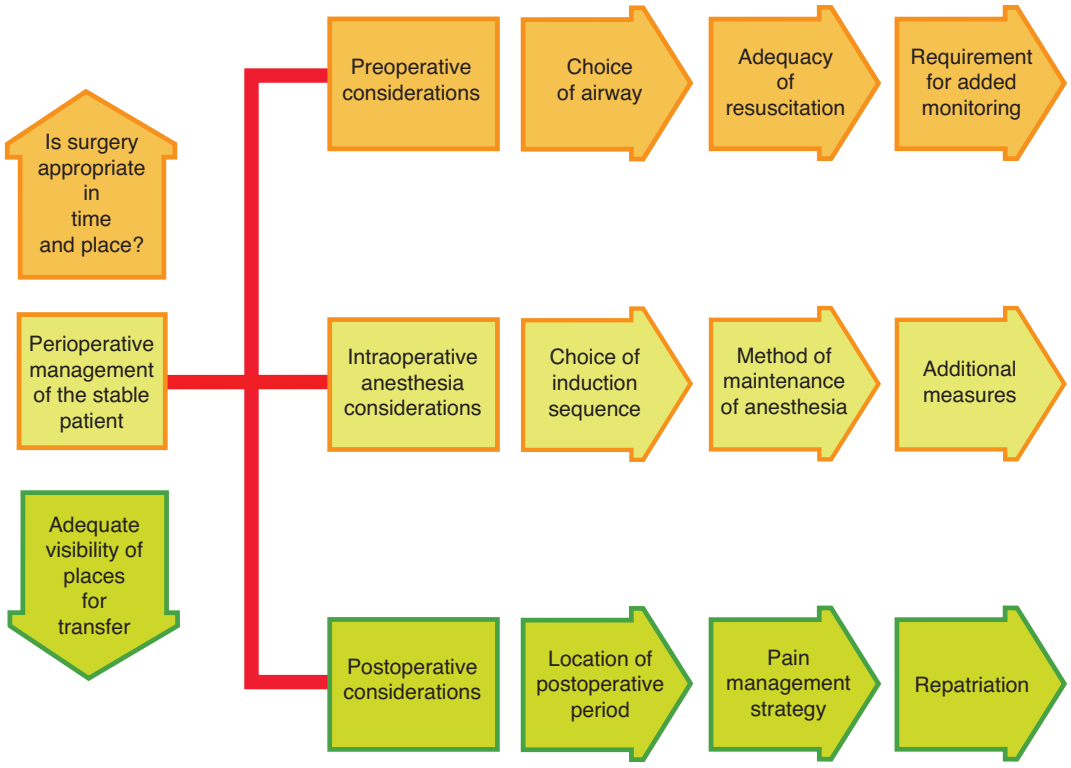

Figure 15-1. Perioperative considerations in a stable trauma patient.

fibroblastic phase of healing when postinjury swelling has diminished.[16] When this is not possible, split skin grafting may be used. Re-look laparotomies are also performed, especially in the presence of laparostomy, as the clinical picture demands, or in the light of radiological findings.

The potential for blood loss with such operations cannot be overemphasized. It is prudent to ensure that blood is available and that large-bore intravenous access is in position prior to commencing the procedure. Equally, the assurance of the stability of imaging, hematological, and biochemical variables will further inform the decision to re-operate. Early visibility of plans to evacuate a given patient elsewhere within the theater of operations or indeed outside it will assist in formulating rational perioperative management plans. Figure 15-1 outlines the major perioperative considerations in a stable patient.

CHOICE OF ANESTHETIC TECHNIQUE

Choice of anesthetic technique is usually dictated by patient presentation and personal preference. The deployed military anesthetist must also take account of the resources available as well as the tactical and strategic contexts. This section explores the potential advantages and disadvantages of each technique in the stable war surgery patient.

Clinical reassessment of the stable patient together with the results of appropriate investigations will normally precede the anesthetic. Whichever technique is selected, optimization of analgesia in the stabilized, nonemergent war surgery patient is highly desirable. Humane considerations apart, pain associated with repeated procedures may increase the care burden in an environment where resources are not unlimited. Furthermore, acute uncontrolled pain has deleterious effects not only on the patient but also on family members and medical staff.[17] A causal link to the appearance of chronic pain problems following combat injury remains to be fully elucidated, although the benefits of timely and effective interventions do appear to reduce longer-term consumption of analgesics.[18]

Intraoperative monitoring will normally be expected to conform to the standards of care provided at a Role 4 hospital.[19,20] Depending on individual national doctrinal stance and equipment scaling, appropriate advanced monitoring modalities such as thromboelastometry and intracranial pressure monitoring are

applicable to anesthesia for stable patients receiving intensive care in order to maintain their continued recovery.

Volatile Gas Anesthesia

An intravenous induction sequence followed by volatile gas anesthesia (VGA) continues to be widely accepted as a safe and practicable choice in the deployed setting. Details of this well-understood technique will not be discussed here; however, it is worth noting that all volatile agents produce dose-dependent depression of myocardial contractility, with the newer agents (desflurane, isoflurane, and sevoflurane) maintaining cardiac output better than older agents. Although there is no absolute contraindication to any volatile agent, nitrous oxide should be avoided to limit bowel and closed-space gas accumulation in the presence of potential pneumothorax, pneumocephalus, and bowel trauma[21] (its availability on deployment is likely to be limited anyway). Concerns have arisen regarding low-flow VGA using sevoflurane, which has been demonstrated to produce nephrotoxic compound A in rats. However, a study in humans was unable to reproduce this effect.[22]

Regional Anesthesia and Neuraxial Anesthesia

Regional anesthesia (RA) and neuraxial anesthesia may be used alone or in combination with general anesthesia or conscious sedation. The physician's imperative to "first, do no harm" is particularly applicable when considering the deployment of these techniques. The military anesthetist should be confident that the casualty does not have coagulopathy of trauma shock. Thromboelastometry, where available, may reveal clinically significant platelet dysfunction in the presence of apparently normal laboratory clotting tests.[23] Sterility should certainly be ensured and adequate postoperative monitoring and care must be available, particularly when in-dwelling catheters are used.

Limb injury has been highly prevalent in war surgery patients during the conflicts in Iraq and Afghanistan.[24,25] Peripheral nerve blockade has many advantages in this patient group, including providing excellent pain relief while reducing the use (and side effects) of traditional opioid-based analgesia. Recent developments in advanced RA techniques and continuous peripheral nerve blockade have been driven by an improved understanding of pain in war casualties and improvements in ultrasound technology. Pioneering initiatives such as the Military Advanced Regional Anesthesia and Analgesia program in the US military have expanded the use of RA to provide pain management not only intraoperatively but during repatriation and well into the postoperative period.[26]

A persisting concern with peripheral nerve blockade is its potential to mask acute compartment syndrome (ACS). This concern has not been borne out in a study of over 100 battlefield casualties, of whom only two developed a delayed ACS requiring fasciotomy as a late presentation (rather than a missed primary presentation) after evacuation to Role 4. A policy of performing fasciotomies prior to prolonged aeromedical transfer when there is a significant chance of developing ACS is recommended, and RA should remain a valid technique.[27] RA techniques have been well-recognized as useful in austere circumstances[28,29] and are discussed in Chapter 22, Regional Anesthesia and Coagulopathy.

Surgeon Rear Admiral G. Gordon-Taylor, a veteran of both world wars, famously pronounced, "for the abdominal wounds of war spinal anesthesia is certain euthanasia."[30] Most modern day military anesthesiologists would probably agree with this statement as applied to patients in the resuscitation phase. In the stable patient, however, neuraxial anesthesia has been successfully employed.[31] Epidural anesthesia, as a particularly effective technique in circumstances where even an anesthetist is unavailable, has been described.[32] Recent operational experiences with epidural anesthesia have largely been in the anesthetic management of stable casualties with bilateral lower limb injuries.

Conscious Sedation

Conscious sedation is commonly employed as an adjunct to analgesia (either systemic or local anesthesia/regional block) to make unpleasant procedures more acceptable. The patients necessarily fall into the stable category, and procedures frequently requiring sedation in the field hospital include repeat dressing changes, drain removal, and dental operations.

The term "sedation" can mean different things to different clinicians (anesthesiologists and surgeons in particular). It has been defined as:

> A technique in which the use of a drug or drugs produces a state of depression of the central nervous system enabling treatment to be carried out, but during which verbal contact with the patient is maintained throughout the period of sedation. The drugs and techniques used to provide conscious sedation . . . should carry a margin of safety wide enough to render loss of consciousness unlikely.[33]

Some clinicians describe a state of "deep sedation." However, a patient who is unresponsive to verbal and physical stimuli may be unable to maintain a clear airway,[34] and such a state should be regarded as

general anesthesia. It is also worth remembering that more deaths occur under sedation than under general anesthesia.[35]

Conscious sedation may be produced with small doses of anesthetic agents such as intravenous midazolam and propofol. Inhalational sedation with 50:50 nitrous oxide: oxygen mixtures is widely practiced in dentistry but may not be available or appropriate in the field hospital for reasons already mentioned. Caution is advised with drug combinations, particularly for doctors without anesthetic training. However, the experience of the authors is that an IV bolus of midazolam (2 mg) combined with small incremental IV doses of ketamine (20 mg) for adult patients undergoing potentially painful dressing changes is safe, well tolerated, and provides an effective analgesia. Successful and safe use of similar techniques have been described in austere circumstances.[36,37]

Minimal monitoring for conscious sedation consists of a pulse oximeter and, most importantly, a suitably trained individual present throughout the procedure with designated responsibility for patient safety. Blood pressure and electrocardiograph monitoring are not necessary unless cardiovascular problems are anticipated. Oxygen therapy should be available, and facilities for resuscitation should be immediately at hand.[34]

After sedation, patients should be cared for in a designated recovery area with properly trained nursing staff. In the case of military patients who have undergone minor procedures, at least 24 hours of light duties should be prescribed before they are allowed to drive, handle a weapon, operate heavy machinery, or resume a decision-making role.

Total Intravenous Anesthesia

Historically, the synergy that exists between military conflicts and medicine is well recognized. Indeed, the origins of the military use of intravenous anesthesia can possibly be traced to the 17th century following the English Civil War.[38] There is also considerable evidence of the use of intravenous ether and barbiturates in the military setting, culminating in the well-publicized use of thiopentone. Over the last 3 decades, ketamine has emerged as an important constituent of the anesthesia sequence, particularly in austere circumstances.

Concurrent and possibly serendipitous events in modern anesthesia, such as the introduction of propofol and the laryngeal mask airway, together with an improved understanding of compartment-based pharmacokinetics, have undoubtedly positively influenced anesthesia management of the stable patient. Manual infusion regimes, both simple and complex, have been recommended and used in total intravenous anesthesia (TIVA) practice. In recent years, the use of population pharmacokinetics to guide the development of devices that can deliver target-controlled infusions (TCI) has been a significant development in TIVA. The introduction of remifentanil, a potent opioid with ultra-short context-sensitive half-time, was equally felicitous, given that the synergy between it and propofol could be exploited clinically. A recent review discusses military applications of TIVA/TCI in greater detail together with suggested regimes.[39]

However, understandable reservations remain about the use of TIVA/TCI techniques during the acute phase of damage control resuscitation and related surgery. These concerns mostly relate to the unquantifiable changes in compartmental volumes that inevitably occur in the presence of major hemorrhagic injury.[40] These changes would, in turn, make it less prudent to place implicit reliance on the conventional pharmacokinetic models currently used in TIVA/TCI practice. Nonetheless, there is considerable benefit to be had in using these techniques in the stable patient, despite the inevitable technological burden associated with sophisticated syringe pumps. These benefits include:

- decreased recovery time with rapid return of cognitive function, thus lessening nursing burden;
- the possibility of seamless transition between sedation and analgesia in the stable but ventilated patient;
- abolition of pollution hazards to healthcare personnel, a concern of particular relevance in military MTFs that are unlikely to have active scavenging of waste gases; and
- the ability to quantify desired sedoanalgesia targets in a given patient in the context of repeat surgical procedures or lengthy patient transfers.

The introduction of "open" TCI pumps has removed the requirement to use custom-made prefilled syringes. Current levels of sophistication of these infusion devices permit administration of remifentanil by TCI. It is incumbent upon the user to be familiar with the critical assumptions employed by the pharmacokinetic models used in such devices.[41] An important example is the difference in the volume of the central compartment between the Marsh and the Schnider models for propofol.[42] While an extensive discussion of these models is beyond the scope of this chapter, it should be noted that dosing strategies for the individual patient require not only the use of the appropriate pharmacokinetic model but also clinical "calibration" by observation of clinical endpoints. Similarly, the use

of either plasma or effect-site (brain) targets for propofol administration must be guided by the clinical suitability and levels of fitness of the individual patient. The benefits of effect-site targeting for remifentanil administration remain to be fully established.

It should also be noted that current TCI systems are "open-loop" systems in that they deliver drug effect based on pharmacokinetic modelling derived from population studies. Although the model-derived predictions of plasma and effect-site concentrations do not necessarily reflect actual tissue concentrations, the authors suggest that clinical dose-response behavior in a given patient is more informative than knowledge of precise tissue concentrations. Such an individualized approach can also allay concerns about the ability of computer-driven models to cope with intra-individual and inter-individual differences in drug handling and clinical behavior. An understanding of this concept coupled with a fuller appreciation of compartmental distribution has led to widespread acceptance of TCI technology.

However, in the United States, the Food and Drug Administration has yet to endorse it for a variety of reasons, especially because these algorithm-based devices require drug approval rather than device approval. These regulatory hurdles have prevented adoption of TCI into mainstream anesthesia practice in the United States.[43,44] However, US military interest in the technique[45] has included formation of the Triservice Research Group Initiative on TIVA (TARGIT) in an effort to increase the use of this technique on the battlefield.[45] Training in TIVA has also been identified as an essential requirement for graduating US anesthesia residents, together with achieving an understanding of the usefulness of TIVA in the combat situation.[46]

Future directions for TCI may include an examination of "closed-loop" systems that incorporate depth of anesthesia (DoA) monitoring to provide biofeedback. Even such systems are likely to encounter significant regulatory hurdles and challenges before translation into routine clinical practice occurs.[47]

MONITORING DEPTH OF ANESTHESIA

The availability of a monitoring system that quantifies brain activity under anesthesia and sedation may increasingly need to be considered even in the context of relatively austere field conditions. Such a capability could provide a window into the effect site of interest—the brain—thus permitting differentiation of two critical anesthetic effects: hypnosis and analgesia. While a discussion of the various monitors is beyond the scope of this chapter, the uses of auditory-evoked potentials, spectral entropy, and a bispectral index have been widely examined, with the latter technology generating the most literature. Although the extensive literature on DoA monitoring has largely been focused on mitigating concerns about awareness under anesthesia, such monitoring may also have a role in determining the quality of anesthesia.[48]

Given the likely turbulent nature of the trauma resuscitative process, such monitoring could add a further layer of reassurance to patient management. It is still unclear whether the various commercially available modalities have sufficient sensitivity and specificity to provide an unimpeachable biological signal. Bruhn et al discuss these important aspects in greater detail.[49] While the debate continues about the ability of such monitoring to meaningfully integrate data derived from both spontaneous and evoked cerebral activity under anesthesia,[50] it has also revealed the need to examine whether excessively deep anesthesia has distant consequences. Monk et al ignited this issue by their prospective observational study in which they hypothesized that, in addition to recognized factors such as comorbidity, excessively deep anesthesia may well have an adverse impact on 1-year mortality.[51] Significantly, the study quantified DoA with the assistance of a bispectral index monitor to derive cumulative deep hypnotic time. The study has generated considerable debate, some of which questioned the need for DoA monitoring,[52] and has also focused attention on the need to reexamine the quality of delivered anesthesia. Improved anesthesia quality may provide longer-term benefits for the stable patient, in addition to the logistical advantages to the MTF of rapid recovery and diminished clinical burden.

CONCLUSION

Medical facilities in support of expeditionary military activity or disaster relief operations are likely to be austere but increasingly rely on highly developed protocol- and team-based working, supplemented by appropriate medical resources, to deliver high quality healthcare. Trauma remains a significant part of the workload of such MTFs.[53] It is incumbent on the part of such establishments to arrange for robust and enduring processes for the continuing care of the stable and the stabilized patient. When such care involves surgery, optimal perioperative management of such patients, particularly in the

areas of anesthesia, analgesia, fluids management, and timely transfer will not only enhance the effectiveness of the MTF but also inform the management of the less stable patient.

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Chapter 16

THE PHYSIOLOGY OF ACUTE PAIN

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INTRODUCTION

The understanding of acute pain physiology has expanded greatly over recent decades, revealing increasing layers of complexity. This chapter aims to simplify and distil current thinking and tackle the central question of, "how do we feel pain?" Any such discussion will necessarily touch upon numerous receptor types and pain pathways, but the chapter will focus on the principles of pain transmission and modulation, charting the journey from peripheral detection to the brain's central experience of pain.

BASIC CONCEPTS

Pain is in itself a complex construct, defined by the International Association for the Study of Pain as an "unpleasant sensory and emotional experience associated with actual or potential tissue damage." According to this definition, an intact nervous system is needed to transmit the signal and an intact consciousness must process it, with physiological and psychological factors able to influence the experience.[1] A concept of a body–self neuromatrix has been proposed in which multiple physiological and psychological inputs are processed to produce the output called pain.[2]

The ability to detect noxious stimuli that are actually or potentially associated with tissue damage is an essential protective defense mechanism; the neural processing of such stimuli is called "nociception" and represents the sensory component of pain. Acute pain can therefore be thought of as beginning with transduction by peripheral nociceptors of a noxious stimulus via an electrical impulse, transmitted to the dorsal horn of the spinal cord, where signals are processed and potentially modulated by descending pathways from the periaqueductal grey (PAG). Signals then ascend via the thalamus to a higher "pain matrix" consisting of the primary and secondary sensory cortex, the insular cortex, the anterior cingulate cortex, and motor regions, including the cerebellum.[1] The limbic system feeds into this matrix, accounting for the modulatory effects of emotional state on pain perception (Figure 16-1).

PAIN MECHANISMS

Peripheral Nociceptors

Peripheral nociceptors are essentially afferent unmyelinated C-fibers and lightly myelinated A-∂ fibers lying in the periphery that connect to the laminae of the dorsal horn of the spinal cord. The most numerous subclass of nociceptor is the C-fiber polymodalnociceptor, which responds to a broad range of physical and chemical stimuli.[3] The sensory component of pain begins with the formation of an action potential in these afferent nerves and its transmission to the dorsal horn. C-fiber pain is classically described as burning or aching, whereas A-∂ fibers, with their increased conduction velocities owing to myelination, transmit sharp or pricking pain and are involved in the protective reflex arc.

Nociceptor Receptors and Ligands

The process of an action potential being generated is complex, with numerous receptors and chemical ligands implicated; however, the underlying principle is simple: all the receptors serve one of two purposes, either to generate the threshold potential required by directly allowing ion movement, or to metabolically facilitate signal transmission, for example by effects on voltage-gated sodium channels.

Transient receptor potential vallinoid (TRPV) channels detect a range of thermal stimuli[4] and can initiate signals purely in response to such stimuli (Table 16-1); however, the majority of receptors are activated by the chemical cascade associated with tissue damage. Injury to the tissue leads to disruption of the cells, allowing their contents to spill into the interstitium. This lowers pH, stimulates the inflammatory response with mast cell activation and degranulation, and induces enzymes such as cyclooxygenase-2. The net result is a cocktail of mediators including prostaglandins, hydrogen ions, bradykinins (BKs), serotonin (5-HT), potassium, adenosine triphosphate (ATP), histamine, nerve growth factor (NGF), and glutamate. These mediators act at a host of receptors to sensitize the peripheral nociceptors, including acid-sensing ion channels, BK-1 and BK-2 receptors, 5-HT_{2A} receptors, P_2X_3 (which binds ATP), and tyrosine kinase A (TrkA) receptors (which bind NGF).

Additional Peripheral Mechanisms

Additional mechanisms serve to perpetuate this painful response to tissue injury: neuropeptides such as substance P are released by the peripheral nerve terminals and contribute to the recruitment of serum factors and inflammatory cells at the site

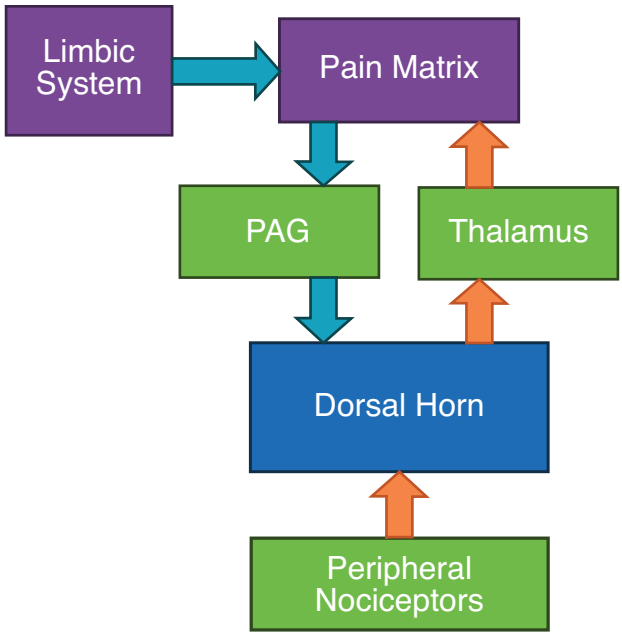

Figure 16-1. Simplified schematic of pain transmission. Orange arrows represent ascending pain pathways and blue arrows modulatory pathways.
PAG: periaqueductal grey

of injury (neurogenic edema).[3] Many inflammatory cells also express TrkA receptors, meaning that the presence of NGF can stimulate these cells to release further 5-HT and histamine. In addition, sympathetic nerve terminals release prostaglandins in response to BK.[1]

The Dorsal Horn

The dorsal horn is essentially an integration center where primary inputs from the periphery can be significantly modified before being transmitted to the cortex. The cell bodies of the nociceptive afferents that innervate the trunk, limbs, and viscera are located in the dorsal root ganglia and project to the dorsal horn, while those innervating the head and neck are in the trigeminal ganglia and project to the trigeminal nucleus. The C-fiber and A-∂ nociceptive afferents predominantly terminate superficially in laminae I and II of the dorsal horn, with some deeper A-∂ projections to wide dynamic range neurons in lamina V, which encode both noxious and innocuous stimuli. A-β fibers transmit light touch or innocuous mechanical stimuli to laminae III and IV and are implicated in gate-control theory, which suggests that impulses in these fibers can reduce onward nociceptive traffic to the brain.[5]

Pain Transmission, "Wind-Up," and Long Term Potentiation

Pain transmission in the dorsal horn between the primary and secondary afferents is predominantly mediated by the excitatory amino acid glutamate and the peptide substance P. Depolarization of the primary afferent terminal results in glutamate release into the synaptic cleft, which binds to postsynaptic ionotropic alpha-amino-3-hydroxyl-5-methyl-4-isoxazole-propionate (AMPA) receptors. The AMPA receptors signal information related to the location and intensity of noxious stimuli.[3] Usually a constant and predictable stimulus-response ratio is maintained during transmission.[6]

However, repeated C-fiber stimulation at the dorsal horn results in a progressively more depolarized postsynaptic membrane and in turn displacement of magnesium ions from *N*-methyl-D-aspartate (NMDA) receptors. The now vacant NMDA receptors can bind glutamate, which, in combination with glutamate's action at metabotropic receptors and substance P acting at neurokinin-1 (NK_1) receptors, leads to the "wind-up" phenomenon. Essentially, a progressive increase or wind-up in output from the dorsal horn occurs in response to each stimulus. This change is evoked by low frequency C-fiber stimulation and manifests as an enhanced postsynaptic response during a train of stimuli.[3]

Associated with higher frequency stimulation, long-term potentiation (LTP) is similar to the wind-up phenomenon in the sense that output from the dorsal horn is increased, but, crucially, the enhanced response outlasts the conditioning stimulus; LTP has been implicated in learning and memory in the hip-

TABLE 16-1

TRANSIENT RECEPTOR POTENTIAL VALLINOID CHANNEL SUBTYPES

Subtype	Stimulus / Ligand
$TRPV_1$	Heat (>42°C), hydrogen ions, capsaicin
$TRPV_2$	Heat (>53°C)
$TRPV_3$	Warm (>32°C)
$TRPV_4$	Warm (>32°C)

TRPV: transient receptor potential vallinoid
Data source: Applied Physiology of Pain. In: Macintyre P, Scott D, Schug S, Visser E, Walker S, eds. *Acute Pain Management:Scientific Evidence*. 3rd ed. Australian and New Zealand College of Anaesthetists and Faculty of Pain Medicine; 2010:1-6.

pocampus and pain sensitization in the spinal cord.[7] LTP is mediated by calcium influx through NMDA receptors activating intracellular kinases; these in turn bring about alterations in ion channel and receptor numbers on the postsynaptic membrane, leading to more efficacious neural transmission.[3]

Through similar cellular processes, repeated stimulation in the dorsal horn also leads to increased sensitivity in secondary afferents in close proximity to the directly stimulated area. This can result in intact tissue anatomically adjacent to the damaged area demonstrating hyperalgesia (increased response following noxious inputs).[1] This secondary hyperalgesia, together with wind-up and LTP, may contribute to central sensitization, which encompasses increased sensitivity to both C and A-β fiber inputs, resulting in hyperalgesia and allodynia (pain in response to previously nonpainful stimuli).[7]

Descending Modulatory Pathways

Signals originating in the cortex, hypothalamus, and amygdala are integrated in PAG in the brainstem, giving rise to a descending pathway that interacts at the synapse of the primary and secondary afferents in the dorsal horn. The pathway is rich in opioid receptors, allowing for modulation by both endogenous and exogenous opioids; 5-HT and adrenaline also function as neurotransmitters in this system.[1] The adrenergic component in particular is inhibitory at the dorsal horn and is likely, at least in part, to account for why combat casualties with very severe injuries, whose systems are flooded with adrenaline, experience little or no acute pain. Serotonergic pathways can be involved in pain inhibition, but they have also been implicated in facilitating pain transmission at the dorsal horn.[8]

Other local inhibitory systems at the dorsal horn that may provide future avenues for pharmacological intervention include the α-4-β-2 nicotinic acetylcholine receptor and cannabinoid receptor type 1.

Role of the Glia

Until recently, the glial cells had been thought to provide a framework to support the neurons performing "housekeeping" and homeostatic functions only[1]; however, current research suggests that glial cell activation also plays a part in nociception due to release of neuroexcitatory products.[9] The role of the glia is arguably more pronounced in the development of chronic pain, but these cells can also affect the transmission of acute pain.

Following peripheral tissue or nerve injury, microglia and astrocytes can shift to an activated state characterized by the release of a plethora of proinflammatory substances including cytokines, chemokines, arachidonic acid, prostaglandins, excitatory amino acids, ATP, and NGF. These glial products have a modulatory effect on acute pain transmission, directly enhancing neuronal excitability and increasing pain-associated neurotransmitter release from sensory afferents. Key to this process is the up-regulation of the number and conductance of excitatory receptors such as AMPA and NMDA, and down-regulation of inhibitory receptors such as γ-aminobutyric acid (GABA) and glial glutamate transporters.[9]

The mechanism of glial activation is a complex process involving many putative transmitters released by neurons in response to injury. One receptor that may be key to the activation process is toll-like receptor 4 (TLR4)[9]; this receptor could represent a target for pharmacological intervention to mitigate the impact of glial cells on both acute pain transmission and the subsequent development of neuropathic pain.

Pain Perception in Higher Centers: The "Pain Matrix"

The spinothalamic tract carries signals from the primary afferent terminals in laminae I and II, via connections in lamina V of the dorsal horn, to the thalamus and then to the somatosensory cortex, conveying information on the site and type of painful stimulus. The spinoreticular and spinomesencepahlic tracts ascend to the medulla and brainstem, allowing integration of nociceptive information with arousal and homeostatic and autonomic responses.[3]

The concept of a higher "pain matrix" aims to explain how different areas of the brain combine to produce the complex experience of pain. The somatosensory cortex is key to the matrix, responsible for the sensory component of pain and allowing comprehension of its location and nature. The insular cortex is thought to have a somatic representation of pain similar to the sensory cortex and seems to provide the affective component of pain; together with the cingulate and prefrontal cortex, it conveys the unpleasantness of pain.[1] Feeding into this matrix is the limbic system, which allows a person's emotional state to impact on pain perception.

It is now clear that the psychological context of the stimulus in terms of anticipation and attention can be as important as the stimulus parameters.[8] Reported pain is less during tasks that require concentration, demonstrating how higher cortical function can modulate pain experience in a top-down fashion. The effect of anticipation, once dismissed as mediating anxiety-linked augmentation of pain only, now ap-

pears important in its own right by causing activation of most of the nociceptive system, although evoking smaller responses than the responses related to pain intensity.[10]

CONCLUSION

Understanding of the physiology behind the experience of acute pain will unquestionably continue to develop in the future. Despite potentially clouding the understanding of basic transmission processes, the complexity already delineated has indicated exciting new possibilities for pharmacological research. The large number of receptor types involved throughout the nociceptive system clearly shows that no one drug will provide all the answers, and a truly multimodal approach to the management of pain must exploit more of these mechanisms, particularly in the periphery and at the dorsal horn.

Acute and chronic pain have traditionally been considered distinct clinical entities, but current thinking views them as a continuum of the same basic process, involving the same nociceptive tree.[8] An understanding of the physiology of acute pain underpins research into how this system begins to function aberrantly in chronic pain. More importantly, the role of acute pain management in manipulating this physiology and preventing the transition to persistent pain must be further investigated. Pain and its treatment are discussed further in the following chapters in section 3 of this volume.

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Chapter 17

WHY PAIN RELIEF IS IMPORTANT: THE PHYSIOLOGICAL RESPONSE

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INTRODUCTION

One of the main reasons we treat pain is to relieve suffering; it is one of our four bioethical principals—beneficence— and one of the most critical aspects of care for a patient. Treatment will improve patients' quality of life, their ability to work, and their physical and emotional functioning. Another reason to treat pain is because it activates complex neurohumoral, neuroendocrine, immune, and psychological responses, known together as the stress response. If severe and prolonged, pain can have deleterious effects on patient rehabilitation and outcome. The adverse physiological effects are legion and the magnitude and duration of the response is related to that of the stimulus.[1] Most experimental data are from studies with combined tissue trauma and the resultant pain. However, data has been collected about pain in the absence of injury using electrical stimulation, which still shows the stress response.[2] Effective pain management can significantly impact upon the physiological response to injury. This chapter will review the mechanisms of the initial pain response and discuss how acute pain management improves long-term outcomes.

THE INITIAL STRESS RESPONSE

The initial tissue trauma or pain elicits the neuroendocrine response with activation of the hypothalamic-pituitary-adrenal axis. This activation is driven by the limbic input to the hypothalamus via the paraventricular nucleus. The pituitary gland secretes adrenocorticotropin (ACTH), growth hormone, vasopressin, prolactin, and endorphins, while stimulation of the sympathetic nervous system increases plasma catecholamines, resulting in tachycardia and hypertension. Sympathetic stimulation also activates the renin-angiotensin system with subsequent increases in plasma aldosterone. This hormonal soup leads to the catabolic process, as mentioned, in proportion to the initial stimulus.

The main responses can be broadly classified into metabolic, inflammatory, hyperalgesic, cardiovascular, respiratory, coagulation, and immune function. The metabolic response can be further divided into protein catabolism, lipolysis, hyperglycemia, and changes in water and electrolyte balance (Figure 17-1).

Key Hormones Released

Cortisol is a glucocorticoid released from the adrenal cortex in response to ACTH secretion. In severe pain and trauma, the normal feedback mechanisms fail, so persistently high levels of cortisol are produced. Cortisol has far-reaching effects resulting in protein catabolism, lipolysis, and carbohydrate metabolism; it promotes gluconeogenesis and has insulin-suppressive effects. It also has antiinflammatory and immune suppressant effects by inhibiting the accumulation of macrophages and neutrophils; it also reduces inflammatory mediators and affects water and electrolyte balance.

Growth hormone released from the anterior pituitary has hyperglycemic effects due to its glycogenolytic and lipolytic actions, as well as effects causing insulin resistance. Growth hormone also has anabolic effects with regard to protein, although these effects are not large enough to counter the massive overall catabolic effects of the stress response.

The effect of insulin, or indeed its lack of effect, contributes to the stress response. Insulin is an anabolic hormone that leads to glucose storage and utilization. In the stress response its release is initially inhibited by the effects of catecholamines, but subsequently resistance to its effects develops due to failure of cellular response.

Increased arginine vasopressin—antidiuretic hormone—release and increased sympathetic activity with catecholamine release occur with the stress response. The responses to these substances are detailed in other parts of this chapter. (There are also increased secretions of other hormones such as glucagon, prolactin, and endorphins, which have less important effects and are outside the scope of this book.)

Metabolic Responses

Protein Catabolism

The initial response includes a net catabolism of protein into amino acids for gluconeogenesis. The process is driven by increased catecholamines, cortisol, glucagon, and interleukins. As the stress response produces an accelerated protein breakdown as well as an inadequate reaction or reduction in total protein synthesis, a negative nitrogen balance results. Skeletal muscle protein is mainly affected, and the loss of up to 0.5 kg per day of lean muscle mass may result in decreased muscle strength, delayed wound healing, and reduced immune function.[3] Albumin production is also reduced, affecting the maintenance of the extracellular volume and leading to edema.

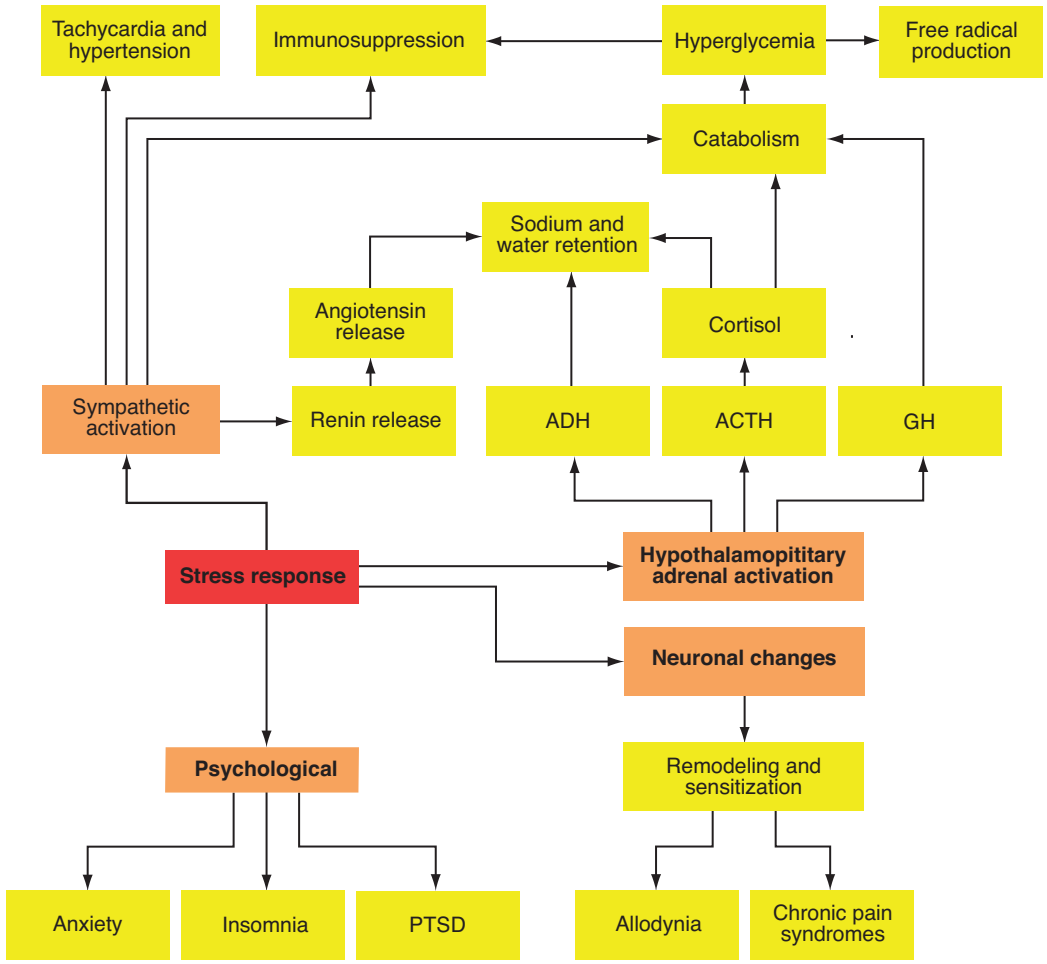

Figure 17-1. Diagram showing various components of the stress response.
ACTH: adrenocorticotropin; ADH: antidiuretic hormone; GH: growth hormone; PTSD: posttraumatic stress disorder

Lipolysis

Lipolysis, with resultant increases in free fatty acids and glycerol, is due to increased levels of circulating catecholamines, glucagon, cortisol, and growth hormones and reduced amounts of insulin. The higher levels of free fatty acids can have a negative inotropic effect, increasing myocardial oxygen consumption—with the possibility of ischemic damage—and possibly increasing free radical production.[4] Glycerol is a gluconeogenic substrate that further contributes to the hyperglycemic state.

Carbohydrate Metabolism

Catecholamine, glucagon, and cortisol also produce hyperglycemia, which is potentiated by an initial lack of insulin secretion followed by insulin resistance. The catecholamines and cortisol promote glycogenolysis and gluconeogenesis. The excess circulating glucose results in protein glycosylation and increased free radical production. The increased protein glycosylation, among other effects, reduces immunoglobulin function, thus increasing risk of infection. The raised levels of free radicals lead to increased mitochondrial dysfunction and eventual cell death. Glucose concentrations greater than 11.1 mmol/L are associated with impaired wound healing, increased infection rates, increased hospital length of stay, and increased mortality.[5]

Water and Electrolyte Balance

Renin is released from the juxtaglomerular cells of the kidneys in response to sympathetic stimulation; this results in angiotensin I being converted to angiotensin II. The increased angiotensin II stimulates the adrenal cortex to release aldosterone, which leads to

increased sodium reabsorption and water retention from the distal convoluted tubules. Arginine vasopressin release from the posterior pituitary results in water retention and potassium excretion. These effects are increased by the mineralocorticoid activities of cortisol, producing water retention, potassium excretion, and sodium reabsorption.

System Responses

Cardiovascular

Pain activates the sympathetic nervous system, which may increase the myocardial oxygen demand through its chronotropic, inotropic, and hypertensive effects. The increased sympathetic demand can also reduce oxygen supply by causing coronary artery vasoconstriction. These two factors along with the hypercoagulable state greatly increase the chance of myocardial ischemia.

Respiratory

Hypoxemia and other pulmonary complications can result from pain. Pain can reduce functional respiratory capacity, producing atelectasis and ventilation–perfusion mismatch. Atelectasis, caused by the inability to take deep breaths, and reduced effective coughing due to pain increase the risk of chest infections.

Immune Function

As previously discussed, immune function is reduced by persistently high levels of not only cortisol but also catecholamines, and to a lesser extent growth hormone and prolactin. These hormones reduce natural killer cell activity, antibody production, and lymphocyte proliferation.[6]

Neurological Systems

Chronic pain is common following trauma and surgery and is a major cause of ongoing patient morbidity.[7] Changes can occur in peripheral nerves, the spinal cord, higher pain centers, or the sympathetic nervous system. Inflammation at the site of injury can result in increased levels of mediators such as bradykinin, monoamines, prostaglandins, and leukotrienes. These chemicals sensitize afferent C fibers, resulting in a reduced threshold for firing. There is also an increase in the numbers of nociceptors. Central sensitization occurs at the dorsal horn, where there is an exaggerated response to C fiber and A-β input. "Wind-up" can occur due to activation of *N*-methyl D-aspartate receptors, leading to chronic pain syndromes and a cycle of pain difficult to treat. Models of the pathophysiology of pain suggest that within minutes of injury, neuronal expression of new genes occurs. This is the initial phase of neuronal remodeling and sensitization.

Early analgesia can prevent or reverse this cascade. Preemptive analgesia may not be possible for trauma patients, although early epidural insertion or continuous peripheral nerve block can often be used. Aggressive analgesia following trauma may reduce both acute and chronic pain by reducing both peripheral sensitization from the injury and central sensitization with its subsequent wind-up.

PSYCHOLOGICAL RESPONSES

Failure to relieve acute pain is associated with undesirable psychological changes, including anxiety, insomnia, and feelings of helplessness and loss of autonomy. Failure to treat acute pain can increase the incidence of chronic pain and posttraumatic stress disorder (PTSD), which are far more difficult to treat.[8] It has been found that pain scores following traumatic injury within the first 48 hour period are strongly associated with the development of PTSD. Indeed, an observational study suggested that the use of morphine in the initial trauma resuscitation can significantly reduce the development of PTSD.[9] Results of studies on the use of substances such as benzodiazepines and β-blocking agents have been inconsistent.[10,11]

Pain is subjective. It has a variable correlation with the extent of tissue injury and is multifactorial in nature. The psychological response is affected not only by adequacy of pain control but also by the patient's psychological resilience, previous pain experiences, culture, anxiety levels, mood, and preparedness. It is also dependent on situational factors. Engel's biopsychosocial illustration of pain demonstrates that pain cannot always be treated purely by analgesic administration because of the complex interaction between the three components.[12] Beecher noted that military patients with similar injuries had lower analgesic requirements compared to their civilian counterparts.[13] This difference was thought to be because the injury was associated with evacuation from the war zone and rehabilitation in a safe environment including families and support for family members. In the civilian setting, on the other hand, recovery from trauma was associated with decreased earning power and an uncertain future with potential for social hardship.

SUMMARY AND PATIENT OUTCOMES

It can be inferred from the physiological response to pain and trauma that adequate acute pain management is essential to modulate the potent stress response. Pain management will aid in attenuation of the multiorgan dysfunction, the catabolic state, hypercoagulability, resultant chronic pain syndromes, and the psychological effects of uncontrolled pain. Rapid effective pain relief at the point of injury is needed to reduce the neuronal remodeling and sensitization that occurs within 20 minutes of the initial trauma. In the chaotic combat environment, pain relief reduces casualties' physiological stress and facilitates evacuation. Analgesia must then be continued throughout the care and rehabilitation process.[14] It has been found that adequate neuroaxial analgesia can reduce protein catabolism, cortisol levels, and hyperglycemia while improving immune function.[6,15]

Studies show a good correlation between inadequate analgesia and the catabolic state, pulmonary complications, immunosuppression, and thromboembolic events. There is also moderate correlation with PTSD, chronic pain states, and mortality.[16]

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Chapter 18

MULTIMODAL ANALGESIA FOR SPECIFIC INJURY PATTERNS

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INTRODUCTION

It is clear from earlier chapters in this book that developments in patient care and especially resuscitation have advanced considerably during the conflicts of the last decade. Similar advances have also taken place in the fields of acute pain management and regional anesthesia. Pain relief in recent conflicts has become an area of acute deployed medicine that has rightly received considerable attention from the chain of command in both the United Kingdom and the United States. Other chapters describe the benefits of analgesia (Chapter 17, Why Pain Relief is Important: The Physiological Response) and the pain medications (Chapter 20, Pain Medications) available to the deployed anesthesiologist. This chapter will consider the application of the combined techniques of regional anesthesia and pain medications to common combat injuries.

BASICS OF MULTIMODAL ANALGESIA

Casualties of military operations sustain the complete spectrum of injuries from the very minor to the most serious and complex injuries imaginable, and a multimodal approach to analgesia is recommended from the start of treatment. The multimodal approach includes using regular doses of simple analgesics with differing mechanisms of action as well as opioids and adjuvants (described in Chapter 20). The World Health Organization (WHO) pain ladder (Figure 18-1)[1] describes the generally accepted approach to prescribing simple analgesics and opioids in painful conditions, with a stepwise increase in strength and dose of opioid as the degree of pain increases. This approach is the foundation of multimodal analgesia. When there are no contraindications, patients should receive regular doses of nonopioid analgesics such as paracetamol (acetaminophen) and a nonsteroidal antiinflammatory drug (NSAID) by the oral or intravenous route as available.

Opioid analgesics and the options for the deployed environment are described in Chapter 20. The use of regular doses of weaker opiates should be guided by the degree of pain a patient is experiencing. In some situations regular doses of codeine phosphate are appropriate; however, as the amount of pain increases, it may be better to begin regular doses of a more potent opioid, such as tramadol, in a lower dose. Regular doses of tramadol should also be considered as part of a multimodal approach for acute pain, especially if there may be nerve injury. Tramadol has a number of actions. It is a μ-opioid receptor agonist, a serotonin-releasing agent, a norepinephrine reuptake inhibitor, an *N*-methyl-D-aspartate receptor antagonist, and a 5-HT_{2C} receptor antagonist. It is metabolized to *O*-desmethyltramadol, which has stronger μ-opioid agonist action (as well as actions on norepinephrine reuptake and 5-HT).[2] Because of its useful spectrum of action, tramadol is particularly appropriate in patients with actual or potential nerve injury.

As the degree of pain increases, it is appropriate to add morphine to the prescription. The route chosen will obviously depend on the amount of pain patients are experiencing and whether they have a functioning gastrointestinal tract. Intravenous morphine will be required for the most severe acute pain and to keep side effects, patient satisfaction, and nursing workload at the optimum Patient-controlled analgesia (PCA) is the recommended approach. Oral morphine, as a liquid or tablet, can be used for lesser degrees of pain as well as for weaning patients from PCA. Ketamine is also very effective in treating acute pain; doses of 10 to 30 mg are a useful starting point. Ketamine works well on its own or in combination with conventional opioids.

ADVANCED TECHNIQUES

PCA is a well established technique available to US and UK military medical services. Its well known advantages of effective analgesia with reduced side effects and less pain breakthrough, along with reduced nurse workload, are as applicable in the deployed setting as in civilian practice. For PCA to work effectively, the patient must be instructed in the technique and understand the concept, so it is usually prescribed to adults only. Current experience is that many local nationals do not understand the concept of PCA even when instructed directly through a translator. Alternatives to PCA include the use of a morphine or ketamine infusion, which may make it necessary for the patient to be cared for in a medium- to high-dependency setting postoperatively.

Adjuvant drugs other than analgesic medications should also be considered. It is important to address phantom or neuropathic pain as well as conventional acute postoperative pain. The adjuvants most often prescribed for injuries that may cause this type of pain are tricyclic antidepressants and anticonvulsants. If nerve injury is obvious or likely, it is appropriate to

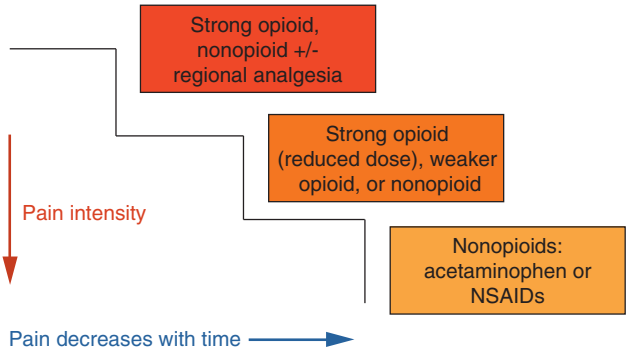

Figure 18-1. Decreasing ladder of acute pain, based on the World Health Organization pain ladder.
NSAID: nonsteroidal antiinflammatory drug
Adapted with permission from: World Health Organization. *Cancer Pain Relief*. 2nd ed. Geneva, Switzerland: WHO; 1996: 15.

start amitriptyline and gabapentin or pregabalin in the early postoperative period.

The use of regional anesthesia in the deployed environment has grown over the last 5 to 10 years. The introduction of portable ultrasound technology and improved regional anesthesia needles and catheters into the deployed environment has made a significant impact. For some injuries single-injection regional anesthetic blocks to a particular nerve or plexus can provide excellent operating conditions and all the analgesia needed. However, many trauma patients require ongoing pain relief, particularly during evacuation, so it is always appropriate to have a low threshold for inserting a catheter. Often combat casualties have multiple injuries, so a mixture of simple analgesia, PCA, and regional blocks are brought to bear (ie, multimodal analgesia).

MULTIMODAL APPLICATIONS

Simple or Single Injuries

Isolated Upper Limb Injury

For many injuries of the upper limb, regional anesthesia via the supraclavicular approach to the brachial plexus is recommended. This technique is often and accurately described as the "spinal" anesthetic of the arm. The approach is particularly suitable for single injections or catheters and can be performed on an anesthetized patient (for postoperative pain relief) or on an awake patient either as the sole anesthetic or prior to a general anesthetic. If the block is performed in the emergency room prior to a general anesthetic, requirements for analgesia are simplified as well. The axillary approach to the brachial plexus is useful for hand injuries; however, even with an intercostobrachial block (which is appropriate with the supraclavicular approach as well), tourniquet pain may be an issue for the awake patient after 1 or 2 hours. The interscalene approach is a useful block for surgery to the shoulder and proximal upper limb.

A working knowledge of individual nerve blocks at the elbow or wrist is valuable for surgery in these areas as well as for rescuing a failed block performed more proximal. The choice of block will depend on a number of factors, including area of injury, access to the limb, and the experience of the anesthetist. As above, the supraclavicular approach is often a good choice.

The decision to use a single injection or to insert a continuous peripheral nerve block (CPNB) catheter will depend on factors previously discussed. Generally, an approach that can be recommended for most isolated limb injuries is that of regional anesthesia alone or, as is quite often the case, general anesthesia with addition of a supraclavicular block (single injection or catheter as appropriate) for postoperative pain relief.

Isolated Lower Limb Injury

For the majority of cases with injuries confined to a single lower limb, anesthesia and analgesia should be provided by blockade of the femoral and sciatic nerve. Some controversy remains regarding the use of regional analgesia in patients with the potential to develop compartment syndrome (CS), as discussed below. For injuries or operations above the knee, femoral and sciatic nerve blocks are effective and appropriate. For injuries below the knee, the sciatic nerve can be blocked in the popliteal fossa above the division into the tibial and common peroneal nerves. Catheter techniques are effective in all of these locations. When short-term pain relief is required below the knee, a saphenous nerve block can be effective without producing the motor weakness associated with blockade of the femoral nerve at the groin.

Abdominal Injuries

A number of approaches to pain relief are required due to the variety of abdominal surgical procedures, ranging from groin incisions for extraperitoneal vascular control to extended midline laparotomy incisions. The choice of regional analgesia technique usually rests between epidural and transversus abdominis plane (TAP) block. Although the thoracic epidural remains the gold standard for abdominal surgery,[3] an epidural may not always provide perfect analgesia

for an incision from xiphisternum (T6) to pubis (T12). When performing a thoracic epidural for a laparotomy incision, many anesthesiologists insert the needle half to two-thirds of the way along the dermatomal range (eg, T7/8 or T8/9). Although catheters placed lower can be bolused to improve spread, this may be associated with greater hemodynamic instability and unnecessary pain between boluses. If patient-controlled epidural anesthesia is available, lower insertion may be acceptable. For incisions below T10, the conventional TAP block is very satisfactory; however, it is unusual for the block to spread much above T9. For upper abdominal incisions, a subcostal TAP block can be performed, but with this approach it is unlikely there will be significant block below T10. A recent paper comparing TAP blocks to epidural analgesia for upper abdominal surgery (incision at or above T10) found little difference in pain scores but an increased opioid consumption in the TAP group, potentially reflecting increased visceral pain.[4]

Although TAP blocks are commonly used as a single-injection technique, they also work well with CPNB catheters.[5] A key factor in the choice between TAP blocks and epidural in the combat casualty is likely to be coagulation status. Clearly, an epidural is contraindicated in the patient with coagulopathy (see Chapter 22, Regional Anesthesia and Coagulopathy of Trauma Shock, for detailed information including guidance on ROTEM [TEM International GmbH, Munich, Germany] results). The use of rectus sheath catheters, placed by the surgeon prior to wound closure, has also received attention recently in civilian practice and may be worth attempting in the deployed environment, particularly in the patient with coagulopathy.[6] Specially designed catheter systems are available and their use is being investigated.

Although PCA alone is not likely to be associated with acceptable pain scores during movement, it may well be required in addition to a regional technique (eg, for controlling visceral pain when TAP blocks have been performed). If an epidural is used with PCA, the local anesthetic solution should be opioid free to avoid the risk of delayed respiratory depression with central and intravenous opioids.

Thoracic Injuries

As with abdominal surgery, the choice of postoperative pain relief for thoracic injuries depends on the operation performed and therefore the incision used. Once again the gold standard is almost certainly a thoracic epidural. As before, concerns over coagulopathy may delay an epidural until any coagulation defect has been corrected, or another approach may be used. Alternative approaches also depend on the incision. Paravertebral injections are one option. Although sometimes used only for unilateral surgery, bilateral paravertebral blocks (both single-injection and catheter) are regularly performed. Single-injection techniques have been well described,[7] as have catheter techniques,[8] although the available space is small and inserting a catheter is not always as easy as an epidural. It is recommended that only 2 cm of catheter be advanced, which does not leave much room for error postoperatively. If more than four dermatomes need to be blocked, more than one injection site is required, which tends to rule out the option of continuous infusions. A simpler alternative is asking the surgeon to place a catheter in the interpleural space posteriorly in the chest cavity. Although pain relief can be extremely effective using this technique, if multiple chest drains are inserted as well the catheter may be adversely impacted. Once again, a combination of a regional technique with PCA is often required.

Compartment Syndrome

CS has been reviewed by UK medical subject matter experts,[9] and more recently a US clinical practice guideline has been published on the subject.[10] CS is a well described complication of severe traumatic limb injury. Definitive treatment is surgical release of compartments,[11] but this treatment is not without complications. Although the decision is obvious in some cases, fasciotomy is not immediately necessary in others. As with any other injuries, optimal analgesia is a requirement. Past controversy has occurred about the correct route of analgesia for limb injuries, particularly in lower limbs. Although regional blocks are a very effective option for analgesia in these cases, some orthopedic surgeons have been concerned that such blocks can mask CS signs and symptoms. The literature contains case reports[12–15] in which CS has been masked by conventional analgesia, including PCA morphine. There seems to be no appetite to restrict the use of morphine in these cases.

Similarly, there is no evidence in the literature that regional analgesia has masked CS more than other forms of pain relief. A review of 28 case reports[16] of CS found classic signs and symptoms of CS in the presence of epidural analgesia in 32 of 35 patients, including 18 patients with documented breakthrough pain. These results strongly suggest that regional anesthesia techniques are not very effective in treating ischemic pain from CS in the presence of epidural analgesia. Good analgesia of any kind could potentially mask CS, so avoiding regional analgesia on this basis is illogical. It is also important to distinguish between dense blocks

with high concentrations of local anesthetic agents and blocks using lower concentrations. In this situation the use of 0.2% ropivacaine is recommended. Monitoring of motor function should be part of the standard observations on any patient with an epidural or CPNB.

The military casualty population differs from the civilian population in the requirement for evacuation, sometimes over long distances. During this period surgical decompression of CS is not possible. It is therefore necessary for attending surgeons to make the decision to decompress prior to transfer. In US facilities, the trauma surgeon and acute pain anesthesiologist should discuss in detail any cases that are at high risk for CS. In UK facilities the Deployed Medical Director may need to be involved. Pain treatments may be withheld briefly for diagnosis of CS. Ultimately the treatment for CS is surgery.

Complex Injuries

A signature injury of the current conflict is traumatic bilateral lower limb amputations, often with upper limb injuries and potentially perineal or abdominal injury as well. The abdomen is often opened in casualties with these severe injuries, usually to gain vascular control. American and British service personnel who sustain injuries of this severity will often be transferred to a Role 4 facility as a matter of urgency. Patients who will remain ventilated on infusions of opioids and sedatives clearly require no regional anesthesia at this stage. However, there are advantages to extubating a ventilated patient as early as possible.[17] Benefits include reduced morbidity (eg, for ventilator-acquired pneumonia) as well as the ability to begin decompression.

Casualties also often complain about long periods of "amnesia" from being sedated (commonly for several days) following their return from the operating room. For patients who can be extubated, it is necessary to provide the best pain relief possible to support maximum respiratory effort in this situation. As mentioned above, the patient's coagulation status is often a major concern, especially if epidural analgesia is being considered. Because many such patients will be sedated and ventilated for hours in the immediate postoperative period, it is important to discuss their management with the intensive care physician, aiming to correct coagulation and then insert an epidural prior to weaning and extubation, which may take place 12 to 24 hours after surgery.

Although discrete nerve blocks are extremely useful in multiply injured patients, the number of blocks or catheters inserted in a single patient should be limited to avoid toxic doses of local anesthetic. As a rule it is recommended that no more than two or occasionally three catheters be inserted**.** For bilateral lower limb injuries, when coagulation issues have been resolved, epidural is the obvious choice. If upper limb injuries are also present, it is possible to insert a supraclavicular catheter for the worst affected side. In this situation opioid-free local anesthetic solutions should be used for the epidural, with additional pain management using PCA morphine.

Injuries to Local Nationals

Injuries to local national civilians and combatants on both sides are common, and despite attempts to remove such patients from the military care system through various "eligibility matrix" policies, experience shows that these patients will be received. Such patients raise difficult ethical decisions (see Chapter 42, Ethical Challenges of Deployed Military Critical Care), but all patients should be treated equally in the provision of adequate pain relief. Additionally, by reducing morbidity it may be possible to speed up their recovery and move these patients into the local medical system, thus relieving pressure on beds in what are usually small medical facilities.

The usual pain relief techniques are appropriate with local nationals; however, as described above, PCA may not be well understood, despite the use of an interpreter. There have also been problems with recreational drug use among local nationals, which can affect pain management. The management of children often causes concern for military clinicians who tend to be doctrinally focused on adults. However, within the bounds of experience and competency, many of the techniques described above may be used in the pediatric population. Epidurals remain appropriate for abdominal and bilateral lower limb injuries. If an epidural is not considered appropriate because of a patient's age and size, TAP catheters or even rectus sheath catheters may be inserted. PCA is unlikely to be appropriate in this age group, but in a medium- to high-dependency setting morphine infusions are appropriate and ketamine infusions have been used. Full regular doses of paracetamol and an NSAID should be prescribed as for adults when there are no contraindications.

Treatment Facilities

Current practices for regional anesthesia may vary according to type of facility or role of care. In the deployed environments, Role 3 facilities are permanent structures where infection control is adequate and the clinical environment is clean. In tented medical facili-

ties (Role 2), practice has differed between UK and US clinical staff, especially over the insertion of epidural catheters. The UK view has been that this environment is more contaminated, so epidural insertion has been avoided. However, there is little evidence for this position, and US staff have continued to insert epidurals in Role 2 facilities with little evidence of harm. The greatest source of contamination is the patient, and use of a standard sterile approach (skin decontamination, gown, gloves, mask, drapes, etc) should minimize risk. As with any clinical procedure, a risk–benefit assessment should be made. There is good evidence of safety for CPNB catheters based on recent experience in a variety of medical treatment facilities. The risk of infection can also be reduced by tunneling catheters, which is recommended.

CASE STUDIES

Local National Combatant With Isolated Forearm Gunshot Wound

The operating room was busy and no tables were free, but surgery was urgently required. It was expected that this casualty would be unlikely to use PCA properly, but his acute pain had to be addressed and his requirement for an anesthetic in the near future considered. A supraclavicular CPNB was performed. The patient was made comfortable, allowing transfer to the ward to wait for operating room space. In due course he was brought to the operating room and his surgery carried out under the regional block.

Local National Child With Penetrating Abdominal Injuries

A 9-year-old local national child was admitted with fragmentation injuries to the abdomen, and an exploratory laparotomy was undertaken. Postoperative pain relief was initially managed with bilateral TAP blocks as single injections under US guidance. These worked well, and the patient was returned to an intermediate-dependency area with bolus intravenous morphine as rescue analgesia. Approximately 12 hours later, the Role 3 acute pain service was asked to review the patient due to large morphine requirements and high pain scores. The pain service team decided to return to the operating room and insert a thoracic epidural. This was performed in the lateral position uneventfully and the patient's pain was well controlled thereafter. This case demonstrates the potential problems associated with single-injection techniques as well as the benefit of a dedicated acute pain service.

SUMMARY

It is clearly important to manage acute pain in a timely and effective fashion for reasons of simple humanity, practicality (ie, easier patient management), reduced morbidity, and reduced incidence of chronic pain and long-term opioid consumption. A decade's experience with combat injuries shows that even relatively advanced techniques, including PCA and regional blocks, are not sufficient when used on their own to provide the degree of pain control these patients require. A multimodal and multidisciplinary approach, however, provides exceptional pain relief in the majority of these casualties.

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Chapter 19

SCORING PAIN

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INTRODUCTION

Although the simple yet vital humanitarian action of pain relief must not be forgotten, the pathophysiological effects of poorly controlled acute pain can affect every body system with potentially catastrophic consequences. Formally scoring and recording pain levels raises awareness and decreases the clinician's subjective input to pain evaluation. Also, the identification of pain aids in its treatment. Pain is multifactorial and subjective in nature, which makes it is difficult and complex to score. No formal standard of pain measurement exists, but a variety of different pain scoring systems have being implemented. Within the military, pain intensity is scored numerically; the US military also uses a questionnaire to determine the effects of pain.

DIFFICULTIES WITH SCORING PAIN

Although pain scoring has been shown to be essential to the treatment of patients, it is difficult to quantify pain and standardize these measurements for a number of reasons. Pain is subjective. It is often difficult to describe even by the coherent, articulate patient. A common language of pain does not exist, either verbal or nonverbal. Different patients communicate their degree of pain in very different ways. The medical language used specifically by pain medicine does not always correlate with the language a patient would use to describe the pain they are experiencing.

There are often additional barriers to communication. Explicit factors are seen in infants, elderly patients, and patients with cognitive or speech impairments. However, some factors are less apparent, such as culture, psychological issues, level of arousal, previous pain experiences, and learnt behaviors in response to pain. Some patients perceive benefit in under- or over-reporting pain.[1]

Culture has long been thought to be a major factor in pain reporting. Most clinical studies show differences in the perception of pain and the behavioral response to pain. However, what actually mediates this difference is still unknown. Additionally, great variation occurs even within cultural groups, depending on gender, age, socioeconomic status, and ties to country of origin. Lipton and Marbach studied facial pain in black, Irish, Italian, Jewish, and Puerto Rican patients. They found that pain perception was the same between different races; however, the emotional response to pain—stoic versus expressive—showed interracial variability, as did the effects of pain upon activities of daily living.[2]

Patients who are in fear of pain—when they experience no respite from their symptoms or experience frequent recurrent pain—can have increased sensation or attention. McCracken found that patients felt greater pain intensity and were more distressed by pain when they had increased vigilance regarding pain.[3]

Classifying pain is difficult, which makes pain scoring difficult. Many different ways to classify pain exist, and some presentations of pain do not fit easily into one category. An example of classification is acute versus chronic, but in between there are many variations: acute on chronic; chronic progressive; episodes of acute pain with significant pain-free periods. Types of pain can also be somewhat arbitrary in their classification—somatic, neuropathic, visceral—and many people experience a combination of these. Others find it very difficult to provide a history to help the physician to distinguish one type from another.[4]

Pain is multifactorial in nature, so scoring must be either complex enough to accommodate the many factors involved, or simplified, without taking into account this multifaceted nature. Of all pain-related factors scored, scoring the intensity of pain has shown the most significant outcomes (see discussion below).[5] However, using intensity alone does not factor in pain's effects on physical and psychological wellbeing.[6]

WHY SCORE PAIN?

Scoring pain minimizes the risk of undetected or poorly managed pain. Pain is the primary reason people seek medical treatment. Characterizing pain is core to the process of finding a diagnosis for pathology. Scoring pain can assist the clinician in determining the pathology and formulating a diagnosis. Untreated pain leads to pathophysiological sequelae that can worsen the patient's physical condition and lead to additional pathology such as chronic pain. In the acute setting, self-treatment of pain leads to poor management of the symptoms and extended time experiencing pain.[7]

Additionally, pain that limits a patient's activities of daily living has significant financial consequences, in terms of direct cost of pain treatment, loss of income if the patient is unable to work, and the government benefits the patient may consequently require. Poor identification of pain and lack of auditing its management will only increase these costs.[8] Pain scoring is necessary to study the mechanisms of pain and the efficacy of treatment methods. By allowing clinicians to audit such measurements, management plans can be implemented using evidence-based medicine.

HOW TO SCORE PAIN

There are two main types of pain measurement: those assessing intensity of pain and those measuring the effects of pain. Pain intensity is related to how much a person hurts. Pain intensity appears to be homogenous in nature; patients can readily rate it and with a good degree of reproducibility.[9] The effects of pain appear to be more complex, wide-ranging, and variable. Effects of pain are dependent on a number of other physiological and psychological states, and thus require a far more complex system of rating.

Scoring Pain Intensity

The main pain intensity scoring systems are verbal rating scales, visual analogue scales, and numerical rating scales. Using a simple scale of pain intensity negates the need for patients to actively report pain, minimizes reporter bias, and provides data for a simple protocol-driven management plan.

A verbal rating scale uses words that describe pain in order of severity. Descriptions at opposite ends of the spectrum are often "no pain" and "most intense pain imaginable"; adjectives between these extremes describe different gradations of pain. Patients read through these words and pick those most appropriate to the intensity of pain they are currently experiencing. Verbal rating scales require little training to administer. Commonly the descriptors of pain intensity are assigned a pain score, which enables ratio properties and makes statistical analysis easier (but assigning numerical scores is controversial because different verbal descriptors do not represent equal changes in magnitude of pain). Verbal scales are generally well received by patients and have the highest compliance rate of all the intensity scoring systems.[10] On the other hand, they also require good understanding of the vocabulary, and words can be interpreted differently. Some patients may not relate the words on the scale to the pain they are experiencing, and the words take time to read and understand.

With a visual analogue scale, the patient marks pain intensity at a certain location on a line (usually 10 cm long), with each end representing the extremes of pain ("no pain" and "pain as bad as it can be"). This scale is easy to perform and has good validity. It is theoretically is the most sensitive of the pain scales given the number of possible responses. However, it also requires a degree of manual dexterity that can be compromised by pain, injuries, and environmental factors. In addition, the scale requires equipment, paper and pen, which are not always available in the field setting. It can also be difficult to compare scores with these scales.

Picture rating scales usually use numerous cartoon faces experiencing different degrees of pain, which can making them easier for patients who do not share the same language as the person assessing the patient. However, like visual analogue scales, they require equipment.

Numerical rating scales require a patient to rate their pain intensity along a numerical scale, with 0 representing no pain. Their validity has been demonstrated in many studies. Compared with other measures of pain intensity, numerical scales correlate well[10] and have been shown to be sensitive. They require little or no equipment (particularly if administered verbally) and therefore can be used in a wide range of environments. Simple and easy to administer, they can be used with a wide variety of patients, particularly those with whom other measures of pain intensity might fail (eg, the elderly, patients with motor impairment or reduced vocabulary). However, unlike visual and verbal scales, they do not have ratio properties, which limits their ability to quantify reductions in pain intensity.

Scoring Effects of Pain

Scoring systems such as the Brief Pain Inventory and the McGill questionnaire assess the multidimensional aspects of pain over a time period. Although these tools have a definite place in pain management and assessment, they do not provide the simplicity and ease of use in a wide range of clinical scenarios, particularly the more austere environments prior to Role 4 care.[11]

TABLE 19-1

UNITED KINGDOM MILITARY PAIN SCORING SYSTEM

Pain Score	Level of Pain	Analgesic Action
3	severe pain	morphine (or other strong opioid agent)
2	moderate pain	weak opioid or NSAID; also consider agents below on analgesic ladder
1	mild pain	paracetamol
0	no pain	none

NSAID: nonsteroidal antiinflammatory drug

Military Scoring Systems

The United Kingdom Defence Medical Services use a numerical scoring system (Table 19-1) for acute pain scoring, which fulfills a number of criteria. The system requires minimal training and is quick and simple enough to be used at all stages in the medical chain, including the point of wounding. It can be used by all ranks and is concise and easy to remember. The system has been adopted by Role 4 medical facilities, thus providing continuity of assessment.[11] It is also easy to record and allows a clear and concise management

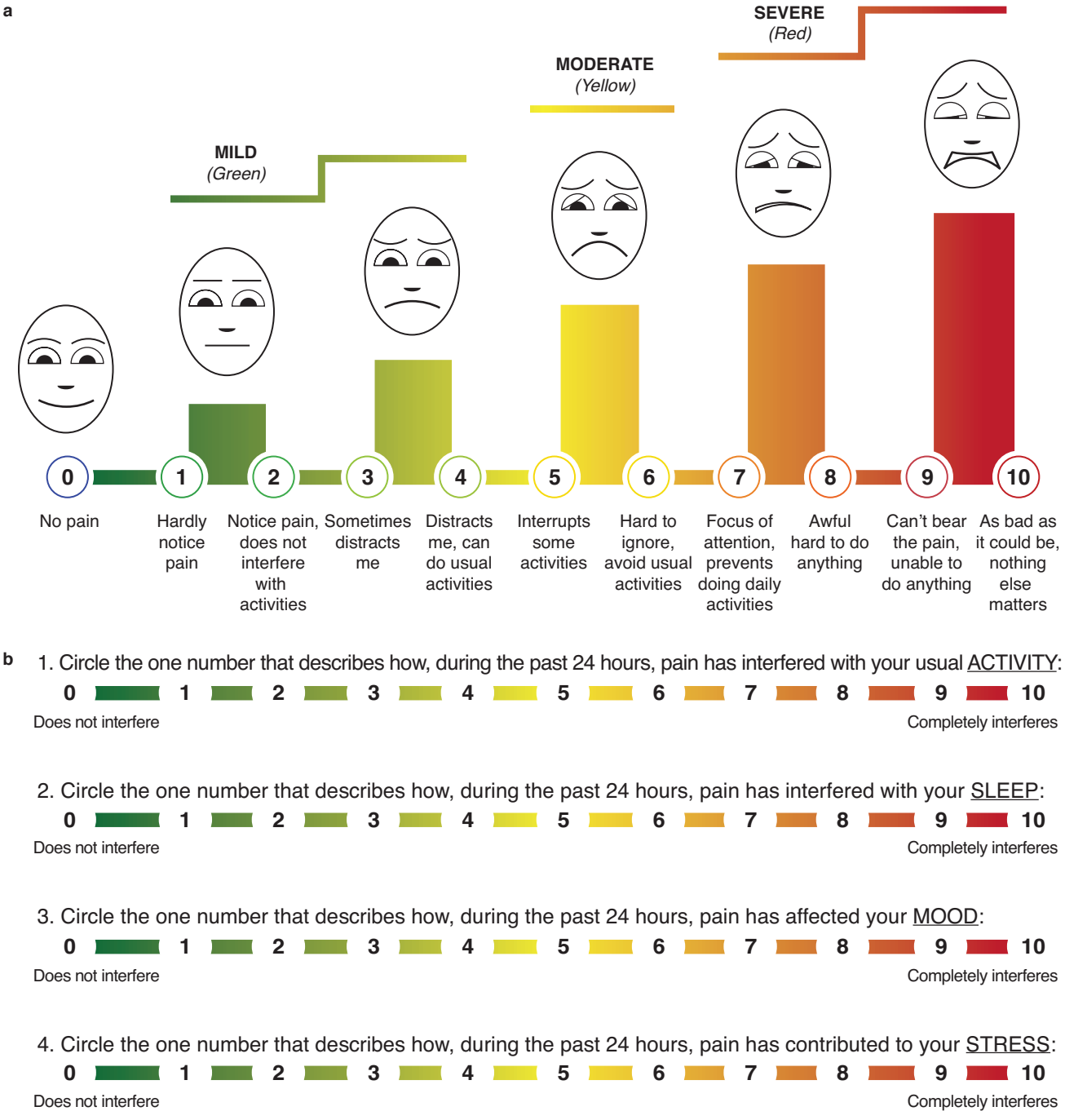

Figure 19-1. US Department of Defense/Veterans Administration pain scale tool (**a**), and supplemental questions (**b**).

plan to be instigated based on the results.

The US military relied on an 11-point numeric rating scale (0: no pain; 10: pain as bad as it could be) for many years as a validated method to evaluate pain. Unfortunately, the scale was found to be inconsistently administered, too subjective, and easily abused or misused. The lack of a standardized pain assessment tool adversely affected pain management throughout the care pathway and resulted in little to no consistent pain data.[12] In 2010 the US Army surgeon general released the Pain Task Force final report,[12] in which the requirement for an improved pain scale was made.

The Task Force subsequently developed a new Department of Defense/Veterans Administration pain scale (Figure 19-1) from these requirements. This tool combines the validated 11-point pain scale used by clinical researchers with a simple green, yellow, and red scale more suitable for combat medical conditions. Furthermore, it grounds each numeral on the 11-point scale with standardized "functional" language.[13] The new tool is expected to greatly enhance clarity for both patients and providers when discussing pain levels and treatment effectiveness throughout the care continuum. The tool also includes supplemental questions for clinicians at all levels (depending on need) to evaluate the biopsychosocial impact of pain in their patients. Questions ask about the impact of pain on general activity, mood, level of stress, and sleep. These questions, combined with the functionally anchored 11-point scale, have the potential to provide a powerful clinical tool in evaluating a patient's pain. The clinician or patient can use the most appropriate form of scoring system in each interaction, tailoring the system to the patient.[14]

WHEN TO SCORE PAIN

Pain can vary over time and should be scored regularly. Pain has been found to affect memory, and retrospective pain scoring (especially over a long period of time) renders it inaccurate. Linton and Gotestam found that if patients are required to retrospectively recall their pain, their current pain influences their pain scoring; regular, well-timed pain scoring is therefore key.[15] Pain should be assessed at regular intervals during the day, particularly after turnover of care providers, and before and after the use of analgesia.

CONCLUSION

Ultimately the primary goal of pain scoring should not be forgotten: the treatment of pain. Whatever pain assessment tool is use, the concern should be not just recording the pain but also reducing it to acceptable levels. The acceptable level has been suggested to be no worse than mild pain, which correlates with significant benefits physiologically and psychologically.[16]

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Chapter 20

PAIN MEDICATIONS

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INTRODUCTION

The practice of acute pain medicine in a battlefield environment must take into account injury severity and location-specific capabilities, including continued patient monitoring. Due to austere conditions and possible physiologic instability, an acute pain medicine physician must have a broad command of various multimodal analgesic pathways and options. Various injuries, whether mild or severe, place soldiers at risk for chronic postsurgical pain.[1] Although no analgesic regimen has been shown to significantly impact the prevalence of chronic postsurgical pain following traumatic injury, a multimodal approach aims to mitigate contributing risk factors. Specifically, patients who experience severe preoperative and postoperative pain are noted to have higher incidences of chronic pain.[1,2] A truly preemptive approach is impossible due to the nature of such traumatic injuries; however, a continuous multimodal approach embraces a theoretically preventive strategy.[3] This strategy utilizes both pharmacologic and interventional modalities aimed at various nociceptive mechanisms (inflammatory, neuropathic, etc), with the secondary benefit of minimizing the side effects of any one therapy. The strategy provides a continuous analgesic pathway that extends through numerous preoperative, intraoperative, and postoperative settings.

Various classes of analgesic modalities are available. However, in a battlefield setting, practical issues such as availability, physiologic status of the patient, and the ability to rapidly transport the patient to a tertiary level of care outside the theater of conflict must be considered. Pharmacologic modalities can roughly be separated into two classes, opioids and nonopioid adjuncts. The discussion below of pharmacologic therapies is intended to apply to the first 24 to 48 hours postinjury prior to transfer to a tertiary care site. Interventional modalities will be discussed in Chapter 22, Regional Anesthesia and Coagulopathy of Trauma Shock.

OPIOIDS

Opioids, particularly morphine, have been the mainstay of analgesic treatment since the 19th century. The range of opioid formulations available makes them versatile for pain control in the setting of limited resources and in patients who may or may not have reliable intravenous access. Although opioids have displayed longstanding utility, overuse accentuates significant limitations in the form of side effects as well as unforeseen effects such as opioid-induced hyperalgesia or acute tolerance. Therefore, acute pain medicine practice on the battlefield has deemphasized the use of opioids as the sole analgesic agent in favor of a multimodal approach. However, opioids still offer the benefit of a diverse array of agents (short or long acting, per os [PO] or intravenous [IV] routes) that can be matched to a variety of injury severities (Table 20-1).

Per Os Administration

Various formulations of PO opioids of both short- and long-acting duration are available. In general, only short-acting oral opioids are utilized in the acute phase of battlefield pain management for mild and moderate injuries. Patients must be able to tolerate oral intake and cognizant enough to communicate about dosing or hand carry medications that can be self administered during transport. Long-acting agents are often unnecessary because they cannot be titrated and patients are rapidly transferred out of theater.

All short-acting PO opioid formulations can be used with similar efficacy. However, older agents such as codeine-based analgesics require biotransformation into morphine to exert their effects, and up to 10% of the general population lack the necessary enzymes to perform this transformation.[4] Furthermore, synthetic agents such as oxycodone, hydrocodone, or oral hydromorphone exhibit better bioavailability, allowing for a faster onset of action when compared to oral formulations of morphine or codeine.[4,5] Also useful are various short-acting agents available in tablet or liquid form. Almost all short-acting agents share similar clinical onset (15–30 minutes) and duration of action (2–4 hours).

One attribute unique to short-acting PO opioids is that they often come in dual analgesic formulations including either a combination of nonsteroidal antiinflammatory drugs (NSAIDs) or acetaminophen. Although each of these nonopioid components is useful within a multimodal analgesic regimen, they limit upward titration of their corresponding opioid due to the potential toxicity associated with exceeding daily maximal doses of NSAIDS or acetaminophen. Thus, the opioid component should be administered separately from the NSAID/acetaminophen component.

Intravenous Administration

IV opioids remain an essential component of battlefield and immediate resuscitation analgesia prior to evacuation. Depending of the severity of injury, intra-

TABLE 20-1

SUGGESTED ACUTE PAIN REGIMEN BASED ON INJURY SEVERITY

	Mild Injury, Conscious Patient	Moderate Injury, Conscious Patient	Severe Injury, Patient Extubated	Severe Injury, Patient Intubated
	Examples			
Medication	Superficial wounds	Fragmentation wounds, gunshot to extremities, distal amputation	Postoperative exploratory laparotomy, proximal single amputation	Proximal double amputation, significant intrathoracic/abdominal injury
Opioids, PO	Oxycodone, hydrocodone, morphine, hydropmorphone, or tramadol	Oxycodone, hydrocodone, morphine, hydropmorphone, or tramadol	Oral opioids as tolerated	Do not use
Opioids, IV	Do not use	Morphine/hydromorphone as needed or PCA	Morphine/hydromorphone as needed or PCA, possibly low-dose fentanyl infusion	Opioid infusion (eg, fentanyl)
Ketamine	Do not use	Infusion or PCA	Infusion or PCA	Infusion
NSAIDs and acetaminophen	Oral acetaminophen + NSAID (eg, celecoxib)	IV or PO acetaminophen + IV NSAID (ketorolac or ibuprofen) or PO NSAID (celecoxib or naproxen)	IV acetaminophen + IV NSAID (ketorolac or ibuprofen)	IV acetaminophen
Gabapentanoid	Gabapentin or pregabalin	Gabapentin or pregabalin	As tolerated	As tolerated (unlikely utility in severely injured ventilated patients)
Lidocaine infusion	Do not use	Do not use	Consider lidocaine infusion	Consider lidocaine infusion
α2-Adrenergic agonists	Do not use	Clonidine included in regional anesthetic if applicable	Clonidine included in regional anesthetic if applicable	Clonidine included in regional anesthetic if applicable

IV: intravenous; NSAID: nonsteroidal antiinflammatory drug; PCA: patient-controlled analgesia; PO: per os

venous opioids can be utilized as a continuous infusion, via a patient-controlled device, or as needed. Mild or moderately injured service members who are awake and alert with pain unrelieved by PO medications are ideal candidates for patient-controlled analgesia (PCA). However, severely injured service members who must remain intubated are often managed with continuous rather than as-needed intravenous opioids, along with continuous monitoring. Patients on PCA who exhibit stable vital signs within a given duration of time do not warrant continuous monitoring during subsequent aeromedical evacuation within or out of theater. Close communication between the transport team and the acute pain service is required to determine appropriate monitoring during transport.

Similar analgesic end points to PO opioids can be achieved with a variety of IV agents. Currently, morphine, hydromorphone, and fentanyl comprise the most common IV opioids used in the battlefield environment. Meperidine use is not recommended because it causes accumulation of detrimental metabolites that may lead to seizures. During the early resuscitative phase (initial operating room visits), fentanyl provides an easily titratable profile and a relatively short duration of action when compared to more hydrophilic agents (morphine or hydromorphone). Notably, it may not be possible to administer IV opioids in severely injured service members until hemodynamic stability is achieved. For postoperative analgesia, either morphine or hydromorphone is appropriate, considering

that morphine may need to be switched to another IV opioid if histamine-related side effects or significant renal impairment occur.

Adverse Events

In general, opioid-related adverse events are dose dependent, although some side effects persist regardless of dosing. In the acute postinjury period, when a patient is hemodynamically stable, opioid-related oversedation and respiratory depression are the most pertinent adverse events. These effects have led to numerous in-flight events when injured service members require intubation. Naloxone (suggested dosing: 40-μg increments up to 400 μg) must be readily available during the acute setting and initial transport to avoid unnecessary hypoxia.

Numerous other adverse events are related to opioids in the acute setting, particularly related to the gastrointestinal system. In mild or moderately injured patients who are awake and alert, nausea, vomiting, and constipation are common occurrences. In addition to deemphasizing opioids via a multimodal approach, rescue modalities such as antiemetics and adequate bowel regimens should be instituted early, if needed. Peripheral opioid antagonists such as methylnaltrexone have received much attention in recent years as an additional adjunct in the setting of opioid-accentuated ileus. However, such agents are probably not necessary within the first 24 to 48 hours of care and can be started if necessary in out-of-theater care centers.

In contrast to the adverse events discussed above, in which opioids detrimentally impact other systems, much attention has been paid to opioid-induced hyperalgesia, when opioids accentuate pronociceptive pathways in the acute setting. While usually not a concern within the first 48 hours postinjury, opioid-induced hyperalgesia serves as an example of how a multimodal strategy may indeed impact analgesic quality at further care sites. Although mechanisms are poorly understood, numerous systems are theorized to be involved: central glutamatergic *N*-methyl-D-aspartate (NMDA) activation, genetic dispositions, descending pathway facilitation, and spinal dynorphins.[6] Recent reports have suggested a role of acute opioid administration with hyperalgesic symptoms in the perioperative setting, especially with the use of remifentanil.[7] While conflicting data exists, a multimodal approach involving NMDA antagonists (noted below) has been indicated as a possible prevention tool for acute opioid-induced hyperalgesia.[6] Opioids cannot be realistically avoided, but the phenomenon of opioid-induced hyperalgesia provides further credence to the utility of multimodal regimens.

NONOPIOID ANALGESICS

N-methyl-D-aspartate Receptor Antagonists

NMDA receptor antagonists are an evolving treatment option in acute pain control. Through excitatory amino acids, the NMDA receptor is thought to play a significant role in acute pain signaling as well as prolonged central sensitization.[8] Although numerous oral agents are available, ketamine, a noncompetitive IV NMDA antagonist, is the most widely used and practical agent in the initial postinjury stages. A dissociative anesthetic, ketamine has a long history of use in the battlefield setting and is increasingly used in subanesthetic doses to provide excellent pain control postoperatively. Ketamine has numerous routes of administration for acute pain control, including PCA, IV infusion, oral dosing, and IV boluses. Ketamine may be used alone as PCA and can also be combined with morphine into an opioid PCA to decrease overall opioid requirements. Although conflicting data exists about its utility in the perioperative setting, a combined morphine/ketamine PCA has demonstrated benefit in patients undergoing thoracotomy and major abdominal surgery.[9–11] Table 20-2 lists suggested dosing.

Ketamine's side effects include nausea, vomiting, dysphoria, excessive secretions/salivation, hallucinations, hypertension, elevated intracranial pressure, and tachycardia. Pretreatment with a benzodiazepine or addition of transdermal scopolamine will minimize incidence of dysphoria and hallucinations. Episodes of nausea and vomiting can be minimized with prophylactic antiemetics. However, side effects of ketamine when used in low doses are not significantly greater than those associated with opioids.[12] In fact, Subramanian et al, in a metaanalysis of perioperative ketamine, reported that the incidence of side effects such as delirium or sedation is equivalent between groups on a PCA alone and groups on ketamine alone.[13] Compared to opioids, ketamine does not cause respiratory depression and in general has much less of a depressant effect on hemodynamics. As mentioned above, ketamine is also opioid sparing and may help avoid long-term postsurgical pain.[14,15] Recent work has suggested that ketamine may also have favorable effects on the incidence of posttraumatic stress disorder.[16] Ketamine's wide therapeutic window in small doses for acute pain control adds safety benefits, especially in wounded personnel during transport in the field, which often requires larger doses of opioids.

TABLE 20-2

SUGGESTED DOSES OF KETAMINE FOR ACUTE PAIN CONTROL

Administration	Steps
Infusion	1. Premedicate with benzodiazepine or scopalamine patch. 2. Bolus: 0.2–0.5 mg/kg over 30–60 minutes. 3. Begin fusion at 0.05–0.3 mg/kg/hr. 4. Titrate to clinical effect. 5. Observe for side effects (nausea, dysphoria, hallucination, excess secretions) and decrease infusion rate if present.
PCA (without opioid)	1. Initiate PCA at 4–6 mg every 10 minutes 2. Adjust bolus and interval as necessary. 3. Use caution if doses exceed 0.5 mg/kg/hr due to high incidence of side effects.

PCA: patient-controlled analgesia

Other agents including magnesium have also been studied in regard to perioperative pain management. Magnesium forms a "plug" within the NMDA receptor, effectively acting as an antagonist. Under physiologic conditions, binding of an agonist and cellular depolarization are required to displace magnesium from the NMDA receptor. IV magnesium has been studied in the setting of abdominal, cardiac, and orthopedic surgery. The data is conflicting, but the predominance of studies demonstrate an improvement in pain scores as well as an opioid-sparing effect.[17] Although rarely used in austere environments, magnesium warrants further study in battlefield-injured service members.

It has been suggested that memantine, an oral NMDA antagonist, may have a significant analgesic benefit in the perioperative period. However, convincing data is scarce, with a lack of strong prospective study designs. Further investigations are indeed warranted into its use in acute pain.

Nonsteroidal Antiinflammatory Drugs

NSAIDs are key adjuncts in multimodal acute pain management. NSAIDs are versatile and available in multiple formulations including IV, oral, and topical. Their lack of respiratory and hemodynamic side effects make them valuable for use in the field. Although weak analgesics, NSAIDs add effective synergy to opioids and other classes of analgesics. Unless an absolute contraindication is present, NSAIDS should be administered around the clock for all battlefield-related injuries.

The majority of NSAIDs exert their action by antagonism of cyclooxygenase (COX) 1 and/or 2, affecting prostaglandin synthesis. Older NSAIDs exert nonselective COX inhibition, resulting in possible antithrombotic and gastric side effects. Newer COX-2 inhibitors are more selective, with reduced gastric side effects and no antiplatelet effects. COX inhibitors including ketorolac and ibuprofen are available in IV form that increases their utility in trauma medicine. However, in patients who are awake, alert, and tolerating PO medication, any of the available COX-2 inhibitors or oral ibuprofen or diclofenac are appropriate.

NSAIDs have a long history of success in the perioperative realm, especially in the orthopedic and abdominal surgery literature.[18] These NSAIDs serve as opioid-sparing agents and display no clinically significant increase in bleeding.[19] However, for critically battlefield-injured personnel (bilateral amputation, head trauma, etc), withholding NSAIDs until hemorrhage is adequately controlled and renal concerns are minimal is the best course of treatment.

Acetaminophen

Acetaminophen exerts its effects by unknown mechanisms; however, it is suspected to act on both central and peripheral pain pathways. Unlike NSAIDs, acetaminophen is devoid of antithrombotic and gastric side effects. Acetaminophen has a longstanding history of opioid-sparing qualities in the perioperative setting.[20,21] Combination with an NSAID leads to synergistic analgesia when compared to either agent alone. While an IV formulation (paracetamol) has been available in Europe for many years, IV acetaminophen has only recently become available within the United States. While either PO or IV routes are appropriate, IV routes serve as a useful adjunct in severely injured battlefield patients. Unless severe liver dysfunction is present, all patients requiring analgesic therapy should receive a derivative of acetaminophen in the

acute phase of treatment and throughout transport.

Acetaminophen has an excellent safety profile; toxicity is very rare when dosing guidelines are followed. Liver toxicity secondary to the metabolite *N*-acetyl-p-benzoquinine imine (NAPQI) has been associated with large doses. Currently, no more than 4 g per day should be utilized.

Anticonvulsants

Anticonvulsant agents such as gabapentin and pregabalin have been studied in numerous settings to determine their perioperative analgesic benefit. Both agents act via presynaptic antagonism of the alpha-2-delta subunit of dorsal horn calcium channels, which become excessively active during various levels of nociception.

Numerous trials have demonstrated an analgesic benefit predominantly in the form of opioid sparing and improved pain scores.[22–24] However, there is conflicting data about decreased postoperative nausea and vomiting and opioid side effects with use of these anticonvulsants[25,26]; notably, trials have documented early increased sedation and dizziness with both agents.[24] Further controlled trials are needed to determine the long-term benefit of such agents, although recent investigations in patients undergoing total knee arthroplasty have suggested a lasting effect of decreased neuropathic pain when such agents are extended beyond the immediate postoperative period.[2]

Although both agents have shown some perioperative benefit, pregabalin demonstrates a more favorable pharmacodynamic profile with greater bioavailability, linear pharmacokinetics, and a faster achievement of therapeutic levels. Dosing regimens vary, but in general gabapentin may be administered as a 600-mg preoperative dose followed by 300 mg three times a day. For pregabalin, a 300-mg preoperative dose may be given followed by 150 mg twice a day in the postoperative period. Common side effects include dizziness, sedation, and possibly edema.

In a battlefield setting, gabapentin and pregabalin can be utilized in patients who are tolerating PO intake. Gabapentin can also be administered as a liquid via nasogastric tube in patients who cannot swallow. In mildly and moderately injured patients, either agent can be used preoperatively and continued during transport in awake and alert patients as a means to spare opioid usage. However, in critically injured patients, such agents probably offer little noticeable benefit and can be held until gut function has returned and full resuscitation has occurred.

α_2-Adrenergic Agonists

α_2-Adrenergic agonists have a long history of analgesic utility in various perioperative regimens. While the major site of action is via spinal pain modulation, α_2 agonists such as clonidine or dexmedetomidine have a complex array of analgesic mechanisms including norepinephrine regulation in the locus cereleus (facilitating descending inhibitory signals), peripheral interaction with afferent neurons, and regulation with nonadrenergic spinal neurotransmitters (acetylcholine, γ-aminobutyric acid [GABA]) that also modulate pain at the level of the spinal cord.[5]

While clonidine (particularly epidural or intrathecal) and dexmedetomidine have demonstrated opioid sparing qualities, their notable side effects of hypotension and bradycardia severely limit their utility in an austere environment. Even in mildly to moderately injured patients who are hemodynamically stable, their utility probably does not outweigh the risk of their common side effects during forthcoming transport where resources and monitoring is limited. However, peripheral use in the form of extending regional anesthetic blocks is a reasonable use of clonidine.[27] Dexmedetomidine has not been studied in a prospective manner to comment on the safety of its use in peripheral regional anesthetics. Doses for nonopioid analgesics are summarized in Table 20-3.

LOCAL ANESTHETICS

IV lidocaine has potential as an adjunct in acute pain therapy. Numerous studies have shown additional utility for pain control with lidocaine versus local anesthesia alone. IV lidocaine has demonstrated antiinflammatory, opioid-sparing, and analgesic properties.[28–30] These benefits could be particularly useful in the severely injured casualty, such as a double amputee, whose coagulation status contraindicates regional anesthesia. Side effects, while uncommon, include tongue or perioral numbness, tinnitus, restlessness, vertigo, concentration deficits, slurred speech, muscle twitching, seizures, respiratory depression, and cardiovascular depression. Patients treated with lidocaine infusions should therefore be continuously monitored.

A recommended regimen for a lidocaine infusion is to start with an initial bolus of 1 to 1.5 mg/kg and initiate continuous infusion of 1.25 to 1.5 mg/kg/hr.

TABLE 20-3

NONOPIOID ANALGESICS FOR ACUTE BATTLEFIED PAIN MANAGEMENT

Medication	PO	IV	Comments
		Aniline Derivative	
Acetaminophen	1 g every 6 hours	325 mg–1g every 4–6 hour	Maximum dose: 4 g/24 hours. No gastric or antiplatelet effects. Hepatotoxic in large doses
		NSAIDs	
Ketorolac	N/A	30 mg, then 15–30 mg every 6 hours	
Ibuprofen	400–800 mg every 8 hours	400–800 mg every 6 hours	Maximum dose: 3,200 mg/day
Diclofenac	50 mg three times daily	N/A	Maximum dose: 200 mg/day for first day, then 150 mg/day thereafter
Meloxicam	7.5–15 mg daily	N/A	Maximum dose: 15 mg/day
Celecoxib	100–200 mg daily	N/A	Selective for COX 2
Naproxen	250–500 mg every 12 hours	N/A	Maximum dose: 1,100 mg/day
		Anticonvulsants	
Gabapentin	100–300 mg every 8 hours. Titrate as needed to maximum dose of 1,200 mg three times daily.	N/A	Optional 600 mg loading dose prior to start of therapy
Pregabalin	75–150 mg every 12 hours. Titrate to maximum dose of 300 mg twice daily.	N/A	Optional 300 mg loading dose prior to start of therapy
		α_2-Adrenergic Agonists	
Clonidine	0.1 mg loading oral dose following by transdermal patch (not applicable within first 24–48 hours postinjury)	0.3 –1 μg/kg bolus	Severely limited by hypotension and bradycardia in acute battlefield setting. Peripheral nerve block dose: 0.5–1 μg/kg in local anesthesia
Dexmedetomidine	N/A	Load dose: 0.5–1 μg/kg over 10–20 minutes (if tolerated). Infusion: 0.2–0.7 μg/kg/h	Limited by bradycardia and hypotension
		Local Anesthetics	
Lidocaine	N/A	Bolus: 1–1.5 mg/kg followed by infusion of 1.25–1.5 mg/kg/h.	Possible option for noninterventional candidate (lack of access, coagulopathy, etc)

COX: cyclooxygenase; N/A: not applicable; NSAID: nonsteroidal antiinflammatory drug

SUMMARY

A multimodal strategy is essential early in the analgesic treatment of wounded soldiers. Opioids are no longer considered the sole agents of analgesia, as evident in clinically significant adverse events and the possibility that they worsen pain. Each patient's analgesic regimen is stratified based on injury sever-

ity (see Table 20-1), and such a multimodal approach should be continued throughout transport. Although pharmacologic modalities are only a portion of analgesic regimens, expertise in their pharmacokinetics and pharmacodynamics is essential in the setting of polypharmacy. Daily attention is required to monitor for analgesic benefit as well as side effects. However, almost all data about the use of this strategy comes from civilian nontraumatic literature, and further prospective trials are needed among injured service members.

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Chapter 21

ADVANCED PAIN MANAGEMENT TECHNIQUES

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INTRODUCTION

Advanced battlefield pain management offers anesthesiologists multiple options for managing perioperative pain from surgery or battlefield trauma. Because combat casualty injuries range from simple to complex, the initiation of pain management options will depend on the patient's injury pattern and subjective pain. For instance, a soldier with a superficial tissue injury may report adequate analgesia with intravenous (IV) acetaminophen and nonsteroidal antiinflammatory drugs (NSAIDs). More extensively injured patients with extremity injuries or traumatic amputations may require morphine patient-controlled analgesia (PCA) combined with an epidural or continuous peripheral nerve block (CPNB). Effective battlefield analgesia requires physician understanding of each patient's physiology, injury pattern, and pain threshold to provide an individualized and effective pain plan while minimizing unwanted side effects of pain medications in the challenging military evacuation environment. On the modern battlefield, technology now allows individualized pain management, which supports casualty evacuation and enhances rehabilitation and recovery of the wounded service member. This level of care requires physicians and nurses at all roles of care who are dedicated to managing pain.

PATIENT-CONTROLLED ANALGESIA

PCA has become commonly used in the deployed military setting in recent conflicts. It involves the use of a pump, which allows the patient to administer a bolus of an analgesic drug, most commonly morphine, via an IV line when he or she requires analgesia. A lockout mechanism controls the time between boluses and helps prevent excessive sedation and respiratory depression. Use of PCA has increased as pumps have become safe, reliable, and robust enough for use in a military environment and during aeromedical evacuation.

Types of Drugs

A range of drugs may be used in PCA, although morphine is the most frequently chosen. Hydromorphone, fentanyl, and ketamine (a nonopioid *N*-methyl-d-aspartate receptor antagonist) may also be used, and antiemetics such as cyclizine or droperidol may be added. Morphine has been the standard choice for PCA in recent deployments due to supply constraints on other drugs, sustainability, and the need for standardization. As new drugs are developed in the future, the use of morphine as the standard PCA drug may change.

Indications

IV dosing of opiates provides analgesia to pain arising from sites throughout the body. While regional techniques provide excellent analgesia to specific anatomical areas, systemic opiate administration provides analgesia to patients with multiple wounds, including four-extremity trauma, thorax, and head and neck, areas that may not be covered by regional techniques.

Contraindications

Allergy to opiates is an absolute contraindication for opiate-based PCA. Patients may need analgesic options other than PCA if appropriate management techniques do not suppress opiate side effects.

Consideration also needs to be given to the ability of the patient to understand the concept of a PCA, which may not be the case for some foreign national patients. In these circumstances, a continuous opiate infusion may be more appropriate when the patient is in the intensive care unit and on continuous monitoring.

The success of PCA depends on the ability of the patient to press the bolus button on the device. Most PCA devices require a certain force to push the bolus button in order to prevent accidental bolus delivery. Therefore, individual consideration needs to be given to patients with bilateral upper limb injuries, cognitive impairment, or neurological deficits.

Benefits

With PCA, patients control their own analgesia. This reduces any delay in provision of pain relief on the ward or evacuation aircraft, ideally keeping the patient comfortable and minimizing escalation of pain. Passing control of analgesia to the patient may also have psychological benefits and reduce the fear of being left in pain.

PCA is technically easy to establish in comparison to other techniques. Although the IV cannula access may be displaced, this is relatively easy to correct, even during an aeromedical evacuation flight.

Complications

The complications of PCA are usually secondary to the use of opiates, and these are well documented (Table 21-1). All patients need to be monitored for these side effects and treated as appropriate. Patients

TABLE 21-1

SIDE EFFECTS OF OPIATES

Side Effects	Management of Side Effect
Respiratory depression	Give O_2. If respiration rate < 8 breaths/min, call on-call doctor or anesthetist and give 400 μg naloxone IV (repeat as necessary).
Hypotension	Give O_2. Lie patient flat. Stop PCA. Give fluid bolus as prescribed. Consider other causes hypotension and call physician.
Nausea and vomiting	Consider antiemetics. Can be scheduled with breakthrough opioids as needed.
Sedation	Monitor respiration rate and BP. Stop further use of PCA. If patient is unrousable, call physician. If restarting PCA, consider increasing demand interval.
Pruritis	Consider chlorphenamine maleate (UK) or diphenhydramine (US) and regular ondansetron. If condition persists, use low-dose naloxone.
Constipation	Consider use of laxatives.
Urinary retention	Observe urinary output and if necessary catheterize.

BP: blood pressure
IV: intravenous
PCA: patient-controlled analgesia
Reproduced from: UK Joint Force Medical Group. *Pain Management at the Role 3 (UK) Medical Treatment Facility.* Bastion Joint Operating Base, Afghanistan: Joint Medical Command; 2010. UK JF Med Gp Standard Operating Instruction.

Figure 21-1. AmbIT Military PCA Pump, currently used by US forces for patient-controlled analgesia, epidural, and continuous peripheral nerve block. Used with permission from Summit Medical Products, Salt Lake City, UT.

on PCA should also receive regular nonopiate analgesia. Acetaminophen and an NSAID (if appropriate) should be prescribed regularly, and antiemetics should also be prescribed as required. Stool softeners should be prescribed to prevent constipation. In the event of respiratory depression, oxygen and naloxone should be readily available to reverse opiate effects.

Pump Settings

The PCA pumps available for United Kingdom (UK) personnel on deployment, Baxter Infusor Elastomeric Infusion System (Baxter, Thetford, UK) are elastomeric devices with a button to deliver a bolus on demand. The standard pumps provide a 0.5-mL bolus with a 6-minute lockout. The concentration of morphine is 2 mg/mL, which provides a 1-mg bolus every 6 minutes up to a maximum of 10 mg per hour.[1]

The United States currently uses the ambIT Military PCA Pump (Figure 21-1; Summit Medical Products, Salt Lake City, UT) for PCA, epidural, and CPNB. This pump is electronic and requires two AA batteries. The basal rate and bolus time intervals should be programmed for each individual patient. Depending on deployment drug inventory, US personnel use morphine or hydromorphone for PCA. In an opiate-naïve patient, the initial hydromorphone PCA dose is 0.2 mg IV every 10 minutes with a 1.2-mg per hour lockout. In the event the patient still reports intolerable pain after 1 or 2 hours, the ambIT pump can be evaluated and titrated appropriately.

If a morphine infusion is required for patients unable to use or understand PCA, then for UK personnel a standard infusion pump is used, the Braun Perfusor (Figure 21-2; B Braun, Melsungen, Germany). (The ambIT pump has a continuous infusion setting as well.) For the Braun Perfusor, the standard morphine concentration is 1 mg/mL in a 60-mL syringe with a clearly defined maximum infusion rate.[1] Patients using this device must be closely monitored for any adverse effects because a continuous infusion may cause unrecognized sedation and respiratory depression.

Figure 21-2. Braun Perfusor pump, used by UK personnel when morphine infusion is required for patients unable to use or understand patient-controlled analgesia. Used with permission from B Braun, Melsungen, Germany.

When to Initiate

PCA can be commenced in the immediate postoperative period once the patient is alert enough to understand how to use the device. PCA should be initiated if the patient does not report adequate analgesia despite scheduled opiate boluses (every 4–6 hours). PCA should also be prescribed for patients being repatriated with either CPNB or epidural in situ in case of in-flight failure of these techniques.

Prior to air evacuation, PCA settings should be evaluated to ensure that the patient reports tolerable pain relief and to minimize side effects such as sedation or respiratory depression. Due to the austere environment of aeromedical evacuation, monitoring in conditions of excessive noise and poor lighting should be considered.

Nursing Guidelines

Patients using a PCA should have clinical observations monitored on an hourly basis for the first 4 hours after initiation. If they remain stable after this period, observations should be carried out every 2 hours for the next 8 hours and then every 4 hours for the duration of the time the PCA is used.[1] In addition to routine physiological observations, pains scores should be monitored and entered on a specific PCA chart.

Clear instructions should be provided to the nursing staff for recognizing and managing complications such as respiratory depression, hypotension, increasing pain, or change in mental status. Nursing staff who work on surgical wards are likely to be familiar with PCA use, although the pump device may vary. Predeployment education about the specific pumps and PCA charts will be necessary. If drugs other than morphine are being used, dosages need to be clearly documented and communicated to ward staff to prevent drug errors. Documentation of the PCA device, the medication infused, and the PCA program should be clearly identified on the patient's evacuation chart and communicated to evacuation personnel.

EPIDURAL ANALGESIA

The use of epidural analgesia in military deployments has increased in recent years. The technique involves inserting a catheter into the epidural space and infusing a solution of local anesthetic to provide a central neuraxial block for pain. Epidurals are being used to provide analgesia for abdominal and lower limb wounds at the field hospital, during aeromedical evacuation, and in the patient's home nation.

Indications

Epidurals provide analgesia for abdominal, thoracic, pelvic, and lower limb wounds.

Epidurals can be inserted in patients with injuries amenable to epidural analgesia and patients woken from general anesthesia. An epidural in the field environment is not appropriate if the patient is to remain intubated and anesthetized or is unable to cooperate with neurological assessments during postoperative management.

Contraindications

Absolute contraindications to epidural anesthesia include patient refusal, infection at the needle insertion site, hypovolemia, elevated intracranial pressure, and allergy to local anesthetics.

Relative contraindications include coagulopathy, sepsis, and vertebral fractures. Severely injured patients presenting with a coagulopathy should not receive an epidural in the acute setting; however, placement may occur once the coagulopathy resolves. Patients with fever and an elevated white blood cell count should undergo further evaluation for bacteremia or sepsis prior to placement. After initiation of antibiotic therapy, epidural analgesia should be considered in patients with clinical improvement (ie, afebrile, declining white blood cell count). Although traditionally considered a contraindication, patients presenting with vertebral spinal fractures should be handled on a case-by-case basis. Vertebral radiographs describing the spinal level as well as the type of fracture

(spinous process, transverse process, vertebral body) may permit placement of an epidural under fluoroscopy or ultrasound guidance.

Benefits

Successful epidurals provide profound analgesia to the abdominal area and lower extremities. Epidurals have the added advantage of treating incisional pain. Additionally, epidurals tend to provide an opiate-sparing effect and thus reduce the side effects of opiate analgesia, particularly respiratory depression, nausea, vomiting, and pruritus. In patients with compromised respiratory function, the reduction in opiate dose associated with an epidural may be of particular benefit in preserving vital capacity. Other benefits include a reduction in the autonomic stress response to surgery, reduced incidence of hypercoagulability, and improved gastrointestinal motility.[2]

Complications

Patients with epidurals should be monitored for risks and complications (Table 21-2). Epidural hematoma and abscess can cause permanent spinal cord injury and require an emergency neurosurgical evaluation. The site of the epidural insertion should be monitored daily for any evidence of infection such as localized redness, swelling, tenderness, or obvious pus. Signs of central neurological infection such as meningitis should be aggressively managed. Epidural hematoma symptoms include increasing motor block, increasing back pain, or a change in bowel or bladder control and should prompt immediate imaging studies and neurologic consultation.

When to Initiate

Insertion of epidural catheters in the elective setting is usually carried out with the patient awake in order to obtain cooperation for positioning and allow the patient to alert the practitioner to any nerve root pain or paresthesia. In the deployed environment, this is unlikely to be an option due to the urgent need for surgery, and epidurals may be inserted at the end of the operation with the patient still anesthetized. This allows analgesia to be established prior to waking the trauma patient from general anesthesia, avoiding the pain stress response and "wind-up" (see Chapter 16, Physiology of Pain). Epidurals can be considered at any time following surgery to improve pain management. The use of ultrasound to identify the spinous process and estimate the depth of the epidural space may be of benefit, especially in the anesthetized patient. The timing of epidural insertion and removal should reflect the latest guidelines for anticoagulation medication.

TABLE 21-2

COMPLICATIONS OF EPIDURALS

Risks and Complications	Management
Dural puncture and postdural puncture headache	Lie patient flat, encourage oral fluids including caffeine. Consider blood patch.
Failure or patchy block	Establish sensory level. Provide top-up dose (anesthetist). Consider patient positioning for patchy or unilateral blocks.
Hypotension	Give O_2 and lie flat. Stop epidural infusion. Give fluid bolus as prescribed. Call anesthetist. Consider other causes of hypotension. Consider use of vasopressors.
Itching (opiate related)	Consider chlorphenamine maleate and ondansetron. If condition persists, use low-dose naloxone.
Nerve damage (needle damage, epidural hematoma, epidural abscess, anterior spinal artery syndrome, or arachnoiditis)	Observe for increasing back pain, increasing motor weakness, or change in bowel or bladder function. Inform anesthetist immediately; patient may need spinal imaging (CT or MRI).
Urinary retention	Consider need for catheterization if not already in situ.
Motor weakness	Consider reducing infusion rate. Also consider epidural hematoma.

CT: computed tomography
MRI: magnetic resonance imaging
Reproduced from: UK Joint Force Medical Group. *Pain Management at the Role 3 (UK) Medical Treatment Facility*. Bastion Joint Operating Base, Afghanistan: Joint Medical Command; 2010. UK JF Med Gp Standard Operating Instruction.

Securing the Catheter

When securing epidurals in the deployed environment, consideration should be given to tunneling the epidural catheter. Tunneling of epidurals has been shown to reduce the chance of catheter displacement,[3–5] which is important when the epidural may be in situ for a prolonged period, including the multiple moves involved in aeromedical evacuation. Although there is some evidence that tunneling of epidurals may reduce

the rate of bacterial colonization,[6] there is very little evidence that tunneling reduces epidural infection rates. Tunneling of caudal catheters has been demonstrated to reduce colonization rates to the same as those of lumbar epidurals.[7]

Skin glue (medical cyanoacrylate adhesives) can also be used to reduce the chance of accidental removal of the epidural catheter[8] and should be considered in conjunction with tunneling to reduce the risk of catheter displacement. The site should then be covered with a clear dressing to allow easy inspection while the catheter remains in situ.

Drugs and Infusion Settings

For UK personnel, the standard infusion is bupivacaine 0.125% infusing at 7 to 15 mL per hour. Because of the increased risk of respiratory depression from combining epidural and IV PCA opiates, the current recommended policy is to use an opiate-free epidural solution. If opiates are added, the infusion would be 0.1% bupivacaine with 2 μg/mL fentanyl.[1] For US personnel the standard infusion is 0.2% ropivacaine infusing at 6 to 12 mL per hour. A typical initial epidural setting includes a basal rate of 8 mL per hour with a 3-mL demand bolus every 20 minutes.

Although these are the standard choices for UK and US epidurals, whichever drug is available may be used if there are supply constraints.

Pumps

Pumps used for an epidural infusion should always be clearly labeled to prevent medication errors, which may include incorrect drug as well as incorrect route of administration. Labeling is of particular importance where infusion devices may have more than one use and are not for sole epidural or nerve catheter use.

The US military currently uses the ambIT PCA Military Pump for epidural infusions as well as for PCA and CPNBs. Deployed UK military personnel currently use Braun Perfusor pumps to provide epidural infusions. The drug is made up in 50-mL syringes (rather than the pre-prepared bags common in civilian practice).

Daily Rounding Considerations

All patients with an epidural should have an epidural chart as well as regular physiological observations. The level of the block should be measured and recorded, and leg strength should also be documented. Pain scores should be assessed and recorded on the epidural chart, and there should be clear instructions about who to contact in the event of side effects, complications, or increasing pain. If there are any signs of infection or epidural hematoma, an anesthetist should be called immediately. If pain is increased an anesthetist may deliver a top-up bolus through the epidural (prior the top-up bolus, the level of the block and sensory deficit should be assessed). Patients with epidurals should also receive regular simple analgesia such as acetaminophen and an NSAID if appropriate to support a multimodal pain management plan.

For patients who are not being evacuated, providers will need to decide when to remove the epidural catheter. Removal will depend on the clinical situation and is likely to be about 3 days after insertion if the catheter is not tunneled. If the patient requires an epidural beyond 3 days, the non-tunneled catheter should be removed and replaced via a tunneled technique. Tunneled catheters have been managed for up to 14 days. In these circumstances, daily examination of the catheter insertion site is very important to detect any evidence of infection.

Nursing Guidelines

Many nursing staff working on surgical wards will be familiar with managing patients with epidurals in situ, although the pumps may be unfamiliar. Predeployment training should help increase confidence using the specific pumps seen on deployment. Epidural charts should also be discussed in predeployment training. Nurses unfamiliar with epidural infusions should receive training in basic epidural management as well as interpretation of epidural charts prior to deployment. The complications associated with epidural use should be emphasized, and the signs and symptoms of a complication should be checked for with each set of nursing observations. In the event of any questions or emergency, clear instructions to contact the duty anesthetist should be emphasized. Prior to deployment, anesthetists should also receive instructions and standard protocols on the pumps to be used during their deployment.

Epidural Equipment Changes

In the next 2 years, UK safety guidelines will call for a change in epidural catheter connectors so they are no longer compatible with IV devices. Currently the pumps and syringes are not specific to epidurals, so this may result in significant changes in the equipment available during deployment.

CONTINUOUS PERIPHERAL NERVE BLOCK

Because of advancements in body armor during the Iraq and Afghanistan conflicts, the vast majority of battlefield injuries now involve extremities. Due to the unpredictable injury patterns caused by combat trauma, CPNB is commonly used to manage acute pain on the battlefield. CPNB can provide postoperative analgesia to multiple extremities by the perineural administration of local anesthetic.

Indications

The opportunity to apply CPNB should be considered when patients present with single or multiple extremity injuries or rib fractures with chest tubes, as well as during abdominal surgery when epidural anesthesia is contraindicated. The application of CPNB therefore requires the acute pain physician to understand the patient's injury pattern as well as the pathophysiology caused by the trauma and resuscitation. Although CPNB is commonly used for one to two extremity injuries, the analgesic plan may be expanded to cover three extremities (Exhibit 21-1).

Contraindications

Compared to epidural analgesia, CPNB has relatively few contraindications. Absolute contraindications include patient refusal, allergy to local anesthetics, systemic infection, and infection at the site of needle entry. Relative contraindications include preexisting neurological deficit and coagulopathy.

Battlefield traumatic injuries may result in neurologic deficits, which may be masked or exacerbated by regional anesthesia. Prior to placing a regional block, the acute pain physician should discuss the injury and neurologic deficit with the surgeon. However, the risk of neurologic injury from CPNB is small, and the benefits of optimal pain control may outweigh the potential risk. Trauma patients with lower extremity fractures at risk for compartment syndrome should have a prophylactic fasciotomy prior to transport. If a patient develops compartment syndrome, the likelihood of CPNB masking ischemic pain is extremely low when a dilute local anesthetic is infused.

Coagulopathy should be considered a relative contraindication for CPNB when the procedure is performed under ultrasound guidance by a skilled practitioner who demonstrates appropriate needle visualization. Trauma patients with a coagulopathy should not receive a deep block such as a lumbar plexus or posterior sciatic nerve block with a nerve stimulator until the coagulopathy is corrected. If a patient requires therapeutic anticoagulation after placement of CPNB, most catheters (with the exception of a lumbar plexus catheter) may remain in place, assuming that a risk-benefit analysis is communicated to the patient and surgeon. Similar to epidural technique, the latest guidelines on regional anesthesia, coagulopathy, and anticoagulation medications should be considered when performing a CPNB.

EXHIBIT 21-1

CONTINUOUS PERIPHERAL NERVE BLOCK OPTIONS FOR MULTIPLE EXTREMITY INJURY

- Epidural + brachial plexus
- Femoral/sciatic +/- brachial plexus
- Sciatic (bilateral) +/- brachial plexus
- Paravertebral +/- brachial plexus or lower extremity
- Brachial plexus (avoid bilateral phrenic nerve)

Benefits

Similar to epidural anesthesia, the goals of CPNB involve achieving a tolerable level of postoperative analgesia while minimizing the side effects of opioids. Recent studies have reported on the systemic effects of uncontrolled pain as well as increased stress response.[9] Compared to epidural analgesia, patients with CPNBs may receive twice daily prophylactic anticoagulants (enoxaparin) and do not require a urinary catheter.

Complications

The complications of CPNB are similar to those of epidural anesthesia and include failed block, bleeding, infection, local anesthetic systemic toxicity (LAST), and nerve injury. Pneumothorax is a potential complication of paravertebral as well as upper extremity blocks including supraclavicular and infraclavicular blocks, although this event is rare. Patients requiring bilateral brachial plexus CPNB should not receive two blocks that will cause bilateral phrenic nerve involvement.

The acute pain physician must guard against LAST during placement and management of CPNB. Proper technique involves slow injection (5 mL every 5–10 seconds) combined with gentle aspiration for blood ev-

EXHIBIT 21-2

SYMPTOMS AND SIGNS OF LOCAL ANESTHETIC SYSTEMIC TOXICITY

- Lightheadedness
- Dizziness
- Acute anxiety
- Vision changes
- Disorientation and drowsiness
- Tremor, shivering, twitching, and tics
- Seizure
- Cardiac arrest

EXHIBIT 21-3

TREATMENT RECOMMENDATIONS FOR LOCAL ANESTHETIC SYSTEMIC TOXICITY

1. Airway management.
2. Seizures: Treat quickly with benzodiazepines. Avoid propofol with cardiovascular compromise.
3. Cardiac arrest:
   a. Epinephrine, small initial doses (10- to 100-μg boluses).
   b. Avoid vasopressin.
   c. Avoid calcium channel blockers and β-adrenergic receptor blockers.
   d. Amiodarone is preferred in presence of ventricular arrhythmia.
4. Lipid emulsion therapy:
   a. 1.5 mL/kg 20% lipid emulsion bolus.
   b. 0.25 mL/kg/min, continued for at least 10 minutes after patient is hemodynamically stable.
   c. Hemodynamically unstable? Consider repeat bolus and increasing infusion to 0.5 mL/kg/min.
   d. Maximum upper dose: 10 mL/kg over 30 minutes.

Data source: Weinberg GL. Treatment of local anesthetic systemic toxicity (LAST). *Reg Anesth Pain Med*. 2010;35:188–193.

ery 5 mL with constant surveillance for any side effect (Exhibit 21-2). An emergency cart with resuscitation equipment and medications including lipid emulsion therapy (Intralipid [Fresenius Kabi, Bad Homburg, Germany]) should be readily available. If the patient develops a seizure, the acute pain physician must provide immediate airway support and supplemental oxygen, and terminate the seizure with a benzodiazepine or propofol (25–50 mg). Patients progressing to cardiovascular collapse should immediately receive advanced cardiac life support and lipid emulsion therapy (Exhibit 21-3).

The incidence of permanent neurologic injury for regional anesthesia has previously been documented at 0.4%.[10] Unique to the battlefield, this adverse sequela may not manifest until after multiple evacuations and catheter removal. If a patient presents with a neurologic injury such as residual numbness or a motor deficit, a focused neurologic history and physical examination should be obtained. A chart review including the regional anesthesia used and operative records should be analyzed to establish preexisting injury, block complication, or use of a tourniquet. Patients should describe their neurologic symptoms in their own words. A brief neurologic examination to confirm motor strength, residual numbness, and reflexes provides an opportunity to map the lesion anatomically with a drawing.

With specific information from the history and physical, the physician should attempt to identify a mechanism of injury that matches the neurologic deficits with the nerves involved. Multiple etiologies of nerve injury exist, including compression, stretch, laceration, needle involvement, and chemical local anesthetic. The traumatic component often complicates the clinical presentation, which should prompt consultation with a neurologist for further neurologic testing. For patients with persistent neurologic deficits who are in the evacuation process, the current acute pain physician must provide detailed documentation and recommend appropriate consultation with neurology, physical medicine, and physical therapy at the next level of medical care.

When to Initiate

In the setting of elective surgery, CPNB placement commonly occurs prior to surgery. Because combat surgical patients may present with hemorrhagic shock, the primary focus should remain on facilitating the start of the surgery and fluid resuscitation. After completion of the surgery and adequate fluid resuscitation, the acute pain physician will have to decide whether to place the block in either the operating room (OR), postanesthesia care unit, ward, or intensive care unit. In an unpredictable mass casualty scenario, the decision to transfer the patient out of the OR should always remain a high priority.

Assuming that the immediate use of the OR is not required, recent developments in ultrasound technology permit an acute pain physician to place CPNB prior to extubation. Although placing a CPNB at this time does not permit the patient to report a transient paresthesia, appropriate needle visualization combined with normal syringe resistance greatly reduces the incidence of a neuronal injection. The underlying rationale for utilizing regional anesthesia prior to emergence from general anesthesia is to prevent severe pain and the detrimental systemic effects of uncontrolled pain.[9] The safety of regional anesthesia in patients under general anesthesia has previously been discussed in the field of pediatric regional anesthesia.[11]

Securing the Catheter

As the battlefield anesthesia environment fluctuates from austere ORs to more modern combat hospitals, the acute pain physician's ability to adequately secure a regional catheter will maintain the functionality and longevity of the catheter. A trauma patient's exposure to harsh weather as well as air evacuation should prompt the provider to minimize the risk of catheter migration. Military anesthesiologists have commonly tunneled catheters for CPNBs lasting more than 3 days. Because CPNBs may be placed outside the OR, tunneled catheters may reduce infections.[6,7] CPNB in the military environment may increase the risk of inadvertent catheter removal because evacuation via airplane or helicopter contributes to catheter migration through provider handling and vibration associated with these aircraft. Multiple techniques are available to secure a catheter, but the recommended process involves tunneling with angiocatheter needle, skin adhesive (eg, Dermabond [Ethicon Inc, Sommerville, NJ]), adhesive spray (eg, Medical Adhesive, Hollister, Libertyville, IL) and a transparent dressing (eg, Tegaderm [3M, St Paul, MN]).[12(ch24)]

Drugs and Infusion Settings

Continuous infusions of local anesthetics may consist of either 0.2% ropivacaine or 0.125% bupivacaine. Buckenmaier and Bleckner[12(ch3)] have previously described infusion rates for standard ropivacaine doses for continuous regional anesthesia at Walter Reed Army Medical Center. In the UK, patients receive single catheter infusions of 0.2% ropivacaine at 10 mL per hour. In the United States, single catheter infusion settings may vary, with a continuous infusion between 8 and 10 mL per hour combined with a patient-controlled bolus rate of 2 to 3 mL every 20 minutes. Multiple catheter infusions require detailed attention to prevent LAST. In patients with two catheters, the continuous infusion may range from 5 to 10 mL per hour, with a patient-controlled bolus rate of 2 to 3 mL every 20 minutes for one of the catheters. Rate and bolus options may vary depending on the patient's pain location. Although there is no exact formula to prevent LAST, total infusions (continuous plus bolus) greater than 20 mL per hour are typically avoided.

Although multiple options exist for delivery of local anesthetic infusion, device selection depends on the respective governing departments' air evacuation safety certification (US Air Force or UK Ministry of Defence). The approved US infusion device for CPNB, as for PCA and epidural catheters, is the AmbIT PCA Military Pump. The approved UK CPNB infusion device is the elastomeric Braun Perfusor pump (Figure 21-3).

Daily Rounding Considerations

The ultimate goal of providing superior analgesic relief with CPNB to combat patients depends heavily on the ability to monitor the catheters each day. CPNB and other advanced pain modalities are best used under the management of a dedicated acute pain service (APS). The APS consists of specially trained anesthesiologists and nurses who are responsible for the day-to-day management of pain within the Role 3 (or higher) military care facility. The US military has established a Joint Theater Trauma System Clinical Practice Guideline on the management of pain, anxiety,

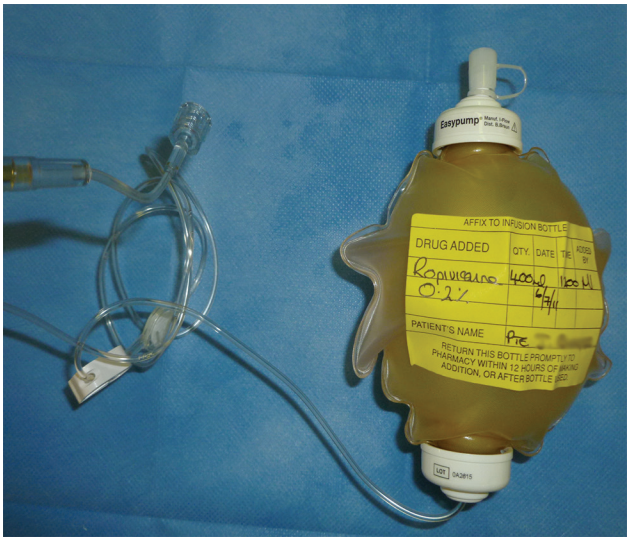

Figure 21-3. Braun Perfusor pump, the approved UK continuous peripheral nerve block infusion device. Used with permission from B Braun, Melsungen, Germany.

and delirium in injured service members that mandates an APS activity at all Role 3 (or higher) military healthcare facilities.[13]

The APS should review the pump infusion settings at each patient encounter. Catheter functionality should initially be assessed by asking the patients how they are doing. The benefits of asking an open ended question will allow patients to declare the severity of their pain without prompting them to immediately comment on their pain score. Depending on the patient's response, the APS can direct the patient interview toward pain scores. If the patient reports pain, the location and quality of the pain must be established to determine the functionality of the catheter. Depending on the severity of the injury, the incision injury may extend beyond the dermatomes of the blocked nerves. The anesthesia provider should consider contacting a surgeon if there is any suspicion of compartment syndrome.

If the patient reports pain in the anatomic distribution of a specific CPNB, the APS must determine if the patient has a sensory deficit with the affected dermatomes. Assuming that the patient does not have a sensory deficit to ice in the affected area, the physician should obtain vital signs every 5 minutes for 15 minutes after the administration of a 10-mL bolus of local anesthetic (1.5% mepivacaine or 0.5% ropivacaine). If the patient reports significant analgesic relief with the bolus, the provider should consider increasing the rate of the infusion by 2 mL per hour. If the patient reports persistent pain and no sensory deficit 15 to 20 minutes after the bolus, strong consideration should be given to catheter removal with potential replacement. Daily considerations for catheter removal should be reviewed; however, the APS should attempt to maintain the catheters as long as the patient has scheduled surgery because catheters can be used to reestablish surgical anesthesia with local anesthetics for repeated surgical interventions.

The APS's ability to monitor side effects and complications is a high priority as the patient is evacuated and obtains a new set of providers. During rounds, a focused chart review should include maximum temperature, coagulation labs (platelets, prothrombin time, partial thromboplastin time) and anticoagulation medications (enoxaparin, heparin, clopidogrel). The transparent dressings must be assessed at least once a day with emphasis on the following:

1. Does the site have any sign of infection such as tenderness, erythema, or purulent discharge?
2. Is there any evidence of blood in the catheter?
3. Finally, symptoms of local anesthetic toxicity such as tinnitus, metallic taste in mouth, central nervous system agitation, or thoughts of impending doom should be excluded (see Exhibit 21-2).

Nursing Guidelines

Prior to or shortly after the beginning of the deployment, the APS should provide a brief orientation to the ward or critical care nurses about regional catheters. Although the APS is directly responsible for the catheters, well-educated nurses will positively impact the patient's analgesic plan. Specifically, nurses should be educated on basic pump features such as turning the device on and off, changing batteries, and resetting the device when local anesthetic is replaced. Even though an APS may visit a patient once or twice a day, the ward nurse who attentively identifies a pump malfunction, suspicious skin rash, or early sign of local anesthetic toxicity will ultimately benefit the patient. The benefits of an established relationship between the APS and nursing team will help identify these issues sooner rather than later.

CONCLUSION

The regular use of advanced analgesic techniques is now commonplace in the deployed field hospital and is responsible in a large part for the improvements in analgesia for injured service personnel.

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Chapter 22

REGIONAL ANESTHESIA AND COAGULOPATHY OF TRAUMA SHOCK

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INTRODUCTION

Acute coagulopathy of trauma shock (COTS) is an ill-defined entity that is induced by tissue trauma, shock, acidemia, hypothermia, and hemodilution. It is often exacerbated by large volume autologous blood transfusion, with further dilution and consumption of coagulation factors. COTS has a very different pathophysiology compared to other forms of coagulopathy such as drug-induced (eg, heparin, low molecular weight heparin [LMWH]), preeclampsia, and bleeding disorders. Aggressive trauma resuscitation in accordance with current best practice is the best defense against undesirable consequences of markedly disturbed coagulation.

DETERMINING WHEN TO USE REGIONAL ANESTHESIA

The trauma patient, particularly following blast or ballistic trauma, with complex injuries requiring multiple dressing changes and operations may benefit from regional anesthesia (Exhibit 22-1). Guidelines have been published by national societies on the management of regional anesthesia in coagulopathy,[1,2] but there is no published evidence specifically on COTS and regional anesthesia.

Therefore, the decision-making process for regional anesthesia in the presence of COTS should focus on two principles: (1) Coagulopathy is dynamic; no fixed numbers show "safe" or "unsafe" conditions. (2) Risks of regional anesthesia need to be weighed against the potential benefit for each patient (Figure 22-1). This decision should include other trauma team members and the patient (when possible).

THE CAMP BASTION PROTOCOL

An acceptable approach has been used at the joint UK/US Role 3 hospital in Camp Bastion, Afghanistan. Anesthesiologists there are encouraged to assess and document coagulation as well as discuss potential risks and advantages of the planned regional anesthetic technique with the patient's trauma team. When available, thromboelastometry offers significant advantages for functional assessment of coagulation alongside traditional laboratory tests. Manufacturers of thromboelastomeric machines provide standard figures to assist with interpretation of results. The standard figures, as is the case with other laboratory standards, are developed from evidence and expert opinion on non-COTS coagulopathy. These figures should be used as supplemental information concerning a patient's coagulation state and no more. However, the addition of thromboelastomeric data has supplemented clinical decisions in COTS patients at the Camp Bastion

EXHIBIT 22-1

POTENTIAL BENEFITS OF REGIONAL ANESTHESIA

- Decreased morphine (or other opioid) requirement, which means less initial pain, fewer side effects of morphine, less time in recovery, and less opioid-associated immune suppression. Also, regional anesthesia is easier to manage on ward.[1]
- Humanitarian; foreign nationals are less likely to communicate their own pain experience.[2]
- During critical care, patient will awaken early, have a shorter stay in intensive care, and have improved respiratory dynamics.[3]
- Better early pain management potentially decreases the severity and incidence of both acute traumatic brain injury and chronic pain.[4]

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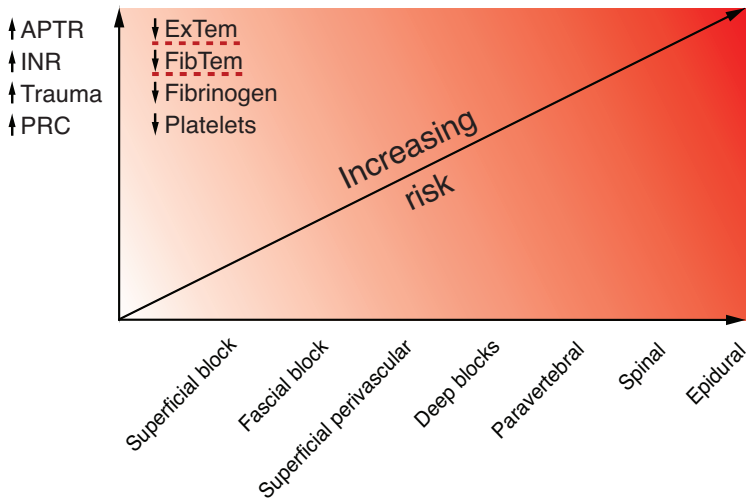

Figure 22-1. The continuum of regional anesthesia risk.
APTR: activated partial thromboplastin time ratio
INR: international normalized ratio
PRC: packed red blood cells
ExTem: platelet- and fibrin-dependent clotting test on thromboelastogram
FibTem: fibrin-dependent clotting test on thromboelastogram

hospital, and since its introduction in May 2010, no bleeding-related complications of regional anesthesia have been reported.

Suggested guidelines for regional anesthesia in the COTS patient (the Bastion protocol) are as follows:

Epidural catheter insertion (also applies to single injection spinal and epidural techniques)

1. Discuss and document the clinical requirement (risk vs benefit) for regional anesthesia (done by two senior clinicians; when possible, the requirement should also be discussed with the patient).
2. After large transfusions associated with use of fresh frozen plasma, epidural insertion should only be performed by a specialist; aim for least traumatic insertion.[3]
3. Insert epidural only when:
   - international normalized ratio (INR) ≤ 1.5 (INR = prothrombin time [test] / prothrombin time [normal]);
   - the activated partial thromboplastin time ratio (APTR) ≤ 1.5[2] (APTR = test / normal);
   - and platelets > 80 × 10^9 / L.[4]
4. If the above measures are acceptable and thromboelastometry is available, the epidural insertion should still be deferred if:
   - clotting time (CT) > 100 s,
   - and maximum clot firmness (MCF) (Ex) < 40 mm or MCF (Fib) < 8 mm. (Expert opinion only; patient must be normothermic.)
5. If the patient is already on a prophylactic LMWH dose, then the epidural catheter should not be placed until more than 12 hours after the last dose. The following dose should be delayed at least 4 hours after insertion.[2]

Epidural catheter removal

1. Remove only when:
   - INR ≤ 1.4,
   - APTR ≤ 1.4,[2]
   - and platelets > 80 x10^9 / L.[4]
2. If thromboelastometry is available, then MCF should be in normal range before removal (no research evidence presently exists to support this recommendation).
3. Catheter must be removed more than 12 hours after LMWH dose.[2]
4. Subsequent dose of LMWH should be at least 4 hours after catheter removal.[2]

Deep peripheral nerve block (single, continuous)

1. Follow epidural catheter insertion and removal guidelines above.[2,5]
2. Be aware of the risk of retroperitoneal hematoma in the lumbar plexus, requiring surgical evacuation.
3. The paravertebral space, which is relatively avascular but incompressible, can be used as an alternative to the neuraxial approach if the benefit outweighs the risk (per expert opinion).

Superficial peripheral nerve block (single, continuous)

1. Bleeding or hematoma related to superficial nerve block placement is not associated with long-term damage,[2] and large case series demonstrate safe removal of continuous peripheral nerve block (CPNB) catheters in patients treated with warfarin, LMWH, and heparin.[5,6]
2. CPNB catheters have been placed in patients receiving therapeutic (high dose) LMWH.[7]
3. Ultrasound use may reduce the risk of accidental vascular puncture.[8]
4. Higher values of INR and APTR as well as lower platelet count can be accepted for placement of CPNBs. There is insufficient evidence to make absolute numerical recommendations; therefore, the decision should be made per a risk/benefit analysis for each patient. Thromboelastometry can be of help in this process.
5. CPNB catheters must be removed more than 12 hours after an LMWH dose.[2]
6. Subsequent dose of LMWH should be at least 4 hours after catheter removal.[2]

Notes

- MCF (Ex), or MCF (ExTem) is a platelet- and fibrin-dependent clotting test on thromboelastogram. An abnormal MCF (Ex) in the presence of a normal MCF (Fib) reflects reduced platelet function. MCF (Fib) or MCF (FibTem) measures fibrin clot only. Low MCF (Fib) denotes fibrinogen or F XIII deficiency.
- Coagulation is dynamic; results should be less than 2 hours old or stable.
- Patient must be normothermic.
- There is no evidence to suggest which thromboelastometry values are safe for epidural insertion. An epidural or deep catheter should NOT be inserted if CT > 100 s, MCF (Ex)< 40 mm, or MCF (Fib) < 8 mm. If parameters are better than these values, clinical discretion must still be applied.
- Increased vigilance, including simple neurological observation and pain team review in accordance with standard procedures, is required after insertion of any epidural or CPNB catheter.
- Clear documentation of discussion and values should be appropriately recorded.

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Chapter 23

ACUTE PRESENTATIONS OF CHRONIC PAIN CONDITIONS

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INTRODUCTION

It is often assumed that battle-related injuries are the leading cause of hospitalization and medical evacuation during combat operations. In fact, as long as accurate figures have been maintained, injuries caused in combat have never been the leading reason for soldier attrition. In World War I, respiratory illness and infections were the most common reason for removal from the battlefield, with combat injuries in third place, and nonbattle injuries (NBIs) ranking fourth. In subsequent conflicts this order changed, and by the Vietnam War NBIs had become the leading cause of loss of personnel from the combat arena, which has continued to be the case into the present time.[1,2]

Among NBIs, the conditions associated with the lowest return-to-duty (RTD) rates are psychiatric conditions, back pain, and other musculoskeletal conditions.[3] A striking feature these conditions have in common is that the farther away from the battlefield they are treated, the less probability there is of successful RTD. With back pain and other musculoskeletal pains, studies have suggested that earlier intervention and treatment, as close to the parent unit as possible, may be associated with an increased RTD rate.[4] For practical purposes, this chapter will deal mainly with common spinal and other musculoskeletal pains.

DEFINITION AND CLASSIFICATION OF PAIN

The International Association for the Study of Pain defines pain as an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage.[5] Pain is generally classified as acute or chronic. Acute pain is a normal response to a physiological insult and serves several important functions. It is a protective mechanism that helps us survive in hostile environments. It serves as a warning sign of imminent danger, and also causes an individual to nurse or rest the affected body part, thus allowing it time to heal. Acute pain is therefore a symptom of an event or disease state.

In contrast, chronic pain serves no such purpose and is not normally associated with ongoing damage; the original physiological response has changed into a nonfunctional pain signal. Acute pain therefore ceases to be a symptom of a disease, and instead becomes a "disease" itself. Chronologically, pain that lasts beyond the usual time necessary for an injury to heal is considered chronic, generally accepted be less than 3 months, although some authorities maintain that 4 to 6 weeks is a more appropriate cutoff between acute and chronic pain.

Etiologically, pain can be classified as either nociceptive or neuropathic, although in reality there is significant overlap between the categories, and many conditions such as spinal pain share characteristics of both. Nociceptive pain refers to the pain that arises from noxious stimuli, and may be somatic or visceral. It is the result of actual or potential tissue damage, and would accurately describe postoperative pain. Somatic pain arising from tissue damage tends to be well localized, and is transmitted via fast myelinated A-δ nerve fibers and slower unmyelinated C fibers. Visceral pain, as the name suggests, arises from internal organs and is generally poorly localized due to convergence. In contrast to neuropathic and somatic pain, it is more likely to be described as "dull, cramping, and deep."

Nociceptors are specific for a variety of noxious stimuli, and include thermal, mechanical, and chemical receptors. A-δ fiber discharge is linearly related to the intensity of the stimulus. The response threshold, and the rate of firing to secondary-order neurons in the dorsal horn, allow afferent signals to be encoded in the central nervous system for processing. Wide dynamic range nociceptors respond to a continuum of stimuli

TABLE 23-1

CLINICAL FEATURES OF NEUROPATHIC PAIN

Clinical Feature	Presentation
Allodynia	Pain caused by stimulus that is not normally painful, eg, light touch, cold
Hyperalgesia	An exaggerated pain response to a normally painful stimulus
Dysesthesia	Altered sensations, eg, sensation of something crawling on the skin (formication)
Hyperpathia	Pain that may occur due to repeated innocuous or noxious stimuli, and which may even be present with sensory impairment
Pain quality	Often described as shooting, burning, or lancinating
Sensation	Frequently accompanied by sensory loss in the distribution of a dermatome or peripheral nerve
Temporal nature	May be paroxysmal or continuous

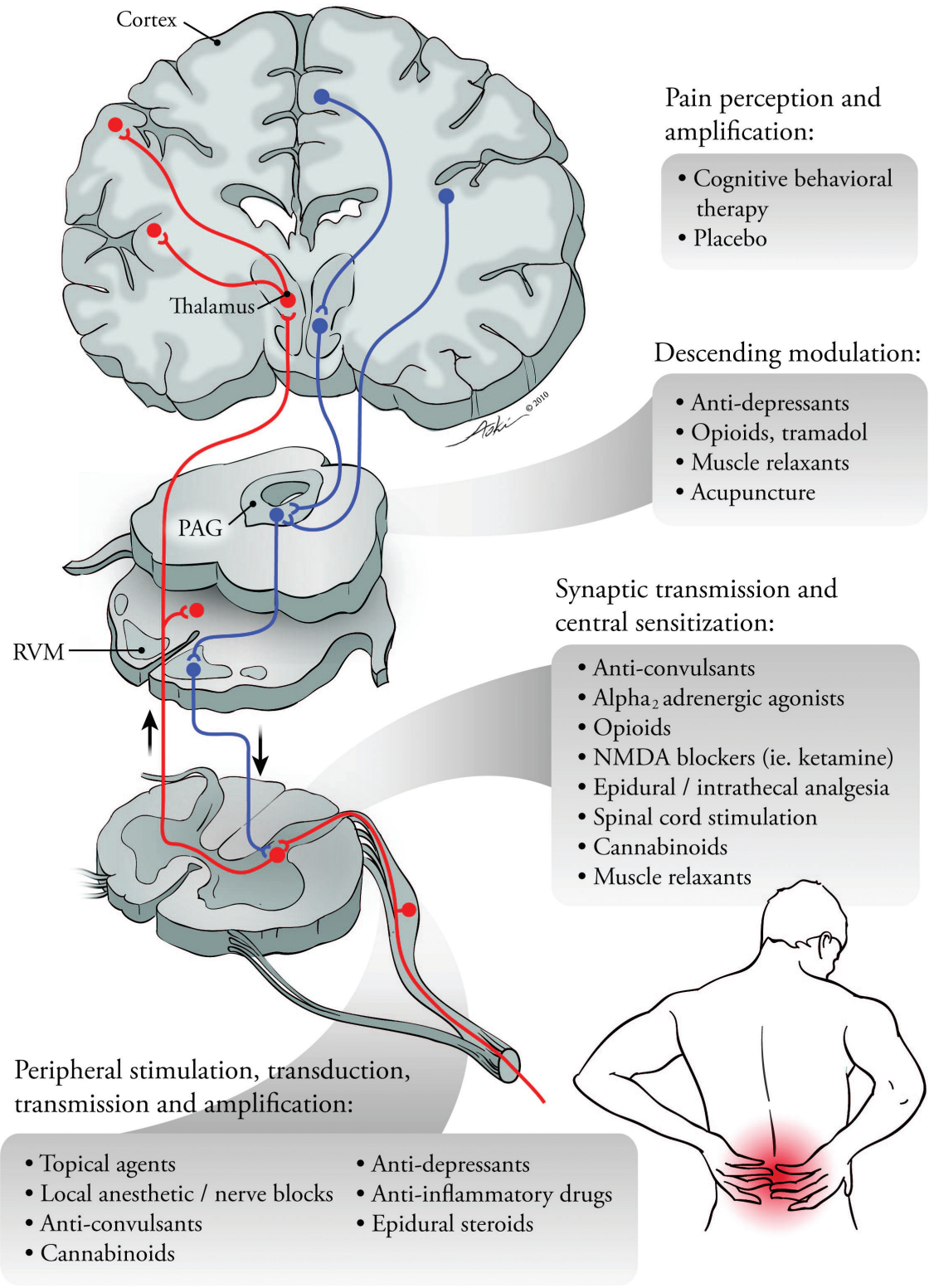

Figure 23-1. Ascending pain pathways (red) and descending modulation (blue), illustrating the sites of actions for various analgesic agents.
NMDA: *N*-methyl D-aspartate; PAG: periaqueductal grayplease; RVM: rostroventral medullaplease
Reproduced with permission from Cohen SP, Raja SN. Pain. In: Goldman L, Schafer AI, eds. *Cecil Textbook of Medicine*. 24th ed. Philadelphia, PA: Saunders; 2011:133–139.

ranging from gentle warmth to tissue-damaging heat. With exposure to any noxious stimulus, a variety of nociceptors are stimulated in various degrees, and their output is summated to produce the subjective pain experience, which includes descending modulation and cognitive, emotional, and psychological input (Figure 23-1).

Neuropathic pain arises from damaged nervous tissue. The injury may occur anywhere along the nociceptive pathway (eg, the pain receptors, peripheral nerves, spinal cord, or central regions of the brain). Neuropathic pain may result from a variety of pathological conditions, including inflammation, trauma, ischemia, and degenerative processes, and persists even in the absence of ongoing disease or physiological insult (eg, diabetic nephropathy). Neuropathic pain is frequently subdivided into peripheral neuropathic pain (eg, diabetic neuropathy) and central pain (eg, phantom pain or spinal cord injury). Table 23-1 lists clinical features of neuropathic pain.

BACK PAIN

In the civilian population, it is estimated that about 10% of consultations with primary care practitioners are related to musculoskeletal pain. The majority of these consultations involve back or spine pain.[6] As one may expect, this problem is also common in the military population, and during training exercises and active deployments, the incidence of back pain rises even higher. About 75% of the consultations for spine pain in the military setting involve low back pain, with the remainder involving neck and mid-back pain problems.

In the civilian population, the strongest predictors for persistent back pain after an acute episode relate to lifestyle (eg, heavy physical activity, sedentary lifestyle, obesity, smoking); profession (eg, low job satisfaction); and psychosocial issues (eg, depression, anxiety, fear-avoidance behavior, poor coping skills, catastrophization [believing something is far worse than it is]). These factors are also present in military personnel and can be exacerbated by military-specific risk factors, such as inadequate support structures, lack of autonomy, concomitant psychological trauma, heavy combat loads, job-related sleep deprivation, austere living conditions, and high-impact landings during airborne, air-assault, and dismounted ground operations.

The management of low back pain accounts for a major part of the practice for military pain physicians. It is estimated that between 50% and 80% of the adult population will experience significant back pain at some time in their life. A significant proportion of these episodes are self-limiting, although recent studies have suggested that over one-third of these individuals may continue to experience pain for up to 12 months and longer from their first presentation, despite a return to their previous work status and function.[7]

Among those who seek medical advice, the majority will not receive a definitive diagnosis. This situation demonstrates that back pain is a symptom rather than a diagnosis. Even in those individuals who go on to receive further work-up and treatment, it is often difficult to correlate symptoms with pathology. For low back, mid-back, and neck pain, multiple studies have demonstrated a high rate of abnormal radiological findings in asymptomatic individuals.[8–10] Military physicians should therefore focus less on identifying a precise cause, which is often not possible, and more on returning an individual to maximal functional capacity, which will reduce the impact on unit effectiveness.

Etiology of Back Pain

There is often a very poor correlation between the actual process of the disease, the signs and symptoms, and the investigations carried out. This is frustrating for both practitioner and patient; the latter may be overly focused on obtaining a precise diagnosis. Within the spine there are numerous structures that can give rise to back pain. For the pain physician, it is much more important to identify or rule out a small number of specific conditions that, if missed, might prove catastrophic than to try pinpointing one particular structure as the principal cause of chronic pain. The potentially catastrophic conditions make up only a small proportion of back pain cases; however, it is vitally important that they are recognized and treated quickly. Their signs and symptoms should be considered red flags (Table 23-2).

Nonspecific or Mechanical Back Pain

Nonspecific or mechanical back pain is more of a description than a diagnosis, and generally implies pain arising from the posterior elements of the spine. Mechanical back pain typically involves the lower lumbar region, but may also be referred into the groin or posterolateral thighs. It is usually confined to above the knee. Although many structures have been suspected of causing this type of pain, the muscles and ligaments are perhaps the most commonly implicated. Multiple studies have demonstrated increased electromyographic activity in low back pain sufferers irrespective

TABLE 23-2

WHAT NOT TO MISS: RED FLAGS SUGGESTING SERIOUS UNDERLYING PATHOLOGY OR NERVE ROOT PATHOLOGY

Red Flag	Possible Underlying Conditions	Individuals at Increased Risk	Associated Signs and Symptoms
Age > 50 years	Metastases, vertebral fractures, herpes zoster, or life-threatening conditions such as aortic rupture or perforated bowel	*Malignancy*: positive family or previous cancer history, positive smoking history, unremitting pain not relieved by recumbency *Zoster*: risk of acute infection and postherpetic neuralgia exponentially increase with age *Vertebral fracture:* h/o fall or other trauma *Abdominal pathology (aortic aneurysm)*: h/o smoking, hypertension, vasculitis, abdominal trauma, positive family history; prior surgery (ruptured bowel)	*Malignancy*: unexplained weight loss, unremitting pain not relieved by recumbency *Zoster*: history of rash *Abdominal pathology*: concomitant abdominal discomfort, peritoneal signs, nausea, and vomiting
Age < 20	Congenital anomalies (eg, spina bifida); early-onset disorders (eg, Scheuermann's disease); conditions associated with substance abuse (ie, osteomyelitis)	*Congenital disorders*: neurological symptoms, positive family history, other congenital abnormalities, systemic disease (eg, diabetes, epilepsy for spina bifida *Substance abuse*: males, depression or other psychiatric condition, poor school or work performance	*Congenital anomalies:* birth marks, overlying skin tags, patches of hair
Trauma	Vertebral fractures, sacroiliac joint pain	*Vertebral factors:* old age, gait abnormalities, osteoporosis, female gender, previous fractures, corticosteroid use, Asian and Caucasian race	Fractures, ecchymoses, peritoneal signs
Systemic illness	Vertebral fractures, spinal infections, metastases	*Spinal infections:* recent infections, intravenous drug abuse, immunosuppression, recent spinal procedures, diabetes, older age	*Spinal infections*: malaise, fever, chills, tenderness, leukocytosis, local signs of infection, elevated ESR
Constitutional symptoms	Metastases, spinal infections	*Spinal metastases:* patient with breast, lung, prostate, or thyroid cancer	See *Spinal infections*, above. Signs of discitis may be subtle; signs of meningitis may be fulminant and include meningeal signs
Immunosuppression or steroid use	May predispose patients to infectious process, malignancy, or vertebral fractures	Patients with prolonged corticosteroid or immunosuppressive drug use (eg, transplant recipients, autoimmune disease). Most common locations for vertebral fractures are mid-thoracic, thoracolumbar junction, and lower lumbar regions	*Vertebral fracture:* focal tenderness, sudden onset, pain worsened by any movement and relieved by lying on back, height loss and deformity
Widespread neurological symptoms	Cauda equina syndrome, myelopathy, multiple sclerosis	Patients with large disc herniations, recent (< 48 hours) spinal procedures, traumatic injury, malignant or benign spinal tumors, spinal stenosis, and inflammatory conditions (eg, ankylosing spondylitis and Paget's disease)	Marked motor and sensory deficits involving multiple nerve roots, gait disturbances, overflow incontinence, saddle anesthesia, and diminished reflexes and sphincter tone
Unrelenting pain	Psychogenic pain/somatoform disorder, malingering, malignancy, life-threatening abdominal pathology	*Psychogenic pain:* h/o depression, anxiety, psychosocial stressors, multiple somatic complaints, drug or alcohol problems	*Psychogenic pain:* Signs of nonorganic pathology (ie, Waddell's signs), changes in appetite or sleep habits, difficulty concentrating and irritability, irrational fears, panic attacks

ESR: erythrocyte sedimentation rate; h/o: history of
Adapted from: Cohen SP, Argoff CE, Carragee EJ. Management of low back pain. *BMJ.* 2008; 337: a2718.

of the etiology, and clinical trials have demonstrated efficacy for muscle relaxants and botulinum toxin in back pain patients.[11–15] Additional evidence for the role of muscular pathology as a contributor to low back pain comes in from numerous studies demonstrating the effectiveness of neuromuscular reeducation and lumbar stabilization.[16]

Significantly, there are generally no signs of nerve root dysfunction in nonspecific back pain. When referred pain is present, it is usually in a non-dermatomal distribution. Despite the numerous tests that have been advocated for low back pain, no single feature in the history or physical examination can reliably identify a particular structure as the primary source of pain.[17] Other causes of nonspecific back pain can include facet joint pain, myofascial pain, sacroiliac (SI) joint pain, and bony pathology (Table 23-3).

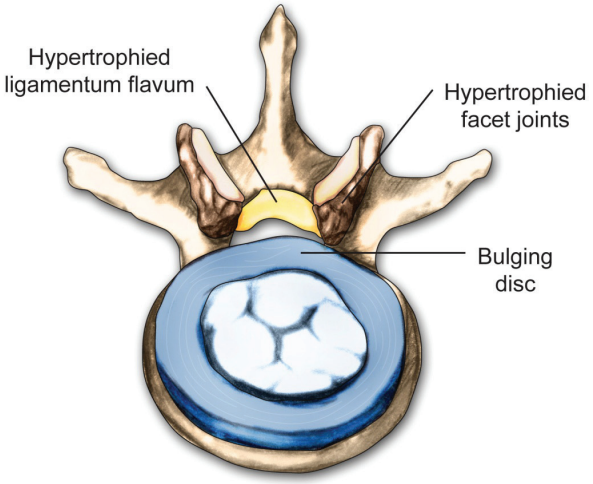

Figure 23-2. Axial view of a vertebral body demonstrating spinal stenosis secondary to hypertrophied facet joints and ligamentum flavum, and bulging discs.
Reproduced with permission from: BMJ Publishing Group Limited [Cohen SP, Argoff CE, Carragee EJ. Management of low back pain. *BMJ*. 2008;337:a2718.]

Facet (Zygopaphyseal) Joint Pain

The facet joints are true synovial joints containing a joint space, cartilaginous surfaces, a synovial membrane, and a fibrous capsule. The capsule is richly innervated, such that any disruption is a potential source of pain. Similar to other synovial joints, the facet joints are vulnerable to the inflammatory and degenerative changes seen with both rheumatoid arthritis and osteoarthritis. The lumbar zygapophyseal joints typically bear between 3% and 25% of the axial load; this burden increases with disc degeneration and facet joint hypertrophy (Figure 23-2).[18] Depending on the particular spinal level, lateral and forward flexion can significantly increase the stress on the joints (Table 23-4).[19] In view of their large load carriages and the repetitive strain associated with military training, service members are at increased risk of developing facetogenic back pain.

Patients with facet-mediated pain typically present with localized pain and tenderness. One study found that paraspinal tenderness to be a strong predictor of successful lumbar facet radiofrequency denervation.[20] Symptoms typically worsen with lumbar motion and load carriage. The pain may radiate to the posterolateral thigh, especially when stress is applied to the facet joints. However, no symptom or provocative maneuver is pathognomonic. Most studies have demonstrated that imaging poorly correlates with symptoms. Plain films and commuted tomography scans may show hypertrophic joints, erosion of endplates, and nonspecific acute and chronic inflammatory changes. There is a general consensus that fluoroscopically guided, low-volume facet joint or medial branch blocks are the most reliable means to identify a zygapophyseal joint as the pain generator. Whereas these injections are associated with a high false-positive rate,[21,22] a recent randomized comparative cost-effectiveness study that included active duty service members demonstrated that utilizing multiple blocks in an effort to reduce the false-positive rate will lower the overall success rate for treatment.[23] Controlled studies have shown that between 50% and 67% of carefully selected individuals will obtain intermediate-term relief from facet joint radiofrequency denervation.[24,25]

Musculature of the Back and Myofascial Pain

The significance of the paraspinal musculature as primary pain generators has not been well elucidated. What is known is that the muscles contain a significant population of A-δ and C fibers (see explanation above), and represent by far the largest surface area in the lumbar region. A-δ and C fibers serve a nociceptive function, and may therefore play an etiological role under stressful conditions. On examination of some patients, it may be possible to identify areas of muscular spasm and occasionally discrete trigger points that respond to targeted injections. In some cases targeted injections may produce dramatic reduction in pain symptoms.

Sacroiliac Joint Pain

The mechanism of injury in SI joint pain is often described as a combination of axial loading and abrupt rotation. Unlike pain from internal disc disruption

TABLE 23-3

FEATURES DISTINGUISHING DIFFERENT CAUSES OF SPINE PAIN

Condition	History	Physical Examination	Diagnosis in Theater	Treatment in Theater
Myofascial pain	Focal neck or back pain, usually after inciting event	Nonfocal neurological exam. Spasm or swelling may be noted	History and physical examination	More than 80% of cases improve spontaneously. Ice and heat may be helpful. Resume activities as soon as tolerated. NSAIDs and muscle relaxants beneficial in short term
Radiculopathy from herniated disc	Usually unilateral pain extending to distal extremity, often in dermatomal distribution. Sensory and motor changes common	Straight leg raising and Spurling's test are sensitive but not specific. Sensory and motor deficits may be present. Reflexes may be impaired	History and physical exam more than two-thirds accurate. CT scan 90% sensitive in detecting disc pathology	Natural course is improvement and recurrence. Epidural steroids may hasten recovery. Weak treatment effect for adjuvants (eg, anticonvulsants and antidepressants). PT and exercise are beneficial
Facet joint arthropathy	Usually symmetrical pain extending to the proximal extremity, head, or groin	No reliable physical signs. Normal neurological exam. Paraspinal tenderness often present	Diagnosis made by local anesthetic blocks. High false-positive rate	Intraarticular injections are effective in only a small percent of patients with acute inflammation. Radio-frequency denervation may provide intermediate-term relief in carefully selected patients. PT and exercise are beneficial. Small effect size for NSAIDs and antidepressants
Discogenic pain from degenerative disc disease	Usually symmetrical pain radiating into proximal extremity, head, or groin. Lumbar pain aggravated by sitting	Pain worsened by forward flexion. Midline tenderness often present. Normal neurological exam	CT scan has poor specificity. Discography not indicated in theater	PT and exercise are beneficial. Small effect size for NSAIDs and antidepressants
Sacroiliac joint pain	Often unilateral pain that frequently occurs after trauma or surgery. Often extends into upper leg, groin, and occasionally lower leg	Tenderness overlying SI joint usually present. Single provocative tests unreliable. Normal neurological exam	Diagnosis made by LA blocks, which have high false-positive rate. Battery of provocative tests have moderate sensitivity and specificity	SI joint blocks may provide short-term relief. Radiofrequency denervation may result in intermediate-term relief in select patients with extraarticular pathology. PT and exercise are especially beneficial. Small effect size for NSAIDs and antidepressants

CT: commuted tomography; LA: local anesthesia; NSAID: nonsteroidal antiinflammatory drug; PT: physical therapy; SI: sacroiliac

TABLE 23-4

MOTIONS ASSOCIATED WITH THE LARGEST INTERVERTEBRAL ANGULATION AND STRAIN FOR THE LUMBAR FACET JOINTS

Facet Joint Level	Movement Associated With Maximal Intervertebral Angle	Largest Strain
L1–2	Right bending	Right bending
L2–3	Left bending	Right bending
L3–4	Right bending	Right bending
L4–5	Forward flexion	Forward flexion
L5–S1	Extension	Forward flexion

Adapted with permission from: Ianuzzi A, Little JS, Chiu JB, Baitner A, Kawchuk G, Khalso PS. Human lumbar facet joint capsule strains: I. During physiological motions. *Spine J*. 2004;4:141–152.

(ie, discogenic pain) and facetogenic pain, which are typically insidious in onset, a specific inciting event can be identified in between 40% and 50% of cases of SI joint pain, most commonly motor vehicle accidents, falls, and repetitive strain from sports.[26–28] In the military population, airborne-related parachute landings and repetitive stress from physical training render service members at increased risk for developing SI joint pain.

SI joint pain is a heterogeneous condition, and can be classified as either intraarticular or extraarticular. Not surprisingly, intraarticular pathology is more likely to occur symmetrically in the elderly, while younger individuals are more likely to present with unilateral extraarticular SI joint-mediated pain. SI joint pain is frequently associated with other musculoskeletal conditions such as trochanteric bursitis and facet joint pain.

Similar to zygapophyseal joint pain, no isolated symptom or physical exam sign can distinguish a painful SI joint from other potential pain generators, although some systematic reviews have found that utilizing a battery of provocative tests[29,30] can accurately identify most cases. In a study by Slipman et al,[31] the authors found that in 50% of patients with SI joint pain, the pain was referred into the lower extremity, and in 28% of cases it extended into the distal leg.

The reference standard for making a diagnosis of SI joint pain is via a low-volume injection, which is also associated with a high false-positive rate.[32] Both intraarticular and extraarticular injections with corticosteroids may provide at least short-term relief to patients with SI joint pain[33,34]; at least one study[35] determined that utilizing both approaches provides superior results. Radiograph guidance should always be used to perform SI joint procedures, because "blind" attempts miss the target in the large majority of cases.[36] In patients who obtain only temporary relief from corticosteroid injections, radiofrequency denervation has been shown in controlled studies to provide intermediate-term relief in a majority of individuals.[37] Because the lateral branches amenable to radiofrequency lesioning innervate extraarticular rather than intraarticular structures, young adults such as service members are especially likely to benefit from denervation.[38,39]

Back Pain and Disc Lesions

In addition to radicular symptoms resulting from prolapse, intervertebral discs can be sources of pain through degeneration. This pain is often referred to as discogenic pain, in contrast to radicular pain, which is the result of nerve root irritation. Discs are attached superiorly and inferiorly to the intervertebral body endplates. Anteriorly and posteriorly, they are attached to the longitudinal ligaments. The discs themselves are made up of a central gelatinous mass, the nucleus pulposus, surrounded by fibrocartilaginous annulus fibrosus. The annulus fibrosus is comprised of 10 to 20 concentric layers of collagen fibers called "lamellae," which pass obliquely between adjacent vertebral bodies and attach to the anterior and posterior ligaments.

As individuals age, there is inevitable loss of disc integrity. As the number of intact lamellae decreases as a result of repetitive strain or acute torsional events, the load borne remains the same, so that eventually the threshold for nociception is reached. Fractures in the endplates can also result in inflammatory cytokines leaking into the nucleus fibrosus. Degenerative discs contain more extensive and deeper nerve in-growth than normal discs. As annular tears develop, the cytokines may come into contact with nociceptors, resulting in chemical sensitization. Given the nature of military service, this process may be accelerated in service members.

The typical presentation of discogenic pain is axial pain, often referred into the lower extremity, which is exacerbated by sitting or forward flexion. In light of the high prevalence rate of degenerative disc disease in asymptomatic individuals, it can be extremely difficult to correlate symptoms with imaging results. Discography, although advocated as a means to identify painful intervertebral discs, is fraught with controversy regarding its prognostic value, high false-positive rate, and uncertainty about whether or not it injures discs.

Back Pain With Nerve Root Compromise

Back pain with nerve root compromise may occur from prolapsed intervertebral discs, degenerative bony lesions, and spinal stenosis. By far, herniated discs are the most common cause of neuropathic back pain in service members, with the peak incidence occurring during the 3rd and 4th decades of life. The two most frequently affected nerve roots are L5 and S1, reflecting the fact that the lowest lumbar disc is most likely to degenerate and herniate. In over one-third of cases, two or more nerve roots are involved. Younger individuals are also more likely than the elderly to note a specific inciting event.

Cauda equina syndrome, although rare, is critically important to identify. It is usually caused by a large, midline disc herniation that impinges upon the sacral nerve roots. This is the reason for the characteristic loss of bladder and bowel control, and absent or diminished perineal or saddle sensation. It may also be accompanied by sciatica, unilateral or bilateral depending on the type of disc herniation, and motor weakness or paralysis. Cauda equina syndrome represents a true surgical emergency and requires immediate referral and medical evacuation to an appropriate treatment facility.

If a normal nerve root is compressed, it may be accompanied by loss of function but is not normally painful. Some studies suggest that previous exposure to inflammatory cytokines is necessary for radicular pain.[40] However, with chronic compression the nerve root becomes inflamed and irritated, and pain can therefore occur. In addition to pain caused by chronic root compression, pain may also result from physical distortion of neighboring anatomical structures such as muscles, ligaments, and joint capsules.

When individual nerve roots are compressed, pain typically occurs in a dermatomal distribution, though there is significant dermatomal overlap and over a third of cases of radiculopathy involve multiple nerve roots. Because the amount of force necessary to herniate a disc varies inversely with the degree of disc degeneration, radicular pain usually involves the back as well as the distal parts of the lower limbs. Systematic reviews have determined that the straight leg raising test is about 85% sensitive and 52% specific in detecting lower lumbosacral radiculopathy.[41] For spinal stenosis, the test's sensitivity is less; for upper lumbar herniated discs, the femoral stretch test may be useful in distinguishing radicular pain from referred mechanical back pain. Whereas acute radiculopathy secondary to a herniated disc will usually resolve spontaneously as the disc retracts, patients often experience recurrences of symptoms.

In individuals with radiculopathy, epidural steroid injections may provide at least short-term benefit, and can be repeated in a series of shots when pain recurs. The transforaminal approach, which directly deposits the injectate over the affected nerve roots and into the ventral epidural space, may be more effective than a conventional interlaminar approach.[42]

Spinal Stenosis

Spinal stenosis can occur in the central part of the spinal canal, the lateral recesses, or the intervertebral foramen. There are many possible causes of spinal stenosis, including disc protrusions, ligamentous hypertrophy, facet joint arthritis, spondylolisthesis, and congenitally short pedicles. Because most of these processes involve chronic degenerative changes, spinal stenosis is much more common in the elderly, and tends to be more chronic and progressive than radicular pain secondary to a herniated disc.

Central stenosis commonly presents as pain in the lower back extending into the lower legs. Extension of the spine can exacerbate the discomfort, while flexion may ease the symptoms. A common observation is that patients find it easier walking uphill or upstairs than down. Lateral recess and foraminal stenosis tend to include pain and discomfort in a radicular distribution, and may or may not be associated with sensory changes or motor dysfunction. Although epidural steroid injections can provide significant relief to patients with spinal stenosis, the duration of benefit tends to be shorter than in individuals whose pain is from a herniated disc.

NECK PAIN

Approximately two-thirds of individuals will experience neck pain throughout their lives. The annual prevalence rate is about 40%, and it occurs somewhat more frequently in females.[43,44] As with back pain, cervical spine pain is often multifactorial in nature, and may be due to problems with bony structures such as the facet joints, intervertebral discs, soft-tissue pathology, and nerve root or spinal cord compression. Post-traumatic neck pain is common, particularly following motor vehicle accidents. As with back pain, neck pain may be the presenting feature of serious underlying systemic disease, which should be excluded by history, examination, and imaging.

Many predisposing factors render military personnel at increased risk for neck pain, and many of them are similar to the factors predisposing individuals to

back pain. Common inciting factors include heavy load carrying (including the burden of combat body armor), abnormal postures, work-related stress, transport in military vehicles with hard suspensions over unpaved roads, and many others.

Cervical Radiculopathy

Cervical radiculopathy results from compression of nerve roots due to degenerative disease or disc protrusion. Symptoms may include neck and arm pain, most commonly unilateral; sensory loss; weakness; and possibly diminished reflexes. Disc prolapse is most common at C5–6 and C6–7. Young, physically active individuals such as military personnel are more likely to suffer an acute onset of symptoms (most likely disc prolapse), whereas in the civilian population the onset is more likely to be gradual. The treatment of cervical radiculopathy is similar to that for lumbar radiculopathy, except that the transforaminal approach to epidural steroid delivery is rarely used due to the higher risk of paraplegia and death with particulate steroids.[45]

Cervical Myelopathy

Similar to radiculopathy, spondylotic myelopathy may occur as a result of disc herniation or bony overgrowth. Cervical spondylotic myelopathy is the most common cause of spinal cord dysfunction in older persons. The aging process results in degenerative changes in the cervical spine, which in advanced stages can cause compression of the spinal cord. Symptoms often develop insidiously and are characterized by neck stiffness; arm pain; numbness, tingling, and weakness in the hands; and clonus. In addition, other features such as bladder dysfunction and gait disturbances may be observed. On physical exam, clonus, hyperreflexia, and other signs of upper motor neuron lesions such as Hoffmann's and Babinski's signs may be present. The differential diagnosis includes other conditions that can result in myelopathy such as multiple sclerosis, amyotrophic lateral sclerosis, and tumors that impinge on the spinal cord. The diagnosis is confirmed by a magnetic resonance imagery scan. Myelopathy is generally progressive in nature, and will not generally resolve spontaneously. Patients with ongoing and progressive disease should be referred for urgent decompressive surgery.

Occipital Neuralgia

Occipital neuralgia is a frequent cause of occipital headaches. It usually describes recurrent pain in the upper neck and occipital region, within the distribution of the greater and lesser occipital nerves. These nerves are derived from the posteriors C2 and C3 nerve roots.

Occipital neuralgia is unilateral in 85% of patients, with the greater occipital nerve being involved more frequently (90%) than the lesser occipital nerve (10%). In approximately 10% of cases both branches are involved.[46] Patients with occipital neuralgia typically describe a unilateral pain characterized by piercing, throbbing, or "electric-shock–like" sensations in the upper neck, back of the head, and behind the ears. Often, the pain begins in the neck and spreads upward. Some individuals experience pain in the scalp, forehead, and behind the eyes. There is usually tenderness overlying the trunk or course of the nerve, which can elicit pain in the nerve distribution. The cause of occipital neuralgia may be irritation or injury to the nerves as a result of overly tight neck or scalp muscles causing compression of the nerve. Some studies suggest trauma to be a common precedent.[47] In one epidemiological study evaluating service members evacuated from Operations Iraqi and Enduring Freedom for headache, 5% had a primary diagnosis of occipital neuralgia, with 46% of these individuals citing physical trauma as the precipitating event.[48] Frequent lengthy periods of keeping the head in a forward flexed position may contribute to occipital neuralgia; however, in most cases no specific cause can be found. The diagnosis of occipital neuralgia is confirmed by nerve block, which in some cases can provide long-standing benefit when corticosteroids are added. In those individuals who fail to obtain sustained benefit, pulsed radiofrequency may provide long-term relief.[49,50]

Whiplash Injuries of the Neck

The most common cause of chronic neck pain is whiplash injury. Whiplash is commonly associated with acceleration–deceleration injuries, which force the neck into hyperextension and flexion, then rebound. A common scenario is a motor vehicle accident where an affected individual undergoes a rear-end impact. One of the earliest studies on whiplash was performed by Severy et al,[51] who demonstrated the importance of phasing differences during acceleration and deceleration between the vehicle and human volunteers subjected to rear-end collisions. The peak acceleration of the vehicle preceded that of the torso, which in turn preceded that of the neck and head. This established that a critical element of whiplash involved inertial loading of the neck, as the torso abruptly moved forward under an initially stationary

head.[51] A review by Bogduk and Yoganandan[52] concluded that in whiplash injuries, instead of the facet joint articular processes gliding across one another, the inferior articular processes of the moving vertebrae chisel into the superior articular processes, resulting in microscopic injury.

The role of the cervical zygapophyseal joints is supported by prevalence studies suggesting that the prevalence of facetogenic pain in individuals with chronic neck pain after whiplash injuries is around 50%.[53,54] If the head is not in the neutral anatomical position during impact, injuries can also occur in the rotational and/or lateral flexion planes. Other structures that may contribute to neck pain after trauma include muscles, ligaments, discs, and the atlanto-axial and atlanto-occipital joints. In one cadaveric study involving rear-end impacts without head rests, injuries to the cervical intervertebral discs were found in 90% of cases, tears of the anterior longitudinal ligament in 80%, tears in the cervical zygapophyseal joint capsules in 40%, and vertebral body fractures in 30%. In the cadavers protected by head rests, no injuries were found.[55]

Early symptoms after whiplash injuries include neck and shoulder stiffness, and occipital pain. There may be localized tenderness on palpation, and reduced range of movement in the cervical spine. Many people complain of headaches. Neurological symptoms are rare and if present may indicate more extensive damage. Imaging is likely to be of little use in the vast majority of cases; with the natural course most people will recover with conservative measures such as nonsteroidal antiinflammatory drugs and physiotherapy. About 10% of individuals develop persistent symptoms. In some of these cases, emotional and psychological distress can be disproportionate to pathology.

TREATMENT OPTIONS FOR BACK AND NECK PAIN

Medical officers in the field do not have the full range of treatment modalities available to the civilian practitioner; however, a number of therapeutic options are available (Tables 23-5 and 23-6). Similar to civilian practice and treatment in garrison, treatment options in theaters of operation will ideally utilize a multimodal approach, albeit with certain considerations. Military pain specialists are primarily deployed as anesthesiologists or physiatrists, so pain management is a secondary mission. Thus at any given time, experienced physicians may or may not be available to provide the full range of interventional techniques described below. The end result is that patients may be seen and treated only if sufficient expertise, time, space, and equipment are available. Future leaders in military medicine should strongly consider recognizing pain medicine as a separate subspecialty so that the availability of interventional pain treatment services is not contingent on the presence of anesthesiologists, physiatrists, or neurologists who may or may not have received adequate specialty training. In the interim, primary care physicians should be capable of triaging pain patients to prioritize treatment for those with a reasonable likelihood of remaining in theater with proper therapy, so as not to unnecessarily overburden already strained resources.

When considering analgesic medications, the same classes of medications used in civilian practice are available to the medical officer, although the choices within those classes may be limited. Simple analgesics such as paracetamol (acetaminophen), along with nonsteroidal antiinflammatories, form the foundation of analgesic treatments. As pain requirements increase, the medical officer may consider the use of opioids, starting with weaker preparations such as tramadol or codeine, and progressing to stronger drugs such as morphine. Most pain physicians believe that opioids are a reasonable treatment for some patients with acute

TABLE 23-5

PAIN TREATMENTS COMMONLY AVAILABLE TO MEDICAL OFFICERS*

Treatment	Examples
Physical therapies	Graded exercise, iontophoresis
Complementary and alternative therapies	Acupuncture, spinal manipulation
Neuromodulation	TENS, spinal cord stimulation†
Injection therapy	Epidural steroid injections, facet joint injections, radiofrequency lesioning, regional anesthesia
Pharmacological interventions	Analgesia based on WHO analgesic ladder
Psychological interventions	Cognitive-behavioral therapy

*Availability depends on the facility at which an individual is treated. Injection therapies are not suited to truly austere combat areas and should be carried out only in appropriate clinical settings.
†Available in garrison only.
TENS: transcutaneous electrical nerve stimulation
WHO: World Health Organization

TABLE 23-6

INTERVENTIONAL PROCEDURES FOR PAIN AVAILABLE IN THEATER IN SUITABLE ENVIRONMENTS

Technique	Injectate Volume*	Fluoroscopy	Comment
Cervical ESI	2–4 mL	Yes	Risk of permanent injury or death, especially with transforaminal approach. Use of local anesthetic controversial
Interlaminar lumbar ESI	3–5 mL	Strongly advised	Fluoroscopy associated with increased likelihood of injectate in target area
TFESI	2–3 mL	Yes	Superior outcome compared to the interlaminar approach
Facet joint injection	Cervical 1 Lumbar 1-2	Yes	Good outcome only in carefully selected patients with acute symptoms
Facet joint RF denervation	0.5–1 mL before lesioning	Yes	Moderate evidence for relief lasting > 6 months
Greater and lesser occipital nerve blocks	2–4 mL	No	Can be difficult to distinguish from referred cervical pain
SI joint injection	2–4 mL	Yes	Greater likelihood of placing injectate in target area with fluoroscopy
SI joint RF denervation	0.5–1 mL before lesioning	Yes	Targeted levels include L5–S3 and sometimes L4 and S4. More effective for extraarticular pathology
Piriformis injection	2–8 mL	Yes	Presentation may be similar to radicular pain, although straight leg raising test is likely to be negative. Injection of local anesthetic may lead to sciatic nerve weakness

*Injectate volume is the total volume and usually comprises a mixture of long-acting (depot) corticosteroid and local anesthetic.
ESI: epidural steroid injection; RF: radiofrequency; SI: sacroiliac; TFESI: transforaminal ESI

spinal pain episodes. However, long-term use should be balanced against the proven adverse effects of these drugs, such as impaired cognition and reduced reaction time, attention, balance, and memory, especially in the period following initiation of treatment. Individuals on long-term opioids require close clinical supervision, as do those individuals who fit into the demographic and clinical profile of young, combat-hardened service personnel, who may be suffering from comorbid physical and psychological illnesses that predispose them to an increased risk of misuse and diversion. Studies suggest that younger individuals such as service members may develop tolerance at faster rates than the elderly.[56]

Other drug treatments include the use of tricyclic antidepressants and anticonvulsants such as gabapentin and pregabalin. In very carefully selected patients with clear-cut neuropathic pain, the number needed-to-treat for one patient to obtain clinically meaningful benefit with first-line agents (eg, nortriptyline, gabapentin) tends to range between 2.5 and 4. For spinal pain, the likelihood of success is generally acknowledged to be significantly lower. Currently, only duloxetine, a serotonin-norepinephrine reuptake inhibitor, is the only drug approved for spinal pain. Large metaanalyses have failed to produce strong evidence in favor of one particular group of drugs over another.

SUMMARY

As the nature of combat evolves, the prevention and treatment of NBIs comprise an increasingly important role for medical officers. Although most of these conditions are similar to those encountered in civilian practice, the considerations and implications differ. Differences between treatment in theater and in garrison include limited resources in the former, the subordination of pain medicine to more emergent endeavors (ie, stabilization of combat-wounded personnel), prioritizing treatment outcomes (ie, RTD) over diagnostic specificity, and the need for the rapid realization of treatment

results, which often results in multiple concurrent interventions. In order to optimize treatment outcomes, medial officers in forward-deployed areas should be able to distinguish between patients who may benefit from pain medicine specialty referral, and those who can be effectively treated with conservative measures not requiring evacuation to a level-3 treatment center.

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Chapter 24

THE DEPLOYED PAIN SERVICE

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INTRODUCTION

Pain, the oldest physical affliction of humankind, is an unavoidable consequence of battlefield trauma. Recent advances in pain management have led to a greater understanding of the pathophysiology of pain, yet there is still no panacea for its management. Without the appropriate management of pain, an individual may suffer considerable and unnecessary physical and psychological distress, which may result in deleterious effects upon wound healing, rehabilitation, and mental state. Inadequate analgesia is also associated with a higher incidence of chronic pain complications.[1]

The nature of trauma experienced in the military operational environment may result in severe and varied types of pain. These casualties will benefit from the involvement of advanced pain management techniques and services. It is vital therefore that in addition to simple analgesia, the plethora of sophisticated pain management options available in nondeployed hospitals (Role 4) are not only accessible on deployment, but are also appropriately and effectively implemented.

Safe utilization of advanced analgesic techniques requires a structure enabling continuing medical education, assessment of efficacy, reporting of adverse events or near misses, implementation of guidelines issued by professional bodies,[2] and continuous service development. The acute pain service (APS) team has responsibility for the conduct of this wide range of tasks.

GOVERNANCE

The deployed clinical team has a responsibility to their patients, but at the end of their deployment responsibility is transferred. Short deployments may lead either to "re-invention," with lack of corporate knowledge, or personnel may resist development of services because they are unlikely to see positive outcomes during their tenure. For these reasons the deployed APS is governed external to the deployed environment, by a specialist interest group (SIG) or team who are able to provide a consistent education package, consult subject matter experts for advice, and develop the deployed service over the medium to long term. This structure provides the necessary governance in which the service can develop to the highest standards. The different roles and responsibilities between the deployed APS and the SIG are listed in Exhibit 24-1.

DEPLOYED STRUCTURE: THE MULTIDISCIPLINARY TEAM

Often pain, and its causes and effects, are complex in nature and require a variety of specialists' input for effective treatment; the effective management of pain requires a multidisciplinary approach. At minimum, the administration of effective analgesia requires a licensed prescriber or practitioner and the ability to

EXHIBIT 24-1

RESPONSIBILITIES OF THE SPECIALIST INTEREST GROUP AND THE DEPLOYED ACUTE PAIN SERVICE

Specialist Interest Group

- Develop and maintain an effective standard operating instruction for the deployed pain service.
- Develop the deployed acute pain service in the medium to long term.
- Education: take a lead role in enabling effective predeployment training.
- Audit/research: provide oversight and facilitation as required.
- Subject matter experts: provide advice and assistance as required.

Deployed Acute Pain Service

- Implement an effective pain management strategy.
- Deliver clinical acute pain service.
- Provide ongoing education to deployed personnel.
- Audit key performance indicators in pain management.
- Identify areas for service improvement to the special interest group.

supply, dispense, administer, and monitor the effects of therapy. A deployed APS, however, requires broader representation to fulfil its wider responsibilities, including education, audit, service development, and research, in addition to the fundamental role of pain assessment and management.

The composition of the multidisciplinary team may be influenced by the size of the deployed footprint and its capability, but broadly should include the following elements:

- **Lead clinician.** Often a specialist in anesthesia (the management of acute pain being within the remit of all anesthetic practitioners). Familiarity with advanced pain management techniques and an appropriate skill set, including management skills, equips the anesthesiologist to perform this role.
- **Pain nurse.** A dedicated member of the nursing cadre with advanced training in pain management techniques. The pain nurse is an essential component of the pain team, effective in providing a link between nursing staff and clinicians. The pain nurse role involves direct patient care with assessment of patients and pain management, as well as education, the conduct of audit, and liaison with all members of the pain team.
- **Ward nurse/pain management champion.** Each hospital ward should identify a nurse pain management "champion" to serve as the ward liaison to the APS and the primary resource for ward nurse pain education and support. The ward nurse pain champions are a valuable resource for outcomes feedback on APS pain management programs.
- **Pharmacist.** An integral member of the pain team, responsible for supplying and dispensing medications. Clinical care includes reviewing prescriptions and providing advice and information to the clinical team.
- **Physiotherapist.** The role of the physiotherapist is two-fold: (1) Functional mobility is associated with reduced postoperative complications, and the physiotherapist has a role in assessing pain during therapy, ensuring pain is managed effectively to allow compliance with treatment. (2) Additionally, specialist techniques such as transcutaneous nerve stimulation, acupuncture, and massage may be accessible through deployed physiotherapy. While such techniques may not be appropriate to major injuries from battlefield trauma, the provision of these type of services may impact upon force generation, assisting in retaining soldiers in theater who otherwise might be aeromedically evacuated for further care.
- **Deployed aeromedical team.** Evacuation of patients to points of definitive care may require utilization of aeromedical assets. The aeromedical team, who must have an open dialogue with the APS, influences pain management techniques used in the field hospital environment to provide continuity of analgesia during the evacuation phase. Analgesic regimens should incorporate a secondary mode in the event of failure of the primary mode in long evacuation flights. Replacement of advanced analgesic catheters in flight is challenging and often not achievable. Electromedical equipment must be certified air-worthy to fly on military aircraft.
- **Command representation.** Usually the responsibility of the senior nursing officer, representation by the hospital command chain at APS team meetings provides oversight of current issues relating to pain within the facility, and can influence members of the hospital administration, personnel, and equipment supply in relation to delivering pain service.

This list of team members is not exhaustive, and the management of pain also involves broader disciplines such as mental health, religious support, and welfare services. The important positive psychological impacts these services can bring for a patient must not be overlooked in the management of pain; however, accessibility may vary depending on the nature of the deployed operation.

CLINICAL FRAMEWORK: THE STANDARD OPERATING INSTRUCTION AND CLINICAL PRACTICE GUIDELINE

A pain management system is multimodal in utility and multidisciplinary in composition. For any such system to operate at its most effective, it is imperative that each component understands both its relation to, and the functioning of, the other component parts. All members of the pain service must understand their role and that of their colleagues, and have the same expectations regarding any intervention. For this reason a clinical framework or guideline is necessary for an effective pain management service.

A standard operating instruction (SOI) or clinical practice guideline (CPG) should address roles and

responsibilities and provide continuity in assessing pain, managing pain, prescribing medications, and using advanced analgesic techniques. In addition, a clinical framework may be extended to provide for theater-specific requirements such as the management of pain in children or local nationals when facilities at forward places of care may influence decisions and techniques.

The ownership of the SOI/CPG should reside with the SIG, which is able to update the SOI with developments in techniques and procedures and deliver these updates to deploying troops through timely predeployment education packages. The SOI/CPG in place at the medical treatment facility should align with the lead nation for the facility. For example, the United Kingdom (UK) is lead nation of the multinational clinical team at the UK Medical Treatment Facility at Camp Bastion, Afghanistan, and its clinical SOIs were delivered by the UK and conform to UK clinical governance.

PREDEPLOYMENT TRAINING

For medical units, an element of predeployment training includes clinical and moulage training in teams. For pain elements, there are two aspects of predeployment training:

1. Providing education on pain management, SOI/CPGs, and equipment.
2. Enabling the pain team to form and develop its role prior to deployment.

Pain Education

While every member of the deploying force should be current in their clinical practice, when the force is assembled from a disparate cohort there will be local differences to this practice, not only from one hospital to another but also from one nation to another. For example, some staff may have a background in which pain scoring is measured from 1 to 10, and others may be more familiar with a 0-to-3 system. Examples of international differences may be as simple as drug names (eg, paracetamol/acetaminophen).

Predeployment training in pain management can range from formal lectures to informal workshops (Figure 24-1) and should cover the intended pain management standard procedures for use in deployment, including familiarization with equipment not previously encountered.

Team Training

The pain team on deployment would be considered small in relation to comparable civilian acute pain services. Although led by a consultant in anesthesia, it relies heavily on a pain nurse and a link nurse identified in each key clinical area (emergency department, operating room, critical care unit, and wards). Identifying team members prior to deployment enables them to develop working relations both within the team and with other members of the deploying medical facility.

Figure 24-1. The author conducting continuation pain training, Operation Herrick (UK deployment to Afghanistan, 2002–present); 2011.

Figure 24-2. Deployed pain service meeting, Operation Herrick (UK deployment to Afghanistan, 2002–present); 2011.

TEAM ROUNDS AND MEETINGS

The primary responsibility of the deployed pain team is to conduct daily pain rounds and provide consultation and intervention as required for the management of acute pain. The APS should also maintain a routine presence in general surgical rounds. The benefits of conducting a regular pain round include providing oversight of all pain management and specialist input when advanced analgesic techniques are utilized. Rounds also provide a sense of pain management continuity that surgeons and other specialists appreciate, engendering confidence in the activities of the APS. Daily pain rounds not only raise awareness of pain, its assessment, and its treatment within the clinical team, but also reassure the patient that pain is a focus of care, not to be ignored or poorly managed.

Pain management group meetings (Figure 24-2) should be a regular feature of an enduring medical operation. This forum provides an environment to identify areas of good practice and areas of concern where improvements may be made. Any reported serious untoward incidents involving pain management should be identified, investigated, and be reported on locally and through the relevant reporting chains. The need for any ongoing training may be identified and an implementation plan agreed to, and audit utilized to clarify areas requiring service improvement or to assess the effect of service improvements.

CONCLUSION: ENABLING CHANGE

Many ideas and innovations in acute pain management are generated within the operational environment. When personnel return to their home duty posting, these ideas are often not developed and disappear with the departing personnel. The collective role of the pain management group is to consolidate these innovations, develop them locally as applicable, and report back to the lead governance structure by way of the SIG. In this way corporate memory is retained and service development continues, improving and refining the deployed management of pain.

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Chapter 25

PREHOSPITAL ANALGESIA

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INTRODUCTION

Historically, analgesic interventions in the prehospital environment were of secondary importance in the management of trauma patients. Aggressive pain control was even considered detrimental to injury diagnosis on arrival to the hospital. An introduction to a 1981 paper on prehospital analgesia stated, "Any agent that interferes with the patient's normal pain response may frustrate the physician attempting to make a diagnosis," and "a suitable agent . . . should be quick-acting and short-lived in order to preserve the pain response for diagnostic purposes in the ED."[1] Compounding this deemphasis on pain control was a prevailing belief that seriously injured casualties suffered little, as the oft-quoted statement by Dr Henry K Beecher suggested: "severe wounds in soldiers are often associated with surprisingly little pain."[2] As a US Army physician serving overseas during World War II, Beecher reported that up to 75% of battle-wounded soldiers deferred analgesia.

Fortunately, medical thinking in both regards is moving beyond these antiquated paradigms. Little controversy now remains that point-of-injury treatment of pain, before transfer to a definitive care facility, is a desirable and in fact medically beneficial goal. Holbrook et al, for example, found that use of morphine in US military personnel immediately after combat-related trauma was associated with lower rates of posttraumatic stress disorder (PTSD).[3] What remains undecided is the ideal analgesic regimen for trauma patients, a medical demographic largely defined by their heterogeneous and complex afflictions. A standardized pain regimen may aid one patient immensely while resulting in catastrophic complications for another. Therefore, valid concerns about the potential for adverse reactions to medications in this vulnerable population do exist and bear consideration. Both objectives of blunting nociception and avoiding exacerbation of the physiologic insults of trauma thus shape the development of any widely applied prehospital pain control algorithm.

AN IDEAL BATTLEFIELD ANALGESIC

Treatment of pain in the battlefield setting involves unique and demanding circumstances unlike any other medical scenario. Environmental challenges of heat, cold, aridity, moisture, dust, and sunlight exposure threaten the stability of medical materials. Various routes of medication administration must be available because victims of polytrauma may have multiple traumatically amputated limbs or severe hemorrhagic shock; either situation confounds intravenous (IV) access. Traumatically disrupted gastrointestinal organs make oral administration ineffective at best, life-threatening at worst. Intraosseous (IO) access, especially by sterno-manubrial approach, is thus experiencing heightened interest for its availability in even critically wounded patients. Methods of delivery should be as straightforward as possible because all friendly forces in combat are potential caregivers. In a tactical setting, medical care may be administered by the casualties themselves, a medically naive service member, a trained medic, or even a credentialed independent provider.

Once suitable for the demands of the battlefield, an analgesic intervention for traumatic injury must produce a clinically significant reduction in pain, proportionate to the severity of wounds sustained. Pain relief should have a rapid onset, measurable in minutes, with a rarity of adverse effect at clinically efficacious doses. Hemodynamic stability should be maintained, if not augmented, given the threat of hypovolemic or obstructive shock. Optimally, medications would not impair airway reflexes or minute ventilation, although a reduction in respiratory rate and tidal volume is consistent with adequate pain control. Besides preservation of cardiopulmonary function, medications given for combat analgesia should have minimal deleterious side effects, such as excessively altered mental status, disabling motor block, platelet inhibition, emetogenic potential, increased intracranial pressure (ICP), allergic reaction, or interference with expected effect of other medications. Other desirable qualities include a large therapeutic index, low interpatient pharmacokinetic variability, and arguably an amnestic effect.

Exclusive to military operations is the need to preserve the fighting force even in the face of excruciating pain. If adequate pain control for wounds will impair a service member's operation of a critical weapon system or fighting position, the analgesia could be withheld for the survivability of the unit. Therefore, analgesics that will not remove an otherwise mission-ready service member from the fight should be in the provider's armamentarium.

MODALITIES

In the search for the optimal pain intervention regimen on the battlefield, evidence in the form of randomized clinical trials is lacking. Best practice must be extrapolated from retrospective or observational

studies, established civilian trauma pain management, and other data not wholly representative of the combat trauma population.

Narcotics

Narcotics have been in use for military pain management since the American Civil War, testimony to their effectiveness in treating acute pain. Once available only at the field hospital, single-use morphine "syrettes" were developed by the US Navy for use by individual service members; their use was reported by Major Charles Wilson as early as 1941.[4] Intramuscular (IM) morphine has since been the historical "gold standard" in battlefield analgesia, but IV, intranasal (IN), and transmucosal preparations of various opioids have been explored in recent years. Smith et al studied 204 trauma patients given IV morphine or IV fentanyl during helicopter evacuation.[5] The medications were equally effective, with both groups achieving a decrease in mean pain scores from 80 mm to approximately 55 mm on the visual analog scale (VAS), which uses a 100-mm line to score pain. Neither group achieved pain scores below 40 mm, which is considered mild pain. Doses used in this study were 4 mg IV morphine and 50 μg IV fentanyl, with an average of two subsequent doses during transport to the hospital. No statistically significant difference in adverse effects was identified.

In a double-blinded, randomized, but small clinical trial, Galinski et al compared prehospital administration of IV morphine and IV fentanyl, at doses of 0.1 mg/kg and 1 μg /kg, respectively. Both treatments were effective at reducing pain by approximately 40 mm on the VAS, and there was no significant difference in pain relief or incidence of side effects.[6] Bounes et al conducted a randomized, double-blind, out-of-hospital trial comparing strict sufentanil or morphine regimens for adult patients with severe traumatic acute pain. While pain control in the sufentanil group was superior at 9 minutes after institution of treatment, the difference was negligible at 15 minutes.[7] Overall, administration of IV narcotics for patients in the prehospital setting suffering from moderate to severe pain appears safe and effective in studied dosing schemes.[8–11] No IV formulation of narcotic, however, holds the distinction as the "best" IV opioid for acute trauma patients.

For patients in whom IV access is impractical or impossible, alternative routes of administration are an option. Rickard et al found no significant difference between prehospital use of IN and IV fentanyl.[12] IN fentanyl was dosed at 180 μg, with subsequent doses of 60 μg, while IV morphine was given in 2.5-mg to 5-mg doses. Each reduced verbal rating scores (VRS) of pain by approximately 4 points on a scale of 0 to 10. Karlsen et al evaluated 903 patients who received IN fentanyl in a prospective observational study, noting a median pain score reduction of 3 points, with no serious side effects or naloxone requirement.[13] Both studies involved patients with nontraumatic, presumed cardiac pain, confounding their application to battlefield settings, but they illustrate that IN administration is a viable option when necessary. More proven in the operational environment is oral transmucosal fentanyl citrate (OTFC), which is formulated as a "lollipop" for placement on oral mucosa for absorption and systemic effect. Buccal absorption produces quick onset of analgesia, while gastric and intestinal absorption afford sustained analgesia. OTFC use in 286 military casualties over 7 years demonstrates satisfactory reduction in verbal-numeric rating scale scores, averaging a 4.8-point reduction in 15 to 30 minutes.[14] Only one patient in this series required naloxone due to hypoventilation, and that was after receiving 3,200 μg of oral fentanyl and 20 mg of morphine.

In 2010, Park et al reviewed 21 studies encompassing 6,212 patients who received various forms of prehospital analgesia, with most data focused on the use of opioids, specifically morphine, fentanyl, alfentanil, and tramadol. These studies represented a mixture of patient populations, such as civilians with traumatic injuries and acute medical patients; three studies specifically examined military injuries. Park et al concluded that opioids overall achieved satisfactory pain levels (defined as less than or equal to 30 mm on the VAS) in approximately 35% of patients by 10 minutes, and 70% by 40 minutes. No patients in this systematic review required ventilatory support, only two required naloxone, and cardiovascular instability related to opioid administration was uncommon.[15]

This review succinctly concludes that narcotics of various formulations have an acceptable efficacy and safety record when used for traumatically injured patients at the studied doses. Any attempt to improve upon opioid analgesia onset and intensity must be balanced against the very real untoward effects of narcotics. Concerns about aggravating hypovolemic shock or hypercarbia resulting in intracranial hypertension are well founded, especially in the deployed combat scenario. In any setting, use of narcotics for severe pain assumes the risk of potentially life-threatening respiratory depression.

Nonsteroidal Antiinflammatory Drugs

Although not the mainstay treatment for management of severe traumatic pain, nonsteroidal antiinflammatory drugs (NSAIDs) and acetaminophen serve important roles in battlefield pain management. They

supplement the analgesia of narcotics without contributing to the risks of respiratory depression or hypotension, and may be readily dispersed to nonmedical personnel. The risks of NSAID-related gastrointestinal bleeding or acute renal injury are remote in the typical healthy service member, and acetaminophen has a minimal side effect profile in appropriate doses.[16] Furthermore, NSAIDs and acetaminophen are ideal sole agents to address minor ailments that could otherwise impact a soldier's mission-readiness.

Data is absent concerning acetaminophen or NSAID effectiveness for prehospital treatment of combat-injured patients; rather, most studies address orthopedic ailments in the emergency department setting. Viallon et al administered 1,000 mg of oral acetaminophen to 571 emergency department patients with musculoskeletal injuries, ranging from sprains to dislocations to fractures, showing that pain scores improved on average by 27/100 mm on the VAS after 1 hour.[17] Impressively, 1,000 mg IV acetaminophen demonstrated equivalent analgesia to 10 mg IV morphine in a randomized, double-blind study of adult patients with isolated limb traumatic injury.[18] Ibuprofen, ubiquitous in the military world, and acetaminophen were both found to reduce VAS scores by a mean of 20/100 mm within 1 hour in the emergency department when administered for acute musculoskeletal pain, but the two drugs did not show synergistic analgesia when given together.[19]

In contrast, a Cochrane database systematic review of ibuprofen and acetaminophen administration for postoperative pain management found the combination of an NSAID and acetaminophen to be more effective than ibuprofen alone. Groups compared included patients experiencing acute perioperative pain or migraine. Higher dosing strategies of ibuprofen plus acetaminophen (versus placebo or ibuprofen alone) increased the percentage of patients achieving 50% of maximal pain control at 6 hours and significantly lengthened the amount of time until further rescue medication was needed.[20] In postoperative pain control and acute musculoskeletal injuries, ketorolac and diclofenac have demonstrated analgesia comparable to weaker opioids.[16] Proving efficacy of NSAIDs or acetaminophen for combat injuries will be challenging even if such as study is attempted, but the paucity of side effects and the likelihood of some analgesic benefit make these medications attractive in polytrauma patients.

Inhalational Analgesia

Volatile anesthetics have known analgesic effect, a quality exploited by several countries in the prehospital setting and emergency department. The United Kingdom readily employs Entonox (BOC Healthcare, Worsley Manchester, United Kingdom), a 50/50 mixture of oxygen and nitrous oxide, for administration by emergency medical technicians before the patient arrives at the hospital. A randomized clinical trial conducted by Ducassé and colleagues compared Entonox to an oxygen placebo during ambulance administration.[21] The study enrolled adult patients with moderate acute traumatic pain, a demographic resembling a typical military trauma patient. After 15 minutes of inhalation, Entonox successfully decreased initial pain scores from a median of 6 to 3 or below on a numeric rating scale in 67% of patients.

Although inexpensive and hemodynamically benign, nitrous oxide has several contraindications that restrict its widespread application in the traumatically injured patient population. It is well known to complicate certain traumatic injuries, such as pneumothorax or air emboli, due to its relatively high solubility coefficient when compared to nitrogen.

Methoxyflurane, a halogenated ether, was removed from the US and Canadian markets for unacceptable risk of hepatotoxicity and dose-dependent nephrotoxicity when used at general anesthetic doses. In low concentrations of up to 0.5%, however, patients may enjoy the benefit of pain relief with minimal risk of hepatic or renal damage. Buntine et al showed a mean reduction in VRS scales of 2.47 in 83 patients receiving methoxyflurane during ambulance transport, with 72.3% of patients reporting satisfaction with the level of pain control.[22]

Middleton et al reviewed the prehospital pain regimens for 52,046 patients to compare IV morphine, IN fentanyl, and inhaled methoxyflurane.[23] Only 59.1% of patients who received methoxyflurane had a 30% or greater reduction in their pain, as measured on a 0 to 10 verbal-numeric rating scale. This analgesic efficacy was statistically inferior to the respective 81.8% and 80.0% of patients receiving IV morphine or IN fentanyl with the same threshold of pain relief. With IM, IN, and transmucosal preparations of other medications answering the need for analgesia when IV access is not available, the expansion of inhaled halogenated ether use in a prehospital setting is unlikely.

Ketamine

The *N*-methyl-D-aspartate (NMDA) antagonist ketamine was first synthesized in the 1960s from phencyclidine in an attempt to decrease incidence of delirium while retaining the dissociative anesthetic quality.[24] The potential for battlefield pain management was quickly recognized, and ketamine came into use

by the US military during the Vietnam War. Ketamine is now experiencing a resurgence of interest for analgesia on the modern battlefield thanks to a deeper understanding of its favorable hemodynamic effects in the trauma patients, relative preservation of airway reflexes and the carbon dioxide response curve, and multiple available routes of administration.

The historically cited adverse effects of ketamine in the traumatically injured patient must be addressed in the context of recent academic skepticism, these concerns being increased ICP, increased intraocular pressure (IOP), and distressing psychotic symptoms. The former was addressed in a review of five randomized prospective studies, including patients with traumatic brain injuries, in which ketamine infusions for sedation showed no statistically significant increase in ICP and possibly an increase in cerebral perfusion pressure.[25] Halstead et al challenged the second concern by measuring IOP in otherwise healthy children without ocular injury who received ketamine for procedural sedation in the emergency department. IOP was not statistically increased after giving ketamine in average doses of 1.6 mg/kg.[26] Lastly, ketamine use was associated with a decrease in PTSD in burned active duty service members despite more extensive burns and longer stays in the intensive care unit in the ketamine-treated group.[27]

Barring these specific controversies, IV and IM ketamine has an excellent safety record when used outside a medical facility. Bredmose et al found no episodes of hypoxia or loss of airway patency related to ketamine administration in 1,030 prehospital clinical encounters.[28] A systematic review by Jennings et al evaluated six studies, finding that ketamine delivered effective relief for acute traumatic pain in the prehospital setting, either as monotherapy or by reduction in morphine requirement.[29] When compared directly against the "gold standard" of morphine in a prehospital prospective study, ketamine delivered equivalent reductions in VAS pain scores as morphine, lower rates of emesis, but increased risk of hallucinations and agitation.[30] Also, 57 of 169 patients with head trauma who received ketamine in this series suffered no demonstrable declines in mental status. If both drugs are available for point-of-injury care, some data supports superior analgesia with coadministration of ketamine and morphine over morphine alone, with mean VRS reductions of 5.6 versus 3.2, respectively.[31] The ability to administer ketamine via the IN route needs further exploration, but preliminary investigation in nine patients suggests efficacy for point-of-injury use.[32] While it is premature to conclude that ketamine should completely replace narcotics as the foundation of moderate to severe combat trauma pain management, its theoretical and proven qualities appear closely suited to the needs of today's battlefield medicine.

CURRENT MILITARY PRACTICE

The US military has widely adopted the Tactical Combat Casualty Care (TCCC) model for training its service members to prevent battlefield deaths with simple, life-saving procedures. The guidelines are regularly reviewed and updated as new data are published, most recently on 28 October 2013 (http://www.usaisr.amedd.army.mil/assets/pdfs/TCCC_Guidelines_131028.pdf). While TCCC recommendations cast a wide net over battlefield medical care, only the pain management aspects will be summarized in this discussion. Casualties are quickly dichotomized into mission-capable and disabled patients by the attendant medical provider. Personnel with minor wounds who are able to meaningfully contribute to combat operations are administered 1,300 mg oral acetaminophen every 8 hours and 15 mg oral meloxicam once daily. Meloxicam was selected for its relative cyclooxygenase-2 selectivity.[33] Ideally, all service members carry these medications as part of their issued first aid kits and may self-administer them when hurt.

Seriously injured trauma casualties may receive acetaminophen and meloxicam if they can tolerate oral medications, but the TCCC algorithm then stresses escalation to narcotics and ketamine. Patients with moderate to severe pain, not suffering from hemodynamic shock, and without evidence of respiratory depression, receive an 800 μg OTFC lozenge/lollipop. TCCC guidelines suggest taping the OTFC lozenge-on-a-stick to the patient's finger, so that if the patient becomes excessively sedated, the drooping arm will pull the lozenge from the mouth and prevent further narcotization. A second lozenge may be used directly following the first in the event of inadequate analgesia.

If a patient is suffering moderate to severe pain and is at risk for hemodynamic or pulmonary instability, ketamine is the first-line treatment. When IV or IO access is available, the qualified medical provider on scene administers 20 mg of ketamine every 20 minutes. Alternatively, ketamine may be injected IM in 50-mg aliquots or sprayed IN as a 50-mg dose every 30 minutes. Ketamine dosing is halted upon attaining satisfactory analgesia, or in the event of nystagmus, ventilatory compromise, or agitation. TCCC allows for ketamine dosing for patients with ophthalmic injuries or significant traumatic brain injury, acknowledging the controversy over increased IOP and ICP, respectively.

IV or IO morphine remains on the algorithm as an alternative to OTFC, given in 5-mg doses every 10 minutes, titrated to pain control, with monitoring of respiratory depression. The availability of naloxone is strongly encouraged when administering any narcotic, however, and use of ketamine and narcotics is reserved for combat medics or paramedics, the latter being members of the special operations community who have received advanced medical training.

SUMMARY

Even in the 21st century modernized battlefield, the optimal pain regimen for military trauma victims is unclear. While randomized clinical trials and certainly placebo controls are impractical for research in a combat zone, higher quality data elucidating best care practices for this particular population are needed. There remains a paucity of information, and what is available is complicated by wide variation in the patient, provider, and environment. The combat medical provider is strongly encouraged to utilize current guidelines pursuant to their level of expertise, but critically apply them as each unique trauma scenario dictates. Potential side effects must be recognized and averted if possible. Treatment algorithms should be routinely scrutinized for updates based on new evidence and shifting paradigms. No service member should suffer unnecessarily after injury, but control of pain must never take priority over life-saving interventions, which are paramount in combat casualty care.

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Chapter 26

COMBAT TRAUMA OUTCOMES TRACKING AND RESEARCH

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INTRODUCTION

Military pain management was ushered into the modern era with Friedrick Sertürner's discovery of the "sleep-inducing factor" of the poppy, which he named after the Greek god of sleep and dreams, Morpheus, and is now known as morphine.[1,2] By the American Civil War (1861–1865) and the Franco-Prussian War (1870–1871), the use of morphine in managing battlefield trauma was widespread, so much so that opioid addiction among wounded veterans was common and termed "soldier's disease."[3] Despite the life-threatening side effects associated with morphine, its effectiveness in managing complex battlefield trauma was beyond dispute. Although the consequences of opioid monotherapy for pain, particularly addiction, were well known, the lack of viable alternatives left military physicians with little choice because these medications were essential for managing the war wounded. Following World War I (1914–1918), Ernest Bishop, MD, professor of medicine at the New York Polyclinical Medical School, commented "that opiate addiction is and will be one of the medical problems of this war is recognized and must be openly met."[4] He suggested that opioid addiction was unavoidable because opioid use was the only way to manage battlefield pain, and learning to manage addiction was the only humane and rational response to the problem.[4]

Between World War I and World War II (1939–1945), research into pain mechanisms and management remained rudimentary. For most physicians at the time, pain was considered an unfortunate and unavoidable consequence of wounding or surgery, and many considered anesthetic agents unnecessary and possibly a hindrance to recovery.[5] Opioids remained the primary, and often sole, solution for managing pain in World War II. The growing number of opioid-related deaths during this time prompted the first large-scale study on pain management practice within the US military.[6] John Bonica, MD, a military anesthesiologist working at Madigan Army Hospital in Washington, was inundated with pain management cases from World War II and frustrated at the general lack of understanding within the medical community concerning pain management. This situation defined Bonica's career for the next 20 years as he crusaded for multidisciplinary pain clinics, promoted the medical specialty of pain medicine, and wrote his seminal text, *The Management of Pain*.[7] Bonica's efforts provided a foundation for pain medicine development, but military and civilian medical communities were slow to adopt changes in traditional opioid-based pain management practices. Morphine remained the primary drug for war trauma pain in World War II, the Korean War, and the Vietnam War, although early innovators began to change that paradigm. Captain J Markowitz used spinal analgesia to great effect on World War II prisoners of war in Thailand who required amputations in austere medical conditions.[8] Gale Thompson pioneered the use of regional anesthesia in Vietnam War wounded, improving operating room efficiency and patient analgesia.[9,10] In the following decades, others such as Alon Winnie, who developed many of the peripheral nerve block procedures used today, commented on the value of regional anesthetic catheters on the modern battlefield that "would allow analgesia to last as long as necessary."[11] While these were significant steps forward in improving pain care for the wounded warrior, within the military medical establishment pain remained a consequence of war and wounding that was poorly understood, subjectively diagnosed, and difficult to treat. In military culture, pain was expected to be endured, as evidenced by common expressions such as "no pain, no gain" and "pain is weakness leaving the body."

Following the terrorist attacks on the United States on September 11, 2001, and the onset of the Afghanistan and Iraq conflicts, reliance on morphine as the sole battlefield analgesic had essentially remained unchanged from the 19th century. Research data on the impact of pain on the combat casualty was nonexistent. The stagnant evolution of pain management methods likely relates to a general lack of understanding of the impact poorly managed pain has as a disease process involving both the peripheral and central nervous systems.[12] In previous conflicts, the wounded tended to remain static for days or even weeks in theater until they were stable enough for transport. Managing pain exclusively with morphine was likely a viable strategy in this situation because static patients could be appropriately monitored and the drug correctly titrated. This has not been the case in contemporary conflicts because the current paradigm for casualty management relies on rapidly evacuating stabilized casualties by air out of theater within hours to days. The exclusive use of morphine in this challenging, relatively austere aeromedical environment has not been ideal because of the inherent challenges in patient monitoring and the potentially life-threatening side effects associated with opioid medication.

Perhaps even more concerning has been the continued paucity of data from modern conflicts on the impact of pain following wounding. In a systematic review of prehospital analgesia, Park et al[13] noted a general lack of evidence to inform pain management

practice in these environments, despite broad search and inclusion criteria. The wide-ranging lack of understanding about battlefield trauma pain is made more poignant with improved understanding of the relationship between chronic pain, posttraumatic stress injury, and traumatic brain injury (termed the "polytrauma clinical triad").[14] Additionally, the impact of poorly managed pain on the US general population, estimated at $100 billion annually in increased healthcare expenses, lost income, and lost productivity, was seen by the US military as a significant unmet military healthcare problem that lacked a comprehensive strategy to address deficiencies.[15] In response to this emerging issue, the US Army surgeon general, Lieutenant General Eric B Schoomaker chartered the Pain Management Task Force in August 2009 to review current military pain practice and make recommendations for a pain management strategy "that was holistic, multidisciplinary, and multimodal in its approach, utilizes state of the art/science modalities and technologies, and provides optimal quality of life for soldiers and other patients with acute and chronic pain." The Pain Management Task Force report was published in May 2010 and determined that the general lack of military pain data was causing "difficulty in making responsible decisions on the myriad of possible treatment modalities."[15] The surgeon general of the British Defence Medical Services, Lieutenant General L Lillywhite, made improvements in wounded warrior pain management one of his main efforts in 2007, suggesting the United Kingdom (UK) military was coming to similar conclusions from their experience.[16]

With this historical perspective, which clearly indicates a general lack of data on pain in combat wounded, the ensuing chapter will focus on recent successes and developing efforts to study pain in combat casualties and build a new approach to military pain management for the 21st century.

JOINT THEATER TRAUMA REGISTRY

Following the Vietnam War (1955–1975), many key advances in military trauma care were transferred into civilian medicine and became the modern trauma system seen today. Civilian proponents of modern trauma systems recognized the need for outcomes measurement to justify the expense of these systems through improved survival and outcomes.[17] In an effort to mimic civilian success in trauma system development, the Joint Theater Trauma Registry (JTTR) was developed as a battlefield database designed to inform processes resulting in "the right patient, to the right place, at the right time, to receive the right care (R4)."[18] It would be difficult to overestimate the impact the JTTR has had on battlefield trauma care in current conflicts. Perhaps most notable have been the 27 evidence-based clinical practice guidelines that have been developed from the data generated by the JTTR. As important as the JTTR has been to refining the military medical response to battlefield trauma, it has not provided additional insight into the impact of pain following wounding. Pain data was not even part of the JTTR until 2007, and the information is limited to visual analogue scale (VAS) data. Clearly this information, though valuable, is insufficient to use for practice recommendations for pain management. As noted in the Pain Management Task Force report, a system for obtaining actionable pain data from the battlefield, throughout evacuation, at home, and into recovery is needed if evidence-based improvements are going to be made to battlefield pain management.

MEASURING PAIN

Due to the subjective nature of pain, its measurement as an indicator of severity or clinical success with treatment has always been difficult. Since the 1970s, most clinicians have accepted the VAS as the preferred method for measuring pain and determining pain relief.[19] Outside of a research protocol that consistently controls how pain is measured, there is rarely uniformity in how clinicians use VAS pain scores when managing patients, making it problematic to compare VAS pain scores among facilities or groups of providers. Beyond the universal paucity of pain data from the present conflicts is the general lack of agreement in how to consistently measure pain in wounded soldiers.

The UK sought to establish a pain-measuring system that was simple, consistent with the World Health Organization's "pain ladder," and easily administered by all levels of providers in austere medical environments. The UK has selected a simple 0-to-3 pain scale (see Chapter 19, Scoring Pain, Table 19-1) with examples of possible therapeutic interventions based on the pain ladder.[20] Scores of 2 or 3 are considered unacceptable and prompt pain intervention.

The United States wanted to retain the familiarity and scientific value of the 0-to-10 VAS, but ground these values with functional anchors to provide consistency in scale administration, enhance clarity for patients and providers, and provide a common bench-

mark for comparing treatment effectiveness. There was also a desire to evaluate the biopsychosocial influence of pain through its impact on general activity, mood, stress level, and sleep. This resulted in the Defense and Veterans Pain Rating Scale (DVPRS; Chapter 19, Scoring Pain, Figure 19-1). The DVPRS was developed to provide a standardized method for pain assessment that would be easily adapted to US military databases, useful across all roles of care (eg, medics, ward nurses, primary care providers, and pain specialty care), and consistent with current validated pain research tools. Based on initial validation studies, the US military is in the process of applying the DVPRS standard throughout all roles of care.[21]

COMMUNICATION AND PAIN MANAGEMENT THROUGH THE ROLES OF CARE

One of the more significant barriers to improving pain management on the modern battlefield has been communication between roles of care throughout the evacuation chain. Early in the recent conflicts, continuous peripheral nerve block (CPNB) was identified as valuable analgesia in the preponderance of extremity wounds.[22,23] While detailed records of care were maintained at each role along the evacuation chain, this information did not routinely travel with the patient beyond an air evacuation summary document, preventing pain data collection and hindering the introduction of pain management innovation beyond morphine. As a result, few manuscripts on pain levels or management innovation in combat wounded, beyond small surveys and reviews, exist from the current conflicts.[24–26] The lack of scholarly publication on pain care for wounded service members represents a missed opportunity to advance the science of pain care and prevent providers from having to start over in the next conflict.

The increased use of regional anesthesia on the battlefield illustrates how enhanced communication supports advancements in pain management. The use of CPNB catheters requires daily review by health professionals supplied with sufficient information on CPNB pain infusion. Initially, this information was passed between pain specialists within the evacuation chain via e-mail. Although e-mail was successful, it was an unsatisfactory way to transmit this sensitive information. The United States has developed military-sanctioned electronic pain notes that allow proper communication between providers, although this system is used inconsistently. As capability and complexity of pain management techniques continue to evolve, medical communication systems will need to be developed with sufficient bandwidth to support military medicine into the 21st century.

ACUTE PAIN SERVICES AND ROLE 3: AN OVERDUE REQUIREMENT

Pain relief following trauma or surgery remains a significant medical challenge despite contemporary understanding of the detrimental impact inadequate pain management has on rehabilitation and recovery.[27] Civilian healthcare providers in most developed societies have recognized the benefits of an interdisciplinary team approach to supervising and administering analgesic medications and techniques provided by acute pain services (APS) within the hospital setting. Many anesthesia accreditation bodies, such as the UK Royal College of Anaesthetists and the Australian and New Zealand College of Anaesthetists, require APS as a prerequisite for training programs.[28] Although many agree that APS improves patient pain relief and results in enhanced appreciation of its recovery benefits, optimal APS structure and cost effectiveness remain ill defined.[29,30] Nevertheless, most anesthesiologists understand that more sophisticated pain management plans that include medications beyond morphine or that integrate sophisticated techniques such as CPNB require an interdisciplinary team approach, which is most easily embodied through APS.

Although the need for more general improvements to pain practices on the battlefield was recognized earlier, the first use of CPNB in the current conflicts occurred in 2003.[31] Although CPNB was an exciting innovation in battlefield pain care at the time, it was used in combat support hospital (CSH) conditions where patient-controlled analgesia or analgesics beyond morphine were unavailable. Among the many lessons learned since 2003 is the realization that battlefield pain care must operate in a continuum that begins at the point of injury, extends through the battlefield and evacuation system to home, continues in home-country medical centers, and stretches into the rest of the veteran's life.[32] The problem with pain management on the modern battlefield was that it has been considered the responsibility of every healthcare provider within theater; this meant that since everyone was responsible, no one was held accountable.

Military providers have always done their best to provide exceptional pain care, despite minimal guidance and a lack of sophisticated equipment. Through improvisation, they have often overcome the disadvantageous conditions to improve pain care standards. Yet these efforts have been unsustainable and dependent on innovative providers; therefore, casualties' pain management has depended on which providers

were deployed to their locations when they were injured. In addition to inconsistency in pain care, the lack of a clinical pain infrastructure (eg, APS) with defined personnel made standardization of pain care and data collection challenging in the field environment.

UK and US military anesthesiologists began rectifying this situation through a collaborative acute pain research and care initiative that involved the deployment of a US Army APS to a UK-commanded CSH in Camp Bastion, Afghanistan, in 2009.[33] The APS was composed of a physician trained in acute pain medicine as well as pain nurses within each care ward of the CSH. It was outfitted with a pain medicine augmentation chest that included pain infusion pumps, regional anesthesia equipment, a portable ultrasound machine, and other specialized equipment. The Camp Bastion CSH leadership prioritized pain management and made it a key indicator of care quality during this deployment. During this effort, approximately 455 trauma cases were managed (average 5.62 daily) at the CSH, and the APS staff served as the facility pain consultants on many of these cases based on injury severity or specific issues with pain. Of the 71 casualties managed by the APS, 51 (71.8%) received regional anesthesia, though all wounded under APS care were managed with individualized multimodal analgesic care plans (Table 26-1). In this series, the average percentage of improvement in pain, based on the service members' recall estimate at point of injury to air evacuation, was 51.9% (± 31.2). The realities of battlefield medicine precluded the establishment of a control group for this combat-injured population, but the significant pain relief brought about by the APS is undeniable. Of greater significance was the emphasis on pain management in daily rounds and routine CSH leadership meetings that prioritized this care issue.

TABLE 26-1

FREQUENCY OF INTRAVENOUS AND ORAL ANALGESIC ADMINISTRATION

Medication	No. of Patients	Frequency of Patients (%)
Paracetamol (IV)	66	93.0%
Diclofenac (IV)	59	83.1%
Morphine (IV)	30	42.3%
Oramorph SR* (PO)	19	26.8%
Codeine (PO)	5	7.0%
Ketamine (IV)	5	7.0%
Ketorolac (IV)	5	7.0%
Ibuprofen (PO)	4	5.6%
Tramadol (PO)	4	5.6%
Acetaminophen (PO)	1	1.4%
Amitriptyline (PO)	1	1.4%
Co-codamol (PO)	1	1.4%
Methocarbamol (PO)	1	1.4%

*Manufactured by Xanodyne Pharmaceuticals, Newport, KY.
IV: intravenous
PO: per os (by mouth)
Reproduced with permission from: Buckenmaier C 3rd, Galloway KT, Polomano RC, cDuffie M, Kwon N, Gallagher RM. Preliminary validation of the Defense and Veterans Pain Rating Scale (DVPRS) in a military population. *Pain Med.* 2013;14:1–14.

A survey of healthcare providers was conducted to evaluate their perceptions of the value added by a CSH APS to wounded warrior care. The purpose of this investigation was to provide meaningful data for the British Defence Medical Services and the US Department of Defense to guide future plans and policies for APS deployment.[33] A majority sample of 70 UK and US military healthcare providers at Camp Bastion during the APS pilot completed the survey instrument. The survey tool consisted of 12 items designed to represent concepts and impressions of APS outcomes, complexity of care, decision-making, satisfaction, pain-management education, and areas needing improvement. The survey demonstrated a high degree of enthusiasm for the CSH-based APS concept, with the majority of respondents agreeing that wounded soldiers managed with APS consultation reported decreased levels of pain (64.8%) and obtained greater relief (73.9%). Furthermore, the majority (73.5%) agreed that, overall, the APS had a significant impact on patient outcomes.

Although the survey demonstrated casualty care improvements after the CSH APS was put into place, this activity remains the exception rather than the accepted standard. The United States has established a Joint Theater Practice Guideline for pain and sedation[34] that establishes the requirement for APS in US CSHs; however, it will take a general command emphasis within the military medical system to make this policy a reality in the next conflict.

THE FUTURE OF PAIN MANAGEMENT ON THE BATTLEFIELD

If there is anything beneficial about war, it is that war is a catalyst for medical innovation and advancement. In terms of trauma pain management, recent conflicts validate this sentiment. Pain practice has changed tremendously since the beginning of the present conflicts, with the introduction of new medications,

techniques, technologies, and provider emphasis on pain management. The key challenge is to ensure that these lessons are not lost for the next war and that they become part of military medical culture. Collecting and analyzing pain data from present conflicts is essential.

The following conditions should become a focus for military medical planners if improvements in wounded warrior pain management are to become the standard:

- Pain education must become a routine component of medical training at all levels.
- Pain measurement using a common tool, such as the DVPRS, must become a routine part of all casualty assessment.
- Pain measurement data must be collected. It should be used as a marker of care effectiveness within the CSH and throughout the evacuation chain.
- The JTTR should collect DVPRS data on all casualties.
- The APS should become as integral to a medical facility's function (CSH) as the surgery or medical services.
- The APS must be staffed with physician (usually an anesthesiologist) and nursing assets that are dedicated to and specifically tasked with handling pain issues within the institution.
- The APS should become the accepted conduit for introducing novel pain management strategies in future conflicts.
- Communication (preferably secure and electronic) between roles of care in the evacuation chain must include casualty pain data.
- Provisions for specialized pain equipment sets must become a routine component of the CSH.
- The incidence, intensity, and management of pain on the battlefield must become a research priority, as should alternatives to opioid use for pain management.

All the components exist for establishment of APSs in Role 3 through home-based facilities within the US and UK military medical systems. Once established, the APS will serve as the conduit for trauma pain data flow, research, and innovation. As John Bonica observed in *The Management of Pain*, "The proper management of pain remains, after all, the most important obligation, the main objective, and the crowning achievement of every physician."

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Chapter 27

RECEIVING THE CRITICAL CARE PATIENT

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INTRODUCTION

This chapter provides the deploying intensivist guidance to care for patients admitted to the intensive care unit (ICU) within the first 24 hours of admission. Care in this setting must be delivered through teamwork involving surgeons, anesthetists, nurses, physiotherapists, and other paramedical practitioners. It is paramount that this team operates holistically to stabilize the many physiologic challenges encountered in the deployed military setting.

Casualties present with a wide variety of pathology in addition to the expected polytrauma. Trauma patterns will differ from those encountered in civilian practice. Combat injuries will consist of primarily penetrating and blast injuries, and there may also be a requirement to care for local civilians, including children. Unlike fit and healthy soldiers, civilians may have significant comorbidity.

Patients may be admitted to the ICU following surgery or prior to surgery if the patient workload exceeds operating room (OR) capacity. These latter patients may therefore require ongoing resuscitation and stabilization. The concept of damage control (originally a US Navy term), denoting the resuscitative and surgical procedures, performed swiftly, that are required to save life and limb–as opposed to longer and more definitive surgical techniques–has been widely adopted in recent conflicts and is practiced throughout the whole care pathway from the emergency department (ED), through radiology, OR, and into the ICU. Never was the concept of an ICU "without walls" more appropriate than in the deployed military setting.

This chapter will focus on the military population. Primary topics include resolution of hemorrhagic shock and the potential for respiratory failure due to trauma. Management strategies are focused on oxygen delivery, avoidance of tissue hypoperfusion, and prevention of the systemic inflammatory response progressing to multiorgan dysfunction.

ADMISSION HISTORY AND PHYSICAL EXAMINATION

History

Critically ill patients admitted to the Role 2 and 3 deployed hospitals may arrive either by land or air transport. Initial history consists of "MIST-AT": mechanism of injury, injuries sustained, symptoms and signs, treatment given–age (adult/child), and time of injury. In patients who are awake, an "AMPLE" (allergies, medication, pregnancy, last eaten) history may have been taken prior to intubation. Once the situation allows, further attempts should be made to gather a more detailed history, which may necessitate the use of interpreters and family members.

Examination

Depending on the patient's consciousness level, however, further history may be limited. Irrespective of the origin of the patient (ie, Role 1 vs point of wounding), a full primary survey must be carried out. This typically takes place in the ED. However, if the injuries dictate immediate admission to the OR, bypassing the ED, then primary and secondary surveys may need to be undertaken in parallel with surgery. Occasionally, if the capacity of the ED and OR is overwhelmed, patients may be admitted directly to the ICU. It is therefore important to understand the degree to which history and examination have been undertaken and to perform repeated triaging and assessment, using the <C>ABCDE (catastrophic hemorrhage, airway, breathing, circulation, disability, exposure) approach. Repeated assessments are particularly important because patients' physiology will change rapidly in the initial hours postadmission.

Thus a "head-to-toe" and "back-to-front" examination is required. This examination should include review of all radiology films and laboratory data in consultation with the trauma team surgeons to ascertain all initial injuries. The initial physical examination, which serves as the baseline reference point for further therapy, should follow Battlefield Advanced Trauma Life Support guidelines of <C>ABCDE (Figure 27-1). The examination must include a basic neurologic assessment covering reflexes, motor power, and mental status, as well as an otoscopic ear examination and funduscopic eye examination. Critically ill patients in the ICU are at risk of developing serious neurologic complications including ICU psychosis, septic encephalopathy, critical illness polyneuropathy, entrapment neuropathies, compartment syndromes, cerebral edema, intracerebral hemorrhages (related to coagulopathies), cerebral ischemia (related to hemodynamic instability), and cerebral embolism. These conditions are principally detected and diagnosed by clinical examination (computed tomography [CT] scanners are not always available). Furthermore, these conditions are frequently masked in sedated patients. If the patient does not respond to a noxious stimulus, the sedation must immediately be stopped to facilitate further neurologic evaluation. It should be policy

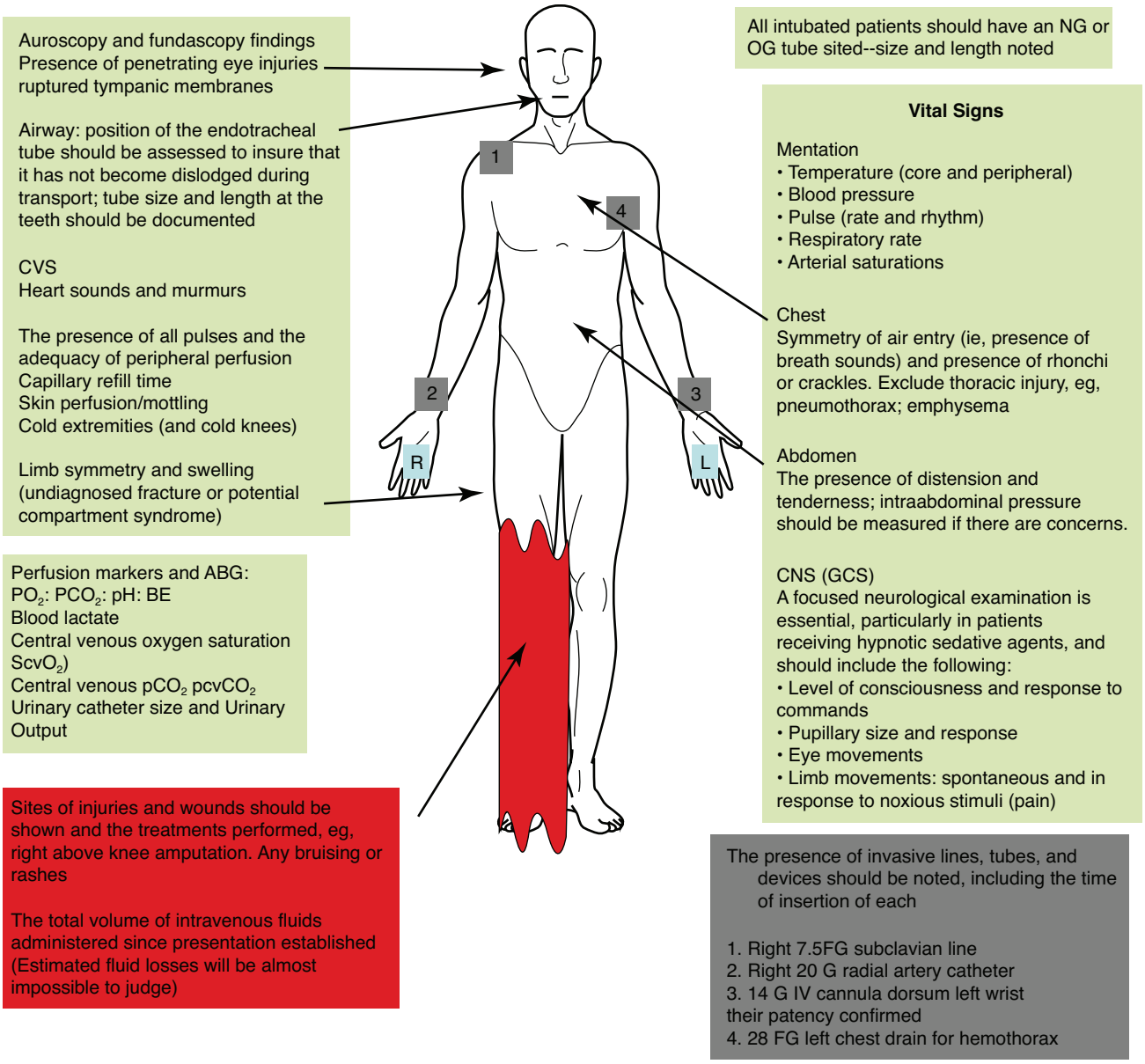

Figure 27-1. The initial physical examination and documentation, following Battlefield Advanced Trauma Life Support guidelines of <C>ABCDE (catastrophic hemorrhage, airway, breathing, circulation, disability, exposure).
ABG: arterial blood gases; BE: base excess ; CNS: central nervous system; CVS: cardiovascular system; FG: French gauge; G: gauge; GCS: Glasgow coma score; IV: intravenous; NGT: nasogastic tube; OGT: orogastric tube

that patients require daily awakenings to determine neurologic status and allow reevaluation of the need for sedation.

All findings must be documented. Initial and on-going reassessment is critical to appropriate care of trauma patients. This is especially true in patients who have undergone damage control surgery or whose injuries are being managed nonoperatively. Patients who undergo damage control surgery will often have more complicated management plans and require more resources. Nonoperative management of hemodynamically stable victims of both penetrating and blunt abdominal trauma has become more common. These approaches require a greater commitment to on-going patient reassessment and monitoring. Depending on the maturity of the operation and size of the hospital facility, access to radiological investigations such as ultrasound and CT will be invaluable in assisting the clinical team in the decision to follow a conservative treatment plan so that OR resources may be more efficiently utilized.

INVESTIGATIONS

Hematology

Hematology investigations for critically ill patients consist of a full blood count, including platelet number, coagulation screen or a thromboelastogram, urea, electrolytes, and regular blood glucose estimations. In massively transfused patients, K^+, Ca^{2+}, and Mg^{2+} should be checked to ensure they are within normal range. In patients with substantial tissue trauma, a creatine kinase check should be requested due to the risk of rhabdomyolysis and subsequent renal failure. In these patients ensuring normovolemia and an adequate urinary output is essential. Other investigations should be guided by the injury mechanism, eg, an electrocardiogram and troponin in those patients with chest injuries. Oxygenation and oxygenation index should be assessed in all patients (usually by pulse oximetry and blood gases when appropriate).

Imaging

A chest radiograph to ensure correct placement of lines and tubes is warranted on admission but should otherwise be performed only as clinical circumstances dictate. A CT traumagram is an invaluable tool but may not be available in all deployed environments. Where it is, close liaison with a radiologist is essential. Ultrasound is increasingly used in the ICU[1] and may rapidly provide information on hemodynamic status. Thus it is increasingly important that clinicians become familiar with the various ultrasonic techniques prior to deployment.

MANAGEMENT PLAN

Following the history and physical examination, and review of the available laboratory data and all radiology, a management plan should be formulated. This plan should involve input from all staff involved in the patient's care. Specific questions that should be addressed are:

1. What is the status of this patient's intravascular volume? Has hemodynamic stability been achieved or is continued resuscitation required? Does this patient have evidence of impaired tissue or organ perfusion?
2. Does this patient have an acute lung injury (ALI)? Does this patient require ongoing ventilation?
3. Does this patient display evidence of systemic inflammatory response, sepsis, or multiple organ dysfunction?
4. Have all the presenting injuries been identified (more likely if the patient has been admitted directly to the ICU without a trauma scan due to logistical reasons)? Is the patient at risk of developing other life-threatening conditions associated with the presenting injury pattern that may present later in the clinical course (blast lung, compartment syndromes, and renal failure)?
5. What is the discharge plan? This will depend upon the nationality of the patient and the tactical situation. The management plan may differ depending on the length of time that the patient remains in the deployed environment.

HEMODYNAMIC CONSIDERATIONS

Adequacy of Resuscitation

Early posttraumatic mortality is determined by the initial traumatic impact and success of early resuscitation. The single goal is to prevent, detect, and treat tissue hypoperfusion. Ideally, markers of resuscitation adequacy should be obtainable without specialized equipment and easy to interpret.[2] Metabolic parameters show the most usefulness in assessing resuscitation from shock.[3] The arterial base deficit (negative base excess), serum lactate, and central venous oxygen saturation [$ScvO_2$] are all readily available measures using standard blood-gas analyzers. A subtle marker of tissue hypoperfusion that is also readily obtainable is the central venous to arterial CO_2 difference [$P(cv\text{-}a)CO_2$].[4] In the presence of a $ScvO_2$ greater than 71%, a $P(cv\text{-}a)CO_2$ greater than 0.7 kPa suggests ongoing tissue hypoperfusion. Although these markers assess oxygen delivery and tissue hypoxia globally and not regionally, they provide the most consistent measures of shock severity and resuscitation response. Serum pH is a useful measure but is too easily influenced by respiratory factors and renal function to provide a reliable pure marker of tissue perfusion.

The goal of fluid replacement is best achieved by determination of fluid responsiveness. High blood to fresh frozen plasma (FFP) ratios are the norm in the early stages of resuscitation in trauma. In the later

stages of management in the ICU, the use of appropriate volumes of crystalloid solutions to avoid tissue hypoperfusion is the goal. Currently no single monitor of cardiac output has been shown to affect outcome in the trauma patient population, and therefore such monitors are not used in deployment (although this may change in the future). In the meantime, determination of fluid balance is clinically led, and care must be taken to avoid under replacement (risking acidosis and pre-renal failure) or over replacement (risking tissue edema, exacerbation of reperfusion injury, and dysfunctional leukocyte adhesion). Clinical measures include monitoring trends in hemodynamic variables and urine output in response to fluid challenges. However, these measurements have drawbacks. The endocrine response posttrauma includes fluid retention, so the intensivist should not pursue an unrealistic urinary output.

Measurements of urinary osmolality and Na^+ are simple and can be effective as a guide to the need for additional fluid. It is essential to avoid "dry-land salt-water drowning" syndrome. [5] Excess fluid and hypervolemia may induce an increase in natriuretic hormone levels; this causes destruction of the endothelial glycocalyx layer, altering the oncotic gradient across capillary endothelium and leading to increased loss of fluid into the interstitium. Compounded with the immobility from sedation and associated decreased lymphatic flow, the result is worsening tissue edema. Current practice, while supporting an aggressive fluid strategy in the management of shock, requires a change to a conservative restrictive fluid policy once resuscitation has been achieved. It is thus important for the intensivist to recognize this phase change.

Volume Status of the Cardiovascular System

Evidence supports the use of hemodynamic parameters for assessing fluid responsiveness, based on cardiopulmonary interaction during positive pressure ventilation.[6] The resulting transpulmonary pressure changes cause variations in venous return, blood pressure, and stroke volume. The amplitude of this variation is inversely proportional to volume status. Thus, hypovolemia is characterized by larger swings in blood pressure and stroke volume. Clinicians have subjectively used this method for a long time by observing swings in the arterial or plethysmographic waveform (Figure 27-2). Response to simple tests such as passive leg raising[6] (Figure 27-3)

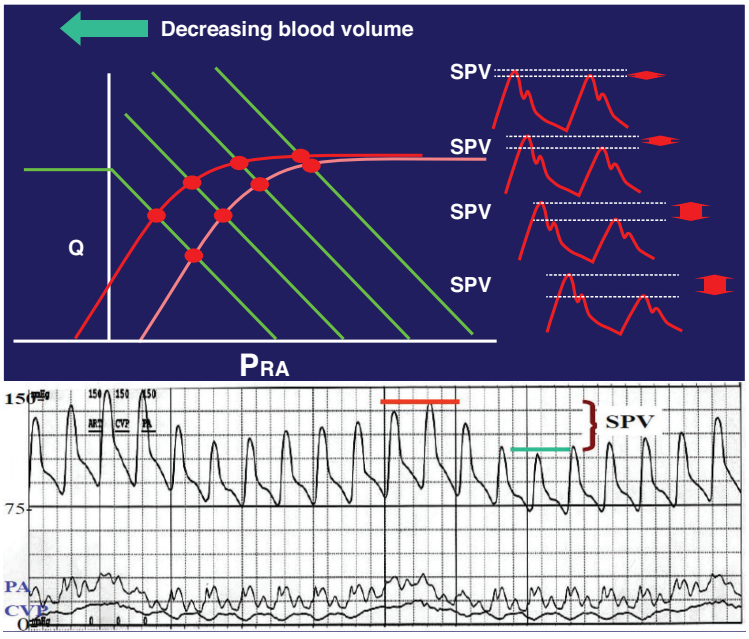

Figure 27-2. Effect of hypovolemia on systolic pressure variation (SPV). The red line indicates normovolemia; the green line indicates hypovolemia. With increasing hypovolemia there is a greater variation in systolic blood pressure between mechanical inspiration and mechanical expiration. The bottom chart shows pulmonary artery pressure (PA) and central venous pressure (CVP). ΔSPV > 10%–15% => fluid responsive.
Δ: difference; PRA: pressure of right atrium; Q: cardiac output

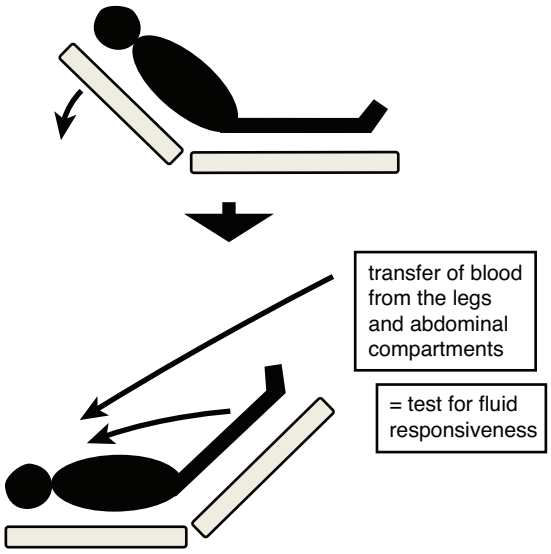

Figure 27-3. Passive leg raising test for fluid responsiveness. Approximately 150 to 200 mL in blood volume is autotransfused from the peripheral to central blood compartment. A systolic pressure variation greater than 15% implies the patient is fluid responsive.

may be useful in assessing fluid responsiveness, but this test is not possible in patients with traumatic lower limb amputations. In such patients the end-occlusion test[7] may be employed. Systolic pressure variation provides useful information on filling status when a patient is mechanically ventilated with a tidal volume greater than 7 mL/kg and in sinus rhythm (Exhibit 27-1).

Targets for blood pressure in adults should not be set arbitrarily; rather, they should be guided by adequacy of end-organ perfusion.[8] In the head-injured patient, in the absence of intracranial pressure, monitoring mean arterial pressure should be set at a higher level (> 80 mm Hg is recommended). Focused transthoracic echocardiography may be employed if available. Fluid replacement should be targeted to ensure that inferior vena caval diameter is over 1.5 cm and diameter variation with respiration is less than 20%.

In the first 24 hours of management in the deployed ICU, fluid should be administered as required, typically as boluses, and the response assessed. Maintenance fluids are not required and may serve only to cause overload and worsen tissue edema. A considerable volume of fluid is given in administering drugs (sedation, analgesia, antibiotics), and for every unit of blood and FFP, 150 mL of crystalloid is administered. Blood product replacement should be guided by the presence of coagulopathy.

The stress response to the injury or surgery causes antidiuresis and oliguria, mediated by vasopressin (antidiuretic hormone), catecholamines, and the rennin-angiotensin-aldosterone system (RAAS). Water and salt are therefore retained in the extracellular space even in the presence of overload. The excretion of excess sodium takes days and appears to be dependent on the passive and permissive suppression of the RAAS rather than any positive action of atrial natriuretic peptide. Salt and water retention is exacerbated by resuscitation with saline-rich fluids and results in peripheral and visceral edema. Following surgery, even when the serum osmolarity is reduced by administration of hypotonic fluid, the ability to excrete free water is limited because the capacity of the kidney to dilute, as well as to concentrate, the urine is impaired. Hyperchloremia from chloride ion retention causes renal vasoconstriction and reduced glomerular filtration rate, further compromising the ability of the kidney to excrete sodium and water.[9] In more seriously ill surgical patients who are catabolic with increased urea production, sodium and chloride excretion competes with excretion of nitrogen; again, much of administered sodium, chloride, and water is retained as interstitial edema. This is exacerbated by endogenous water production from fat and protein breakdown, which is increased (1 kg of either will yield approx 1 L water). Sodium reabsorption is linked to increased H^+ and K^+ excretion caused by RAAS activation and metabolic acidemia related to

EXHIBIT 27-1

MARKERS OF ADEQUATE RESUSCITATION

- Stable hemodynamics without the need for vasoactive or inotropic support
- Serum lactate ≤ 2 mmol/L
- Normal renal function (urinary output > 1 mL/kg/h)
- Core to periphery temperature gradient < 3°C
- $P(cv\text{-}a)CO_2 < 0.7$ kpa (appears to be a useful tool to identify persistent hypoperfusion when goal-directed therapy is associated with a $ScvO_2 \geq 71\%$)
- Normal coagulation (Hb > 100 g/L)
- Normothermia
- No hypoxemia or hypercapnia (not applicable when lung-protective ventilation is in use)

hypoperfusion and tissue trauma. Potassium depletion, due both to RAAS activity and the cellular loss of potassium that accompanies protein catabolism, further reduces the ability to excrete a sodium load. A sustained increase in systemic capillary permeability (as occurs during severe inflammatory responses of trauma and sepsis) allows albumin and its attendant fluid (18 mL for every gram of albumin) to leak into the interstitial space, also worsening interstitial edema. This increase in capillary permeability results in intravascular hypovolemia with further sodium and water retention by continuing activation of the RAAS and secretion of vasopressin. This "relative" hypovolemia is often associated with hypotension and is worsened by further fluid administration. Vasoactive drug support is required.[10]

Fluid balance and urine output are vitally important in the daily and ongoing evaluation of the ICU patient. Even more important is the cumulative fluid balance. Major efforts should be expended in ensuring a neutral or slightly negative fluid balance, especially once the patient is out of the resuscitation phase.

ACUTE LUNG INJURY

Acute Lung Injury, Blast Lung, and Pulmonary Contusion

Many patients develop an ALI following massive transfusion (26%), multiple trauma (16%), and pulmonary contusion (50%). ALI is thought to be the uniform expression of a diffuse and overwhelming inflammatory reaction of the pulmonary parenchyma to either a direct injury to the lung or an indirect lung injury (eg, sepsis). Blast lung (and pulmonary contusion) is a direct injury with initial diffuse bleeding within the lung (causing hypoxemia), which then progresses to an inflammatory state within the lung. ALI has the potential to progress to acute respiratory distress syndrome (ARDS).

Blast lung rarely presents alone; it is often associated with other blast-related traumatic injuries. In current conflicts blast lung results primarily from improvised explosive devices (IED). Any thoracic abnormalities on plain radiographs or CT scan should heighten the clinical suspicion of blast lung if associated with a history of blast exposure. Examples include focal or diffuse opacifications that are clearly not penetrating fragments, pneumothoraces, pneumopericardium, or pneumomediastinum. Clinical symptoms of blast lung include shortness of breath, hemoptysis, and cough associated with hypoxemia (although this may be delayed). Other injuries tend to obscure the lung injury, which may not manifest for several hours.

Management of blast lung is exactly the same as for any patient with ALI/ARDS. General management considerations for all patients include ventilation, sedation, nutrition, and infection control. For patients at high risk of ALI, smaller tidal volumes are more appropriate provided atelectasis and excessive acidemia (due to hypercapnia) are avoided or minimized. The patient with damaged lungs is at an increased risk of morbidity and mortality due to fluid mismanagement and misapplied ventilation.

Ventilation Strategies

Patients with traumatic limb amputation will invariably have received a massive transfusion. They require a lung-protective ventilatory strategy both in the OR and the ICU. It is essential that these fragile lungs do not suffer further (iatrogenic) damage. However, this strategy is not without drawbacks (Exhibit 27-2).

The goal of ventilation for patients with ALI or ARDS is to oxygenate the vital organs but minimize further lung damage (caused by stretching or shearing forces induced by the ventilator) by using a lung-protective ventilatory strategy. Below are suggestions for ventilation; however, ventilator settings must be guided on an individual patient basis.

EXHIBIT 27-2

PROS AND CONS OF LOW-VOLUME* VENTILATION IN PATIENTS AT RISK OF ACUTE LUNG INJURY

Pros

- Proven mortality benefit in those with ARDS
- Decreased cytokine production
- High V_T associated with ALI/ARDS

Cons

- Hypercapnia may cause raised ICP, myocardial depression, and pulmonary hypertension
- Atelectasis if inadequate PEEP is used
- Patients may require more sedation and use of muscle relaxants

*V_T < 6 mL/kg

ALI: acute lung injury; ARDS: acute respiratory distress syndrome; ICP: intracranial pressure; PEEP: positive end-expiratory pressure; Vt: tidal volume

- Plateau airway pressure < 30 cm H_2O, and set positive end-expiratory pressure (PEEP) at a level to maintain an SpO_2 > 90%.
- Tidal volume no more than 6 mL/kg (lean body weight); lower if possible.
- Permissive hypoxemia may represent a reasonable strategy in the presence of severe lung injury, but minimizing oxygen consumption rate will be required. Target PaO_2: 7 kPa (pO_2 = 50 mm Hg).
- Pursue a strategy of permissive hypercarbia. A target pH taking into account any metabolic acidemia of as low as 7.1 may be acceptable. To counter a low pH the respiratory rate should be altered (up to 30 breaths/minute) in preference to increases in tidal volume.
- Advanced ventilatory techniques such as airway pressure release ventilation may be appropriate to enable alveolar recruitment maneuvers, but may not be available.
- Aim for an FiO_2 < 0.5 or the lowest FiO_2 consistent with an acceptable blood oxygen level.
- Prone positioning may be beneficial if the appropriate use of FiO_2, PEEP, recruitment maneuvers, and neuromuscular blockade has failed to achieve adequate oxygenation.
- Endobronchial toilet in the presence of pulmonary hemorrhage may be required if clot obstruction is contributing to hypoxemia. Segmental obstruction on chest radiograph or CT should prompt careful consideration of the potential risks and benefits of performing endobronchial suction/bronchoscopy.
- United Kingdom Clinical Guidelines for Operations recommends considering recombinant factor VIIa (rFVIIa) in pulmonary hemorrhage related to blast lung.[11]

Sedation

Currently combination treatment with propofol and fentanyl (or remifentanil) is the mainstay of sedation. Benzodiazepines should be used with caution (because of the high incidence of delirium) and barbiturates reserved for treatment of intractable seizures. Sedation should be titrated to the Richmond Agitation-Sedation Scale (RASS) score, unless there are indications for deeply sedating the patient, eg, severe closed head injury with evidence of raised intracranial pressure. The RASS has been shown to be a reproducible and reliable instrument with high inter-rater reliability. Sedation breaks should be considered. The daily interruption of sedative administration allows accumulated sedative agent to dissipate, permits the patient to recover consciousness for assessment purposes, and facilitates recovery from the sedated state. There is a lower incidence of post-traumatic stress disorder in the civilian population with this approach.

Reduction in sedation and a spontaneous breathing trial should be performed as early as possible. The question of awakening patients who are to be transferred may arise. This decision must be discussed with the aeromedical team. If the tactical tempo and patient pathology permit, it may be preferable to extubate patients prior to aeromedical evacuation, provided adequate analgesia is established. Transferring ventilated patients to a Role 4 hospital carries risks that can be minimized if the patient is awake and stable; however, these patients must be truly stable with no risk of deterioration in-flight.

Nutrition

The maturity of the military operation will dictate the sophistication of caloric replacement therapies available. In general, the mainstay of treatment is to start enteral nutrition once resuscitation has been completed. Close liaison with the surgical team will facilitate the placement of feeding tubes at initial surgery and the subsequent timing of nutrition initiation. Parenteral feeding remains controversial (even in the civilian setting) and is outside the parameters of the first 24 hours of care.

Although most service personnel are premorbidly physically fit, adequate, balanced nutrition in austere environments can present challenges. Additionally, the nutritional state of any local nationals presenting to the field hospital may vary widely. Following injury, polytrauma patients are catabolic, and providing sufficient protein, fat, and carbohydrate often remains a problem into Role 4. For this reason, if feeding can be commenced in the first 24 hours, it should be.

Infection Prevention and Control

In the military setting, traumatic wounds are often penetrating and heavily contaminated. Dirt and debris must be subject to extensive debridement as soon as possible. Coverage with empirical antimicrobials should be provided as early as possible, following locally generated clinical guidelines for operations. Within the ICU setting, signs of sepsis should be treated aggressively in a multidisciplinary manner; further debridement is often more successful than stronger antibiotics. If the tactical tempo allows, changing invasive lines placed before the ICU admission should be the rule.

MANAGEMENT PROBLEMS FOLLOWING DAMAGE CONTROL SURGERY

The goals of therapy in the ICU after damage control surgery and hemorrhagic shock are well-defined:

1. correct metabolic acidosis;
2. restore normothermia;
3. reverse coagulopathy; and
4. ensure adequate oxygen delivery and consumption.

Correction of the physiologic dysfunction is necessary prior to returning to the OR. The components of the "lethal triad" (items 1–3) act synergistically. Of the three, coagulopathy is most affected by the presence and severity of the other two. Preventing or attenuating the severity of coagulopathy depends on limiting ongoing tissue injuries and hypoxia, and preemptively transfusing clotting factors and platelets. In injured patients arriving from the OR following damage control resuscitation, disturbances in coagulation may be observed at a temperature of 35°C. The contributing factors of coagulopathy should therefore be aggressively addressed, in an effort to minimize their additive effects, until normothermia is restored. To treat any hypothermia, all intravenous fluids (especially blood products) should be warmed and forced-air warming blankets at 42°C for the patient should be used. Additionally, the efficiency and overall activity of most clotting factors are substantially reduced in an acidic environment (pH < 7.40). In patients with ongoing hemorrhage, resuscitation with blood, FFP, and platelets, as well as tranexamic acid, Ca^{2+}, and rFVIIa, may be required. There is no place for colloid or crystalloid use. Similarly, there is no place for hypertonic saline except in the management of raised intracranial pressure. Use of thromboelastogram (ROTEM [TEM International GmbH, Munich, Germany]) and blood counts will assist in treatment guidance. Repeated clinical and lab assessment, especially with respect to volemic status, is essential.

PATIENT DISCHARGE

The maturity and tempo of the operation will dictate the options for patient discharge. United Kingdom, US, and coalition patients whose level of care cannot be swiftly reduced will need to be returned to their host nation via a critical care team. This is covered in more detail elsewhere (see Chapter 38, Air Transport of the Critical Care Patient). Patients who can be transferred to the ward will need detailed documentation of their present and future care and appropriate handover to ward staff. Wherever possible, the ICU staff can continue providing outreach care and support to the wards, especially when epidural analgesic techniques are employed.

SUMMARY

Military traumatic injuries have multiple presentations; however, their ICU management is relatively straightforward and is directed toward reversing the lethal triad. The importance of serial reevaluation cannot be overemphasized. Teamwork among all providers is also essential.

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Chapter 28

DAMAGE CONTROL PHILOSOPHY IN CRITICAL CARE: PATIENT MANAGEMENT AND ORGAN SUPPORT

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INTRODUCTION

Following ballistic trauma, there are two clear goals of damage control during intensive care:

1. To recognize and treat the complications of all primary injuries.
2. To identify and manage the complications of therapy and prevent iatrogenic injury.

Ten years ago, United Kingdom (UK) and US military intensive care was provided as part of a sequential package following initial resuscitation and damage control surgery. Operative procedures during this stage were limited to 60 minutes so that surgeons focused on "life and limb" interventions, controlling bleeding, and removing contamination. Following temporary wound closure, the patient was resuscitated in critical care, with targeted interventions against the lethal triad of acidosis, hypothermia, and coagulopathy. With corrected physiology, the patient could later be returned to surgery for second-look procedures.

Experience gained in Iraq and Afghanistan has led to refinement of treatments and development of damage control resuscitation. Surgeons now follow an integrated model of care involving permissive hypotension, hemostatic resuscitation, and damage control surgery.[1-4] In combination with massive transfusion,[5,6] targeted warming, and near-patient testing of coagulation and arterial blood gases, damage control resuscitation is so successful that patients can potentially emerge from surgery normothermic, euvolemic, and with normal blood gases and clotting profile. These techniques have allowed surgeons to extend operating times and perform more surgical procedures, in particular vascular repairs, extensive debridement, and the external fixation of fractures, in a single episode of surgery.

Despite these advances, significant challenges remain for the deployed intensivist. Even with intraoperative resuscitation, changes in coagulation and volume status will likely develop due to ongoing consumption of clotting factors and third space losses. Not all patients have initial resuscitative surgery in Role 3 facilities, and consequently blood products and near-patient testing may be limited. These patients will require transfer to a Role 3 facility for further investigation, resuscitation, and management. During major incidents, patients may need direct admission to the critical care unit until an operating room table becomes available. Finally, those managed in Role 3 need appropriate preparation before transfer to tertiary services for second-look and definitive care. Consequently, critical care can be involved at any point on the damage control resuscitation and evacuation pathway.

Despite the complexity of their injury patterns and the large transfusions given, patients with major trauma can be saved if simple rules are followed. Clinicians must avoid assumptions, be meticulous, and follow a structured approach with an active, regular review process. Care must be proactive and targeted to prevent the lethal triad. It is good practice to follow the "ABCDE" approach, as explained below, and to remember the goals of treating all primary injuries and preventing iatrogenic insults.

AIRWAY

For any patient requiring mechanical ventilation, it is mandatory to use the correct size endotracheal tube, insert it to the correct length, and inflate the cuff to the correct pressure. Furthermore, the tube must be securely attached to the patient, mindful of the patient's injury pattern. Failure to apply these rules can cause a number of iatrogenic complications.

Currently, cuff pressures are rarely measured prior to admission to intensive care, and particularly with out-of-hospital intubations, cuffs are likely to be overinflated in the field to quickly secure the airway. Consequently, it is mandatory to check cuff pressures on admission to intensive care to limit long-term sequelae from mucosal ischemia such as subglottic stenosis.

Unrecognized endobronchial intubations can cause segmental lung collapse, which limits gas exchange and mimics hemothorax radiologically. In the presence of multiple injuries, these radiographic findings can lead to the unnecessary placement of chest tubes. Children are particularly at risk of endobronchial intubation because of their shorter airways.

Care should be taken when securing endotracheal tubes to ensure that they do not migrate. Local injuries may make it necessary to use adhesive tape, commercial tube holders, or sutures. Tape ties must not be allowed to obstruct neck veins or cause pressure areas.

BREATHING

Patients requiring mechanical ventilation should be managed with a protective lung strategy to prevent injury from excessive volume or pressure. To prevent such injuries, tidal volumes must be limited

to a maximum of 6 mL/kg. Carbon dioxide clearance is facilitated by increasing respiratory rates (up to 30 breaths per minute), although in the absence of a concurrent metabolic acidosis, hypercapnia can be tolerated providing the pH remains above 7.25. All patients should have end-tidal CO_2 monitoring as part of their respiratory care package.

Patients should be ventilated with 5 cm of positive end-expiratory pressure (PEEP) to prevent basal atelectasis, but PEEP can be increased if oxygenation is difficult. $Pa0_2$ greater than 8 kPa is an acceptable target for most patients.

A chest tube with suction system or Heimlich valve is mandatory for patients with a pneumothorax who need aeromedical evacuation,[7] but all chest drains should be checked to ensure they are correctly positioned in the chest cavity and that pneumothoraces and fluid collections are draining appropriately. Regular checks should ensure that the drains are intact, without disconnection, and that any suction is applied within safe limits. All drains should be removed at the earliest opportunity to reduce the risk of infection.

CIRCULATION

Maintaining Blood Pressure

Damage control resuscitation includes permissive hypotension (blood pressure targeted to a palpable radial pulse), hemostatic resuscitation, and damage control surgery. Permissive hypotension is permitted only during the first hour until control of massive hemorrhage has occurred and is not usually a feature of critical care.[2] Hemostatic resuscitation is a strategy using blood and blood products as primary resuscitation fluids. The UK policy is to give blood, fresh frozen plasma, and platelets in a ratio of 1:1:1, unit for unit. Patients receive doses of tranexamic acid and cryoprecipitate as guided by rotational thromboelastometry (ROTEM [Tem International, Munich, Germany]), and ionized calcium is kept above 1 mmol/L, monitored by arterial blood gas analysis. This policy is followed from point of wounding through critical care.

Clotting activity is known to fall by 10% for every 1°C fall in core temperature; consequently, it is important to correct hypothermia to interrupt the lethal triad. Blood and blood products (excluding platelets) are given through high volume infusion equipment (examples include the Level One Fast Fluid Warmer [Smiths Medical, Ashford, Kent, UK], or the Belmont Rapid Infuser [Belmont Instrument Corp, Billerica, MA]). The warming capabilities of these devices, in combination with forced air warming blankets and heated mattresses, ensure that patients achieve normothermia. Considerable care should be taken not to overheat patients, since the vasodilatation associated with warming might accentuate relative hypovolemia.

Guided by hemoglobin, lactate, bicarbonate, and base deficit, together with central venous pressure (CVP) and mean arterial pressure (MAP), blood products are often given in large volumes. As a consequence, infusions of vasopressors are seldom required in the operating room because most patients can be resuscitated to euvolemia. Despite concerns that stored blood might contribute to the metabolic acidosis of hypovolemic shock,[8,9] experience shows that the acidosis usually resolves during resuscitation. Failure to correct acidosis typically results from a missed or overwhelming injury.

Advanced cardiac output monitoring is usually not required in the operating room or intensive care units, but clinicians should avoid fluid overload once hemorrhage has been controlled. Although pulmonary edema can be readily treated by increased PEEP and doses of furosemide, lung contusions are common in trauma patients. The combination of mechanical injury (contusion) with iatrogenic insult (edema) can cause hypoxemia, which is poorly tolerated by those with concurrent head injury. Efforts to improve oxygenation in the injured lung by increasing PEEP can lead to barotrauma and raised intracranial pressure (ICP). Pulmonary edema is probably the only indication for furosemide in the first 24 hours of admission.

Complications of Fluid Administration

Patients who have received large volumes of blood products are at risk from hyperkalemia, arrhythmias, and cardiac arrest. Although these concerns are reduced by administering calcium as part of the transfusion protocol, calcium does not eliminate these risks. The presence of tented T-waves on electrocardiogram (Figure 28-1) indicates dangerous hyperkalemia and should be treated acutely with 10 units of insulin with 50 mL of 50% dextrose. Electrocardiogram changes (tented T waves, ST segment depression, and widened QRS > 0.12 sec) are expected to resolve and plasma potassium levels return to the normal physiological range following treatment. It is important to avoid episodes of hypoglycemia when managing hyperkalemia, and consequently arterial blood gases should be taken to measure both the response of glucose and potassium to this intervention.

Arbitrary targets for MAP (> 60 mm Hg) and CVP (>10 cm H_2O) can be set, but must be associated with improvements in metabolic function. Ventilated and sedated trauma patients tolerate hypovolemia poorly

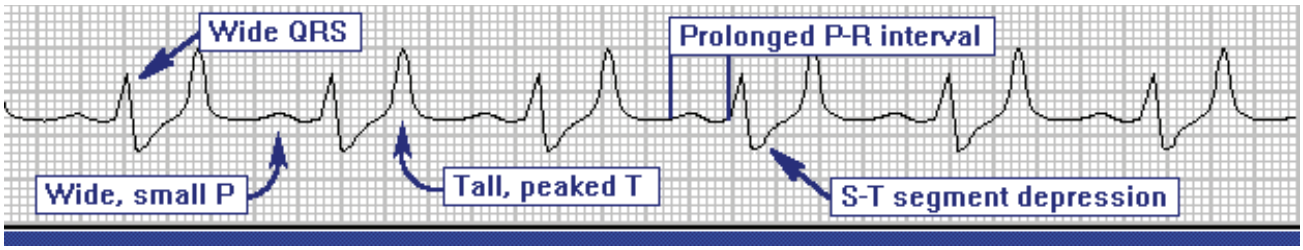

Figure 28-1. Changes in the T wave (tall, peaked, or tented) give the earliest indication of hyperkalemia. Other changes that might be seen include wide, flat, or absent P waves; prolonged P-R interval; S-T segment depression; and widened QRS complexes.

because positive pressure ventilation reduces venous return and cardiac output. This effect is compounded by the use of sedative drugs, which are negatively inotropic and vasodilating in effect. Following the completion of surgery and return to the intensive care unit, it may be necessary to use a low-dose vasopressor and inotropes to achieve a suitable MAP, provided the patient is euvolemic.

A urine output of 1 mL/kg/hour is desirable, but the stress response may reduce this amount by increasing water retention in the kidneys. If the patient's acid base, urea, and creatinine are normal (or correcting), there is no need to increase urine output with fluid boluses, and furosemide should not be administered. In clinical scenarios where rhabdomyolysis and myoglobinuria are demonstrated, it is desirable to have a good urine output, but the restoration of euvolemia and MAP are appropriate means to achieve this, rather than loop or osmotic diuretics.

Avoiding large volumes of crystalloid solutions during resuscitation has a number of advantages. The negative impact of these solutions on coagulation, acid base balance, and renal function (hyperchloremia) is avoided. Anecdotally, there appears to be less tissue edema in patients who have had hemostatic resuscitation, reducing the incidence of bowel dysfunction and abdominal compartment syndrome.

Compartment Syndromes

Compartment syndromes result from increased pressure in a closed body compartment, eg, tension pneumothorax, pericardial tamponade, and increased ICP, all conditions that are observed in the victims of ballistic trauma. Extremity compartment syndromes can occur following any mechanism of injury but are most common following fractures. Inflammation and edema following injury (and fluid resuscitation) cause swelling and increased pressure within fascial compartments. Microvasculature is compressed, causing ischemia and rhabdomyolysis, with subsequent lymphatic and venous drainage obstruction, resulting in venous congestion. If left untreated, compartment syndrome can have devastating consequences such as muscle necrosis, ischemic contractures, infection, and delayed union of fractures. For this reason, fasciotomies are often performed electively in patients with an injury pattern placing them at risk. When a fasciotomy is not performed, the intensivist must be alert to the possibility of compartment syndrome, because muscle necrosis may have commenced by the time peripheral pulses are lost.

Abdominal compartment syndrome (ACS) is described as intraabdominal pressure greater than 20 mm Hg associated with new onset end-organ dysfunction or failure.[10] Patients who have had an emergency laparotomy for trauma are at high risk for developing ACS even with a laparostomy. ACS has many causes, including intraabdominal packs; post-resuscitation visceral edema; intraperitoneal, retroperitoneal, or pelvic bleeding; and the failure of conservative measures to treat solid organ injury. Intraabdominal hypertension can affect the cardiovascular, respiratory, renal, alimentary, and central nervous systems. Raised intraabdominal pressure impedes venous return to the heart and causes sequestration of blood in the lower extremities. The diaphragm is pushed cephalad, decreasing thoracic compliance, and increased airway pressures are required for mechanical ventilation. Additionally, functional residual capacity falls, and ventilation perfusion mismatch impairs oxygenation. In the face of massive transfusion, these changes could be misinterpreted as acute lung injury.

Oliguria or anuria despite aggressive fluid resuscitation is a typical sign of ACS. The mechanisms for these conditions include direct compression of the renal parenchyma, decreased renal perfusion secondary to a fall in cardiac output, and increased water and sodium retention through activation of the renin angiotensin system.

ACS increases ICP by increasing intrathoracic pressure and impeding venous return from the brain. Gut

perfusion falls due to reduced cardiac output and increasing splanchnic vascular resistance, causing tissue ischemia.

The significant consequences of ACS and the danger of mistaking it for other conditions mandate that it be identified at the earliest opportunity by measuring intrabdominal pressure in all at-risk patients. ACS can occur in the absence of intraabdominal pathology, representing whole body ischemia reperfusion injury, and is associated with resuscitation from massive shock, eg, following multiple extremity fractures, traumatic amputations, pelvic fractures, and penetrating chest injury.

Although successful in treating coagulopathy and acidosis, hemostatic resuscitation is not without attendant risks. Hyperkalemia, pulmonary edema, and urticarial reactions are often seen. Transfusion-related acute lung injury is also a risk, and later complications such as infection and thromboembolism are currently being assessed. Therefore, it is important to use blood products appropriately and recognize the transition between active volume resuscitation against hemorrhage and the vasodilating, systemic inflammatory response syndrome when vasopressors may be more beneficial.

DISABILITY

The presence of traumatic brain injury (TBI) considerably complicates the management of trauma patients, but damage control principles can be used to limit secondary brain insults. Periods of hypotension are poorly tolerated by patients with TBI, and consequently permissive hypotension should not be practiced during initial resuscitation. In the field critical care unit, it is unlikely that invasive ICP monitoring will be available (although at the time of writing this situation is under review for UK forces). The intensivist must therefore make an assumption about ICP based on computed tomography imaging, pattern of injury, and clinical findings. Evidence suggests that neurological outcomes are worse if cerebral perfusion pressure falls below 60 mm Hg, so MAP must be targeted at 80 to 90 mm Hg, assuming an ICP of 20. This MAP level contrasts with the lower blood pressures tolerated in the absence of TBI, and may require the earlier use of vasopressors. Vasopressors can complicate the management of metabolic acidosis because they may be required before euvolemia has been achieved and can contribute to tissue ischemia through their vasoconstricting effects.

Patients should be adequately sedated and nursed head up (30°), ensuring that endotracheal tube ties do not obstruct venous drainage of the head and neck. Patients should be ventilated to normocapnia ($PaCO_2$ of 4.5–5.5 kPa, with $PaO_2 > 10$ kPa). Maintaining this ventilation may be challenging, necessitating protective lung strategies that might result in a degree of respiratory acidosis. In this potentially no-win situation, the intensivist must balance the consequences of increased ICP against the risk of acute lung injury and acute respiratory distress syndrome.

Furthermore, in the case of a lung contusion or edema, the clinician must weigh the risk of increased PEEP on ICP against an acceptable target for PaO_2. Efforts to improve the mechanics of ventilation should be considered, including muscle relaxation and relieving intraabdominal hypertension.

The intensivist must be cautious not to over-warm the patient while attempting to limit the coagulopathic consequences of hypothermia since pyrexia is known to elevate ICP. In the absence of ICP monitoring, mild degrees of hypothermia may represent the favorable risk.

Fluid and electrolyte disturbances secondary to TBI can be profound, and the clinical goal should be maintaining a euvolemic patient with normal sodium (145–150 mmol/L) and electrolytes. Doses of mannitol or hypertonic saline should not be given indiscriminately but only when clinically indicated. Hypoglycemia and hyperglycemia should be avoided in TBI.

Some patients may need to wear hard cervical spine collars because of real or anticipated injury. These devices can obstruct neck veins and cause local pressure areas and infection. Consequently, they should be removed in ventilated sedated patients and replaced with sand bags and tape, adjusted as necessary when the patient is turned as part of pressure area care.

ENVIRONMENT

Record-keeping and Imaging

Patients often receive definitive treatment at facilities remote from their initial resuscitation, so it is imperative that meticulous records are kept, including notes on injury patterns, investigations, procedures, drugs, and fluids administered. All patients should travel with a copy of these notes and any imaging performed.

Patients normally have a whole body computed tomography scan as part of the trauma screen to identify all injuries. Where practicable, the scan is done prior

to surgery, but in cases of refractory shock it may be delayed until after damage control surgery. It is vital that the critical care team have a full written report of all imaging, and that all surgical teams are made aware of the findings.

Gut

Damage control laparotomy for bowel injury can result in multiple resections, with blind ends left stapled and the bowel in discontinuity. Enteral nutrition is contraindicated, but a nasogastric tube should be left in situ to drain gastric collections. An alternative supply of calories can be provided with a 10% dextrose solution at 40 mL per hour. If enteral nutrition is expected to be delayed for a considerable period(> 72 hours), total parenteral nutrition should be considered, but it must not be commenced through dirty trauma lines. Blood glucose should be measured and kept within the range of 4 to 10 mmol/L. This range provides sufficient margin of error to limit iatrogenic hypoglycemia, while protecting against the adverse effects of hyperglycemia, especially in TBI. If the bowel is continuous and there are no primary anastomoses, enteral nutrition should start at the earliest opportunity.

Gastric Protection

All patients should receive gastric ulcer prophylaxis with H_2 receptor antagonists, and if the bowel is in continuity, prokinetics and aperients as indicated.

Venous Thromboembolism Prophylaxis

Trauma patients are at increased risk of embolic events and should use mechanical devices such as stockings and compression boots as allowed by their injury patterns. Low molecular weight heparins are indicated once there is no risk of bleeding and in the absence of TBI.

Intravascular Lines

Following initial resuscitation and surgery, any peripheral and central lines that are not being used should be removed to limit infection, prevent clots, and preserve veins for later use. Patients should be examined to ensure that all intraosseous devices have been removed, and all central lines should be transduced to confirm that they are placed in the venous system. Trauma lines sited in the emergency department and during initial surgery are by definition "dirty," but it is current practice to leave these in situ until second-look procedures at the tertiary facility.

Position

All patients should be nursed head up (30°) with attention to pressure areas, because this position aids ventilation and helps reduce microaspiration and ventilator-associated pneumonia. Similarly, spinal patients nursed supine should be managed by tilting the bed 15° to 30° head up.

Topical Negative Pressure Dressings

Care should be taken to ensure that topical negative pressure dressings are functioning correctly and that any measured losses are factored into the daily fluid balance. Although any active bleeding is expected to be controlled by surgery, ongoing drain losses associated with physiological changes should prompt urgent surgical review.

Transfer

To reach tertiary services, most patients require long journeys by air. During the flight, patients must be actively managed as an extension of their intensive care[7] (see Chapter 38, Air Transport of the Critical Care Patient).

Drugs

Any antibiotics and pain medications should be given according to local guidance and policy.

Regional Techniques

Once coagulopathy is controlled, peripheral nerve blocks and epidurals can be sited to treat acute pain and reduce the long term sequelae of chronic pain syndromes.

SUMMARY

The immediate survivors of ballistic trauma often have complex and life-threatening injuries. However, by using an integrated, targeted, and cooperative approach to trauma care, initiated early after wounding, many patients recover despite high injury severity scores.[11] Subsequently, the ability of these patients to accept and live with disability and their desire for rehabilitation vindicates the substantial medical assets deployed in their care.

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Chapter 29

MECHANICAL VENTILATION OF THE TRAUMA PATIENT IN THE FIRST 24 HOURS

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INTRODUCTION

Trauma presents an immense global problem to both military and civilian populations. It is the most common cause of death in the first 3 decades of life and the third most common cause of death overall in the civilian population. Injury to the chest contributes to 30% of all combat-related deaths in the current military conflict in Afghanistan and 56% of lethal civilian trauma.[1]

Trauma to the chest can be classified according to whether the mechanism of injury is penetrating or blunt. Conditions common to both mechanisms of injury include hemothorax, cardiac tamponade, pneumothorax, rib fractures, and flail chest.[2] Pulmonary contusion is common in blunt chest trauma, but may also occur in association with high-velocity penetrating injuries. The passage of a shock wave through the pulmonary tissue leads to microscopic disruption at the alveolar-air interface.[3] This results in alveolar hemorrhage and parenchymal damage, which becomes maximal at 24 hours. Blast lung injury is an extreme form of pulmonary contusion and should be borne in mind when lung function deteriorates in the absence of signs of external thoracic injury. As the blast wave passes, there is a rapid expansion of the gas-filled alveoli, leading to secondary explosions within the lung.

Regardless of the mechanism of direct injury to the lung, the pathophysiology consists of an initial injury to the parenchyma followed by damage to the alveolar-capillary barrier due to inflammation. Inflammatory infiltrates, capillary leak, atelectasis, and later fibrosis all combine to cause a loss of compliance.[4] Injury to the lung can also be incurred by a variety of extrapulmonary insults, including sepsis, aspiration pneumonia, multiple trauma, and massive transfusion.[5,6] The relationship between severe injury to the lung and other organ failures in polytrauma is even more complicated. It has been suggested that lung injury can be both the consequence and cause of multiple organ failures.[7]

The terms acute lung injury (ALI) and acute respiratory distress syndrome (ARDS) have been developed to represent the severity of lung dysfunction without specifying the etiology. The American-European Consensus Conference on ARDS standardized the definition of both terms on the basis of the following: (1) acute onset of respiratory distress; (2) bilateral infiltrates on frontal chest radiograph; (3) absence of left atrial hypertension; (4) a pulmonary capillary wedge pressure of 18 mm Hg or less; (5) severe hypoxemia, which is defined as a PaO_2 to fraction of inspired oxygen (FIO_2) ratio up to 300 mm Hg in ALI and up to 200 mm Hg in ARDS.[8]

The wide range of severity in lung injury makes a robust system for concomitant escalation of ventilatory support necessary. This chapter will describe how mechanical ventilation can be safely initiated and appropriately adjusted in the military environment.

PRINCIPLES OF SAFE VENTILATION

The mainstay of managing lung injuries is mechanical ventilation, which replaces or assists the function of the respiratory system. Mechanical ventilation may in itself exacerbate the initial injury by direct mechanical damage, induction of surfactant failure, or stimulation of pulmonary and systemic inflammatory cytokines.[9] Improved understanding of the pathophysiology of ARDS has led to the development of safer ventilatory practices.

Tidal Volume

Traditionally, the primary aim of mechanical ventilation was to ensure oxygenation and control arterial carbon dioxide levels. To this end, tidal volumes of up to 15 mL/kg of ideal body weight were employed.[10] A large clinical study by the ARDS Network demonstrated that conservative ventilation using tidal volumes up to 6 mL/kg together with a limit of maximum plateau pressure to 30 cm H_2O conferred a highly significant improvement in 28-day mortality when compared with traditional ventilation strategies.[11] This ventilation strategy is now widely employed to prevent adjuvant ventilator-induced lung injury (VILI), but the strategy's main drawback is hypoxemia. The degree of hypoxemia that can be safely tolerated remains contentious, but it is clear that a more aggressive oxygenation strategy is necessary in the presence of significant tissue hypoxia manifesting as hemodynamic instability, lactic acidosis, and organ dysfunction.

Monitoring and Optimizing Ventilation

Modern microprocessor technology incorporated into ventilators provides a wide range of monitoring software to optimize interaction between the patient and the ventilator. Clinically useful options include pressure-volume loops, real-time inspiratory and expiratory flow profiles, measurement of intrinsic positive end-expiratory pressure (PEEP), and expiratory capnography. As discussed above, the plateau pressure is a prime target in the strategy of protective

ventilation. However, it should be remembered that the plateau pressure value depends on PEEP and respiratory system compliance. Dynamic flow-volume and flow-pressure loops have become increasingly popular, but they are inferior to static assessments and may underestimate the lung volume at which the upper inflection point is reached.

Positive End-Expiratory Pressure

Increasing PEEP may lead to recruitment of collapsed alveoli and improve the shunt fraction and PaO_2. If the recruitment potential is low, an increase in PEEP may not only have a marginal effect on the shunt fraction, but may also contribute to over-distension of open alveoli and increase the risk of VILI. The potential for recruitment can be identified by the following methods:

1. After a 30-minute trial of increased PEEP, minimal potential for recruitment is identified by minimal change or worsening PaO_2, increased dead space, and worsening compliance.
2. Assessing the shape of the pressure-time curve during delivery of a constant flow inflation can allow estimation of compliance. Worsening compliance is depicted by an upward concavity of the curve, and improved compliance is depicted by a downward concavity. Improving compliance during inflation suggests that the potential for recruitment is high and that an increase in PEEP may be efficacious.
3. Assessing the pressure-volume curve during inflation will yield the pressure at which the lung begins to inflate. Maintenance of PEEP above this lower inflection point will keep recruitable alveoli open at the end of expiration and prevent repetitive recruitment and de-recruitment during the ventilation cycle.
4. Assessing the intrapleural pressure with an esophageal balloon may allow a more precise setting of PEEP above the intrapleural pressure and prevent alveolar collapse. However, the constraints with this measurement technique include artifact or noise from cardiac contractions, compression of the esophagus by mediastinal contents in the supine patient, and nonuniformity in the distribution of pleural pressure; any of these occurrences renders the single site of pressure measurement in the esophagus highly inaccurate.

The optimum level of PEEP and how to determine it remain controversial. Three randomized controlled trials have evaluated modest versus high levels of PEEP in patients with ALI and ARDS.[12–14] There was no survival advantage for a higher level of PEEP, but there were lower rates of refractory hypoxemia and use of rescue therapies. Post hoc analysis of this combined data showed that high PEEP levels conferred less benefit and more adverse effects to patients with mild lung injury when compared to patients with ARDS. This finding highlights the importance of making a comprehensive assessment of the potential benefits and risks of high PEEP on an individual basis.

MODE OF VENTILATION

The choice of mode of ventilation primarily depends on local factors, such as the technology available and experience of the physician. A mechanical ventilator employs a flow or pressure generator to deliver an inspiratory volume. Termination of inspiration occurs when inspiratory time, airway pressure, or tidal volume is achieved. Expiration is passive. Cycling to the inspiratory phase usually depends on time but may be triggered by the patient.

Volume-Preset Ventilation

The guarantee of a tidal volume in this mode is attractive, but it is the least forgiving mode and may lead to high airway pressures, gas trapping, and cardiovascular compromise. Additionally, there is no added flow reserve to compensate for leaks. The relatively low rates of flow generated at standard settings may result in homogenous ventilation and perpetuate any existing mismatch of perfusion and ventilation. Modifying this system by adding a pressure limit and increasing the maximum inspiratory flow to over 100 L/min results in a safer and more flexible variant, but it is essentially analogous to pressure-control ventilation. Pressure-regulated volume control is a hybrid mode in which peak inspiratory pressure is minimized for a given preset tidal volume. This mode confers additional safety but has limited ability to compensate for large or variable leaks.

Pressure-Preset Ventilation

The pressure generator system delivers a constant pressure, but flow decreases during the inspiratory

phase. The high initial inspiratory flow rates allow distribution of gas according to local expiratory time constants. Lung units with low levels of intrinsic PEEP are preferentially ventilated early in inspiration, and those with higher levels receive later and overall less ventilation. Together with the intrinsic pressure limit, a protective ventilation strategy is employed with improved matching of ventilation to perfusion. Commonly used pressure-preset modes include pressure-control ventilation and spontaneous variants, such as airway pressure-release ventilation (APRV) and biphasic positive airway pressure ventilation (BIPAP).

Modern ventilators permit a descending ramp of flow with volume-preset modes, which produces a result similar to the pressure-preset modes. The differences between pressure- and volume-preset modes are now relatively minor, and more emphasis is placed on ensuring that the following are tailored to each patient: tidal volume, plateau airway pressure as an estimator of average alveolar pressure at the end of inspiration, PEEP, and inspiratory time.

EXHIBIT 29-1

CONTRAINDICATIONS FOR POSITIVE PRESSURE VENTILATION

- Cardiac or respiratory arrest
- Decreased consciousness
- Hemodynamic instability
- Trauma or surgery to the face
- Severe upper gastrointestinal bleeding
- Inability to protect the airway
- Increased airway secretions
- Risk of gastric distension and aspiration of gastric contents

Inverse-Ratio Ventilation

This mode allows a period of inspiration of up to three times greater than the period of expiration. Additional recruitment of alveoli occurs due to the elevation of mean airway pressure and intrinsic PEEP. However, a number of controlled clinical studies have reported minimal or no benefit in patients with ARDS using this mode when compared to conventional ventilation strategies.[15–19] In fact, inverse-ratio ventilation may confer a significant risk of hemodynamic compromise.

Spontaneous Ventilation

In minor cases of pulmonary contusion, supplemental oxygen by mask coupled with good analgesia and chest physiotherapy may be sufficient to prevent hypoxemia. Noninvasive positive pressure ventilation has been described for trauma patients, but there are a number of criteria that preclude its use in this group (Exhibit 29-1). Noninvasive positive pressure ventilation can be applied via a tightly fitting facial mask or nasal pillows. The two modes available apply either a continuous level of positive airway pressure (CPAP) or alternate between two levels of positive pressure during the respiratory cycle (BIPAP).

Modern mechanical ventilators can also permit spontaneous ventilation as part of invasive ventilatory support. A wide range of modes are available to facilitate spontaneous ventilation, but those in most common use are BIPAP and APRV. APRV is a system that intermittently varies two levels of CPAP with a very short expiratory time. The high airway pressure maintains adequate alveolar recruitment and together with the F_{IO_2} determines oxygenation. Alveolar ventilation is determined by the timing and duration of the pressure release together with the fraction of the cycle dedicated to low airway pressure. An active exhalation valve allows the patient to breathe spontaneously throughout the ventilator cycle. A time ratio for high to low airway pressures of up to 9:1 can be used, which maximizes recruitment. However, if too little time is allocated to low airway pressures, expiration may be incomplete. Crossover studies reported lower inflation pressures, improved oxygenation, and lower sedation requirements when this mode was compared with a pressure-preset mode.[20–25] Two small randomized clinical trials suggested APRV was superior to conventional ventilation.[26,27] However, this finding may not be relevant to current clinical practice because the definition of conventional ventilation is now markedly different.

Invasive BIPAP ventilation can be used along a spectrum from total mandatory ventilation to a single level of CPAP, through various intermittent mandatory and patient-triggered options. The benefits of this mode include an inherent pressure limit, reduction in shearing forces attributed to a half-sinusoid inspiratory flow pattern in spontaneous modes, additional recruitment of alveoli afforded by spontaneous variation in the depth of breathing, compensation for leaks, a pressure-generator gas distribution profile, and lower sedation requirements.

Independent Lung Ventilation

Independent lung ventilation (ILV) allows independent management of the lungs in the presence of the asymmetrical pulmonary pathologies listed in Exhibit 29-2. It is an obvious extension to the practice of one-

EXHIBIT 29-2

ASYMMETRICAL PULMONARY PATHOLOGIES

- Airway protection in massive hemoptysis or purulent disease
- Unilateral lung disease with marked mismatching of ventilation to perfusion, including:
  - o Acute respiratory distress syndrome
  - o Pneumonia
  - o Pulmonary contusion
  - o Pulmonary hemorrhage
- Bronchopleural fistula
- Severe unilateral airway obstruction

lung anesthesia, and its application to the critical care setting is attributed to the introduction of the double-lumen endobronchial tube (DLT). The new generation of disposable polyvinyl chloride DLTs is suitable for prolonged use because the cuff is designed to contain a high volume and confer low pressures. However, high pressures of up to 80 mm Hg can be generated if the cuff is inflated beyond the sealing pressure. The potential for displacement of the DLT during prolonged management is well recognized and a major drawback to its widespread application. Left-sided tubes should be placed when possible because the right upper lobe bronchus is relatively easily occluded with small displacements of a right-sided DLT.

ILV can involve either one or two lungs. The former involves blocking one of the lungs to control and contain the spread of harmful fluid and secretions while the other lung is ventilated. The latter involves administration of different modes of ventilation to each lung using independent ventilator circuits. Initial ventilator settings should correspond to the practice of safe ventilation, but with adjustment for lung volumes of 55% on the right and 45% on the left. The lungs can be inflated synchronously or asynchronously. In theory, synchronization may prevent unfavorable mediastinal shift, but there is no evidence that this is a significant problem in clinical practice. Synchronization with the cardiac cycle has also been proposed, but this is of physiological interest rather than clinical importance.

In practice, effective ILV mainly involves asynchronous ventilation because of the heterogeneous nature of the majority of lung injuries. Meticulous attention to monitoring the airway pressures of each lung is required to prevent barotrauma to the less diseased lung. PEEP is applied in amounts inversely proportional to the compliance of the lung with the aim of equalizing the functional residual capacities. Equalization of the tidal volumes and end tidal CO_2 is one of many possible criteria that may indicate suitability for conversion to conventional ventilation. Although there are reports of successful use of ILV in the setting of ARDS and other bilateral lung injuries, it remains controversial.[28–30]

High-Frequency Ventilation

High-frequency technology has been utilized in thoracic and airway surgery since the early 1970s, but its role in the critical care setting is yet to be defined. Implementation of high-frequency jet ventilation and high-frequency percussive ventilation has been associated with reduced VILI[31,32] and improved oxygenation,[33–35] respectively. However, use of these devices requires a high level of technical expertise that is currently available only in thoracic centers.

High-frequency oscillatory ventilation (HFOV) is currently available at United Kingdom (UK) Role 4 facilities and has been used in cases that are refractory to conventional ventilation. The oscillators can operate at frequencies up to 3,000 breaths per minute because an active expiratory phase is incorporated. A high mean airway pressure is employed, and the ventilator oscillates the gas delivered to pressures above and below this pressure. Oxygenation depends on the mean airway pressure and F_{IO_2}, whereas elimination of CO_2 depends on the frequency and amplitude of the oscillating pressure. Small tidal volumes are delivered because a large proportion of pressure is dissipated in the proximal airways. Lower volume delivery coupled with high mean airway pressure facilitates alveolar recruitment without the risk of over-distension. The following ventilator settings should be applied initially and then titrated according to requirements: F_{IO_2} of 1.0, frequency of 10 Hz, mean airway pressure of 5 cm H_2O above the current ventilator settings, cycle volume of 100 mL, and a base flow of 20 L/min.

Small observational studies have shown that HFOV is both effective and safe in the management of adult patients with severe ARDS.[36–39] A systematic review of randomized controlled trials comparing HFOV to conventional ventilation in adults and children with ALI or ARDS has shown improved oxygenation with a concomitant decrease in mortality.[40] Separate assessment of the data from the adult trials demonstrates an improvement in oxygenation with HFOV without a benefit to mortality. This ambiguity prompted the initiation of two large multicenter randomized controlled trials designed to compare HFOV with conventional positive pressure ventilation.[41,42] Both trials recruited a broadly similar set of patients with moderate to se-

vere ARDS. The High Frequency OSCillation in ARDS (OSCAR) Trial demonstrated no difference in mortality at 30 days for patients treated with HFOV compared to those treated with conventional ventilation.[41] The Oscillation for Acute Respiratory Distress Syndrome Treated Early (OSCILLATE) trial was stopped after 548 of the planned 1,200 patients had undergone randomization to HFOV or conventional ventilation because there was an excess mortality of 12% in the former treatment group.[42]

On further analysis, patients managed with HFOV had a higher requirement for inotropic agents when compared to those treated with conventional ventilation. This may be a consequence of an increase in after-load for the right ventricle by virtue of high intrathoracic pressures during HFOV. It may be significant that the OSCAR trial used lower airway pressures during HFOV and did not demonstrate this excess in mortality or a high requirement for inotropic agents. Although the OSCILLATE trial suggests that HFOV is injurious, there are many confounding factors. In particular, the mortality rate in the group managed with conventional ventilation is very low. It is possible that the conventional ventilation strategy of high PEEP and low lung volumes is particularly effective. As a consequence, the authors of both trials do not recommend HFOV for the routine care of patients with ARDS. In accordance with this recommendation, the ventilator is not currently used in the deployed military hospitals.

ADJUNCTS TO MECHANICAL VENTILATION

Neuromuscular Blocking Agents

Administration of neuromuscular blocking agents (NMBAs) has been shown to improve oxygenation in patients receiving a protective ventilation strategy for ALI or ARDS. It is postulated that this effect may be attributed to improved synchronization with the ventilator, thereby facilitating accurate adjustment of tidal volume and pressure levels. A concurrent reduction in pro-inflammatory cytokines has also been seen in this setting. The suggestion that NMBAs may modulate inflammation requires more research. A recent multi-center trial has reported that the use of NMBAs improves oxygenation and decreases mortality at 90 days, increases number of days outside the critical care unit, and increases number of days without mechanical ventilation.[43] Despite these promising findings, it should be appreciated that the use of NMBAs in the critical care setting confers the risk of prolonged weakness from myopathy, especially if high-dose corticosteroids are concurrently administered.

Recruitment Maneuvers

A variety of techniques have been described to achieve a transient increase in transpulmonary pressure and thus recruit collapsed alveoli. There are many reports of an initial improvement in oxygenation followed by rapid resolution of this beneficial effect within 20 minutes of the maneuver. However, application of higher levels of PEEP after the recruitment maneuver may sustain the effect.[44,45] The potential for recruitment of alveoli varies widely among patients, and there is no formula for the duration, pressure, or frequency of recruitment maneuvers. The most common complications are hypotension and desaturation. Barotrauma and induction of arrhythmias are relatively rare events.[46]

Prone Positioning

A number of trials have demonstrated an improvement in oxygenation when patients with ALI or ARDS are placed in the prone position.[47–50] This effect has not been translated into a definite improvement in mortality. However, a subsequent metaanalysis showed that the method conferred a benefit to mortality in patients with severe ARDS.[51] The possible mechanisms involved in the improvement in oxygenation are recruitment of collapsed alveoli[52]; redistribution of ventilation toward the dorsal regions in an attempt to match perfusion[53]; and elimination of compression of the lungs by the heart.[54] The problems associated with prone positioning include dislodgment of tracheal tubes and intravascular catheters, increased intraabdominal pressure, facial edema, and ophthalmic injury. However, the technique has been safely applied to trauma patients.

Nitric Oxide

Inhaled nitric oxide causes selective vasodilation of the pulmonary blood vessels in ventilated lung units and may improve matching of perfusion to ventilation. Although the therapy has been shown to improve oxygenation, it has not been translated into clinically beneficial outcomes.[55] It is not available in the deployed setting.

Conservative Fluid Management

Alveolar-capillary injury in ARDS is particularly

characterized by pulmonary edema. The edema has a negative impact on the mechanics of ventilation. Patients with ARDS typically accumulate one liter of fluid per day when managed with a conventional strategy of fluid administration. Although several randomized controlled trials have indicated that a more conservative approach to fluid management may confer clinical benefit,[56,57] a recent large multicenter trial has reported only a modest improvement in oxygenation.[58] There is evidence that more efficacious fluid removal using diuretics, with concomitant colloid infusion, confers an improvement in oxygenation during the early phase of ventilatory management.[59,60] However, an ideal strategy has yet to be elucidated and current emphasis remains on balancing the ventilatory benefits with hemodynamic requirements.

EXTRACORPOREAL MEMBRANE OXYGENATION

The goal of extracorporeal membrane oxygenation (ECMO) is to support gas exchange while allowing a reduction in the intensity of mechanical ventilation. A veno-venous or veno-arterial catheter is utilized to remove blood from the patient and circulate it through an artificial lung back to the patient. In 1972, Hill and colleagues first successfully applied the system for managing pulmonary failure secondary to trauma.[61] Subsequently, trials have failed to demonstrate any benefit to survival when compared to mechanical ventilation.[62–68] Although the most recent trial demonstrated that management with ECMO improved survival of patients with severe ARDS, the lack of standardized management of the control group makes definite conclusions difficult.[69] Coupled with the significant risk of complications, the ECMO's role in treating patients with refractory hypoxemia is likely to remain controversial in the UK.

Only a few facilities around the world have the capability of providing this therapy. In the military environment, the United States has the capability to deploy adult ECMO support teams. However, recent technological advances have yielded devices that could be more readily used in the military environment. The Lifebridge (Ampfing, Germany) B2T "Bridge to Therapy" system is a miniaturized ECMO system weighing only 18 kg that can deliver flows of 6 L/min. The Novalung (Heilbronn, Germany) system uses arterial pressure to drive blood across a membrane to remove CO_2 only. Although it offers fewer treatment options than ECMO, it is easier to manage and is compact, weighing only 653 g.

TRANSFER VENTILATORS AND THEIR LIMITATIONS

Intubated patients require continued airway protection and ventilation during transfer. Transfer ventilators (also commonly referred to as transport ventilators) are usually specially designed for this purpose. Although a manually ventilated patient, using a bag and simple oxygen tubing, could be safely transferred for a very short period of time, the safer and more conventional method is to use a mechanical ventilator. This becomes essential, for example, in head-injured patients who require more accurate and stable ventilatory control. Transfer ventilators vary greatly in design and complexity from a simple intermittent blower to a ventilator with more advanced ventilatory modes; some are even able to support noninvasive ventilation. Many designs are in use by aeromedical evacuation teams around the world.

Transfer ventilators are expected to be operated in different and changing environments, whereas normal intensive care unit ventilators are set up and operated in a stable situation. A transfer ventilator may be moved with the patient three or four times. In the modern military environment this may entail exposure to extremes of temperature, humidity, and barometric pressure. In addition, equipment must be robustly built to withstand austere environments without being physically damaged. Transfer ventilators must be securely mounted for tactical flight maneuvers.

Oxygen delivered to the patient is usually expressed as an inspired concentration (F_{IO_2}), which stays the same with changes in altitude, if nothing else changes. The partial pressure of the inspired oxygen (P_{IO_2}) will decrease with an increase in altitude. If a ventilator does not compensate for altitude change, the medical team will have to increase F_{IO_2}. For example, on ascent from sea level to 7,000 feet, with a starting F_{IO_2} of 0.4, P_{IO_2} decreases from about 300 mm Hg (40 kPa) to 234 mm Hg (31 kPa). F_{IO_2} would have to be increased from 0.4 to 0.52 to compensate. Airway pressures and delivered volumes may also change with altitude changes unless the ventilator compensates. In recognition of this potential hazard, each ventilator used for military aeromedical transfer is assessed in the controlled circumstances of a hypobaric chamber. The ventilator also undergoes tests to ensure that it is compatible and safe for use in a military aircraft.

Alarms in intensive care unit ventilators are visual and audible. In the aircraft environment, particularly in helicopters, audible alarms might not be heard,

and greater reliance is placed on visual alarms. Visual alarms on the ventilator should be in addition to visual capnography and pulse oximetry alarms on a separate monitor. The alarm system should be able to warn of the common and potentially dangerous situations of breathing circuit disconnection or endotracheal tube dislodgment during patient transfers in dark and noisy environments. Alarm lighting should not interfere with aircrew night vision equipment in a combat situation.

The requirements for adequate oxygen and electricity supplies for the ventilator are no different for ground or air transfers and are usually ensured by carrying spare oxygen cylinders and batteries. For longer transfers, the ability to use aircraft oxygen and electricity is an obvious advantage, but must be part of the larger process of equipment/aircraft integration. If high-pressure oxygen supply and batteries fail, there should be provision for manual ventilation with low-pressure oxygen, or air as a last resort. Decreasing cabin altitude might be necessary to maintain adequate oxygenation.

An ideal transfer ventilator would have the following characteristics:

- small size and low weight;
- wide range of operating temperatures and altitudes appropriate for proposed environments;
- ability to compensate for altitude changes with oxygen concentration, airway pressure, and delivered volumes;
- ability to ventilate complex patients (a number of different modes);
- long battery life and ability to use aircraft electricity supplies;
- fail-safe mode (ability to maintain operation when electricity supply fails);
- ability to use aircraft oxygen supplies and oxygen from different sources and connectors;
- controls and displays that are visible at night and are compatible with aircraft operations;
- alarms that are noticeable in a noisy environment; and
- ability to be mounted securely with the patient.

Although the ideal ventilator for all conditions does not yet exist, transfer ventilators have been used successfully for thousands of critical care air transfers despite their limitations. Only in exceptional circumstances should the deployed physician use a ventilator other than one designed for transfer, particularly in an aeromedical setting. Each user should be appropriately trained to understand the limitations of the ventilator, in particular the longevity of its power source. Alternate means of ventilation should be immediately available.

CONCLUSION

Patients with severe lung injury can present significant challenges to the military intensivist. The primary aim is to employ a ventilatory strategy that minimizes further injury to the lung. Rescue therapies may be considered in patients who develop refractory hypoxemia, but because of the lack of conclusive evidence and definitive guidelines, physicians must have a comprehensive understanding of the capabilities and drawbacks of each therapy.

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Chapter 30

VENTILATION FOR TRACHEAL DISRUPTION AND BRONCHOPLEURAL FISTULA

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INTRODUCTION

Tracheal disruption (TD) and bronchopleural fistula (BPF) are rare but serious complications of both blunt and penetrating thoracic trauma. The incidence of TD and BPF in thoracic trauma is between 0.5% to 2%, respectively.[1] In most cases of traumatic BPF, the injury is unilateral, with the majority occurring at the proximal right bronchus, within 2 cm of the carina.[2,3] The most common tracheal injury is a tear near the carina. In both cases, the injury pattern is likely due to the role of shear forces on the airways near the relatively fixed carina. Nontraumatic etiologies of TD include complications of endotrachial, thoracostomy, or tracheostomy tube placement and central venous catheter placement. BPF can be caused by ventilator-induced barotrauma in illnesses such as acute respiratory distress syndrome.[4,5]

DIAGNOSIS

Though anatomically different injuries, TD and BPF share some attributes of diagnosis and management. During initial stabilization, TD or BPF must be suspected in patients with blunt or penetrating chest trauma who deoxygenate, become difficult to manually ventilate, or have absent lung sounds despite placement of needle or tube thoracostomy. In the trauma team's assessment, suspicion for BPF should be increased in patients with respiratory signs and symptoms who display thoracic, cervical, and facial subcutaneous emphysema, pneumothoraces not reduced with chest tube drainage, ventilation that worsens with chest tube suction, or persistent air leak with chest tube drainage.[6–11] When BPF is suspected, fiberoptic bronchoscopy is the initial diagnostic modality of choice because it both permits evaluation of the cervically immobilized trauma patient and facilitates their intubation.[1,7–9,11] Intubating a patient with TD or BPF requires care that the endotracheal tube (ETT) is placed distal to the disruption to avoid the risk of rapid tension pneumothorax and death. For more proximal TD, this may be accomplished with a standard ETT. For distal TD, double-lumen intubation is necessary to protect both lungs from bleeding distal to the injury. In BPF, either one-lung (with a long ETT) or two-lung (with a double-lumen ETT) intubation is required.

Early diagnosis and surgical repair is critical for preserving lung function.[6,7] Late repair after months or years is feasible, with good operative outcomes reported, but usually at the expense of a significant loss of lung function distal to the lesion due to atelectasis, scarring, or infection.[10,12]

VENTILATION CONSIDERATIONS

If the airway is adequately protected with a single or dual-lumen ETT distal to the injury, there are no special ventilation considerations for tracheal disruption unless repair of a distal defect requires one-lung ventilation. In this case, the ventilation strategy will be similar to that for BPF. For patients with a standard ETT, providers must be vigilant for signs of hypoxia, subcutaneous emphysema of the neck and chest, and discordance between inspiratory and expiratory pressures, indicating that the ETT may have moved proximal to the lesion or that the wound has extended distal to the end of the ETT.

Ventilation for BPF and TD requiring one-lung ventilation for surgical approach is broken down into two phases: (1) acute intraoperative management and (2) postoperative care. To care for combat casualties with this injury, an understanding of the modes and risks of transport ventilation is also important. ETT selection and placement is intimately connected with the ventilatory management of patients with BPF. The goals of acute operative airway management are: (*a*) prevent damage to the uninjured lung by secretions from the injured lung, (*b*) ensure adequate ventilation of the patient through the operative procedure, and (*c*) facilitate the surgical repair of the affected bronchus.

Preventing Further Injury

Anatomic separation of the lungs with the ETT protects the uninjured lung from blood and secretions.[6,13] Methods most supported in the literature include bronchial intubation of the uninjured lung with a long ETT or double-lumen ETT placement with the bronchial lumen in the uninjured lung.[8,11] Intubation is ideally performed under direct bronchoscopic visualization in an awake patient or with bronchoscopic evaluation and adjustment, if needed, of an ETT placed by direct or fiberoptic laryngoscopy.[1,8,11,14] If available, a double-lumen ETT is preferred because it also permits physiologic separation of the lungs if the air leak becomes severe enough to require different ventilator modes or settings for each lung.[3,6,13]

In BPF, air leak during positive-pressure ventilation can be up to 25% of minute ventilation.[5,15,16] This

physiology is worsened by ventilator maneuvers that increase the gradient from the lung across the pleural space such as higher inspiratory or positive end-expiratory pressure (PEEP), tidal volume (V_T), increased inspiratory flow, and increased inspiratory time (including breath hold and inverse-ratio).[16] Subacutely, unilateral BPF can create differences in lung physiology between the injured and uninjured lung that can be great enough with large defects to require two-lung independent lung ventilation (2L-ILV), separate ventilator management of each lung to achieve adequate oxygenation and ventilation.[13,14]

Ventilator Settings

The initial approach to ventilator settings is to provide low V_T (6–7 mm Hg/kg) and allow hypercapnia by maintaining P_{CO_2} below 60 mm Hg and pH at 7.3 or greater.[13,17] PEEP should be low, ideally 5 mm Hg, to minimize expiratory air leak.[16] Goal plateau pressure (Ppl) should be less than 26 mm Hg for optimal compliance and gas exchange.[13,18] If the patient cannot be ventilated at a V_T that maintains Ppl at desired levels, switching to pressure-controlled mode with the same target Ppl has been shown to improve ventilation and reduce peak airway pressure.[19] In patients with refractory respiratory acidosis, persistently elevated peak airway pressure, or oxygenation difficulties, salvage modes include high-frequency ventilation (HFV) or 2L-ILV (synchronous or asynchronous).[11,13,20–22] Some studies have found that in a selected population with BPF/TD and otherwise healthy lungs, HFV improves oxygenation at lower mean airway pressures than conventional mechanical ventilation.[20,23,24] As with Ppl in conventional mechanical ventilation, the goal for mean airway pressure in HFV should be less than 26 mm Hg. With 2L-ILV, initial V_T should be administered in a ratio of 55% on the right lung and 45% on the left, then adjusted to meet airway pressure and ventilation goals.[21] Outcomes with 2L-ILV are similar whether synchronous or asynchronous ventilation is used.[22,25] The former is easier to set up because it does not require specialized ventilator synchronization software or connecting cabling between the ventilators.

An advantage of HFV is that ventilation is achieved through an ETT with the cuff deflated.[6,26] This can facilitate surgical repair and healing by removing cuff ulceration as a risk to the wound, but does not provide the protection of anatomic separation of the lungs. More useful for facilitating surgical repair is a two-lung ETT or traditional long ETT cannulation of the uninjured lung, with the inflated cuff anatomically isolating that lung from contamination. Ventilation of the uninjured lung allows deflation of the injured lung to improve surgical access.[1,3,8] In large or bilateral disruption of the trachea, intraoperative cannulation of the injured lung distal to the disruption with a sterile ETT may be necessary.[8,9] Care must be taken to avoid barotrauma when ventilating a lung segment perioperatively, and the anesthesiologist may be required to manually ventilate to achieve low enough airway pressure.[9] There are case reports of cardiac bypass and extracorporeal membrane oxygenation being used in cases of severe bilateral disruption.[9,27]

Postoperative Care

The goals of postoperative ventilatory care are to reduce strain on the operative repair and prevent complications such as pneumonia and atelectasis. The optimal method for protecting the repair is extubation, since spontaneous respiration places the least strain on the airways. Nevertheless, most multisystem trauma patients must remain intubated postoperatively. Goals for postoperative ventilator management are to use the lowest V_T possible, maintain Ppl under 26 mm Hg, and consider modes that encourage spontaneous respirations, such as intermittent mandatory ventilation (IMV).[20] If two-lung IMV was used perioperatively, criteria for converting to single ventilator mode are V_T equalization to within 100 mL and compliance within 20%.[13] Epidural analgesia with fentanyl or bupivacaine has been found in some series to improve lung mechanics and ventilation and may encourage more spontaneous respiration.[28,29]

Aggressive pulmonary toilet is mandatory to reduce the risk of postobstructive pneumonia. Bedside bronchoscopy is often required to clear secretions below the anastomosis.[1,30] Patients with TD and BPF are at high risk for postoperative pneumonia and should receive prophylactic broad-spectrum antibiotics, which can be discontinued when they are extubated and clearing their own secretions.[6,11]

Aeromedical Evacuation

Air transport of patients with BPF, whether repaired or not, should be approached with great caution. Actual V_T and PEEP delivered can increase greatly with altitude, posing a serious risk to an operative repair or of air leak to an unrepaired BPF or TD.[31] Delay in transport until extubation of a repaired BPF is ideal. If air evacuation must be attempted, a thorough understanding of the transport ventilator settings and fluctuations of V_T and PEEP to both sustained and abrupt changes in altitude, as well as scrupulous monitoring of the patient's ventilatory status and chest tube air leak, are essential.

SUMMARY

TD and PBF are rare but serious complications of thoracic and cervical trauma. Suspicion for these injuries should be raised in patients with thoracic or neck injuries who are difficult to ventilate or have absent lung sounds despite needle or chest tube decompression, persistent air leak, or subcutaneous emphysema of the face, neck or thorax. Prompt recognition is essential for appropriate airway management and because early repair is associated with improved long-term lung function. Airway management and ventilation depend on the location of the injury. Proximal TD may be managed with the placement of a standard ETT distal to the injury. TD closer to the carina and BPF will usually require placement of a dual-lumen ETT to protect the lungs from bleeding and one-lung ventilation for surgical exposure. Postoperative ventilator management of BPF requires care to re-expand the injured lung without disrupting the surgical repair. Aeromedical evacuation of patients with BPF, whether repaired or not, is risky. It is preferable to delay evacuation until extubation after surgical repair.

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Chapter 31

RENAL SUPPORT IN MILITARY OPERATIONS

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INTRODUCTION

Renal failure is a serious condition under any circumstances, linked significantly to increased mortality, although whether this relationship is causative or associative is unclear. Combat-related renal failure is uncommon, but potentially more serious than in the civilian context, due to the limitations of available treatment during deployment. This chapter covers renal support in military operations, including current options for support and future equipment development.

RENAL SUPPPORT IN THE DEPLOYED SETTING

History

Acute kidney injury (AKI), previously known as acute renal failure (ARF), following combat trauma was noted only infrequently until World War II, principally because most casualties succumbed to hypovolemic shock before they could develop AKI. During the war, civilian casualties with crush syndrome following bombing, and military casualties with AKI who had been successfully resuscitated, were increasingly described in the medical literature.[1] Following pioneering work during World War II by Willem Kolff, renal replacement therapy (RRT) developed significantly in the Korean War, principally through the work of Paul Teschan and colleagues.[2] Throughout the Vietnam War, a deployed renal team was based in Japan with forward projection to the Philippines and Saigon, equipped for both peritoneal dialysis (PD) and hemodialysis (HD). Many renal failure cases in Vietnam were due to "medical" causes (such as malaria), and the lower incidence of trauma-related AKI compared to previous conflicts was ascribed to improved initial resuscitation. In this conflict, "medical" AKI was predominantly treated with PD, while traumatic/surgical AKI was treated with HD.[3]

Incidence and Etiology

Although common in previous conflicts (up to 20% of severely wounded soldiers), the incidence of AKI in modern combat injuries is low (0.5% of all postoperative casualties in Korea surviving at least 48 h, 0.17% in Vietnam, and even lower in subsequent conflicts).[4,5] This decrease is likely to be multifactorial. Improved body armor reduces the number of severe torso injuries. Active field dressings, improved buddy-aid training, tourniquets, and intraosseous needle use allow for more rapid and effective control of hemorrhage. Additionally, improved initial resuscitation with helicopter-based evacuation to high quality deployed medical facilities allows for definitive surgery and restoration of circulating volume to help prevent AKI.

The most common pathology of renal failure related to combat trauma is acute tubular necrosis (ATN). ATN may be multifactorial in origin, but hypovolemia and hypotension are probably the two most common causative factors. ATN tends to develop over several days, in contrast to traumatic rhabdomyolysis, in which the development of renal failure and life-threatening metabolic complications can occur within hours of injury.

Prevention of Acute Kidney Injury and Renal Failure

With appropriate treatment, the natural history of AKI is recovery to dialysis-free function. Fewer than 10% of patients require long-term renal support following an acute episode of AKI. Ensuring adequate circulating volume replacement and renal perfusion are mainstays in prevention of AKI. Historical reports of maintaining patients for up to 2 weeks with oligoanuria and recovery of renal function demonstrate that meticulous attention to fluid balance, low protein intake, and general care (Borst or Bull regimes[6,7]) can be beneficial even in the absence of sophisticated supportive measures.

Indications for Renal Support

Until relatively recently, multiple definitions for ARF existed. The RIFLE (risk, injury, failure, loss, and end-stage disease) criteria[8] (Table 31-1) are increasingly used to standardize definitions and compare both treatments and thresholds for treatment with RRT. The term "acute kidney injury" is preferable to "acute renal failure."

Traditional indications for acute RRT are fluid overload, acidosis, or hyperkalemia. Additional indications are other severe electrolyte disturbances, and some cases of drug overdose or poisoning. Increased urea and creatinine levels are less acutely a reason to provide RRT. The rate of rise of potassium in an anephric patient depends on body size/muscle mass, metabolic/catabolic rate, tissue injury, and effects of medical therapies administered, but it is typically 0.5 to 3 mmol per liter per day. Likewise, the rate of creatinine rise is variable, but an adult male rendered anephric may show rises of between 90 and 250 mmol per liter

TABLE 31-1

RISK, INJURY, FAILURE, LOSS, AND END-STAGE KIDNEY DISEASE (RIFLE) CLASSIFICATION

Class*	Glomerular Filtration Rate Criteria	Urine Output Criteria
Risk	Serum creatinine × 1.5	< 0.5 mL/kg/h × 6 h
Injury	Serum creatinine × 2	< 0.5 mL/kg/h × 12 h
Failure	Serum creatinine × 3, or serum creatinine ≥ 4 mg/dL with an acute rise > 0.5 mg/dL	< 0.3 mL/kg/h × 24 h, or anuria × 12 h
Loss	Persistent acute renal failure = complete loss of kidney function > 4 weeks	
End-stage kidney disease	End-stage kidney disease > 3 months	

*RIFLE class is determined based on the worst of either glomerular filtration criteria or urine output criteria. Glomerular filtration criteria are calculated as an increase of serum creatinine above the baseline serum creatinine level. Acute kidney injury should be both abrupt (within 1–7 days) and sustained (more than 24 hours). When the baseline serum creatinine is not known and patients are without a history of chronic kidney insufficiency, calculating a baseline serum creatinine using the Modification of Diet in Renal Disease equation for assessment of kidney function, assuming a glomerular filtration rate of 75 mL/min/1.73 m^2, is recommended.

per day (1–3 mg/dL/d).

Efforts to discover markers of renal dysfunction predictive of the requirement for RRT (eg, cystatin C, neutrophil gelatinase-associated lipocortin, kidney injury molecule-1) currently lack sufficient sensitivity and specificity. Historically these markers have not been easily available to the deployed clinician; however, bedside tests are now available and efforts are being made to assess such markers' suitability as a panel of tests.

Acute life threatening complications are most likely to arise as a result of hyperkalemia and acidosis. Medical therapy (Table 31-2) should be the mainstay of management unless timely evacuation to an adequately appointed facility is impractical. Once started, renal support may be required for days or weeks. This requirement must be considered in the decision to provide RRT, particularly in cases involving local nationals or others with limited eligibility according to treatment matrices. These decisions may be ethically difficult.

Management Options

Broadly speaking, renal support may consist of PD, HD, or hemofiltration. Perhaps more significant than any postulated clinical benefits are the requirements of each mode for logistic support, transport, and on-going maintenance, as well as requirements for trained staff (Table 31-3). PD is least efficient, and has traditionally been relatively contraindicated for patients following abdominal trauma and laparotomy. Nonetheless, a small number of cases have been reported in which field-rigged dialysis systems have been successfully employed to provide PD following combat injures when evacuation was impractical or delayed.[9,10] Table 31-4 lists one option for constructing such a system.

Continuous RRT (CRRT) is usually delivered using a veno-venous technique of hemofiltration (CVVH). CVVH requires large bore venous access using specialized catheters, along with filters, circuits, and specialized replacement fluids, in addition to skilled staff. These considerations, along with the frequent requirement for anticoagulation, make CRRT difficult to provide on a routine basis in the initial period following battle injury, especially if experienced staff are unavailable. Anticoagulation requires careful deliberation, balancing the risk of precipitating further bleeding against the risk of clotting in the filtration circuit. Heparin-free techniques using predilution at the filter and alternative agents such as prostacyclin are used in standard practice, but evidence in the setting of immediate postcombat trauma is lacking. CRRT is the most frequently employed mode of renal support used in civilian critical care, especially if patients remain hemodynamically unstable.

Traditionally, HD requires a pure water supply in large quantities (up to several thousand liters daily), along with skilled staff and equipment designed for single use. Recently, technical advances in home dialysis have produced equipment that requires small volumes of water (as low as 10 liters daily) and can be safely managed by relatively unskilled assistants. The equipment is portable and sufficiently compact and lightweight to be carried by helicopter and existing transport aircraft. However, this new equipment is not presently standard military stock.

Current US Army doctrine provides for a hospital augmentation dialysis team; however, these teams have not been deployed to Iraq or Afghanistan since 2001 (principally because the need for such augmentation has not been reached), and the doctrine is being revised.[11] United Kingdom forces doctrine has equipped the Royal Air Force (RAF) with deployable CVVH capability. Two modules are held at the Tactical Medical Wing at RAF Brize Norton to provide a global

TABLE 31-2

MEDICAL THERAPY FOR ACUTE KIDNEY INJURY AND HYPERKALEMIA*

Drug	Dose	Action
Calcium chloride 10%	10 mL every 20 minutes until electrocardiograph normal or 50 mL maximum	Increases threshold potentials, stabilizes cell membranes against depolarization
Calcium resonium	30 g enema and 15 g PO every 8 hours with lactulose 10–20 L every 6 hours	Binds potassium in the gut, preventing absorption
Salbutamol nebulizers	5 mg (2.5 mg in presence of heart disease)	Reduces extracellular potassium levels by increasing cell uptake of potassium
Dextrose insulin	25 mL 50% dextrose and 10 IU rapid acting insulin (such as actrapid or humulin) over 15 minutes Consider 20% dextrose 1,000 mL and 100 IU actrapid at 2 mL/kg/h	Insulin facilitates glucose entry to cells, with accompanying potassium shift from plasma
Sodium bicarbonate	50–100 mL 8.4% bicarbonate over 15 minutes via central venous catheter or 200–400 mL 1.2% peripherally	To correct acidosis (enhances effects of insulin and shift of potassium to intracellular space)

*Consider hemodialysis if : potassium > 7.0 mmol/L; pH < 7.1; uremia > 45 mmol/L or blood urea nitrogen =126 mg/dL; or pericarditis is present.
IU: international unit
PO: per os, by mouth
Data source: UK Ministry of Defence. *Clinical Guidelines for Operations*. Joint Doctrine Publication 4-03.1. London, England: MOD; June 2010.

TABLE 31-3

COMPARISON OF RENAL SUPPORT MODES

Characteristic or Requirement	Peritoneal Dialysis	Hemodialysis	Hemofiltration
Systemic anticoagulation required	No	Usually	Usually
Purified water supply required	No	Yes	No
Efficiency of solute clearance	Poor	High	Moderate
Specialized replacement fluid required	No	Yes	Yes
Can be field rigged	Yes	No	No
Logistic support requirements	Low	High	Intermediate

CVVH capability. Currently the concept of use entails flying the module along with supporting staff to the patient, providing RRT on the ground, and then returning the patient to the United Kingdom when stable. RRT cannot be undertaken during flight in part due to equipment power requirements, but also because the gravimetric analysis of fluid balance is disturbed by vibration. Following the loss of the air-bridge in 2010 as a consequence of volcanic dust, a third RRT module was deployed into theater in case of delays in returning critically ill patients. This module is now a Permanent Joint Headquarters asset and has been used once successfully to stabilize a soldier prior to strategic air evacuation.[12]

Outcomes

In World War II, mortality rates from renal failure exceeded 90%. After the initiation of HD in Korea, trauma-related renal failure mortality fell to 68%, and remained at this level during the Vietnam War. "Medical" renal failure carried a much lower (approximately 10%) mortality rate, even if treated with the less efficient PD. In modern conflicts, AKI is so uncommon that an accurate attributable mortality rate is difficult to measure, since most reports are of small case series only.

TABLE 31-4

CONSTRUCTING A FIELD-EXPEDIENT PERITONEAL DIALYSIS SYSTEM*

Equipment Required	Procedures
IV tubing with roller clamp Mask, gown, cap Disinfectant Diagnostic peritoneal lavage catheter (chest tube or any other tube device with distal portholes may also be used) Drainage bag (emptied IV fluid bag or blood collection bag) Fluid for dialysis: • Lactated Ringer or Hartman solution can be used as a simple field expedient dialysate (both closely mimic the electrolyte profile of stock dialysate). • Glucose can be added to the dialysate to create a 1.5%–4.5% glucose solution. • 50 mL of 50% dextrose solution added to each liter of lactated Ringer solution will produce an approximately 2.5% glucose solution.	Catheter insertion: • The PD catheter can be placed using techniques employed in paracentesis or diagnostic peritoneal lavage. • The method used will depend on the training of the physician, comfort with the different techniques, and available equipment. • Placing a larger catheter/drain such as a chest tube is best accomplished using an open technique with visualization of tissue planes to minimize risk of bowel perforation. Management of PD: • Once the infusion/drainage device is placed, the dialysate can be infused. • Once the lactated Ringer solution bag is spiked with infusion tubing, air should be bled from the line prior to connecting to PD catheter. Up to 3 L of dialysate can be infused. • Dwell time should be 1–4 hours. • After an adequate dwell time has elapsed, the catheter can be connected to the drain bag. The drain bag should be placed in a dependent position (on floor) and the dialysate collected in the drain bag. • Multiple exchanges may be necessary each day as needed to correct the renal failure and fluid overload.

*It is important to note that sterile technique is vital to prevent peritonitis. Field conditions make this challenging, but diligence in this matter can prevent morbidity and mortality for the patient. Electrolyte measurement should be repeated after PD to follow the electrolyte changes and renal function.
IV: intravenous
PD: peritoneal dialysis
Data source: Givens M. Tricks of the trade. *Ann Nav Emerg Med.* March/April 2010:21.

SUMMARY

Combat-related ARF is rare in modern conflict, especially as an isolated injury, but it is important to be aware that it is associated with high mortality rates. The incidence of AKI over succeeding days is not well recorded in terms of frequency, severity, or outcome. As with other forms of renal failure, avoidance is better than post-hoc treatment. Adequate, rapid resuscitation and replacement of circulating volume is a key component of management. In the majority of cases, medical therapy for acute life-threatening disorders in the deployed situation can stabilize a patient's clinical condition pending transfer for formal RRT. A high positive fluid balance, progressive acidaemia, or rapidly rising serum potassium levels would indicate red flags for upgrading the urgency of evacuation.

RRT is best provided in large, well-appointed facilities out of theater, which relies on rapid, reliable evacuation systems. Failing such evacuation capabilities (and contingent upon eligibility matrices), future operations, especially insertion and contingency operations, may involve a small number of patients requiring in-theater renal support. Options for such management may be field-rigged PD or CVVH, depending on exact circumstances. Portable HD machines may provide a practical option in the future, although no military-specific information about this equipment is available to date.

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Chapter 32

DIAGNOSIS AND MANAGEMENT OF HYPOTENSION AND SHOCK IN THE INTENSIVE CARE UNIT

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INTRODUCTION

Shock is a state of impaired tissue oxygenation and perfusion that can be caused by decreased oxygen delivery, poor tissue perfusion, or impaired oxygen utilization. Hypotension is a sign of shock and an indicator of advanced derangement, requiring immediate evaluation and management. For example, in hemorrhagic shock, hypotension is not present until greater than 30% of blood volume has been lost. Although hypotension and shock are not synonymous, the goals of treatment are the same: to restore the body's oxygen balance and correct hypoperfusion. This chapter will address the categories of shock, initial evaluation of a hypotensive patient, general principles of shock management, and management for specific causes of shock.

PATHOPHYSIOLOGY AND CLINICAL PRESENTATION

Shock represents a state of hypoperfusion that can be the final pathway for a number of conditions. Hypoperfusion from any cause results in an inflammatory response. A normal physiologic compensation to improve perfusion of vital organs is sympathetic vasoconstriction resulting in an elevated diastolic pressure, narrow pulse pressure, and peripheral hypothermia. There is also a sympathetically mediated tachycardia that helps maintain cardiac output. Hypoperfusion also causes an acidosis induced by lactate production and resulting in compensatory tachypnea as the body attempts to offset the resulting acidosis. The other major effect of the acidosis is a rightward shift of the oxyhemoglobin curve,[1] allowing more of the oxygen that is bound to hemoglobin to be released. Additionally, there is increased shunting of blood to the most vital of organs—the heart and the brain—because of the opening of arteriovenous connections to bypass capillary flow.[2]

As these compensatory mechanisms begin to fail, the clinical signs and symptoms of shock become evident. The most commonly discussed signs of shock are hypotension, altered mental status, and oligura, but dysfunction of any end organ can result. Laboratory abnormalities include lactic acidosis, elevated base deficit, hypoxia, elevated blood urea nitrogen and creatinine, elevated liver-associated enzymes and bilirubin, and coagulation abnormalities. Lactic acidosis and base deficit are more sensitive indicators of severity and prognosis than are blood pressure and urine output (these will be covered in greater detail later in this chapter).[3,4]

CATEGORIES OF SHOCK

It is helpful to place shock in one of the following four distinct categories: (1) hypovolemic, (2) cardiogenic, (3) distributive, and (4) obstructive. Hypovolemic shock can result from hemorrhage or other forms of intravascular fluid loss such as capillary leak, gastrointestinal losses, or renal losses. Its hemodynamic profile is significant for increased heart rate (HR), decreased cardiac output (CO), increased systemic vascular resistance (SVR), decreased cardiac filling pressures, decreased pulse pressures (PPs), and decreased central venous oxygen saturation (ScvO2). Simply stated, the circulatory system cannot maintain adequate blood flow and the body is compensating by increasing HR in an effort to increase CO and SVR to maintain perfusion. On physical exam, one would expect to see pallor and flat neck veins.

Cardiogenic shock is most often caused by a myocardial infarction, but it can have other causes such as myocardial contusion. Like hypovolemic shock, its hemodynamic profile shows increased HR, decreased CO, increased SVR, and decreased ScvO2. It differs, however, in that cardiac-filling pressures, central venous pressure (CVP), and pulmonary artery occlusion pressure are elevated in cardiogenic shock. In this state, the volume is available, but pump failure causes inadequate blood circulation. Physical exam is significant for distended neck veins, pulmonary edema, and a possible S3 gallop.

Distributive shock is often referred to as high output or hyperdynamic shock because, unlike the other forms of shock, the cardiac output is normal to elevated. The loss of vascular tone that defines distributive shock results in decreased SVR and an increased pulse pressure caused by decreased diastolic pressure. Many causes of distributive shock exist, including early septic shock, neurogenic shock, and anaphylactic shock.

Obstructive shock shares the hemodynamic profile of cardiogenic shock, and the two are often lumped together. The most significant difference between the two is the cause. Obstructive shock is caused by impaired cardiac filling as in cardiac tamponade, or excessive afterload as in a massive pulmonary embolus. Management lies in relieving the obstruction, which is often readily treatable if identified, but can be fatal if not detected.

One must be cognizant that the categorization of

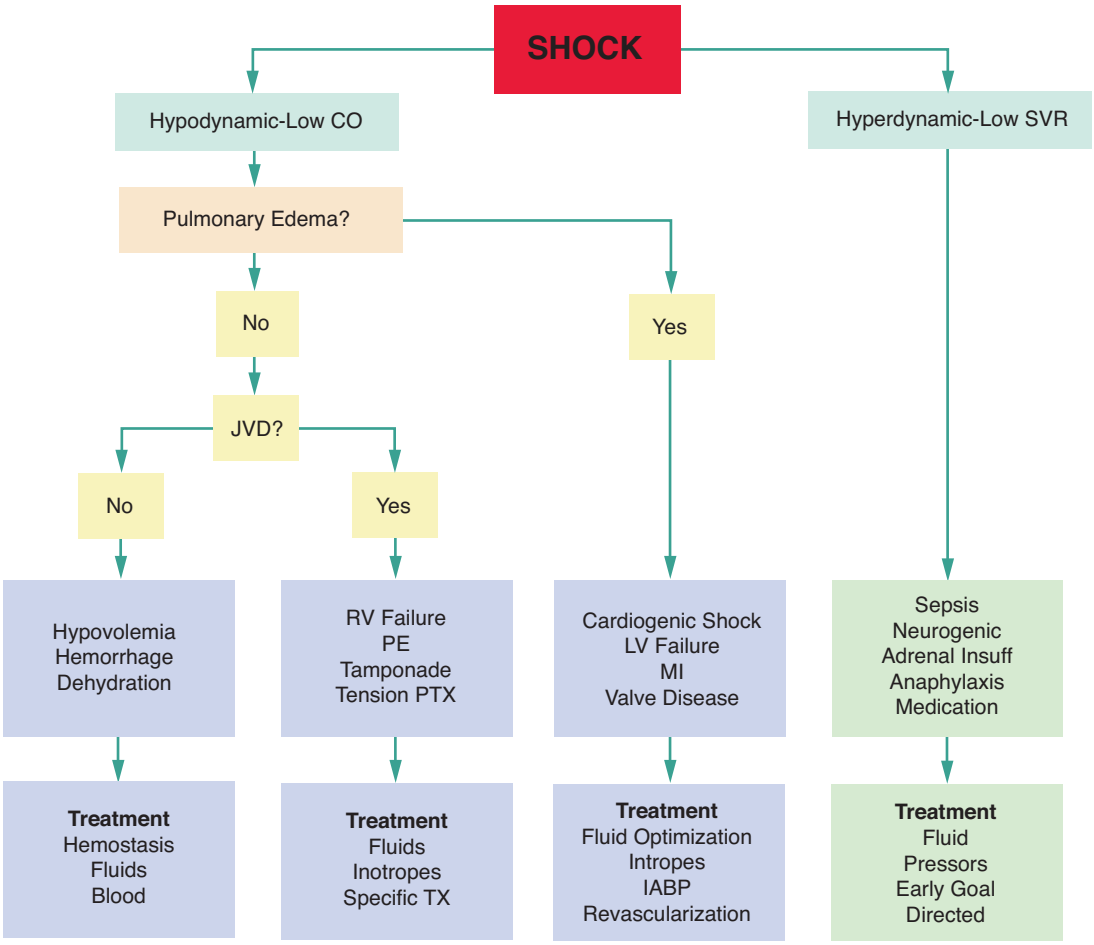

Figure 32-1. Diagnosing and treating various forms of hypotension.
CO: cardiac output; IABP: intra-aortic balloon pump; JVD: jugular vein distension; LV: left ventricular; MI: myocardial infarction; PE: pulmonary embolism; PTX: pneumothorax; RV: right ventricular; SVR: systemic vascular resistance; TX: therapeutics

shock is not always clear cut and overlap often occurs. For example, while septic shock is considered distributive shock, there is often a large component of hypovolemia present from third spacing of fluid. Alternatively, a thoracic trauma patient may suffer hemorrhage, causing hypovolemic shock, but may also have cardiac tamponade or a pneumothorax, resulting in obstructive shock. Additionally, shock is a dynamic state so the dominant component may change over time or with treatment. An overview of the causes and treatments of the various forms of shock can be seen in Figure 32-1.

GENERAL DIAGNOSTIC APPROACH FOR HYPOTENSION

As with any medical illness, diagnosing the source of hypotension should begin with a history and physical examination. The importance of a thorough but focused physical exam must not be underestimated. Vital signs should be obtained. Airway, breathing, and circulation should be immediately assessed and the patient must be fully disrobed and inspected, front and back. Specific findings that may guide the investigation are vital signs, level of consciousness, appearance of neck veins, auscultation of cardiac and breath sounds, sources of external bleeding, assessment of possible internal sources of bleeding, and neurologic status.

Noninvasive vital signs are not adequate to determine the severity of illness or injury. Tachycardia, tachypnea, and hypotension are highly concerning findings, but they likely represent an advanced stage of disease. Consequently, invasive monitoring and laboratory evaluation should be obtained.[4] CVP and ScvO2

can assist with determining the type of shock. Arterial catheterization may be helpful in maintaining a more accurate blood pressure as well as in determining respiratory variation of pressures. There is no apparent benefit to using a pulmonary artery catheter.[5] Lactate and base deficit are important values to obtain.[3,4] They will not assist in determining the cause of hypotension, but they will aid in assessing severity and adequacy of resuscitation. Base deficit has been shown to correlate with greater fluid requirements, ongoing blood loss,[6] and mortality.[7] Lactate has been shown to correlate with the development of multiorgan failure.[8]

Although obtaining a thorough history is not a requirement when assessing a hypotensive critically ill patient, at a minimum one must be aware of allergies and medications. Hypotension can be caused by anaphylaxis or may result from narcotic or sedating medications. Additionally, withdrawal of a chronic medication, such as glucocorticoids, can cause hypotension. If possible, obtaining a thorough trauma history may allow for elucidation of occult injuries.

Radiography is very important in the critically ill trauma patient. Radiographic imaging of the C-spine, chest, and pelvis is generally obtained as part of the initial trauma evaluation, but should be considered in a hypotensive intensive care unit (ICU) patient. A chest radiograph could reveal a pneumothorax, suggest a hemothorax or pericardial effusion, or identify pneumonia in a septic patient. C-spine fractures raise concern about neurogenic shock, and a pelvic fracture may lead to investigation for intraperitoneal hemorrhage. Although these films may guide therapy, it is imperative that obtaining them does not delay any necessary treatment. For example, if a tension pneumothorax is suspected, immediate decompression should be performed without X-ray film confirmation.

The use of ultrasound (US) as a diagnostic tool has dramatically changed the evaluation of hypotension in trauma patients over the past two decades. It can be performed rapidly and repeated frequently without a risk of radiation to the patient. It has many benefits in the acute setting. For example, one can determine whether fluid exists around the heart, if there is impaired cardiac contractility after thoracic trauma, or whether free fluid exists in the abdomen after blunt abdominal trauma. In some cases, a diagnosis can be obtained almost instantaneously. For example, visualization of Morison's pouch can demonstrate free fluid in the abdomen and determine the need for surgery. It is currently taught in Advanced Trauma Life Support[9] and recommended by the Eastern Association for the Surgery of Trauma as the initial test to exclude hemoperitoneum.[10] Physical exam is often of limited value in critically ill trauma patients for many reasons, including medication effect, altered mental status, and distracting injuries. Therefore, to improve diagnostic accuracy, many trauma centers routinely include Focused Assessment Sonography for Trauma (FAST) as part of the physical exam.[11]

The FAST exam consists of four sonographic views to evaluate for pericardial and peritoneal free fluid: (1) pericardial, (2) perisplenic, (3) perihepatic, and (4) pelvic.[11] This exam is most helpful when free fluid is identified. A negative exam is less helpful because of a lower sensitivity. Therefore, the Eastern Association for the Surgery of Trauma guidelines recommend repeat exams and at least 6 hours of monitoring before accepting a negative exam.[10] Similarly, Advanced Trauma Life Support recommends a repeat exam in 30 minutes.[9] The pericardial view allows for identification of a pericardial effusion, but if there is concern for myocardial contusion, a formal echocardiogram should still be obtained.

An extended FAST (eFAST) exam, which includes evaluation of the pericolic gutters and the pleural space, can also be performed. Evaluation of the pleural space with US allows for identification of hemothoraces and pneumothoraces more rapidly than chest radiographs and also has a greater sensitivity.[11] Although a pneumothorax is more easily seen with US, it is more difficult to determine its size this way.[11] As with many traumatic injuries, pneumothoraces are dynamic conditions and repeat exams should be considered. It is also possible to use US to ensure drainage of a pneumothorax. Although not part of the eFAST exam, US can also be used to guide fluid management during resuscitation by measuring the size and collapsibility of the inferior vena cava.[12]

Limitations to the use of US include altered windows caused by obesity, subcutaneous air, or other injuries or dressings. Specific risk factors exist that increase the likelihood of a nondiagnostic US, existence of an injury missed by US, or requirement for a computerized tomography (CT) scan despite US findings.[11] These factors include persistent abdominal pain, seat belt sign, abdominal wall contusion, pulmonary contusion, hematuria, rib fractures, spine fractures, and pelvic fractures. Although false negative rates for screening US in patients with blunt abdominal trauma are low (1%), the risk increases to more than 6% for high-risk patients.[13,14] For a trauma patient with the risk factors listed above, a CT scan should be the initial diagnostic test unless the patient is too unstable for transport to a CT scanner.[15]

CT scans are the most definitive, highest fidelity, noninvasive test for the hypotensive trauma patient.[15] It is important to remember, however, that no unstable patient should go to the CT scanner. Other risks as-

sociated with CT scanning include contrast allergy, contrast nephropathy, and excessive radiation. Because of these risks as well as logistical concerns, CT scans cannot be repeated with the ease of US. Consequently, when scanning, one should consider whether the results would alter management and ensure that all areas of interest are scanned at once.

Diagnostic peritoneal lavage (DPL) was a common step in the evaluation of the hypotensive trauma patient before the evolution of US. DPL remains a reasonable option if US is unavailable, equivocal, or inconsistent with the clinical picture. However, DPL will alter future physical and radiographic exams and it cannot be repeated. Although there are no absolute contraindications to DPL, prior abdominal surgery, abdominal infections, coagulopathy, obesity, and second- or third-trimester pregnancy are all relative contraindications.[16]

GENERAL PRINCIPLES OF MANAGEMENT OF THE HYPOTENSIVE INTENSIVE CARE UNIT PATIENT

The specific treatments for the various causes of shock may differ, but the overall goal in treating hypotension and shock is to restore oxygen balance and improve tissue perfusion. To do this, one must increase blood pressure, increase cardiac output, optimize oxygen delivery, and decrease oxygen demand. In general terms, fluids and vasopressors are used to increase blood pressure. Fluids should be the initial treatment, with vasopressors added only if the patient is unresponsive to fluids. The point at which vasopressors should be added differs based on the type of shock and will be addressed as such. Fluids and inotropes can be used to increase cardiac output. Oxygen delivery is further optimized by increasing hemoglobin and oxygen supply, and oxygen demand is decreased through the use of sedation, analgesia, and antipyretics. To assess progress, monitoring of arterial blood pressure, pulse oximetry, CVP, urinary output, acid base status, lactate, and base deficit are recommended.[4,17] The trends of the values obtained are often of more benefit than the baseline values.

Hemorrhage control and fluid resuscitation are the mainstays of the management of shock. If bleeding is the cause of shock, hemorrhage control is more important than resuscitation and surgical intervention should be pursued emergently. While awaiting surgery, fluid resuscitation is essential, but it should not delay surgery. Clarke et al showed a 1% increase in mortality for every 3 minutes of resuscitation prior to surgery.[18] A reasonable method to determine adequacy of hemorrhage control is to give 2 L of normal saline. If blood pressure improves, bleeding is likely controlled. If blood pressure improves only temporarily, there is ongoing blood loss. If there is no response, there is high volume blood loss. Transient responders and nonresponders require surgical intervention.[9] Following control of hemorrhage, the priority shifts to fluid resuscitation. Crystalloids and colloids are equally effective, although crystalloids are less expensive.[19]

Blood products must also be considered in the critically ill trauma patient. The goals should be to improve perfusion and oxygenation and decrease coagulopathy as opposed to targeting arbitrary laboratory values. A restrictive resuscitation standard, as discussed in the Transfusion Requirements in Critical Care trial,[20] does not apply to an actively bleeding patient. It is difficult to assess the exact amount of blood loss in trauma patients, so it is not often possible to directly replace lost blood with blood products. Furthermore, it is important to remember that a hematocrit is not an accurate measure of blood loss in an acutely bleeding patient because hemodilution has not yet occurred. Consequently, red blood cell transfusion is indicated in any patient with evidence of hemorrhagic shock.[21] After initial resuscitation and hemostasis, red blood cell transfusion should be considered for hemoglobin less than 7 g, and one unit should be given at a time.[21]

The end points of resuscitation are highly controversial. Over-resuscitation can lead to reversal of vasoconstriction of injured vessels, dislodging of clots, dilution of clotting factors, cooling of the patient, and swelling of visceral organs, possibly leading to abdominal compartment syndrome. It was previously thought that over-resuscitation would also increase intracranial pressure, but the amount of fluid given during resuscitation does not correlate with intracranial pressure.[20] Conversely, under-resuscitation risks poor cerebral perfusion and hypoxic brain injury.

No optimal algorithm for resuscitation exists. A mean arterial pressure of greater than 65 is often considered a goal, but this is highly debatable.[15,17] An individual's baseline blood pressure must be considered as well as the injury or illness. Another frequently used indicator is urine output, but if kidney injury exists, it may not be a viable option. More appropriate, sensitive, and specific indicators of perfusion are lactate and base deficit. The initial lactate level and the response of lactate to resuscitation correlate with multiorgan dysfunction and death.[3] Additionally, lactate has been shown to be noninferior to ScvO2 as a marker for resuscitation in septic shock.[22] Base deficit is also helpful in

the initial assessment of severity of illness or injury as well as progress over time. Base deficit changes over time are more predictive of survival than pH.[23] Base deficit has also been shown to correlate with risk of multiple organ dysfunction syndrome, development of acute respiratory distress syndrome, need for blood transfusion, development of renal failure, coagulopathy, and hospital length of stay.[24,25] Persistent elevation of either lactate or base deficit should prompt a search for an occult injury or the development of a complication such as abdominal compartment syndrome. A reasonable endpoint of resuscitation is normalization of lactate or base deficit. The role of vasopressors and inotropes varies with the type of shock, so these agents will be addressed more directly in the management of specific shock etiologies.

MANAGEMENT OF SPECIFIC TYPES OF SHOCK

Hypovolemic Shock

Shock in the trauma patient is considered hypovolemic until proven otherwise. The clinical presentation of the patient in hemorrhagic shock changes as the condition progresses. For the patient with less than 15% blood loss (approximately 750 mL), there will be little evidence of shock. As blood loss increases from 15% to 30%, the patient develops tachycardia, tachypnea, and anxiety. It is not until 30% of blood is lost that hypotension develops. At this point, anxiety has progressed to confusion. In the final stage of shock, more than 40% of blood volume has been lost and this condition is life threatening.[26] This development of hypotension is even more concerning in a young, previously healthy patient because he or she can often compensate until the point of hemodynamic collapse.

When the cause of blood loss is not externally apparent, one must consider four primary sites of massive internal bleeding: (1) long bone fractures (a femur fracture can bleed 2 to 3 units of blood into the thigh), (2) pleural cavities (each cavity can hold 2 to 3 L of fluid), (3) abdominopelvic cavity, and (4) the retroperitoneal space.[15] If bleeding is not the cause of the hypovolemia, gastrointestinal losses, urinary losses, third spacing of fluid, and dehydration must be considered.

The treatment for hypovolemic shock is to stop the volume loss and replace the fluid that has been lost. If it is hemorrhagic shock, hemostasis must be achieved, which may require short-term options such as a tourniquet or pelvic fixation, but surgical intervention may be necessary. Additional hemostatic agents are available, most commonly Quikclot powder and dressings (Z-Medica Corporation, Wallingford, CT) that use the inert mineral kaolin to clot blood.[27] Other developing treatments include recombinant factor VII, tranexamic acid, and red blood cell substitutes, but the roles of these agents are not clear at this time. The 2010 European guidelines, however, make weak recommendations to consider recombinant activated coagulation factor VII if major bleeding in blunt trauma persists despite standard attempts to control bleeding and best-practice use of blood components and that antifibrinolytic agents be considered in the bleeding trauma patient.[28] If hemorrhage is not the cause, other sources of volume loss or underlying disease processes must be controlled. Fluid replacement should resemble fluid lost. For massively bleeding patients, blood products must be delivered. High fresh frozen plasma to packed red blood cell and high platelet to packed red blood cell ratios have demonstrated improved survival.[29] Precise optimal ratios have not been well defined, but it appears that ratios greater than 1:2 are beneficial.[29] As discussed above in the general principles section, optimal resuscitation algorithms do not exist and gastrointestinal and third space losses are difficult to quantify. Consequently, resuscitating to a goal of normalizing lactate or base deficit remains a reasonable option.

Cardiogenic Shock

Cardiogenic shock is caused by pump failure resulting in decreased forward flow and tissue hypoxia. In a nontrauma population, this can be caused by myocardial infarctions, cardiomyopathies, and arrhythmias. Cardiogenic shock from trauma can result from myocardial contusion, penetrating injury, or traumatic valve injury. The development of shock from blunt cardiac trauma is rare because blunt cardiac trauma is usually self-limited.[30] It should, however, be considered in patients with mechanisms of injury involving high speed frontal impact, particularly if any injury to the sternum or chest wall is noted. Furthermore, the stress response to trauma causes a catecholamine response, which increases HR, contractility, and myocardial oxygen demand. In the patient with underlying atherosclerosis, this may overwhelm the heart's limited blood flow and lead to cardiogenic shock even if there is no direct cardiac trauma.

If a myocardial contusion or valvular trauma is suspected, a formal transthoracic echocardiogram, or transesophageal echocardiogram if possible, should be obtained. Initial treatment of cardiogenic shock includes reperfusion, treatment of arrhythmias, and optimization of fluid and electrolyte status. Reperfu-

sion is available in a great many US hospitals, but is often not possible in more austere combat environments and may be contraindicated with anticoagulant and fibrinolytic drugs. Percutaneous intervention may not be available, and thrombolysis is contraindicated in a trauma patient with head or facial trauma within the past 3 months or with internal bleeding in the past 2 to 4 weeks.[31] If the patient's trauma was mild and no significant bleeding resulted, thrombolytics can still be considered, but a full risk-benefit analysis must be completed. Trials of fluid should be cautious and responses should be monitored closely. Inotropic support may be necessary. A patient's blood pressure may not tolerate the addition of a dobutamine alone because the drug causes vasodilation, so the addition of norepinephrine or dopamine is frequently required. More advanced treatments, such as balloon pumps or ventricular assist devices, may be necessary but are beyond the scope of this chapter.

Distributive Shock

Many etiologies of distributive shock exist and the treatment for each cause differs. For example, toxins and medication overdoses can result in distributive shock. Although fluid resuscitation is important in this situation, specific antidotes for the toxin will be necessary. Specific toxicology will not be addressed in this chapter. This section will address the treatment of sepsis, anaphylaxis, neurogenic shock, and adrenal crisis.

Sepsis

The term "sepsis" is often used to refer to a disease spectrum that ranges from systemic inflammatory response syndrome to septic shock. Systemic inflammatory response syndrome criteria include hyperthermia (> 38.3°C) or hypothermia (< 36°C), tachycardia (> 90 beats per minute), hyperventilation (respiratory rate >20 breaths per minute or partial pressure of carbon dioxide < 32) and leukocytosis (white blood cells > 12,000) or leucopenia (white blood cells < 4,000).[29] Sepsis is defined as the presence of two or more of these criteria with a source of infection. The diagnosis shifts to severe sepsis when organ dysfunction is evident. The final stage, septic shock, is diagnosed when refractory hypotension is present.[32]

Sepsis is rare in the immediate posttraumatic period. If the cause of hypotension does appear to result from sepsis in the acute setting, a diagnosis of bowel injury should be considered. As a patient's ICU course continues, sepsis becomes a more likely cause of hypotension. Trauma patients at high risk for sepsis include patients with a prolonged ICU stay, dirty wound (eg, dirt, bowel injury), devitalized tissue (eg, crush injuries), and wounds with a high risk of complication (eg, anastomotic leak, pancreatic leak).[30]

The Surviving Sepsis Campaign has created an algorithm for the treatment of sepsis that has changed care in many ICUs. Based on early goal-directed therapy (Figure 32-2), first published by Rivers, the most recent Surviving Sepsis guidelines were published in 2012.[17] The algorithm begins with fluid resuscitation with crystalloid or colloid to a goal CVP of 8 to 12 cm H_2O (12–15 cm H_2O if intubated). If a goal mean arterial pressure of greater than 65 cm H_2O is not reached with fluid resuscitation, vasopressors should be initiated, with norepinephrine and dopamine being the first line agents of choice. Additional resuscitation goals are an ScvO2 greater than 70% and urine output greater than 0.5 mL/kg/h. If the ScvO2 goal is not reached, treatment options include further fluid resuscitation, red blood cell transfusion, or addition of inotropic support with dobutamine. If mean arterial pressure goals are not reached with fluid resuscitation to an adequate urine output and central venous pressure and vasopressor administration is required, 50 mg of hydrocortisone should be given every 6 hours. Adrenocorticotropic hormone (ACTH) stimulation test is not recommended.[14]

While resuscitation is underway, diagnosis and treatment must also be undertaken. Blood cultures should be obtained as well as cultures of other possible sources of infection (urine, cerebrospinal fluid, sputum).[14] If possible, cultures should be drawn prior to antibiotic administration but should not delay antibiotics. Imaging necessary to determine a diagnosis should also be obtained, but again, this should not delay antibiotic administration. Broad spectrum antibiotics (one or more agents directed against suspected organism with good penetration of likely sources) should be initiated within 1 hour once septic shock is suspected. Source control is the next step. All possible sources of infection should be evaluated and managed as necessary. Least invasive yet effective strategies should guide source control, and all potentially infected foreign objects and devices should be removed. Guidelines for management of blood products, mechanical ventilation, sedation, analgesia, glucose, renal replacement, bicarbonate, deep venous thrombosis prophylaxis, stress ulcer prophylaxis, and limiting support are also included but are beyond the scope of this chapter. They can be found at: www.survivingsepsis.org/guidelines.[14]

Anaphylaxis

Anaphylaxis is a severe allergic reaction caused by degranulation of mast cells or basophils. This process

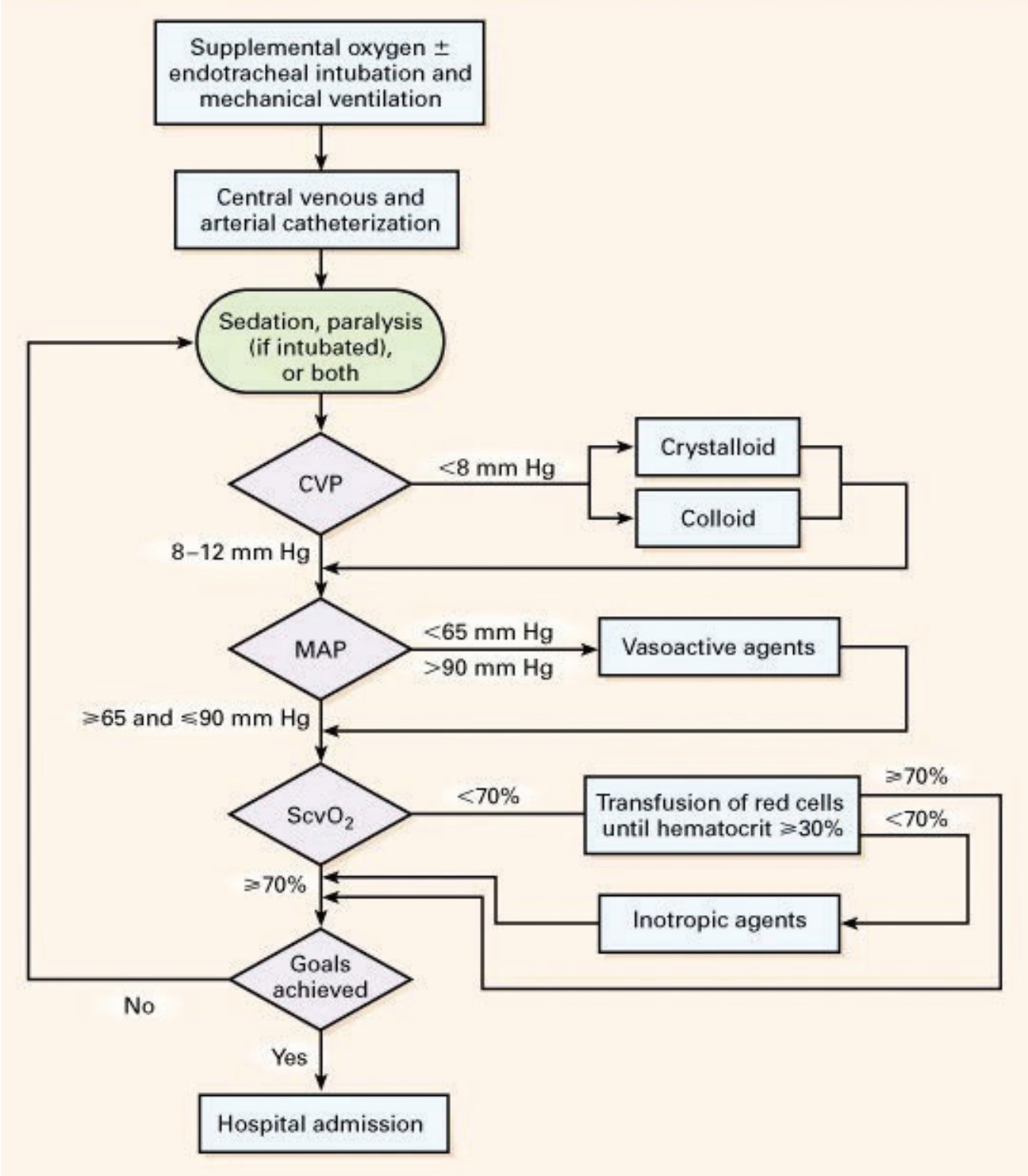

Figure 32-2. Early goal-directed therapy in septic shock.
CVP: central venous pressure
MAP: mean arterial pressure
ScvO2: central venous oxygen saturation
Reproduced with permission from: Rivers E, Nguyen B, Havstad S, et al. Early goal-directed therapy in the treatment of severe sepsis and septic shock. *N Engl J Med*. 2001;345:1368–1377.

is mediated by immunoglobulin E. Anaphylactoid reactions present similarly but are not mediated by immunoglobulin E. Common triggers include foods, insect stings, latex, and medications.

The mainstay of treatment for anaphylactic shock is epinephrine. Intramuscular injection (0.3 to 0.5 mg of 0.1% solution) can be used in mild or moderate cases. Slow, continuous intravenous (2 to 10 μg/min of 0.01% solution)[33] administration is recommended for patients with significant hypotension. Massive fluid shifts can occur with anaphylaxis, and aggressive administration of normal saline should accompany epinephrine. Antihistamines, glucocorticoids, and bronchodilators should also be administered.

Neurogenic Shock

Neurogenic shock can be distinguished from other forms of distributive shock by the relative bradycardia that occurs from loss of sympathetic tone. Neurogenic shock can result from any spinal cord lesion above T6. Penetrating injuries are the most common, but development of a large hematoma with resultant cord compression can also be a cause. Symptoms include hypotension, bradycardia, flaccid paralysis, loss of deep tendon reflexes, and priapism. The goals of treatment are to protect the airway, improve vascular tone, and decrease the potential area of injury by maintaining spinal perfusion. As in other forms of shock, initial treatment is fluid resuscitation, but as hypovolemia is corrected, vasopressors will likely be necessary. Norepinephrine, dopamine, and phenylephrine are all reasonable options. Maintenance of mean arterial blood pressure at 85 to 90 mm Hg for the first 7 days after acute spinal cord injury to improve spinal cord perfusion is recommended.[34] Additionally, atropine may be necessary to combat bradycardia.

Adrenal Crisis

Adrenal insufficiency in the critically ill patient can take many forms. It can be caused by a chronic disease process of the adrenals or of the hypothalamic-pituitary axis, or more acute causes such as medication withdrawal, critical illness, adrenal hemorrhage, hypoperfusion, or direct trauma. Either way, the resulting condition presents with nonspecific findings that can make diagnosis difficult. These findings include weakness, nausea, vomiting, abdominal pain, hypotension, fever, and hypoglycemia. The combined findings of hypotension, hyponatremia, and hyperkalemia should raise suspicion for adrenal crisis, which can be made by completing an ACTH stimulation test. The ACTH stimulation test will not be valid if the patient has received hydrocortisone, so if a patient requires emergent treatment and an ACTH stimulation test is desired later, dexamethasone should be used for steroid replacement. The first line steroid for the treatment of adrenal insufficiency, however, is hydrocortisone, 200 to 300 mg daily, in divided doses.[35] In addition to steroid administration, aggressive fluid resuscitation and determination and treatment of the cause are essential. In the setting of refractory septic shock, an ACTH stimulation test is not recommended, and treatment should be initiated with hydrocortisone at the same dose of 200 to 300 mg daily.[35]

Obstructive Shock

Obstructive shock is the result of an anatomical impediment such as a pneumothorax, pulmonary embolism (PE), or pericardial effusion that causes decreased venous return, excessive afterload, and/or decreased cardiac filling. The treatments for each of these disorders will be addressed independently. Aggressive fluid resuscitation may be necessary to maintain the patient until the obstruction is relieved, but it is strictly a temporizing measure. It is important to note that obstructive shock is likely to significantly worsen with mechanical ventilation. The sedation associated with the intubation process contributes to the condition, but more importantly, the increased intrathoracic pressure that results from positive pressure ventilation can further decrease preload and ventricular filling and exacerbate the condition.

Pulmonary Embolism

The classical findings of PE include dyspnea, pleuritic chest pain, and hemoptysis. In reality, the findings of PE are much less specific and range from dyspnea to cough to wheezing.[36] Patients may even be asymptomatic. Electrocardiogram may show an S wave in lead I, a Q wave in lead III, and T wave changes in lead III, which indicate right heart strain, but more commonly nonspecific ST changes, tachycardia, or a normal electrocardiogram are noted. Chest X-ray (CXR) may show a pleural-based, wedge-shaped defect, referred to as Hampton's hump, or paucity of vascular markings distal to the site of embolus, referred to as Westermark's sign, but the CXR is more likely to be normal. Given the nonspecific findings, it is important to maintain a high suspicion for PE, particularly in a trauma population. Numerous risk factors exist for PE and many of them are relevant to trauma patients. The trauma itself is a risk factor, but venous injury or repair, central venous catheterization, recent surgery, and immobility are also factors common to critically ill trauma patients.

Pneumothorax

Pneumothoraces are the most common injury resulting after blunt thoracic trauma.[11] Patients at risk must be evaluated for equal bilateral breath sounds, equal chest excursion, jugular vein distension, and mediastinal shift. A CXR is a reasonable test when looking for a pneumothorax, but many pneumothoraces are not seen on a CXR and obtaining a CXR could lead to a delay in treatment. US may be a better diagnostic option given its improved sensitivity in trained providers. Blaivas et al showed 98% sensitivity for US compared to 76% for CXR.[37] Additionally, US can be rapidly performed at bedside. Chest CT is another diagnostic option. Regardless of the diagnostic tool used, if suspicion is high and the patient is unstable, the chest should be decompressed without delay for completion of diagnostic tests. In an emergent setting, needle decompression at the second intercostal space along the midclavicular line can be lifesaving. Definitive management with chest tube placement should follow this decompression. It is important to remember that pneumothoraces are dynamic. Repeat evaluation over time may be necessary. An initial negative test or small pneumothorax does not rule out the development of a tension pneumothorax one hour later.

Cardiac Tamponade

Cardiac tamponade results from accumulation of fluid in the pericardial sac and is most commonly caused by penetrating trauma, but it can also result from blunt thoracic trauma.[15] Physical exam is significant for tachycardia, hypotension, muffled heart sounds, jugular vein distension, and elevated CVP. CXR may show a foreign body such as a bullet or other penetrating fragment or may demonstrate a waterbag heart. Electrocardiogram can range from normal to nonspecific ST changes to electrical alternans. The pericardial views obtained in the FAST exam allow for rapid bedside diagnosis of a pericardial effusion, but cardiac tamponade is a clinical diagnosis determined by hemodynamic compromise. Initial therapy consists of volume expansion to improve cardiac filling and cardiac output. This is only a temporizing measure. Definitive treatment is drainage of the pericardial fluid. This can be done by pericardiocentesis or surgery. Pericardiocentesis risks further injury and it may be difficult to drain any clotted blood. It may, however, be lifesaving in the acute setting. Surgical drainage is preferable in patients with potential intrapericardial bleeding or with clotted blood.[38] It allows for complete visualization, more complete drainage, and surgical correction of the source of bleeding. Surgical drainage may not be available, however, so pericardiocentesis—with or without US guidance—may be necessary to prevent hemodynamic collapse. In patients with cardiac tamponade, hemodynamic collapse can be precipitated by positive pressure ventilation (PPV). PPV should be avoided if at all possible, but at the very least, decompensation should be anticipated and optimization of fluid status should be achieved to ensure continued cardiac filling.

SUMMARY

This chapter delineates the various possible causes of hypotension in the ICU and discusses the treatments by category. See Figure 32-2, which provides an overview of this discussion. Although treatments vary based on the cause of hypotension, fluid resuscitation can be lifesaving in all forms of shock. After the patient's airway and breathing have been assured and fluid resuscitation has been initiated, diagnostic tests can be completed to further guide treatment.

It is essential to be vigilant to the patient's physiologic changes over time because shock is a dynamic state. Additionally, one must remember that often more than one cause may be contributing to a patient's shock state. For example, trauma patients can suffer from hemorrhagic shock, neurogenic shock, and obstructive shock simultaneously. Therefore, physical assessment must be rigorous and frequent, and physiologic parameters must be monitored concurrently.

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Chapter 33

DIFFERENTIAL DIAGNOSIS AND MANAGEMENT OF FEVER IN TRAUMA

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INTRODUCTION

Fever is one of the most common physical abnormalities in critically ill patients,[1] and may be caused by infectious or noninfectious etiologies. It is a specific and well-coordinated reaction to a challenge or insult and is part of the systemic inflammatory response. Macrophages and polymorphonuclear leukocytes release endogenous pyrogens such as interleukin-1 that orchestrate a variety of biochemical and physiological responses, of which temperature elevation is only one. Interleukin-1 (and probably other cytokines) stimulates the production of prostaglandins in the fever-mediating region of the preoptic area of the anterior hypothalamus, resulting in fever.[2]

Patients with traumatic injuries have an increased incidence of both fever and infectious complications, resulting in frequent diagnostic workups. A study of 510 critically ill trauma patients found that 79% of patients staying in a surgical and trauma intensive care units (ICUs) for at least 7 days were febrile, and 80% had leukocytosis at some point during their first week of ICU care.[3] A larger retrospective review of 37,448 trauma/neurologic ICU admissions found an overall fever incidence density of 38.2 per 100 days, which was higher than in other surgical, medical, or postcardiac surgery patients.[4] Although the presence of fever always prompts concern about infection, it is recognized that many trauma patients will become febrile despite persistently negative cultures. In these patients, it is thought that the severity of injury frequently leads to increased tissue necrosis, and that the associated stress response causes an increase in granulocytes in the peripheral blood. The increased tissue necrosis and increase in granulocytes lead to subsequent increase in temperature due to the inflammation itself and not necessarily due to an underlying infection.[5]

Surgery, which many trauma patients will undergo, elicits a similar inflammatory response, and both inflammation and macrophage phagocytosis of extravasated blood, common benign postoperative events, have been implicated in a febrile response shortly after surgery.[6] As a result, postoperative fever is a relatively common event immediately after major surgical procedures. A recent prospective observational study of all adult patients (n = 1,032) undergoing inpatient general surgical procedures during a 13-month period at an academic military medical center found an incidence of early postoperative fever (defined as temp >100.4˚F in the 72 hours following surgery) of 23.7%.[7]

Multiple studies of critically ill patients have fairly consistently attributed infectious causes to fever in approximately one-half of cases,[8–10] with pneumonia, sinusitis, urinary tract, bloodstream, wound and skin/soft tissue, and intraabdominal infections being frequent etiologies.[11–13] Noninfectious etiologies are responsible for the remaining febrile episodes. These noninfectious causes of fever may include myocardial infarction, cerebrovascular hemorrhage, thrombophlebitis, drug reactions, transfusion reactions, malignant hyperthermia, heat stroke, pancreatitis, and acute adrenal insufficiency.[1,14–18] Because trauma patients are at increased risk for thromboembolic disease, fever in the presence of a new or worsening oxygen requirement and/or persistent tachycardia should also prompt consideration of pulmonary embolism as a possible cause. A review of 311 patients with angiography-proven pulmonary embolism found a 14% incidence of otherwise unexplained fever (usually low grade).[19]

It is important to note that not all patients with infections are febrile. For unclear reasons, approximately 35% of septic patients are normothermic at presentation and another 10% are hypothermic.[20] Septic patients who fail to develop a temperature have a significantly higher mortality than febrile septic patients. Therefore, patients at risk for infection who manifest unexplained hypothermia should be aggressively evaluated.

In summary, although fever often has a noninfectious etiology, the provider must remain vigilant and appropriately evaluate fever when it represents a new finding or a change in the patient's condition. He or she must also employ appropriate empiric or directed antimicrobial therapy along with such proven measures as wound irrigation, removal of infected foreign bodies, debridement of devitalized tissue, and drainage of abscesses.

INFECTIOUS CONSIDERATIONS

In trauma patients who survive longer than 3 days, infection is second only to severe head injury as the cause of death, and an estimated 37% to 45% of all trauma patients will experience an infectious complication such as pneumonia during their initial hospitalization.[21] Furthermore, there is generalized agreement that war wounds are distinct from civilian traumatic injuries because high-velocity projectiles and blast devices employed as weapons cause a more severe injury than commonly seen in civilian settings, and the accompanying wounds are frequently contaminated by clothing, soil, and environmental debris.[22] Because of these factors and delay before definitive surgery, war wounds have a higher infection potential compared

to civilian injuries, with an incidence of 3.9% in the first 2 weeks after injury reported in one Vietnam-era study.[23] In addition, military trauma patients often have multiple injuries at multiple sites, allowing multiple avenues for possible infections to occur, so it is not at all infrequent for these patients to be infected at different sites with different organisms simultaneously. Furthermore, the subsequent insertion of various tubes and drains allows easy access of organisms in the intensive care environment into normally sterile sites of these already critically ill patients.[24]

A 2003–2004 survey by Yun et al of infections encountered in combat support hospitals in Iraq[25] found that gram-positive bacteria were responsible for most clinical infections in US troops with coagulase-negative staphylococci, accounting for 34% of isolates, *Staphylococcus aureus* for 26%, and streptococcal species for 11%. In contrast, the 732 cultures obtained from the predominantly Iraqi population yielded mostly gram-negative bacteria: *Klebsiella pneumoniae* (13%), *Acinetobacter calcoaceticus-baumannii* complex (11%), and *Pseudomonas aeruginosa* (10%). Both gram-negative and gram-positive bacteria were resistant to a broad array of antimicrobial agents. A similar retrospective review by Petersen et al of the infection patterns of 211 casualties (of which 85% were Iraqi nationals) evacuated to the USNS *Comfort* from the Iraqi theater during Operation Iraqi Freedom[26] found that 26.5% met criteria for infection. Patients with blast injuries, soft tissue injuries, more than three wound sites, loss of limb, abdominal trauma, and a higher Injury Severity Score (ISS) were more likely to be infected. Most infections involved wounds, which accounted for 84% of cases, followed by bloodstream infections (38%) and positive sputum cultures (21%). *Acinetobacter* infections were most common, representing 36% of all wound and 41% of all bloodstream isolates. *Escherichia coli* and *Pseudomonas* species accounted for 14% each, followed by coagulase-negative staph (9%), *Klebsiella* and *Enterobacter* species (both 6%), and *Proteus* species (5%). The remaining cases represented a mixture of *Streptococcus* species and miscellaneous gram-negative bacteria. Overall, 19% of organisms were gram-positive and 81% were gram-negative. Once again, multidrug resistance was common, with *Acinetobacter* isolates exceeding 80% resistance to all drugs tested except imipenem. *E coli* were 85% resistant to ciprofloxacin, and both *E coli* and *Klebsiella* species were very resistant to third generation cephalosporins. However, all of these isolates were carbapenem susceptible. Similarly pooled data of 66 Operation Iraqi Freedom and Operation Enduring Freedom casualties with orthopedic-related trauma treated at Brooke Army Medical Center in 2006 show that of the 26 patients (40%) who received a course of antibiotics, 13 were treated for *Acinetobacter*, 9 for *Klebsiella* species, 6 for *Pseudomonas aeruginosa*, 5 for *Enterobacter* species, and 6 for methicillin-resistant *S aureus*.[27] This and other data suggest that the pattern of causative organisms is similar in the Iraq and Afghanistan theaters.

In patients with negative cultures and without evidence of war injury-related infection, rare but notable febrile infections that have been reported in soldiers returning from Iraq and Afghanistan include tuberculosis, malaria, Q fever, brucellosis, and leishmaniasis. Tuberculosis is endemic in central and southwest Asia; the World Health Organization estimates a prevalence of 149 cases per 100,000 persons in Afghanistan and 45 cases per 100,000 persons in Iraq in2011.[28] The overall deployment-associated conversion rate has been estimated at 2.5%.[29] In Iraq, chloroquine-susceptible *Plasmodium vivax* malaria occurs at low rates (150 cases per year in 2008) and there have been no reported cases among US military forces serving there. Conversely, there were 467,123 malaria cases in Afghanistan, as reported to the WHO in 2008.[30] Transmission is seasonal from June to November, with negligible transmission occurring between December and April. Most cases involve *P vivax*, but *P falciparum* is also transmitted.[29] Although soldiers are routinely issued malaria prophylaxis, noncompliance remains a significant issue. An investigation of an outbreak of *P vivax* among Army Rangers after deployment to eastern Afghanistan yielded a self-reported 52% rate of adherence to mefloquine prophylaxis.[31]

Q fever is a zoonotic infection caused by *Coxiella burnetii* usually acquired through inhalation of infected particle aerosols. Infection typically results from direct contact with the reservoir hosts (commonly cattle, goats, and sheep), but it may also occur after exposure to contaminated manure, straw, or dust kicked up by vehicles. Infection presents acutely as either a self-limited febrile ("flu-like") illness, pneumonia, or hepatitis. Brucellosis, another zoonotic disease endemic to the Middle East, is transmitted to humans through contact with infected animals. *Brucella* bacteria may be ingested, inhaled, or percutaneously inoculated. There are rare reports of brucellosis among deployed US personnel. Visceral leishmaniasis, a protozoan infection usually transmitted by the bite of an infected sand fly, is a form of leishmaniasis that can be asymptomatic, subclinical, or symptomatic and that manifests with chronic fever, pancytopenia, hepatosplenomegaly, and cachexia. In Iraq, visceral leishmaniasis has been mostly reported from the more southern regions, especially among malnourished children.[32]

NONINFECTIOUS CAUSES OF FEVER

The following are potential noninfectious causes of fever in the ICU patient:

- cerebral infarction/hemorrhage
- adrenal insufficiency
- subarachnoid hemorrhage
- deep venous thrombosis
- postoperative fever (48 h postoperative)
- pulmonary emboli
- posttransfusion fever
- hematoma
- drug fever
- gout/pseudogout
- fat emboli
- cirrhosis (without primary peritonitis)
- myocardial infarction
- gastrointestinal bleeding
- pancreatitis
- phlebitis / thrombophlebitis
- acalculous cholecystitis
- ischemic bowel
- intravenous contrast reaction
- aspiration pneumonitis
- decubitus ulcers
- acute respiratory distress syndrome (both acute and late phases)
- neoplastic fevers
- alcohol or drug withdrawal[20]

While many of these will become evident during the workup due to their presentation, a few deserve additional discussion.

Neurogenic Fever

Fever after cerebral insult is common, especially when it results in intraventricular hemorrhage, and is independently associated with worse outcomes in patients when compared to nonfebrile similarly injured individuals. Erickson first described neurogenic hyperthermia in patients after brain surgery or head trauma in 1939.[33] Retrospectively studying cohorts of 40 patients, Albrecht et al observed fever of more than 38.0°C in 70% of patients after subarachnoid hemorrhage and in 68% after closed-head injury.[34] Another retrospective review of 251 consecutive patients with spontaneous supratentorial intracerebral hemorrhage admitted to a neurologic critical care unit showed that fever was present in 19% of all patients on admission and occurred in almost all patients (91%) at least once during the first 72 hours after hospitalization.[35] Refractory high fever (> 42°C) in the immediate aftermath of massive supratentorial[36] or brainstem[37] intracerebral hemorrhage is also well described. The mechanism by which intraventricular hemorrhage may alter hypothalamic function and cause central fever is unknown. Mechanisms proposed include direct hemotoxic damage to thermoregulatory centers in the preoptic region, interference with tonic inhibitory inputs from the lower midbrain that ordinarily suppress thermogenesis, and stimulation of prostaglandin production leading to temperature set-point elevation.[38]

In addition, direct physical or ischemic damage to the baseline temperature control center in the hypothalamus can result in persistent hypothermia or hyperthermia. Patients with such injuries may be hypothermic, with baseline body temperatures as low as 35°C (95°F), or they may be hyperthermic, with baseline temperatures as high as 41.1°C (106°F).[39] Similarly, damage to the hypothalamus can result in inappropriate or uncontrolled intermittent temperature elevations. These patients can have high fevers with relatively minor insults or for no apparent reason.

Hyperthermia of neurologic origin is a diagnosis of exclusion. Patients should be carefully examined and then undergo laboratory testing or imaging to search for a source of infection if indicated. Regardless of etiology, fever should be treated aggressively in head-injured patients because it is independently associated with a poor outcome. Only 1 to 2 degrees of hyperthermia has been shown to be deleterious on outcome in animal models of focal and global ischemia and traumatic brain injury (TBI), and the risk of poor functional outcome is increased with even mild temperature elevation (37.5°C) on admission after ischemic stroke or intracerebral hemorrhage.[40] Initially, sustained fever should be treated with acetaminophen and cooling blankets. Persistent fever that is refractory to acetaminophen and without infectious cause may require adhesive surface-cooling systems and endovascular heat-exchange catheters to maintain normothermia.

Drug Fever

Although the true incidence of this disorder is unknown, drug fever should also be considered in patients with an otherwise unexplained fever, particularly if they are receiving β-lactam antibiotics, procainamide, or diphenylhydantoin.[41] Any drug can cause fever due to hypersensitivity, and some drugs can also cause fever by inducing phlebitis at the site of administration. Drug fever is usually characterized by high spiking temperatures and shaking chills, lack

of appropriate pulse rate response, and a relative bradycardia in the absence of intrinsic conduction defects or beta-blockade, and may be associated with leukocytosis and eosinophilia. A concomitant maculopapular rash helps establish the diagnosis, but the rash is present in only 5% to 10% of cases. Rarely, an increased white blood cell count with a left shift, eosinophilia, a moderate elevation of serum transaminases, or a markedly elevated erythrocyte sedimentation rate (>100 mm/h) are seen.[42] Fever usually resolves in 1 to 3 days but can take up to 7 days to return to normal after the offending agent is removed.[43]

Pancreatitis

Pancreatitis should be considered in patients who have suffered trauma to the epigastrium and have phenomena suggestive of intraabdominal injury. Because the blunt force required to injure the pancreas is significant and penetrating trauma usually injures multiple organs, other organs are also affected when the pancreas is injured. Therefore, multiple organ injury is a red flag suggesting the possibility of a pancreatic injury. Trauma to the pancreas can also occur during damage control or elective operative procedures in the upper abdomen and result in pancreatitis postoperatively. Stern reported that the pancreas was injured more often than was recognized during operative procedures and indicated that the pancreas was particularly vulnerable to injury during operations on the gallbladder with exploration of the common duct, splenectomies, right nephrectomies, pancreatic biopsies, and repair of duodenal ulcers.[44] The diagnosis can be confirmed by elevated serum amylase and lipase levels. Although it lacks sensitivity (75% to 92%) and specificity (20% to 60%), measurement of the serum amylase level is the most widely used method of diagnosing pancreatitis. The advantages of amylase testing are that it is quickly performed, easily obtained, and inexpensive. However, a variety of nonpancreatic conditions, notably injury to the salivary glands or bowel, can also cause increased amylase levels. Lipase levels will also be elevated in pancreatitis, and the test has better specificity (50% to 99%) and sensitivity (86% to 100%) than measurement of amylase.[45] Contrast-enhanced computed tomography (CT) scan provides the best imaging of the pancreas and surrounding structures and may be useful when other diagnostic studies are inconclusive. Direct injury as well as retroperitoneal hematoma, retroperitoneal fluid, free abdominal fluid, and pancreatic edema, all of which frequently accompany injuries to the pancreas, can be visualized. Ultrasound may be a suitable alternative in nonobese patients, with a reported sensitivity of 62% to 95%[45]; however, the pancreas will be obscured by bowel gas in up to 35% of patients.[46]

Acalculous Cholecystitis

Acalculus cholecystitis, the result of gallbladder ischemia and bile stasis, has an estimated incidence of 1.5% among critically ill patients.[47] It is frequently unrecognized, especially in septic patients or in patients recovering from abdominal sepsis, because of the nonspecific clinical signs (pain in the right upper quadrant, nausea, vomiting, and fever) and laboratory workup (leukocytosis and elevated liver enzymes). A high index of suspicion is needed to prevent delays in diagnosis and subsequent disease progression to ischemia, gangrene, and perforation. Right upper quadrant abdominal ultrasound findings such as a gall bladder wall thickness greater than 3 mm, intramural lucencies, gallbladder distension, pericholecystic fluid, or intramural sludge are suggestive but not specific for acute cholecystitis. CT scanning also has a high sensitivity and specificity for these findings and will better depict an inflammatory pericholecystic reaction in the gallbladder fossa. Hepatobiliary scintigraphy is also a sensitive modality in diagnosing acute cholecystitis but is characterized by a high false-positive rate (> 50%) in critically ill patients.[48] The treatment of choice is percutaneous cholecystostomy, which is also the definitive therapy in most patients. Open cholecystectomy is, however, recommended if the abdominal signs, fever, and leucocytosis do not improve within 48 hours of percutaneous cholecystostomy.[20]

Malignant Hyperthermia and Neuroleptic Malignant Syndrome

Malignant hyperthermia (MH) and neuroleptic malignant syndrome (NMS) are rare but should be considered in the critically ill trauma patient when fever is especially high. MH is more common in the operating room than in the ICU and occurs after general anesthesia with volatile inhalational agents and/or succinylcholine (suxamethonium). It can take up to 24 hours after exposure to an offending agent to manifest. It is caused by a mutation in the ryanodine calcium channel of sarcoplasmic reticulum leading to uncontrolled intracellular calcium release and tonic contraction of skeletal muscle with ensuing hyperthermia, acidosis, rhabdomyolysis, and hyperkalemia.[49] Also typical are tachycardia, increased carbon dioxide production, and elevated creatine phosphokinase (CPK) values consistent with muscle injury. Treatment involves external or internal cooling, correction of acidosis and hyperkalemia, and rapid administration of dantrolene.

NMS is a consequence of blockade of dopamine receptors and is usually caused by antipsychotic agents (phenothiazines, thioxanthenes, butyrophenones). It also manifests with muscle rigidity, high fever, and increasing CPK concentrations. It is similarly treated with discontinuation of the offending agent, cooling, supportive care, and muscle relaxation with benzodiazepenes and/or dantrolene. Unlike in MH, because the rigidity and resultant hyperthermia in NMS are centrally initiated, both symptoms can be rapidly controlled with nondepolarizing neuromuscular blockade once the airway is secured.

Serotonin Syndrome

Serotonin syndrome is another often unrecognized pharmacologic cause of fever encountered in the ICU. It consists of a clinical triad of mental status changes, autonomic hyperactivity, and neuromuscular abnormalities and is caused by overstimulation of central 5-HT_{1A} receptors. It always occurs within 24 hours of an increase in dose or addition of a serotonergic agent and is believed to remain unresolved unless the offending agent is discontinued. A variety of serotonergic agents have been implicated in the syndrome, including selective serotonin reuptake inhibitors; tricyclic antidepressants; 5-HT_3 antagonist antiemetics (odansetron, granisetron); metoclopromide; dextramethorphan; fentanyl; pentazocine; tramadol; buspirone; and trazodone; as well as sumatriptan, linezolid, valproate, lithium, and monoamine oxidase inhibitors. Especially severe reactions have been reported with meperidine. Although most patients recover, rare cases can result in death. Treatment consists of discontinuation of the offending agent and administration of benzodiazepines, which have a beneficial effect in moderate cases. Similar to NMS, in severely ill patients with hyperthermia (a temperature higher than 41.1°C) immediate paralysis should be induced with nondepolarizing agents such as vecuronium, followed by orotracheal intubation and ventilation.[50] Succinylcholine should be avoided because of the risk of arrhythmia from hyperkalemia associated with rhabdomyolysis. Therapies such as propranolol, bromocriptine, and dantrolene are not recommended.[51]

Other Causes

Other noninfectious causes of fever in critically ill patients are heatstroke, withdrawal of certain drugs such as alcohol, opiates, barbiturates, or benzodiazepines (often with associated tachycardia, diaphoresis, and hyperreflexia), and blood transfusion (especially platelets). Febrile nonhemolytic transfusion reactions are common and are thought to stem from the formation and/or release of cytokines during the storage of the blood. They are estimated to occur in approximately 3% to 7% of patients receiving red blood cell transfusions and 20% to 30% of those receiving platelet transfusions. On occasion, fevers can approach 40°C (104°F). Also, large isolated hematomas have been reported to result in fever in both adult[52] and pediatric patients.[53]

Atelectasis

Atelectasis is often attributed as a cause of fever in the ICU but conclusive data to support this is lacking. Inducing atelectasis in experimental animals by ligation of a mainstem bronchus does not produce fever.[54,55] Furthermore, Engoren studied 100 postoperative cardiac surgery patients and was unable to demonstrate a relationship between atelectasis and fever.[56] Currently, the role of atelectasis as a cause of fever is unclear; however, atelectasis probably does not cause fever in the absence of pulmonary infection.[20]

WORKUP OF FEVER

The workup of the febrile ICU patient should be directed by the history, physical examination findings, and results of initial diagnostic tests. As always, the evaluation should start with a detailed history, which can help the clinician narrow the differential. A detailed geographical history and the time of onset and duration of symptoms are essential for a complete workup. The history should also include details of visits to farms, caves, and health facilities; consumption of local foods and unpurified water; activities involving fresh or salt water exposure; immunizations and travel prophylaxis received (and compliance with the requirements); as well as sexual activity. A history of contact with ill individuals can be helpful, particularly for localized epidemics (eg, *Legionella*), emerging infections (eg, severe acute respiratory syndrome), or risk assessment for viral hemorrhagic fever.

It is useful to remember that postoperative fever is common within the first 72 hours after surgery. It is usually caused by the release of endogenous pyrogens into the bloodstream, and is usually not of infectious etiology. In these patients, during the first 72 hours and if fever is the only indication, a chest x-ray or cultures are not mandatory, while surgical wounds should be examined daily for infection and a high level of suspicion should be maintained for thromboembolic events

(pulmonary embolism, deep venous thrombosis) or thrombophlebitis.[42] If the patient clinically worsens or remains febrile longer than 72 hours, at which point infection becomes increasingly likely, further investigation is warranted.

Predisposing factors, the type and site of surgery, and underlying comorbidities should be taken into account to help guide the subsequent workup and treatment. The most common infections historically reported in ICU patients are pneumonia, followed by sinusitis, bloodstream infection, and catheter-related infection.[20] Of note, pneumonia is particularly common after upper abdominal surgery or thoracic surgery, wound infections after upper abdominal surgery, and urinary infections after lower abdominal surgery.[57] In all febrile patients, before the initiation of any treatment, at least two blood cultures by separate needles from different sites as well as other appropriate cultures should be obtained.

For patients with fever alone who are otherwise stable, there is usually no need to remove or change all in-dwelling catheters. Patients with worsening sepsis, vasopressor-dependent shock, peripheral embolization, disseminated intravascular coagulation, or acute respiratory distress syndrome should be started on empiric antibiotic therapy after cultures are obtained, and should have all intravascular catheters removed and then reinserted at new sites if indicated.[58] Since up to 20% of central venous catheters are colonized at removal, most unassociated with local infection or bacteremia/fungemia, routine culture of central venous catheters is not recommended. Routine catheter cultures add to microbiology laboratory expense and can lead to unnecessary therapies if interpreted inappropriately. The predictive value of a positive catheter culture is very low when there is a low pretest probability of catheter sepsis, and catheters removed from ICU patients should only be cultured if there is strong clinical suspicion of catheter sepsis.[42]

Although not routinely performed, if the expertise needed for processing is available, quantitative cultures can be drawn from central catheters and peripheral veins if central line infection is strongly suspected and there is no obvious tunnel infection. The diagnosis of line-related sepsis can be made by a colony count in blood cultures drawn from the catheter that is 10 times higher than the colony count in cultures drawn peripherally[59] or by a difference of 2 hours or more in time to positivity between the catheter and peripheral cultures.[60]

In continuous bacteremia, as with endocarditis or intravascular infections, three sets of blood cultures are usually adequate to recover organisms.[61] In addition, a complete metabolic profile including liver function tests is helpful in determining the etiology. A marked increase in alkaline phosphatase and a rising bilirubin can suggest cholecystitis. Unfortunately, this finding is also nonspecific because increased liver enzymes can also be seen in bacteremia and drug-induced fever, and increases in alkaline phosphatase occur with bony injuries. An elevated lipase likewise can be of diagnostic value and may indicate traumatic or drug-induced pancreatitis.

On urinalysis, pyuria, microscopic hematuria, and positive cultures may point to a diagnosis of urinary tract infection. However, a positive urine culture in catheterized patients is not always indicative of infection, and the diagnosis of urinary tract infection as a source of the fever in these patients should be a diagnosis of exclusion. The presence of sterile pyuria should prompt consideration of tuberculosis as well as a search for eosinophiluria, which would suggest a drug-induced interstitial nephritis.

A chest radiograph should be obtained to rule out pneumonia. The presence of infiltrates can make it difficult to differentiate pneumonia from pulmonary contusions and/or infarct, fluid overload, or even congestive heart failure. It is often necessary to obtain a noncontrast CT scan of the chest, especially in patients who are ventilator dependent. If pleural effusions are present, a thoracentesis may be considered to rule out empyema. Sputum collection for Gram stain and culture can be valuable to guide antibiotic choice when pneumonia is present. Stool testing for *Clostridium difficile* toxin should be done in patients who have received antibiotics in the recent past, even when diarrhea is not a prominent symptom; testing of stool for fecal leukocytes is sensitive but not specific for diagnosing pseudomembranous enterocolitis and infection with enteroinvasive bacteria.

If the initial workup is unrevealing, a CT scan of the abdomen and pelvis, with intravenous contrast when possible, can be done to look for intraabdominal abscess, especially in patients who have undergone penetrating abdominal trauma or abdominal surgery. CT scanning can also reveal a retroperitoneal hematoma, cholecystitis, pancreatitis, or colitis suggestive of pseudomembranous enterocolitis. It is important to note that an abscess will take time to organize and form following abdominal injury or surgery, and a study obtained in the first few days after such an injury or surgery will likely show nonspecific intraperitoneal fluid collections and/or residual free air that is unlikely to change clinical management or outcome. There is little data in the literature to guide the clinician on the optimal timing of CT scanning to obtain the highest positive yield. A retrospective study of 53 critically ill surgical patients found that no scan

was positive for abscess prior to the 8th postoperative day and recommends not obtaining a CT scan in the 1st week following abdominal surgery in the workup of sepsis.[62] Abdominal ultrasonography can also be considered. This low-cost noninvasive test can be performed at bedside in the unstable patient, and can detect fluid collections as well as abnormalities of the liver, gallbladder and hepatobiliary system, and the pancreas.

CT scan of the sinuses without contrast may also be done to look for sinusitis, especially in patients with a nasogastric tube or nasotracheal tubes. Sinusitis occurs in approximately 5% of ICU patients and more frequently in neurosurgical patients.[63] If this approach does not lead to a diagnosis, venous Doppler ultrasonography of the lower extremities and/or a thin-cut CT scan of the chest will help to rule out thromboembolic disease in the right setting.

EMPIRIC THERAPY

If an infectious cause of fever is suspected, urgent initiation of empiric antimicrobial therapy is necessary for unstable or high-risk patients while the diagnostic evaluation is ongoing and before culture results are available. Delay of effective antimicrobial therapy is associated with increased mortality from infection.[64,65] Therefore, antibiotic therapy should begin within 1 hour after the diagnosis of severe sepsis or septic shock is considered.[66] The choice of regimen will depend on the suspected infectious etiology and must be broad enough to cover the likely pathogens.

TREATMENT OF FEVER

Providers commonly treat fever due to concern that fever may cause patient discomfort and result in undue metabolic stress in unstable critically ill patients with limited reserve. There is widespread agreement that fever in the presence of TBI is associated with worsened neurologic outcomes, including longer ICU stays, increased intracranial pressure, lower Glasgow coma scale scores, and poorer functional status.[67–69] In the presence of TBI, fever may be associated with increased excitatory amino acid release, increased vasogenic edema, increased intracranial pressure, and increased metabolic expenditure, all of which ultimately result in increased neuronal loss.[70] Because of these effects, it is prudent to avoid hyperthermia in TBI patients through use of acetaminophen and external cooling in the absence of contraindications. Drugs that inhibit platelet function are best avoided, however.

Conversely, there is little data in the literature to support the treatment of fever in nonneurologic critically ill patients given that fever is a normal host response to infection. In a randomized study of 38 febrile surgical ICU patients, use of external cooling resulted in no significant differences in recurrence of fever, incidence of infection, antibiotic therapy, length of stay in the ICU and hospital, or mortality.[71] Similarly, Schulman et al conducted an open, randomized, prospective clinical trial comparing an aggressive fever treatment strategy (acetaminophen for fever > 38.5°C and a cooling blanket added if > 39.5°C) with a permissive strategy (treatment reserved for fever > 40°C only) in patients without brain injury admitted to a trauma unit. Of note, the study was prematurely stopped due to safety concerns after interim analysis revealed an excess mortality rate of 7 of 44 patients (16%) in the aggressive as compared to 1 of 38 (3%) in the permissive group ($P = .06$).[72] Laupland's retrospective review of 24,204 ICU admissions likewise concluded that the presence of fever was not associated with increased ICU mortality and was actually associated with improved survival among the subset of trauma and neurologic patients.[4] Thus, although fever has some harmful effects, it appears to be an adaptive response that helps rid the host of invading pathogens and has been shown to enhance several parameters of immune function, including antibody production, T-cell activation, production of cytokines, and enhanced neutrophil and macrophage function.[73–75] Therefore, in the absence of patient discomfort, cardiac or pulmonary insufficiency, myocardial ischemia or neurologic injury, and if there is minimal potential or actual patient detriment, fever should be considered a normal physiologic response to inflammation and should not actively be suppressed.

SUMMARY

In summary, fever is a common finding in critically ill trauma patients, and may be caused by infectious or noninfectious etiologies. The presence of fever in a critically ill injured patient should prompt a thorough evaluation including a detailed geographic history, physical examination with careful inspection of wounds, and a

search for signs of acute abdomen or occult infections such as sinusitis or perirectal abscess. Blood cultures, chest radiograph, urinalysis, and urine culture should be obtained and will often yield a diagnosis. Unstable or deteriorating patients should be promptly started on empiric antibiotic therapy and should have in-dwelling invasive lines removed or exchanged. Noninfectious causes of fever should also be considered, especially in patients who appear nontoxic despite recurrent high fevers and a workup that does not reveal an infectious cause. Fever should be aggressively treated in patients with head injuries, although it has not been shown to be detrimental in other groups and may be beneficial for patients battling infection.

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Chapter 34

THROMBOEMBOLIC DISEASE AND MANAGEMENT OF ANTI-COAGULATION IN TRAUMA

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INTRODUCTION

Patients who suffer from acute traumatic injuries are at significant risk for venous thromboembolism (VTE) during their recovery period. Traumatic injuries have effects on the physiologic mechanisms responsible for clot formation and resolution, making prevention and treatment challenging. In addition, trauma patients often have injuries that involve and subsequently predispose them to hemorrhage, limiting the physician's ability to prevent and treat VTE.

Patients who are injured in a combat theater have additional risk factors for the development of VTE. Massive resuscitation with component products, whole blood, and hemostatic agents such as factor VIIa and tranexamic acid may increase clotting risk. Patients spend a large part of the first 48 hours after injury traveling back to the continental United States via the air evacuation system. During this time they are intubated and sedated, sometimes paralyzed. Extubation often must be delayed, even when it may be appropriate clinically, in order to maintain adequate control of the patient during transport, because options to make interventions during flight are limited. Although prophylaxis is often administered en route, it may be withheld for precautionary purposes in patients who have recently been resuscitated in an operating room.

After the first 48 hours, patients can spend weeks traveling to and from the operating room for wound debridement and other procedures. Although some surgeons are comfortable continuing chemical VTE prophylaxis during trips to the operating room, others are not, which results in substantial time without chemical prophylaxis. Injuries from improvised explosive devices (IEDs) may involve lower extremity amputations, preventing the regular application of sequential compression devices (SCDs) or other types of mechanical prophylaxis. Finally, when VTE does occur, treatment failure and recurrence rates are high.

PATHOPHYSIOLOGY OF VENOUS THROMBOEMBOLISM IN TRAUMA PATIENTS

The Virchow triad, which consists of blood stasis, endothelial injury, and hypercoagulability, broadly defines the factors that determine the propensity to form clot. Although trauma is a heterogeneous disease, any patient with penetrating trauma and many patients with blunt trauma will experience endothelial injury. The coagulation cascade will be activated at the site of injury, triggered by the exposure of the collagen matrix and basement membrane that support the endothelial lining. Endothelial damage in one area will cause systemic changes, and clot can form in areas far from the primary injury site, mainly the venous system in the lower extremities. The greater the overall damage to the endothelium, the higher is the propensity to form clot.[1]

All patients with major trauma will experience stasis during their recovery period. As mentioned above, patients injured in a combat theater, for any given degree of injury, are more likely to require prolonged intubation, sedation, and paralysis because of the need for rapid air evacuation back to Germany and the continental United States. Although deep venous thrombosis (DVT) can occur within the first 24 hours after injury, rates increase significantly during recovery, reflecting the cumulative effect of longer time periods of immobility.[2–4]

Traumatic injury results in derangements of the coagulation cascade, including an increase in circulating tissue thromboplastin and activated procoagulants and a decrease in fibrinolytic activity. The resulting hypercoagulable state tends to be proportional to the degree of injury and decrease in tissue perfusion.[1] It follows that trauma patients who are not being adequately prophylaxed have high rates of DVT and pulmonary embolism (PE).

Effects of Massive Transfusion, Factor VIIa, and Tranexamic Acid

For research purposes, a massive transfusion is defined as the requirement for more than 10 units of packed red blood cells (pRBCs) during a resuscitation. Recent data from Operation Iraqi Freedom (OIF) shows that 5% of all patients admitted to US combat support hospitals in theater require a massive transfusion.[5] Data from OIF has also shown that during resuscitation a 1:1 ratio of pRBCs to fresh frozen plasma (FFP) is associated with a lower mortality than higher ratios.[5] The result has been a change in focus, where clinical practice guidelines for resuscitations in theater call for limiting crystalloid and increasing the use of whole blood, FFP, platelets, cryoprecipitate, and factor VIIa.

Because of the increases in inflammation, mortality, and multiorgan failure associated with massive transfusion, an increase in VTE among patients who survive their initial injury might be expected.[6] In fact, the need for a massive transfusion is an independent predictor of delayed initiation of chemical prophylaxis, which subsequently increases the risk for VTE during

recovery.[7] Transfusion of pRBCs has been independently associated with VTE in patients recovering from trauma.[4,8,9] In a study of patients in medical and surgical intensive care units, platelet transfusion was independently associated with VTE, although this particular study excluded patients with traumatic injuries.[10]

Although the effects that FFP and cryoprecipitate have on multiple organ failure and lung injury have been well described,[11,12] it is not clear whether either represents an independent risk factor for VTE. One small study found that receipt of FFP was highly associated with VTE, but the patients receiving FFP also received over 10 units of pRBCs, so an independent effect from FFP could not be established.[9] Because military protocols have advocated fixed ratios of blood component products during resuscitation, it has been difficult to isolate the effect that any individual component has on VTE risk in the active duty population recovering from a combat injury.[4]

The CRASH-2 trial, a large randomized study of tranexamic acid use for trauma, did not show an increased risk of vascular occlusion, a composite secondary outcome measure that included pulmonary embolism.[13] A randomized study of factor VIIa use for trauma patients also did not show an increased risk for thromboembolic events,[14] although this study was much smaller than the CRASH-2 trial. Two studies of factor VIIa in US military casualties did not find an increase in thromboembolic events either.[4,15] In a review of thromboembolic complications resulting from the use of factor VIIa at their institution, authors from the Baltimore Shock Trauma Center found DVT or PE in 6 of 285 patients (2.1%) who had received the drug. There was no comparison group available, and this VTE rate seems well within what would be expected in the typical trauma population who had not received factor VIIa.[16]

In summary, pRBCs and platelets have been independently associated with VTE in trauma and critically ill patients, respectively. Although FFP, cryoprecipitate, factor VIIa, and tranexamic acid have not been proven to increase VTE risk, given their mechanism of action and known effects on coagulation, it seems reasonable to assume they increase the likelihood for clot to some degree. A higher VTE rate during recovery should be expected for patients who survive a massive transfusion.

Prevalence of Venous Thromboembolism in Trauma Patients

Early estimates put the rate of DVT in young patients with major trauma and without prophylaxis at approximately 20%; both elderly patients with a hip fracture and patients with head or spinal cord injury have an estimated rate of 40%.[17] Likely due to the inherent heterogeneity of any trauma population studied, DVT rates in other reports vary from 20% to 90%.[1,18] Patient inclusion criteria, imaging modalities used for surveillance and detection, and the presence of prophylaxis will significantly affect overall VTE rates. For similar reasons, PE rates also vary by report, ranging from 4% to 22%,[1] with some estimated rates as low as 0.7%.[19]

Walter Reed Army Medical Center (WRAMC; now Walter Reed National Military Medical Center), where a large proportion of combat casualties are ultimately transferred after being injured in theater, has collected data on VTE events among casualties from OIF and Operation Enduring Freedom (OEF). Over a period of 18 months, from September 2009 through March 2011, data on 506 patients was recorded. No systematic screening was performed, but 46 patients (9.1%) had a documented VTE , and 18 (39.1%) of these events occurred during the initial air evacuation prior to the patient being admitted to WRAMC.[4]

Data from clinical studies have identified specific risk factors that predispose patients to the development of VTE.[1,8,20,21] The typical combat casualty requires surgery and central venous catheterization, which both increase the risk for VTE in all hospitalized patients.[20] In trauma patients specifically, the following factors have been independently associated with VTE in individual studies or metaanalyses: age, blood transfusion, surgery, lower extremity fracture,[4] long bone fracture, spinal fracture, pelvic fracture, spinal cord injury, delay in initiating chemical prophylaxis, and increasing injury severity score (ISS).[8,21] It is not clear at this time how each factor interacts with the others to provide a cumulative risk score for a given patient.

PREVENTION OF VENOUS THROMBOEMBOLISM

Chemical prophylaxis with unfractionated heparin (UFH) or low-molecular weight heparin (LMWH) is the primary method of prophylaxis for most hospitalized patients.[20] Trauma patients, and especially those injured on the battlefield via high-velocity gunshots or IEDs, pose a unique challenge because they can initially present with a hypocoagulable state. Given modern surgical techniques that involve damage control, packing of wounds, and early evacuation, physicians are often hesitant to start any therapy that might

increase the risk for bleeding. Therefore, this chapter will discuss mechanical methods of prophylaxis (the sequential compression device [SCD]) and prophylactic inferior vena cava (IVC) filters as alternatives to heparin for prophylaxis. Much of the discussion will be based on two major position papers, the Eastern Association for the Surgery of Trauma (EAST) guidelines for prevention of VTE in trauma patients, published in 2002,[21] and the American College of Chest Physicians (ACCP) guidelines published in 2008 for prevention of VTE in all hospitalized patients.[20]

Mechanical Prophylaxis

An SCD is a dynamic device that fits like a sleeve over an extremity, usually the calf or thigh, and provides intermittent compression generated by an external compressor. SCDs have been shown to affect two components of the Virchow triad. They reduce stasis by increasing blood flow in the extremity they are worn on, and they increase fibrinolysis, thereby reducing blood coagulation. Of note, both effects seem to decline rapidly once SCDs are removed, implying that continuous, uninterrupted use is required for optimal benefit.[21]

Studies assessing the efficacy of SCDs in hospitalized patients and in trauma patients in particular have been inconsistent. The EAST guidelines[21] cite a lack of level I and II evidence, and use level III evidence to conclude there is no evidence that SCDs prevent VTE when compared to no prophylaxis. They note that there is some data to support the use of SCDs in head-injured patients. A recent metaanalysis of patients requiring neurosurgery would support this benefit, but the studies included in the analysis were not made up of trauma patients.[22]

A metaanalysis published in 2006 also concluded that SCDs have no benefit over placebo. This analysis included only two randomized controlled trials (RCTs)[23] with a combined total of 562 trauma patients. Three other RCTs and 13 observational studies were reviewed, but methodological issues in each study and differences between studies prevented meaningful interpretation and pooling of the data.

The 2008 ACCP guidelines also note the lack of good evidence for efficacy. They discuss the additional concerns that up to a third of all trauma patients will have a contraindication to SCDs due to extremity injuries, and nursing and patient compliance with SCDs tends to be poor. In summary, they recommend the use of SCDs for all trauma patients with a contraindication to chemical prophylaxis.[20]

The US military has adopted this approach for in-theater casualties,[24] which seems prudent for the following reasons: (1) a significant number of patients injured in theater will have an initial contraindication to chemical prophylaxis; (2) there is physiologic data and rationale to support the use of SCDs; and (3) SCDs seem to have few side effects provided they are not applied to an injured extremity. In addition, in studies of critically ill or traumatically injured patients who were screened for events, 3% to 10% of patients who develop DVT have the clot detected on their initial, day 1 ultrasound.[7,9,10,25] Delay in the initiation of chemical prophylaxis of more than 4 days from the date of injury results in three times the risk of VTE compared to institution of chemical prophylaxis within the first 48 hours.[7] SCDs would seem a safe and potentially effective "bridge" to chemical prophylaxis to reduce this risk.

Unfortunately, many patients with IED injuries will not be able to use SCDs on their lower extremities. Although the data supporting the use of SCDs on the upper extremity for prevention of VTE is particularly weak,[21] for the reasons listed above, this approach would also be reasonable for the high-risk trauma patient with no other options for prophylaxis.

Chemical Prophylaxis

Because several studies have shown that UFH may not be sufficient for prophylaxis in trauma patients, the EAST guidelines recommend LMWH be used. These guidelines list nine studies that evaluated the use of UFH for patients recovering from major trauma, including a metaanalysis showing no reduction in VTE events when UFH is used for prophylaxis.[26] Although the 9th consensus ACCP guidelines cite this same report, many of the studies suffer from small sample sizes, and they note the possibility of type II error.[27]

LMWH has become the treatment of choice for high-risk trauma patients.[20,21] This is mainly due to its safety and ease of use, and because a high-quality RCT showed superiority to UFH. Data from service members evacuated to WRAMC with traumatic injuries also show that LMWH specifically is associated with a reduced risk for VTE.[4] The following discussion reviews the individual studies and consensus guidelines that recommend LMWH for trauma patients.

In a study of 442 trauma patients comparing a LMWH to a mechanical method of prophylaxis (SCD), only one patient in the group randomized to Lovenox (Sanofi, Bridgewater, NJ) 30 mg, subcutaneous, twice a day, suffered from DVT.[28] There was no significant increase in bleeding in the Lovenox group, implying that LMWH would not increase bleeding risk more than using SCD alone. However, the SCD group in this study also had a low rate of VTE that was not signifi-

cantly different than the LMWH group. Knudson[29] also compared Lovenox 30 mg, subcutaneous, twice a day, to mechanical prophylaxis and found no difference in VTE rates, although the number of events across both groups was very small. Knudson found no significant increase in bleeding in the LMWH group.

Stannard[30] compared two different prevention protocols using Lovenox 30 mg, subcutaneous, twice a day, in 200 orthopedic trauma patients. One group started Lovenox within 48 hours of admission, while the other started mechanical prophylaxis and delayed Lovenox for 5 days. For the entire population, 22 of 200 patients (11%) experienced DVT. There was no statistically significant difference between groups.

In a prospective cohort study of high-risk trauma patients (mean ISS = 19.5) receiving the LMWH dalteparin at a dose of 5,000 units daily, rates of DVT and PE were 3.9% and 0.8%, respectively. Of the 16 patient deaths, none was judged as being due to PE or late hemorrhage.[31] In an RCT that enrolled orthopedic trauma patients and compared two different doses of the LMWH nadroparin, 3 of 215 patients (1.4%) had DVT after 10 days, and 5 of 150 (3.3%) had DVT after 6 weeks. Major hemorrhage occurred in 10 of 283 patients (3.5%).[32]

In an RCT with 344 patients with trauma and an ISS greater than or equal to 9, Lovenox 30 mg twice a day was significantly more efficacious than UFH 5,000 units twice a day for reducing DVT.[33] LMWH was not associated with an increase in bleeding rates or blood transfusions when compared to UFH. Patients in this trial underwent surveillance and evaluation for symptoms with a combination of ultrasound and confirmatory venography. Patients in the LMWH group had 40 total (31%) and 8 proximal DVTs (6.2%), still a significant number.

Four other trials have compared UFH to LMWH.[34–37] The three that were randomized found that there were fewer VTEs in the LMWH group, but only one found a statistically significant difference.[36] The average sample size for these trials was less than 50 patients. The fourth trial was a before-and-after comparison that was carried out after hospital protocol was switched from LMWH to UFH three times daily for trauma patients.[37] Among a total of 476 patients, no difference in VTE rates between the two regimens was found.

More recently, a small study of surgical intensive care unit patients, 85% of whom suffered from trauma, found that anti-Xa levels were subtherapeutic in 50% of patients receiving Lovenox 30 mg, subcutaneous, twice a day, for prophylaxis.[38] For the entire group, 26% had a VTE during their hospitalization, and those with subtherapeutic levels of anti-Xa were significantly more likely to experience a VTE. Given these findings, along with the high rates of VTE for patients on LMWH in the Geerts,[33] Holley,[4] and Stannard[30] studies, the search for a better chemical agent or method to prevent VTE in trauma patients continues.[18] In the meantime, Lovenox 30 mg, subcutaneous, twice a day, has the most data to support safety and efficacy and should remain the agent of choice for prophylaxis for the trauma patient.

Neuraxial Blockade

Given the high doses of analgesic medications required for pain control in the multitrauma OEF/OIF patients and the known side effects of narcotics, early placement of epidural and peripheral nerve catheters (collectively referred to as neuraxial blockade [NAB]) is being recommended.[39] For those patients who receive an epidural or deep peripheral block, there is a small but real risk of hematoma. Outcomes after epidural or spinal hematoma are largely dependent on the speed with which the resulting neurologic deficits are recognized and the blood is surgically evacuated.[40] For the intubated, sedated, and often paralyzed combat casualty being flown across multiple time zones, new deficits can easily be missed.

It is clear that the presence of anticoagulation increases the risk for hematoma, though the consequences are much worse for epidural and deep peripheral blocks (at noncompressible sites).[40] Based on case reports published in the literature and adverse drug reports from LMWH manufacturers, risk factors for spinal hematoma in the presence of LMWH prophylaxis have been identified. LMWH twice per day was associated with an increased risk, as was the use of nonsteroidal antiinflammatory drugs or other anticoagulants in addition to LMWH prophylaxis. Because LMWH twice daily is the recommended treatment dose for trauma prophylaxis and aspirin is often used when a traumatic vascular injury requires grafting, neuraxial blockade can limit treatment options in the trauma patient. Only one study has specifically looked at the effect that extended periods of NAB have on VTE rates for hospitalized trauma patients.[41] Though chemical prophylaxis was reduced in accordance with guidelines, there was no increase in VTE rates in the group receiving NAB.

Inferior Vena Cava Filters

The use of an IVC filter for VTE prophylaxis in the trauma patient is controversial. Both the ACCP and EAST guidelines acknowledge that there is no high quality evidence to support their use. A recent metaanalysis could not find any randomized trials to evaluate their use.[42] According to the EAST guide-

lines, observational studies that compare outcomes to historical controls support the use of IVC filters as prophylaxis for the high-risk trauma patient with contraindications to chemical prophlaxis. The ACCP did not feel there was sufficient evidence to make such a recommendation. Although removable filters are discussed by both, neither feels that they alter the risk–benefit ratio sufficiently to change their general conclusions about prophylactic filter placement at this time.

The metaanalysis[42] noted that of the observational studies assessed, only two recorded DVT as an outcome. Neither screened for DVT; they only recorded symptomatic events. Although a statistically significant difference was not found, the number of events was small, and one study did show more DVTs in the filter group. The two observational studies did find a significant decrease in PEs favoring the prophylactic filter group. In their conclusions though, the meta-analysis authors note that because most of the studies were more than a decade old, they did not follow current guidelines for chemical VTE prophylaxis. If they had, it is not clear what the effect of prophylactic filter placement would have been. Therefore, the authors concluded that they could not recommend for or against their placement.

SUMMARY

Acute trauma patients have a high rate of VTE if prophylaxis is not instituted. The average combat casualty who is critically injured is at particularly high risk, and poses unique challenges. Chemical prophylaxis should be started as soon as it is considered safe to do so. Until it is safe, mechanical prophylaxis should be used. A careful risk–benefit assessment must be done for each patient before NAB is started, but limited data in the combat-injured military population show that placement does not increase VTE rates. In the appropriate, high-risk patient with contraindications to chemical prophylaxis, an IVC filter could be considered.

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Chapter 35

INTENSIVE CARE UNIT SEDATION IN THE TRAUMA PATIENT

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INTRODUCTION

Critical illness can be a traumatic and anxiety-provoking experience. A multitude of factors can contribute to patients' anxiety in the intensive care unit (ICU), including the constant disruptions and stimulation from alarms, mechanical ventilation and the inability to speak, multiple healthcare providers, frequent vital-sign checks, continuous ambient light, inadequate analgesia, and the associated sleep deprivation, all of which can lead to anxiety and increased stress.[1] The stresses of ICU admission have been associated with a 4% to 15% rate of posttraumatic stress disorder among ICU survivors.[2,3] Combined with posttraumatic stress disorder among veterans returning from Iraq and Afghanistan, the rate of which has been estimated at 17%,[4] resultant morbidity may be significant. Appropriate use of sedative agents may decrease some of these stresses by providing anxiolysis and amnesia and improving tolerance to mechanical ventilation.[5] Additionally, sedation reduces the stress response and improves tolerance of routine procedures performed in the ICU.[6]

While the patient is sedated and undergoing transport, routine monitoring should consist, at a minimum, of continuous pulse oximetry and electrocardiography readings, and regular blood pressure and respiratory rate monitoring.[7–9] Additionally, depending on patient factors, monitoring with more invasive devices, including taking intraarterial blood pressure,[9] central venous pressure, pulmonary arterial pressure, intracranial pressure, and, potentially, capnography, may be beneficial.[10] An additional supply of sedatives should be available when sedated critically ill patients are being transported.[11]

SEDATION SCALES

A sedation scale is critically important because its use has demonstrated fewer instances of over sedation,[12] more precise sedative dosing, shorter duration of mechanical ventilation, and less use of vasopressor therapy.[13] Use of a validated sedation assessment scale was recommended in the 2002 Society of Critical Care Medicine (SCCM) clinical practice guidelines for the sustained use of sedatives in the critically ill adult.[7] Despite this recommendation, survey data show that sedation scales are used by only approximately 50% of intensivists[14] and in a similar percentage of consecutive patients receiving

TABLE 35-1

THE RICHMOND AGITATION-SEDATION SCALE

Score	Term	Description	
+4	Combative	Overtly combative, violent, immediate danger to staff	
+3	Very agitated	Pulls or removes tube(s) or catheter(s); aggressive	
+2	Agitated	Frequent nonpurposeful movement, fights ventilator	
+1	Restless	Anxious but movements not aggressive or vigorous	
0	Alert and calm		
−1	Drowsy	Not fully alert, but has sustained awakening (eye opening/eye contact) to voice (>10 seconds)	Verbal stimulation
−2	Light sedation	Briefly awakens with eye contact to voice (<10 seconds)	
−3	Moderate sedation	Movement or eye opening to voice (but no eye contact)	
−4	Deep sedation	No response to voice, but movement or eye opening to physical stimulation	Physical stimulation
−5	Unarousable	No response to voice or physical stimulation	

Procedure for RASS Assessment

1. Observe patient
   - Patient is alert, restless, or agitated. — Score 0 to +4
2. If not alert, state patient's name and say to open eyes and look at speaker.
   - Patient awakens with sustained eye opening and eye contact. — Score −1
   - Patient awakens with eye opening and eye contact, but not sustained. — Score −2
   - Patient has any movement in response to voice but no eye contact. — Score −3
3. When no response to verbal stimulation, physically stimulate patient by shaking shoulder and/or rubbing sternum.
   - Patient has any movement to physical stimulation. — Score −4
   - Patient has no response to any stimulation. — Score −5

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mechanical ventilation in another study.[15] No specific recommendation has been made by the SCCM favoring any of the myriad scales available. Important features of a sedation scale include rigorous multidisciplinary development; ease of administration, recall, and interpretation; well-defined discrete criteria for each level; assessment of agitation; demonstration of interrater reliability for relevant patient populations; and evidence demonstrating validity in the population in which it is to be used.[16] Several sedation scales, including the Richmond Agitation-Sedation Scale (RASS; Table 35-1),[17] Ramsay Sedation Scale,[18] Sedation Agitation Scale,[19] Motor Activity Assessment Scale (MAAS),[20] and Adaptation to the Intensive Care Environment (ATICE) instrument[21] offer many of these desired characteristics.

Most sedation assessments operate in a similar fashion. First, the patient's level of consciousness is observed to evaluate wakefullness. If the patient is not awake, verbal stimulation is usually the next step, followed by physical stimulation if the patient does not respond to the verbal cue. In the Ramsay Sedation Scale, RASS, and ATICE, a position on the scale is assigned given the patient's response to increasing levels of stimulation. Some scales, like the RASS and ATICE, also incorporate the patient's ability to follow commands. The opposite of sedation, agitation, is important to measure because it may compromise care, raise metabolic requirements, and increase morbidity and mortality.[15] The Sedation Agitation Scale, MAAS, RASS, and ATICE all identify and grade the patient's degree of agitation.[17,19–21]

The RASS has been validated, used in critically ill trauma patients, and shown to correlate well with other methods of monitoring sedation.[17,22] Its scoring system includes a positive number corresponding to restlessness or agitation and a negative number corresponding to sedation, with the higher absolute value correlating with a more extreme behavioral state. This system is intuitive and allows for easier evaluation and recall. The three-step assessment can be done quickly, usually in 30 to 60 seconds, and has favorable interrater reliability.[16] The author recommends the RASS as the first-choice sedation scale in clinical settings.

SEDATIVES

The ideal sedative should have a rapid onset of action and recovery after discontinuation, a predictable dose response, analgesic benefit, and a neutral effect on hemodynamics; it should not result in accumulation, respiratory depression, delirium, or associated toxicity.[5] Because no such sedative exists, providers must choose the most appropriate drug based on medication availability, pharmacokinetics, pharmacodynamics, the patient's comorbidities, and institutional protocols (Table 35-2).

Benzodiazepines

Benzodiazepines (diazepam, lorazepam, and midazolam) are the most commonly administered sedatives.[23] They potentiate the effects of γ-aminobutyric acid (GABA) and suppress the central nervous system,[24] resulting in hypnosis, anxiolysis, muscle relaxation, amnesia, and anticonvulsant activity.[25] Benzodiazepines lower the cerebral metabolic rate of oxygen consumption and decrease cerebral blood flow, but do so in a normal ratio. Midazolam has been shown to be safe and effective in sedating patients with head trauma.[26] As single agents, benzodiazepines do not possess analgesic properties; however, they are known to have an opioid-sparing effect related to modulation of the anticipatory pain response.[27]

Delirium is a side effect more commonly associated with the use of benzodiazepines compared to other sedatives,[19,28] which is important because delirium has also been associated with higher mortality, longer lengths of hospital stay (including time in the ICU), and longer duration of mechanical ventilation.[29,30]

Midazolam

When used as a bolus, midazolam is rapid and short-acting, with an onset of 2 to 5 minutes, making it ideal for rapidly sedating acutely agitated patients.[7] It is a water-soluble benzodiazepine with a half-life of 3 to 12 hours and a large volume of distribution.[7,31] Its primary site of metabolism is the liver, where it oxidizes (via the cytochrome P450 enzyme system) to several water-soluble metabolites that are then renally cleared.[32] The only pharmacologically significant metabolite of midazolam is α1-hydroxymidazolam, an active metabolite with 20% less potency than midazolam and a half-life of approximately 1 hour.[33] Like its parent compound, α1-hydroxymidazolam is a potent central nervous system depressant and can accumulate significantly. Critically ill patients are particularly susceptible to midazolam accumulation and its products of metabolism because of their increased volume of distribution, lower albumin, and more frequent impairment of renal and hepatic function.[7,34] Midazolam should be used for less than 72 hours to avoid accumulation and prolonged sedation; longer use can lead to unpredictable awakenings and increased time to extubation.[7]

TABLE 35–2

PHARMACOLOGY OF SELECTED SEDATIVES

Drug	Onset (min)	Half-life (hours)	Active Metabolites	Special Considerations	IV Dose (ID or LD)	Continuous Infusion Dose
Midazolam	2–5	3–12	+	Accumulates in renal failure	LD: 0.01–0.05 mg/kg q10 min	0.02–0.1 mg/kg/h
Lorazepam	5–20	10–20	–	Propylene glycol toxicity	ID: 0.01–0.1 q2–6h	0.01–0.1 mg/kg/h
Propofol	0.5–2	1.5–12	–	Elevated triglycerides, pain on injection, PRIS	10–30 mg titrated for rapid sedation	5–75 μg/kg/min
Dexmedetomidine	2–20	2	–	Bradycardia, hyper/hypotension, no respiratory depression	LD: 0–1 μg/kg over 10 min	0.2–1 μg/kg/h

ID: intermittent dose; IV: intravenous; LD: loading dose; PRIS: propofol infusion syndrome

Data sources: (1) Ostermann M, Keenan S, Seiferlin R, Sibbald W. Sedation in the intensive care unit: a systematic review. *JAMA* . 2000;283(11):1451–1459. (2) Jacobi J, Fraser GL, Coursin DB, et al. Clinical practice guidelines for the sustained use of sedatives and analgesics in the critically ill adult. *Crit Care Med*. 2002;30(1):119–141. (3) Shapiro BA, Warren J, Egol AB, et al. Practice parameters for intravenous analgesia and sedation for adult patients in the intensive care unit: an executive summary. *Crit Care Med*. 1995;23:1596–1600. (4) Gilliland HE, Prasad BK, Mirakhur RK, Fee JP. An investigation of the potential morphine sparing effect of midazolam. *Anaesthesia*. 1996;51:808–811. (5) Haefely W. The biological basis of benzodiazepine actions. *J Psychoactive Drugs*. 1983;15:19–39. (6) Arcangeli A, Antonelli M, Mignani V, Sandrone C. Sedation in PACU: the role of benzodiazepines. *Curr Drug Targets*. 2005;6:745–748. (7) Sanchez-Izquierdo-Riera JA, Caballero-Cubedo RE, Perez-Vela JL, Ambros-Checa A, Cantalapiedra-Santiago JA, Alted-Lopez E. Propofol versus midazolam: safety and efficacy for sedating the severe trauma patient. *Anesth Analg*. 1998;86:1219–1224. (8) Ghoneim MM, Mewaldt SP. Benzodiazepines and human memory: a review. *Anesthesiology*. 1990:172(5):926–938. (9) Pandharipande P, Pun B, Herr D, et al. Effect of sedation with dexmedetomidine vs lorazepam on acute brain dysfunction in mechanically ventilated patients: the MENDS Randomized Controlled Trial. *JAMA*. 2007;298(22):2644–2653. (10) Spina S, Ensom M. Clinical pharmacokinetic monitoring of midazolam in critically ill patients. *Pharmacotherapy.* 2007;27(3):389–398. (11) Mandema JW, Tuk B, van Steveninck AL, Breimer DD, Cohen AF, Danhof M. Pharmacokinetic-pharmacodynamic modeling of the central nervous system effects of midazolam and its main metabolite alpha-hydroxymidazolam in healthy volunteers. *Clin Pharmacol Ther*. 1992;51:715–728. (12) Devlin J, Roberts R. Pharmacology of commonly used analgesics and sedatives in the ICU: benzodiazepines, propofol, and opioids. *Crit Care Clin*. 2009;25:431–449. (13) Arroliga AC, Shehab N, McCarthy K, Gonzales JP. Relationship of continuous infusion lorazepam to serum propylene glycol concentration in critically ill adults. *Crit Care Med*. 2004;32(8):1709–1714. (14) Ativan injection (lorazepam) [package insert]. Lake Forest, IL: Hospira Inc; 2012. (15) Newman L, McDonald J, Wallace P, Ledingham I. Propofol infusion for sedation in intensive care. *Anaesthesia*. 1987;42:929–937. (16) Dyck J, Ikeda K, Morita K, et al. The pharmacokinetics of propofol vs. age. *Anesthesiology*. 1991;75:A315. (17) Propofol [package insert]. Wilmington, DE: Astra Zenica; 2005. (18) Gerlach AT, Dasta JF. Dexmedetomidine: an updated review. *Ann Pharmacother*. 2007;41:245–254. (19) Gerlach AT, Dasta JF, Steinberg S, Martin LC, Cook CH. A new dosing protocol reduces dexmedetomidine-associated hypotension in critically ill surgical patients. *J Crit Care.* 2009;24:568–574. (20) Videira R, Ferreira R. Dexmedetomidine and asystole. *Anesthesiology*. 2004;101:1479. (21) Precedex [package insert]. Lake Forest, IL: Hospira Inc; 2011.

Erythromycin, itraconazole, diltiazem, and other drugs that are known to interfere with the cytochrome P3A4 can lead to a prolonged effect as well by interrupting midazolam metabolism.

Lorazepam

Lorazepam has a slower onset of action (5–20 minutes) than midazolam, making it less useful for sedating an acutely agitated patient. Additionally, its longer half-life (10–20 hours[7,32,34]) makes infusions less titratable. Therefore, lorazepam is often used as a sedative in intermittent boluses rather than a continuous infusion. If it is infused, intermittent boluses and a relatively constant infusion rate are recommended.[7]

Propylene glycol (PG) is used as a diluent to increase the solubility of lorazepam and diazepam. Either of the benzodiazepines can lead to PG toxicity, but toxicity has been reported most commonly in the ICU related to high-dose lorazepam infusions.[35] The presenting symptom of PG toxicity is usually a hyperosmolar gap metabolic acidosis. Toxicity can then progress to acute tubular necrosis and renal failure, lactic acidosis, intravascular hemolysis, cardiac arrhythmias, seizures, and central nervous system depression.[7,34,35] Toxic doses of PG can occur quickly when lorazepam is used for continuous sedation and in large doses approaching the maximum recommended dosage of 0.1 mg/kg/h.[7] The daily maximum dose of PG considered to be safe is 25 mg/kg. With each 2-mg vial of lorazepam (2 mg/mL) containing 664 mg of PG per milliliter of lorazepam, a heavily sedated, critically ill patient can easily receive more than the recommended daily amount, resulting in toxicity.[36] Patients with hepatic or renal insufficiency

are at an increased risk of PG accumulation because the liver metabolizes 55% and the remainder is excreted, unchanged, in urine.[35]

Unlike the other benzodiazepines commonly used in the ICU, lorazepam undergoes glucuronidation in the liver to inactive metabolites that are renally cleared. Both midazolam and diazepam have active metabolites that can accumulate in a critically ill patient with renal impairment. In patients with liver failure, metabolism of midazolam and, to a lesser degree, lorazepam are affected.[7]

Propofol

Propofol is a hydrophobic intravenous anesthetic in an emulsion of egg phospholipid and glycerol that has been used as a sedative in the ICU since the 1980s.[37,38] The mechanism of action occurs at several receptors, but its main mechanism of action is similar to that of benzodiazepines because it potentiates GABA activity. However, propofol has also been implicated in sodium channel blockade and the endocannabinoid system, making its mechanism of action quite unique.[39–41] The GABA activity is likely the most clinically significant compared to sodium channel blockade and endocannabinoid effects.

Propofol has a rapid onset, reaching peak effect in 90 to 100 seconds.[41] It has a short duration of action and is the recommended sedative when rapid awakening is desired.[7] This was demonstrated in a trial by Kress et al in which sedation was interrupted on a daily basis to allow patients to wake up. The patients receiving propofol showed no significant difference between the intervention and the control groups in the total dose of the drug, owing to its rapid offset.[42] Propofol's metabolism primarily occurs in the liver, where it is conjugated to inactive metabolites that are then renally cleared. In patients with renal or hepatic disease, propofol clearance is not significantly affected; however, in critically ill patients, propofol clearance is delayed compared to the general population.[43,44]

With propofol's conjugation to inactive metabolites, its use in patients with renal failure is not as concerning as with other sedatives that produce active metabolites; however, there are certain propofol side effects providers should be aware of. The most common side effect is a dose-related hypotension from a vasodilatory response.[45] This can be profound in patients who are hypovolemic, including those with trauma or sepsis. Unlike other sedatives, the lipid-based emulsion can support rapid bacterial growth, and multiple cases of bacterial sepsis related to propofol contamination have been reported[31]; therefore, strict aseptic technique should be employed when using propofol.[46] Depending on the manufacturers' formulation, a preservative is added to prevent bacterial growth, with ethylenediaminetetraacetic acid or sodium metabisulfite being the most common. In the ICU, once the propofol vial has been spiked, the infusion should be commenced and completed in 12 hours. At that time, any unused propofol and all tubing should be discarded.[45]

Because propofol is an emulsion, there are certain factors to remember when using it as a sedative. Propofol's formulation accounts for 1.1 kcal/mL and should be considered a source of calories from fat.[45] Hypertriglyceridemia is of concern with propofol infusions and occurs in up to 18% of those receiving it for continuous sedation. Hypertriglyceridemia is associated with high-dose infusions of propofol, hypertriglyceridemia at baseline, and parenteral nutrition lipid administration.[47] After 48 hours, triglycerides levels should be monitored.[7] Propofol has also been linked to pancreatitis. In a study of 159 patients sedated with a propofol infusion, Devlin and colleagues found that of the 18% of the patients that developed hypertriglyceridemia, 10% of those also developed pancreatitis.[47] Propofol's lipid nature also exhibits immunosuppressant effects by depressing neutrophil function.[48] However, the clinical significance of this is undetermined.

One rare complication of propofol use is a constellation of metabolic derangements and organ system failures that is referred to as propofol infusion syndrome (PRIS). It was first described in 1992 in a case series of five pediatric patients sedated in the ICU who died after developing increasing metabolic acidosis associated with bradyarrhythmias and progressive myocardial failure. The patients had been receiving high-dose propofol infusions at greater than 83 μg/kg/min for more than 48 hours.[49] PRIS is rare and has an unknown incidence. It is now known to occur more commonly in children, but can also occur in adults. Risk factors for PRIS are airway infection, severe head injury, propofol infusion (> 48 hours at a dose > 5 mg/kg/h), increased catecholamine and glucocorticoid serum levels, and low carbohydrate stores.[50] Its most prominent clinical characteristics, based on reviews of cases, are metabolic acidosis, cardiac dysfunction, hyperkalemia, hyperlipidemia, elevated creatinine kinase, rhabdomyolysis, myoglobinemia, myoglobinuria, and acute renal failure.[51,52] PRIS carries a very high mortality rate, estimated to be 30% in a retrospective review of the FDA's MedWatch database of 1,139 patients who were suspected to have PRIS.[53] When there is prolonged need for sedation and propofol doses must be increased to maintain constant sedation, or if metabolic acidosis sets in during a propofol infusion, consider using an

alternative means of sedation and do not rule out a PRIS diagnosis if the clinical situation dictates.

PRIS treatment mainly involves supportive care. First and foremost, the propofol infusion must be stopped immediately. Hemodynamics should be supported. PRIS-associated bradycardia is often resistant to catecholamines and external pacing. Hemodialysis or hemofiltration is recommended to eliminate propofol and its potentially toxic metabolites.[54] Extracorporeal membrane oxygenation has assisted in the survival of several patients with PRIS[55–57] and may serve as a last-resort therapy.

Dexmedetomidine

Unlike the benzodiazepines and propofol that act on the GABA receptor, dexmedetomidine is a nonselective $\alpha 2$ agonist. It has 7- to 8-fold higher affinity than clonidine for the $\alpha 2$ adrenergic receptor and an $\alpha 1$ to $\alpha 2$ selectivity ratio of 1600:1.[57–59] The use of dexmedetomidine in the ICU has increased significantly. In 2001, 2% of patients received sedation via intravenous infusion of dexmedetomidine. This proportion increased to 7.2% by 2007.[60] Dexmedetomidine provides sedation and anxiolysis by interacting with receptors in the locus ceruleus, and analgesia through receptors in the locus ceruleus and spinal cord.[61] Two significant benefits of dexmedetomidine are its lack of respiratory depressant effect[61,62] and the ability to wake patients and have them follow commands while intubated and sedated.[61,63] In a phase III study, the most common adverse reactions associated with dexmedetomidine were hypotension (30%), hypertension (12%), nausea (11%), bradycardia (9%), and dry mouth (3%).[57] The most clinically significant side effects of hypotension and bradycardia are related to sympatholysis and more frequently occur during administration of the loading dose.[64–66] The sympatholytic effect of dexmedetomidine can be significant, progressing from bradycardia to asystole.[67,68] In patients with preexisting hypovolemia, it has the potential to cause pronounced hypotension.[69] Hypertension is usually seen with high or loading doses and is caused by peripheral vasoconstriction.[69,70]

For sedation in the ICU, the recommended dosage of dexmedetomidine is a 1 μg/kg loading dose over 10 minutes, followed by an infusion of 0.2 to 0.7 μg/kg/h. It is approved for sedation in the ICU for less than 24 hours.[69] In clinical practice and trials, dexmedetomidine is routinely started with or without a loading dose, infused as high as 1.5 μg/kg/h, and continued for up to several days.[60,62,71,72] Despite a longer duration to a goal level of sedation, many clinicians often forego the loading dose to avoid the potential hemodynamic abnormalities of hypotension, hypertension, or bradycardia.[62,73] In 2009, Gerlach proposed a loading-dose-free protocol in which the infusion dose was based on the RASS score and titrations were made no more frequently than every 30 minutes. The protocol was effective and decreased the rate of hypotensive episodes from 68% to 16% compared to historical controls.[65] Several clinical trials[28,71,74] used maximum dosages in the range of 1.4 to 1.5 μg/kg/h. In these studies, dexmedetomidine was compared to propofol or benzodiazepines and was found to be safe and effective based on the studies' individual criteria. Where dexmedetomidine was found to be less effective was in a small subset of patients in whom the goal was to achieve a deep plane of sedation; there it did not perform as well as propofol or benzodiazepines.[74]

Clonidine is well known for its withdrawal syndrome, which is characterized by rebound hypertension, irrespective of the route of administration.[75] Because of dexmedetomidine's similar mechanism of action, it was originally approved only for short term (< 24 hours) sedation out of concern for similar withdrawal effects. Since its original approval, multiple trials have shown dexmedetomidine to be safe for sedation lasting greater than 24 hours (median duration of therapy ranged from 40 hours[74] to 5 days[28]).[71,76] In the trial by Shehabi et al,[76] 20 adult patients in a combined ICU received dexmedetomidine for a median time of 71 hours (range of 35 to 168 hours). Initially there was a 16% reduction in mean systolic blood pressure and 21% reduction in heart rate, which occurred over the first 4 hours, followed by insignificant changes thereafter. Following abrupt cessation, systolic blood pressure and heart rate were monitored for 24 hours and found to rise by 7% and 11%, respectively. This and other trials have not shown any evidence of a withdrawal syndrome associated with dexmedetomidine.[73,74,76]

Most studies have shown dexmedetomidine to be less commonly associated with delirium than benzodiazapines.[28,71,77] These studies all found significantly decreased rates of delirium or more delirium-free days when patients were sedated with dexmedetomidine compared to benzodiazepines or propofol. However, Ruokonen et al[74] found the contrary. They found a higher rate of delirium in the dexmedetomidine group (43.9%) compared to the propofol group (25%; $P = 0.035$). The authors reported that the dexmedetomidine group had more delirium assessments performed (106 vs 84) because of the interactisve nature of the dexmedetomidine sedation, but the overall rate of positive assessments were the same (17% vs 17.9%; $P > 0.05$).

HEAD INJURY

The mainstay of treating head-injured patients revolves around preventing and treating elevated intracranial pressure (ICP). When caring for these patients, sedation is frequently necessary to control ventilation, treat shivering, and prevent agitation, which can all contribute to transient elevations in ICP. Multiple sedatives can be employed in this population. Sanchez-Izquierdo-Riera et al[26] demonstrated the safety and efficacy of propofol and midazolam in severe trauma patients. Approximately 58% of the patients in their study sustained head trauma. They concluded that propofol and midazolam were both safe and noted no differences in ICP, cerebral perfusion pressure (CPP), or jugular venous saturation. The only difference noted was the time to wakefulness, which was significantly shorter in the propofol group. This is consistent with the SCCM recommendation that propofol be the drug of choice when rapid awakening is desired.[7] In addition to propofol's short duration of action, it has positive neurologic effects, including reducing ICP after traumatic brain injury and decreasing cerebral blood flow and metabolism.[78,79]

Kelly et al[78] compared a regimen of morphine alone to propofol with morphine to evaluate the propofol's safety. However, they also evaluated clinically relevant factors such as CPP, ICP, treatment-related adverse events, and neurologic outcome at 6 months. Despite the propofol arm having a higher incidence of poor prognostic indicators, including lower initial Glascow coma scale scores, older average age, and a higher rate of cistern compression on computed tomography scanning, the mean daily ICP and CPP were similar between the two groups, with the propofol arm having a lower ICP on day 3 of the infusion. At 6 months after injury, the propofol arm had more favorable neurologic outcomes (52.1% vs 47.4%) and a lower mortality rate (17.4% vs 21.1%). In a post hoc analysis, the authors compared the outcomes of high-dose propofol (> 100 μg/kg/min for > 48 hours) to low-dose propofol and found that, despite there being no difference in ICP or CPP between the two groups, there was a significant difference in the neurologic outcomes. At 6 months after injury, the high-dose group had 70% favorable outcomes (defined as a good neurologic recovery or moderate disability) compared to 38.5% in the low-dose group ($P < 0.05$). However, because of the risk of PRIS, high-dose propofol regimens are not recommended.[80]

Similarly, Chiu and colleagues[81] examined 104 head-injured patients who were either in a propofol or nonpropofol arm. They found that the mean ICP for the first 3 days was 17 mm Hg in the propofol group and 33 mm Hg in the nonpropofol group ($P = 0.17$). Over the first 5 days in the ICU, the mean CPP provided similar results as the ICP. The CPP was 71 mm Hg in the propofol arm and 43 mm Hg in the nonpropofol group ($P < 0.001$). The rate of survival was higher in the propofol arm (81.8% vs 46.7%, $P < 0.001$). These findings, in addition to other studies, contributed to joint guidelines published by the Brain Trauma Foundation and the American Association of Neurologic Surgeons for managing severe traumatic brain injury, which recommend propofol as the sedative of choice when managing ICP, but not in an attempt to improve mortality or 6 month outcome.[80]

DAILY INTERRUPTION OF SEDATION

Kress and colleagues showed that daily interruption of sedative infusions was associated with positive outcomes.[42] Patients treated with infusions of propofol with morphine or midazolam with morphine whose infusions were interrupted at the discretion of clinicians in the ICU were compared to those whose infusions were interrupted on a daily basis until the patient was able to answer three or more of four simple commands. The latter group demonstrated a significant reduction in the duration of mechanical ventilation (4.9 vs 7.3 days, $P = 0.004$), ICU length of stay (6.4 vs 9.9 days, $P = 0.02$), and number of diagnostic tests to assess mental status changes (9% vs 27%, $P = 0.02$), and no difference in self-extubations or other complications (4% vs 7%, $P = 0.88$). There was no difference between the propofol and midazolam groups, except for a lower total dose of midazolam and morphine. As a result of fewer ventilator days and a shorter ICU length of stay, daily sedation interruption has been linked to a lower rate of ICU complications related to a shorter length of stay.[82]

Daily interruption of sedation was also employed by Carson and colleagues.[83] In this study, sedation regimens of either a propofol infusion with morphine or intermittent lorazepam boluses with morphine were both interrupted on a daily basis. The propofol group showed a significantly shorter duration of mechanical ventilation (5.8 vs 8.4 days, $P = 0.04$). Overall, daily interruption of sedation has shown great benefit with little harm[42] and has been incorporated into the practice of many intensivists.[84] Despite daily interruptions of sedation being associated with increased levels of catecholamines, patients with coronary artery disease showed no evidence of increased ischemia during the

interruptions of sedation.[85] However, caution should be taken when interrupting sedation in certain patient populations, such as those with unstable cervical spine injuries in whom patient ventilator asynchrony could lead to coughing and potential exacerbation of neurologic injury.

RESTRAINTS

More than 70% of ICU patients may experience some degree of agitation during their ICU stays[86,87] that often coincides with mental status changes. As a result, patients may be unable to understand why certain therapies are ongoing, leading to patient-initiated treatment interference that can be self-injurious. The literature contains multiple reports of fatal self-extubations and removal of intravascular devices,[88,89] making the possibility of patient interference even more concerning. Before resorting to restraint use, clinicians should evaluate whether treatment of a physiologic perturbation (eg, hypoxia, hypercarbia, sepsis, or hypotension) would obviate the need for restraints.[88] After deciding restraints are necessary, the choice between employing pharmacologic or physical restraints must be made. Simple measures such as assuring patients are adequately sedated, as discussed above, can obviate the need for physically restraining a patient. When physical restraints are chosen, they should be the least invasive possible (hand mitts vs restraining all extremities to the bedframe), the need for the restraint selected should be continually evaluated every 8 hours, and complications from the restraint should be checked for every 4 hours. The restraints should be discontinued as soon as they are deemed unnecessary.[88]

SUMMARY

The increasing understanding of sedatives and their ramifications over the last 2 decades has made the sedation of critically ill patients more complex. We now have an improved understanding of delirium and which sedatives may increase its already high rate of occurrence in the trauma population. The importance of preventing delirium is also better understood as it has been shown to increase morbidity, mortality, and length of stay in the ICU, as well as worsening outcomes overall. Appropriate sedatives should be chosen with a thorough understanding of their side effect profile, and preparations must be made to deal with the possible consequences. A sedation scale should be employed to prevent the sequelae of oversedation. With the already high rates of posttraumatic stress disorder in the war wounded, maintaining the proper depth of sedation is vitally important to prevent additional posttraumatic stress related to ICU care.

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Chapter 36

NUTRITIONAL SUPPORT IN THE INTENSIVE CARE UNIT

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INTRODUCTION

Nutritional support in military critical care patients has a relatively limited evidence base from which to build recommendations. Most of the evidence for the caloric goals, timing, and route of feeding comes from the civilian setting.[1–3] The evidence informing these civilian guidelines is of variable quality and should be interpreted with caution. Some apparently sensible expert recommendations, such as early institution of parenteral nutrition (PN), have been shown to be harmful when formally tested.[4] Key points from the literature have been extracted, tempered by clinical experience caring for both military and civilian patients in deployed military hospitals and civilian teaching hospitals during the recent conflicts.

The majority of military patients in the current conflicts have been admitted to the critical care unit with complex polytrauma resulting from blast or ballistic injury. Figure 36-1 shows a complex ballistic trauma patient transferred to a Role 3 hospital following damage control surgery at a forward surgical Role 2 facility. Military patients tend to be young and active, with a greater proportion of their body mass made up of muscle than in the general population. They can therefore be expected to have a correspondingly higher basal metabolic rate. Additional complexity is introduced by the limited resources in the austere military environment, the absence of PN products in most deployed settings, the high frequency of damage control surgery, and the short time before evacuation to advanced medical facilities in the patient's home country. General recommendations are further complicated by the diversity of host nation casualties, including soldiers, civilians, and children.

NUTRITIONAL REQUIREMENTS

The nutritional requirements for intensive care patients include various components: carbohydrate, protein, fat, trace elements, and vitamins. Patients who are fed longer than a short period of time may also require fiber supplementation to prevent constipation. Exactly how much of each component a patient needs may be estimated using various formulae, none of which are proven to affect outcome in polytrauma patients. Alternatively, the energy expenditure of the patient may be measured by indirect calorimetry, which requires specialized equipment, and feeding prescribed based on this measurement. There are also simple formulae based on estimated ideal body weight, which may prove more practical in austere settings. A pragmatic approach is to recommend 20 to 30 kcal of energy per kilogram of body weight and 1.2 to 2.0 g of protein per day,[5] with an increase in the amount of protein and calories for patients with more severe trauma or burns, discussed further below.

INITIATION OF NUTRITIONAL SUPPORT

Military trauma patients rapidly lose muscle bulk and weight, almost as soon as they are admitted to the intensive care unit. Some of the muscle loss may be due to inactivity; data from aerospace research demonstrate that inactivity leads to marked reduction in muscle protein synthesis.[6] However, most of the muscle and weight loss is due to the catabolic state experienced by critically ill patients, during which they cannot efficiently metabolize fat stores, and instead break down muscle proteins.[7] Infantry soldiers and combat support troops make up the majority of casualties and are typically lean, with little body fat reserve. Feeding is believed to reduce the extent of muscle catabolism, but we do not know exactly how many calories should be delivered at various stages in the patient's journey from the battlefield to hospital discharge, and we do not yet have an effective strategy to prevent patients from losing muscle bulk during their stay on the critical care unit. Delays in initiating feeding in the field hospital have been demonstrated by a prospective evaluation of American casualties evacuated from a combat support hospital in Iraq.[8]

Two groups have carried out extensive reviews of the literature relating to feeding in critically ill patients. Guidelines from the European Society for Parenteral and Enteral Nutrition (ESPEN) recommend enteral feeding for critically ill patients unless they are likely to be eating and drinking normally within 3 days.[3] The Canadian clinical practice guidelines (CCPGs) for nutrition support in mechanically ventilated, critically ill adult patients recommend early enteral feeding within 24 to 48 hours of admission.[2] However, the majority of patients in the trials that informed these guidelines were medical or surgical rather than trauma patients.

An international multi-center study by Cahill of almost 3,000 patients showed that only 60% of criti-

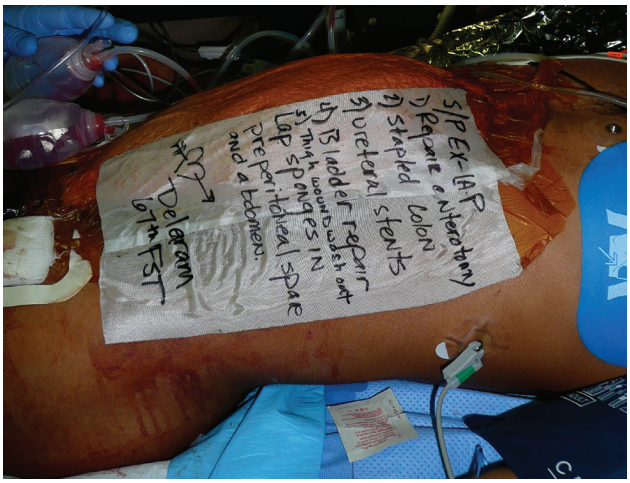

Figure 36-1. Complex ballistic trauma patient on arrival in a Role 3 hospital following damage control surgery at a forward surgical Role 2 facility. The notes on his abdominal dressing list the numerous procedures he has had as an additional safeguard during a period of high patient throughput.

cally ill patients meet their caloric and protein targets, as defined by the CCPG, and that the average time to initiating nutrition was 46 hours.[9] Heyland demonstrated, in 638 patients in 59 intensive care units across Canada, that using the CCPG as a guide makes it more likely that patients will meet their caloric requirements; however, a significant proportion of patients do not meet their target, and the average time to feeding initiation was 1.6 days.[10]

Does meeting these nutritional targets affect outcome? We do not know for sure, but a study of 243 critically ill patients, which measured actual energy expenditure using indirect calorimetry, found a marked reduction in the likelihood of death in female patients in whom energy expenditure was matched with calorie and protein delivery.[11] It is not clear why male patients did not benefit, and fewer than 10% of the patients had suffered traumatic injuries, so these findings may not be directly applicable to military trauma populations. Other authors have also used indirect calorimetry to guide therapy, with similar outcomes: Scheinkestel, in a study of 50 critically ill patients, showed that the delivery of appropriate amounts of calories and protein resulted in mortality lower than predicted by the severity of illness.[12] Again, the findings may not be directly applicable to military trauma patients because the study patients were older adults with a mean age of 53.

In summary, there is reasonable evidence (albeit not relating to trauma patients in particular) and strong consensus that enteral feeding should be started as soon as the patient is stable in the critical care unit. Although some guidelines[2] recommend waiting up to 3 days to initiate enteral nutrition, the authors begin feeding as soon as possible and preferably within the first few hours after injury. Delivery of adequate calories and protein is important because it may improve outcome.

ENTERAL AND PARENTERAL ROUTES

Parenteral feeding is more difficult to achieve than enteral, requiring a dedicated central venous access port, and it is associated with more infective complications in most studies, although no increases in mortality have been seen. Historical studies of PN demonstrated high morbidity rates, predominantly due to infection, possibly from infection of the line used to deliver the nutrient-rich solutions, or due to immunosuppression associated with parenteral feeding. Both the CCPG and the ESPEN guidelines therefore recommend using the enteral route to feed all patients unless there are compelling reasons not to do so,[2,3] such as discontinuity of the gastrointestinal tract. These guidelines also recommend considering a combination of both enteral and parenteral feeding if insufficient feed is delivered by the enteral route, as do other authors.[2,3,13] These recommendations are based on limited evidence, however.

There is little published data to support very gradual upward titration of the rate of enteral nutrition. In the authors' institutions, the practice is to start feed at half of the target rate, then increase feeding to the target rate within 4 hours if tolerated. Starting feed at the target rate is also a reasonable option.

Concerns about gut ischemia are cited as reasons not to feed patients who require significant doses of vasopressor drugs to maintain their blood pressure. There are anecdotal cases of such ischemia occurring; however, the authors have not seen any cases, and this rare occurrence seems unlikely in a previously fit military population. We would generally feed all patients unless they are on very large doses of vasopressors.

Researchers have suspected that the high morbidity attributed to PN in historical studies may prove to be lower in modern populations due to interventions that reduce line infection.[14] Morbidity would be lower if the infection were due to mechanical factors associated with the line rather than hyperglycemia or poorly understood immunosuppression related to receiving intravenous glucose and lipids. The suggestion that harmful effects of PN are related to outdated practices (and of historical relevance only) is refuted by recent

strong evidence that exclusive PN causes increased morbidity when delivered within the first 48 hours of admission to intensive care. Delaying PN until day 8 after admission appears to be a safer strategy, leading to a shorter length of stay and less infection.[4] In summary, parenteral feeding should be used only when the enteral route is not possible after a week of critical illness, or clearly impossible for anatomical reasons. Figure 36-2 provides a pragmatic and simplified approach to determining which feeding route to use.

SPECIFIC CONSIDERATIONS

Types of Enteral Feed Preparations

Enteral feed preparations available on the market include a large variety of proprietary formulations with a caloric content ranging from 1 to 2 kcal/mL. Higher calorie feeds are indicated for patients who have higher energy requirements or electrolyte abnormalities, or in the chronic setting when the patient may be unable to tolerate a large volume of feed. There are also preparations containing varying amounts of insoluble and soluble fiber, ranging from low-residue feeds with almost no fiber, to high-fiber feeds aimed at preventing constipation. More specialized feeds are available for patients with chronic renal failure (high protein content), Crohn's disease (elemental feeds), and various inborn errors of metabolism (metabolically appropriate content), but these are outside the focus of this chapter. Feeds containing shorter peptides rather than the whole protein molecules found in standard feeds have no evidence of benefit.[3]

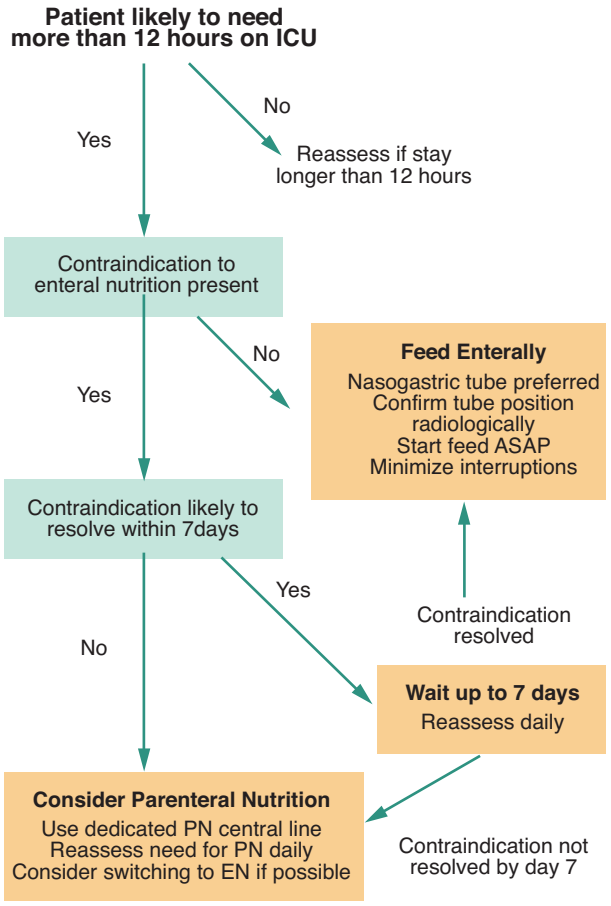

Figure 36-2. Flowchart for deciding between enteral and parenteral feeding.
ASAP: as soon as possible
EN: enteral nutrition
ICU: intensive care unit
PN: parenteral nutrition

The authors aim to feed military polytrauma patients 35 kcal per kilogram of ideal body weight for the first 48 hours after injury. In practice, for a 70-kg patient this rate will approximate to 100 mL/h of feed containing 1 kcal/mL in the first 24 to 48 hours. Feed is increased in the following days if tolerated. Caloric targets are based on extrapolation from civilian patients. It should be noted that in civilian patients high calorie delivery has been associated with poor outcome, albeit with a low level of certainty that the high caloric load was responsible.[3] We have not seen evidence of adverse outcomes in military patients. The ongoing United Kingdom Surgeon General's Casualty Nutrition Study may provide additional information on the caloric requirements of military casualties.[5]

Most deployed military critical care units stock only one type of feed, containing 1 kcal/mL. Logistic considerations argue against the provision of multiple preparations, and therefore this feed is used for all patients who require enteral feeding, including children. In summary, enteral feed should be delivered with the aim of supplying sufficient calories and protein based on ideal body weight and adjusted for severity of injury.

Immunonutrition

Immunonutrition refers to supplementing enteral or parenteral feed with various compounds that have theoretical or proven benefits in critical illness. Supplements that have been subjected to trial in critical care patients include glutamine, fish oils, and trace elements.

Glutamine is made in large amounts in healthy individuals, but in critical illness glutamine levels

drop, due either to reduced production or increased consumption.[15] Trials comparing enteral glutamine supplementation with standard feed have been carried out in trauma patients by two groups. Houdijk studied 72 polytrauma patients and demonstrated a reduction in pneumonia and sepsis in the intervention group, with no effect on mortality.[16] Brantley carried out a study (published in abstract form only) also in 72 trauma patients that demonstrated a reduction in length of hospital stay of just over 1 day for the intervention group and no reduction in mortality. Infection rates were not reported.[17] Novak et al[15] carried out an extensive review of trials of glutamine in critically ill patients, the majority of which did not allow firm conclusions, but some evidence of benefit for high-dose glutamine in surgical and critically ill patients was found, with the possibility that glutamine may reduce length of stay and infective complications in the former group and possibly reduce complications and mortality in the latter group. The ESPEN and CCPG guidelines recommend the use of feed containing glutamine in patients with burns and trauma.[2,3]

The purported benefits of fish oils and various other polyunsaturated fatty acids include reduction of inflammation by altering the balance between proinflammatory and antiinflammatory parts of the eicosanoid pathway.[18] A well-conducted trial of fish oil supplementation in 173 critically ill patients showed no effect on inflammatory mediators or outcome[19]; however, analysis of the literature for the ESPEN PN guideline suggests these supplements do have some effect on length of stay in the intensive care unit.[20] In subgroups of intensive care patients with acute respiratory distress syndrome (ARDS), enteral feed containing fish oils has been shown to reduce length of ventilation and length of intensive care stay.[21] The ESPEN and CCPG guidelines differ on fish oils, with ESPEN recommending fish oil supplementation in patients who have had gastrointestinal surgery, mild sepsis, trauma, or ARDS, but not severe sepsis.[3] The CCPG guideline reserves their use for patients with ARDS.[2]

Trace elements are thought to improve healing, especially in burn patients and those with gastrointestinal disease. Enteral and parenteral feeds typically contain a range of trace elements and vitamins. There is no strong evidence that additional trace elements are beneficial, except in burn patients.[3,20] Recently some manufacturers have started adding fish oil or omega-3 vegetable oils to their standard feeds.

In summary, glutamine supplementation appears to be helpful in trauma patients. Immunonutrition with feeds containing fish oil, while showing some promise, should be reserved for subgroups of patients with ARDS, and trace element supplementation may benefit burned patients.

Enteral Feeding After Abdominal Surgery

A period of fasting, followed by the gradual reintroduction of fluids and then solids, was for many years an integral and unchallenged part of postoperative care.[22] This approach was felt to allow resolution of the "inevitable" postoperative ileus, reduce the risk of vomiting and aspiration, and prevent anastomotic complications. There is no evidence to support this practice. Several well conducted, civilian randomized trials of early enteral nutrition,[23–32] which have been eloquently summarized in two recent systematic reviews,[30,33] do not show an increase in anastomotic leak rates. However, the evidence is stronger for lower gastrointestinal anastomoses[34] than for upper gastrointestinal anastomoses. Only four of the trials included patients who had undergone upper gastrointestinal or hepatobiliary surgery,[23,25,31,32] and even the total number of such patients was too small to allow for a meaningful metaanalysis. A recent nonsystematic review on early oral nutrition after elective upper gastrointestinal surgery, however, concluded that, despite little direct evidence, early feeding will most likely prove to be equally safe after gastric, and possibly also esophageal surgery, as it is after other types of gastrointestinal surgery.[35]

Only one study, also conducted in the civilian setting, specifically investigated the effect of early enteral nutrition in abdominal trauma patients. This study showed a reduction in septic complications, but was too heterogeneous and underpowered to detect differences in anastomotic complications.[36,37]

In summary, although there is little direct evidence from the trauma or military setting, it seems reasonable to extrapolate the findings of civilian primary studies and systematic reviews to a recommendation that gastrointestinal anastomoses and repairs, including those in the upper gastrointestinal tract, are not a contraindication to early enteral feeding.[1]

Postpyloric Feeding

Intragastric feeding using nasogastric tubes is associated with complications such as gastroesophageal reflux that may result in aspiration and delayed gastric emptying, which can result in failure to attain caloric goals. Postpyloric feeding is conceptually attractive, but no good evidence shows it to be advantageous. Further work is required to demonstrate the benefits and relative advantages of the nasoduodenal and nasojejunal routes, and new devices are needed to

ensure that catheters can be placed quickly, accurately, and consistently to prevent delays in the initiation of feeding.[38] Infrequently, intraoperative placement, at the time of the index laparotomy—although not always straightforward because of technical difficulties—may have a role. The route chosen is dependent on the clinical setting, but intragastric delivery is usually more physiological and convenient than postpyloric feeding, and thus the preferred route for the initiation of nutritional support.[1]

Surgical Access to the Gastrointestinal Tract

Direct access to the gastrointestinal tract, through a gastrostomy or jejunostomy, is used in two distinct populations of trauma patients: (1) those who are expected to require nutritional support in the short term only, whose caloric requirements may not be met with nasogastric or postpyloric feeding, and (2) those who will require long-term nutritional support, such as patients with traumatic brain injuries.[1] Gastrostomy placement for long-term nutritional management is outside the scope of this book and will therefore not be discussed further.

The volume of evidence for the short-term use of feeding jejunostomies is small and the quality of studies poor. Furthermore, no reports are specifically from the military setting. Early studies by Dunn and Moore describe small series of civilian patients with a combination of blunt and penetrating abdominal injuries who underwent jejunostomy formation at the time of their initial operation.[39,40] A subsequent randomized study by the same group compared early enteral nutrition using a jejunostomy with controls who received no supplemental nutrition for 5 days and revealed a decrease in septic complications in the jejunostomy group.[41] However, given that most patients in the control group received no nutrition at all (some were given PN), the study actually identifies the benefits of enteral nutrition, rather than jejunostomy. Holmes et al[42] retrospectively analyzed 222 trauma patients who underwent early feeding jejunostomy insertion, and reported a major complication rate of 4%. Eddy et al[43] also found a significant complication rate relating to the use of jejunostomies in trauma patients.

In summary, there is little evidence to recommend the formation of feeding jejunostomies for short-term nutritional support as part of the initial surgical management of patients with abdominal injuries. Nasogastric, or indeed nasoduodenal or nasojejunal access, is more convenient and probably associated with fewer complications. Many of the original reports of feeding jejunostomy use were published in the 1980s, and it is possible that feeding jejunostomy use has declined with the increasing utilization of damage control surgery, since jejunostomies tend to interfere with temporary abdominal closure.

Enteral Nutrition After Temporary Abdominal Closure

The damage control approach is now widely accepted as the standard of care for trauma patients with abdominal injury and severe physiological derangement, and has been utilized extensively in the recent conflicts in Afghanistan and Iraq.[44,45] Damage control laparotomy often entails temporary abdominal closure, to expedite transfer to the intensive care unit, facilitate repeat laparotomy, and prevent abdominal compartment syndrome. Although the "open abdomen" is a testament to the success of modern trauma surgery, it has also been accompanied by new challenges, including nutritional management. Patients who require damage control surgery often have multiple significant injuries, and therefore accentuated metabolic responses. Conventionally, however, these patients were often not fed enterally until after fascial closure, because exposure of the bowel was theorized to promote ileus and intestinal edema, which was thought to be exacerbated by enteral nutrition, thus delaying or preventing fascial closure, or leading to aspiration and pneumonia.

Few studies of enteral feeding in patients with temporary abdominal closure exist. Three civilian case series compare patients managed with early enteral nutrition to those for whom enteral nutrition was introduced late.[46-48] All three studies are retrospective and nonrandomized, and although the groups appear well matched, there is a risk of bias from unmeasured factors. Allowing for these limitations, Collier et al, in a study of 78 trauma patients, reported earlier fascial closure in patients who were fed enterally within 4 days of admission.[47] A further study of 100 trauma patients with hemorrhagic shock, from several facilities, found no difference in mortality, incidence of multiorgan dysfunction syndrome, duration of ventilation, intensive care unit or hospital stay, or fascial closure rates between those given early and late enteral nutrition, but it did report a lower incidence of pneumonia in those managed with early enteral nutrition, and early enteral nutrition remained independently associated with a reduction in the incidence of pneumonia on stepwise regression analysis.[48] A third, more recent but smaller study of 23 trauma patients also showed no differences in fascial closure rates or mortality, but also no difference in the incidence of pneumonia, in patients with an open abdomen who were given early enteral nutrition.[46]

In summary, there is no evidence that enteral feeding of patients with an open abdomen delays fascial closure or prolongs time spent in intensive care, and there is some evidence that it reduces the incidence of ventilator-associated pneumonia. Unless other contraindications are present, enteral nutrition should therefore be established early in patients with an open abdomen.[1]

Continuation of Enteral Nutrition During Repeat Operations

The widespread use of damage control surgery, and the nature of military wounds in general, has resulted in increasing numbers of "relook" or "take-back" operations, involving both the abdomen and other body regions such as amputation stumps. Many Role 3 trauma patients who are not eligible for transfer to Role 4 are returned to the operating theater every 48 to 72 hours during the initial 7 to 10 days of their hospital course. If patients are already intubated and ventilated in the intensive care unit prior to returning to the operating theater, and there are no plans for extubation immediately after reoperation, the question arises as to whether enteral feeding should be discontinued preoperatively and intraoperatively. Adherence to standard fasting guidelines intended for elective surgery will result in lengthy interruptions to feeding and failure to attain caloric goals.

To the authors' knowledge, there is no evidence or guidance, from the military or civilian setting, to inform a recommendation on this subject. Preoperative fasting is intended to reduce the risk of aspiration. Unless dislodged during transfer, patients with a cuffed endotracheal tube are arguably not at increased risk of aspiration, regardless of whether in the operating room or intensive care unit. It would therefore appear reasonable to continue enteral nutrition, certainly for extraabdominal procedures not involving manipulation of the airway, and possibly also for intraabdominal surgery.[1]

SUMMARY

Nutritional support is a broad and complex subject, and current practice is supported by a limited evidence base. Most of this evidence relates to critically ill patients in general, rather than surgical or trauma patients, and virtually all of it originates from the civilian setting. Military trauma patients, and the deployed environment, bring unique challenges and pose unique questions, which current guidelines cannot fully answer, necessitating a degree of extrapolation and pragmatism.

Despite these caveats, certain interventions can be recommended with confidence:

- Enteral feeding is superior to parenteral feeding, and should be started as early as practicable.
- The nasogastric route offers the benefit of being simple to use and facilitates early feeding.
- Early feeding does not adversely affect outcomes following gastrointestinal anastomosis, and is safe when the patient has an open abdomen. It does not delay closure of the abdomen and may reduce the incidence of pneumonia.
- Enteral feeding should be continued when patients are returned to the operating theater for surgery (other than airway or hollow-viscus procedures) in the days after the initial injury.

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Chapter 37

MANAGEMENT OF INFECTION AND SEPSIS IN THE INTENSIVE CARE UNIT

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INTRODUCTION

From an infection perspective, the intensive care unit (ICU) in a deployed military facility is in many ways no different from that in a civilian hospital. Patients are admitted who are acutely ill, as a result of either a primary community-acquired infection or an infection secondary to another event such as trauma. Management decisions are based on the clinical features of the disease process, specialized investigations including laboratory testing, and a knowledge of local disease epidemiology. However, some differences exist between the two settings, and this chapter explores how careful consideration of all the factors involved may lead to improved decision making.

Areas that will be covered include:

- sources of infection, including trauma and community-acquired infections;
- the "sepsis syndrome" and its clinical management;
- determination of likely pathogens involved, including knowledge of the local epidemiology of infectious diseases and microbial resistance patterns, and laboratory investigations; and
- control of the spread of microorganisms, including infection control and antibiotic policies.

SOURCES OF INFECTION

Although it is easy to become focused on current conflicts and the clinical spectrum of patients managed in ICU, predominantly trauma related, it is important to remember that these conditions may not be typical for all military conflicts. For example, in the early stages of the 2003 campaign in Iraq, the majority of ICU admissions were patients with severe illness caused by acute infective pneumonia, and relatively few battle casualties occurred.[1] During operations in Afghanistan some patients have been managed in the ICU as a direct result of a number of different community-acquired infections. These infections have included cases of *Streptococcus pneumoniae* bacteremia secondary to pneumonia, *Neisseria meningitidis* meningitis, Crimean-Congo hemorrhagic fever, rabies, tetanus, and *Escherichia coli* bacteremia secondary to urinary tract infection (AD Green, written communication, August 2013). Although the saying "common things occur commonly" remains true, it is important to reflect that by implication "rare things occur rarely" and still plan accordingly.

For other patients in intensive care, there can be significant challenges in trying to determine whether clinical infection is present (ie, a disease process) and if so, determine the likely etiological cause. For many patients the normal indicators of disease are heavily modified or obscured by the underlying medical condition and are unreliable means for assessment. Examples include temperature, pulse and respiratory rates, and peripheral white blood cell count. Laboratory markers of an acute phase response such as C-reactive protein may be helpful, but they can be modified by nonspecific responses to inflammatory conditions including trauma, and must be judged over time and in context. Microbiological investigations may also be misleading, since a patient's flora changes rapidly when normal physical barriers are compromised and antimicrobial agents are used.

Colonization and Infection

During recent conflicts there has been understandable concern over the isolation of multidrug-resistant (MDR) bacteria from injured personnel in deployed hospitals and the limited antibiotic options available to combat these organisms. Clinicians have a very low threshold for starting broad, aggressive therapy following isolation of these organisms from patients' samples. However, the bacteria isolated often colonize only, without causing disease, and the broad-spectrum antimicrobials prescribed will only further alter the patient's flora, selecting for the most resistant organisms.

Healthy people have a variety of microorganisms that inhabit their skin and mucous membranes. This flora can be split into (1) resident flora, a fixed variety of microorganisms that is normally age- and patient-dependent and will reestablish itself following a disturbance, and (2) transient flora, a mix of nonpathogenic and potentially pathogenic microorganisms that inhabit the skin or mucous membranes for hours to weeks and may cause illness.[2] A variety of factors may change this normal flora, with the use of broad-spectrum antimicrobials and the nosocomial introduction of new microorganisms in the healthcare setting being of particular importance. These two factors may be responsible for the MDR Gram-negative organisms isolated from patients along the evacuation chain in Iraq and Afghanistan.[3]

Once patients are colonized with these MDR bacteria, the big challenge is differentiating between simple colonization by the bacteria and infection causing disease. Contamination can be defined as the presence of

nonreplicating organisms in a wound, and colonization as the presence of replicating organisms.[4] Infection is a clinical diagnosis and indicates the presence of replicating organisms with host injury, often with invasion of the bacteria into tissue.

The presence of host injury and infection, rather than colonization, can often be difficult to determine by clinical examination. Traditional clinical signs of wound infection include inflammation, discharge of pus, and abscess formation. Many wound-scoring systems, especially those focusing on chronic wounds, now include other more subtle signs of wound infection such as delayed wound healing, pocketing at the base of the wound, abnormal smell, and discoloration.[5] The United Kingdom surgical site infection surveillance schemes are based on the US Centers for Disease Control and Prevention definitions from 1992, with superficial or deep incisional wounds having to fulfill specific criteria to be classified as infected.[6]

Wounds

In a deployed setting, infected wounds are a major source of sepsis. Following the loss of the protective layer of skin, open wounds will be colonized with microbes. This wound colonization is not necessarily a bad thing, with the presence of low levels of microbes able to accelerate the wound healing process by increasing the inflammatory response and local blood flow.[7] There then exists a spectrum from colonization, through local infection or critical colonization, to invasive infection.[8] The progression to critical colonization is often characterized by a wound that has no signs of tissue invasion but is not healing as expected.[9–11]

Ventilators

Other major causes of sepsis in the deployed ICU are nosocomial infections from catheter lines and pneumonia following intubation and ventilation. In both cases the normal anatomical barriers to infection—an important part of the innate immune system—have been disrupted.

In a study of ventilator-associated pneumonia (VAP) in Operation Iraqi Freedom, Landrum and Murray found the most common isolated organism was *Acinetobacter* species, followed by *Klebsiella pneumoniae*, and then *Pseudomonas aeruginosa*.[12] Although many factors can lead to ventilator-associated pneumonia, and it is a common complication seen in civilian practice, of particular note in this study was that the rates of VAP and the number of resistant isolates were reduced following the introduction of targeted infection control measures.

Intravascular Lines

Intravenous catheter lines can become contaminated at various points, in particular the catheter hub/infusion tubing junction and at the point of insertion into the skin. Many risk factors for line-associated bacteraemia exist, in particular alteration of the patient's cutaneous microflora (most commonly by antibiotics or colonization with an epidemic strain carried by hospital personnel), active infection at another site, and failure of the healthcare provider to wash his or her hands.[13] The excessive use of broad-spectrum antibiotics combined with poor infection control practices—both of which are very difficult to avoid in a deployed setting—can lead to increased catheter line infection rates and patient morbidity.

Biofilms

The bacteria that cause such concern are able to thrive in the hospital environment due to a number of virulence mechanisms that increase the disease-causing potential of the organism. Certain virulence factors are not found in all bacteria of a species, but only in disease-causing subtypes; for example, strains of *Streptococcus pyogenes* that contain the gene for the M1 protein are associated with more invasive disease and necrotizing fasciitis.[14] In catheter line infection, ventilator-associated infection, and wound infections, the development of a biofilm is a key virulence mechanism. Bacteria produce a biofilm to protect themselves. Biofilm is an extracellular polysaccharide matrix that forms once the colonies reach a particular size. The bacteria are able to detect the size of their colony, develop a mature biofilm, and respond to factors such as nutrient availability by a process called quorum sensing—communication between bacteria using signalling molecules.[15] The biofilm provides mechanical protection to the bacteria, preventing antibiotic penetration and the patient's phagocytic cells from attacking the colony.[16] Bacteria in a biofilm are substantially more resistant to antibiotic treatment than planktonic bacteria (floating outside a biofilm); therefore, although an organism may appear sensitive to a drug in the laboratory, bacteria with biofilm will not be affected and the patient will not improve despite antimicrobial therapy.[17,18] The ability of *Acinetobacter baumannii* to survive so well in hospital environments is due to many virulence factors, especially its ability to form a biofilm on a variety of biological and abiotic surfaces.[19] This ability to survive in the hospital environment was demonstrated in a cluster of VAP cases in Canadian soldiers injured in Afghanistan. The source of the isolate was thought to be environmental, from

the Kandahar military hospital, and the *Acinetobacter baumannii* isolate in four soldiers was indistinguishable from an isolate found growing on a ventilator air intake filter.[20]

SEPSIS

"Sepsis" has been defined by a consensus agreement between the American College of Chest Physicians and the Society of Critical Care Medicine.[21] The definition has been accepted internationally and is used by the global Surviving Sepsis Campaign initiated in 2004. The definition states that sepsis is the presence of a systemic inflammatory response syndrome resulting from infection (Exhibit 37-1). Severe sepsis exists when organ dysfunction develops. When a patient becomes hypotensive despite adequate fluid resuscitation, he or she is in septic shock.[22] Septic shock represents a state of vasopariesis and maldistribution of fluid rather than fluid deficit. Early fluid resuscitation is a temporizing measure that may mitigate poor organ perfusion while vasopressor therapy, appropriate antibiotic therapy, and source control are instigated.

Managing the Patient: The Surviving Sepsis Campaign

The Surviving Sepsis Campaign promotes a set of guidelines agreed upon by an international editorial board following a comprehensive literature review, most of which are directly transferable to the military setting.[23] The guidelines divide management of the septic patient into three parts: (1) the initial resuscitation bundle (Exhibit 37-2), occurring over the first 6 hours; followed by (2) a sepsis management bundle (Exhibit 37-3) extending up to 24 hours; and (3) other supportive therapy (Exhibit 37-4). Although described in sequence, the interventions recommended by the guidelines are implemented concurrently and as soon as possible.

The Resuscitation Bundle

Achieving the initial resuscitation bundle reduces mortality in sepsis by 50%, and treatment should begin as soon as severe sepsis is recognized and before the patient arrives at the ICU. Obtaining cultures before antibiotic administration provides the best chance of

EXHIBIT 37-1

CRITERIA FOR THE SYSTEMIC INFLAMMATORY RESPONSE SYNDROME

Two of the four following parameters in the presence of inflammation:

- temperature (> 38°C or < 36°C)
- white blood cell count (< 4 or > 12 x 109 [or > 10% immature forms])
- tachycardia (HR > 90 bpm)
- tachypnea (RR > 20 min^{-1} [or a $PaCO_2$ < 32 mmHg/4.3 KPa])

bpm: beats per minute
HR: heart rate
$PaCO_2$: partial pressure of arterial carbon dioxide
RR: respiratory rate

EXHIBIT 37-2

THE RESUSCITATION BUNDLE

Bundle Element	Notes
1. Measure serum lactate.	
2. Obtain cultures prior to administering antibiotics.	Obtain all relevant cultures: blood, sputum, urine, tissue, pus, CSF.
3. Administer broad-spectrum antibiotics.	As soon as possible and preferably within the hour.
4. Treat hypotension and/or a lactate > 4 mmol/L.	Administer fluid boluses while patient is fluid responsive and subsequently begin vasopressors to maintain MAP > 65 mm Hg.
5. Obtain source control.	Surgical debridement, percutaneous drainage, or removal of invasive lines.
6. Determine targets for ongoing use of fluid and vasopressors.	Achieve a CVP of > 8 mm Hg, an $ScvO_2$ of > 70%, or an $ScvO_2$ of > 65% and a urine output of 0.5 mL/kg/h.

CSF: cerebrospinal fluid
CVP: central venous pressure
MAP: mean arterial pressure
$ScvO_2$: central venous oxygen saturation
SvO_2: mixed venous oxygen saturation

EXHIBIT 37-3

THE SEPSIS MANAGEMENT BUNDLE

Intervention	Notes
Fluid therapy	Give 1,000 mL/500 mL fluid challenges with crystalloid/colloid respectively to achieve a CVP of 8 mm Hg.
Vasopressors	Use norepinepherine or dopamine to achieve an MAP of 65 mm Hg. Vasopressin 0.03 units/min. Add epinephrine if hemodynamics are deteriorating.
Inotropic therapy	Use dobutamine in presence of myocardial dysfunction (as demonstrated on trans-thoracic echo when deployed).
Steroids	Add hydrocortisone to a maximum dose of 300 mg/day.
Recombinant human activated protein C	Consider in adult patients with multiple organ failure and no contraindications.

CVP: central venous pressure
MAP: mean arterial pressure

EXHIBIT 37-4

OTHER SUPPORTIVE THERAPY

Intervention	Notes
Blood product administration	Target hemoglobin of 7.0–9.0 g/dL. Only correct deranged clotting with fresh frozen plasma if bleeding or invasive procedures are planned.
Mechanical ventilation	Nurse patient with the head up 45°. Provide a tidal volume at 6 mL/kg (predicted body weight) with a peak pressure of ≤ 30 cm H_2O. Tolerate hypercarbia. Use a sedation and weaning protocol including sedation holds and spontaneous breathing trials.
Glucose control	Keep blood glucose < 150 mg/dL (8.3 mmol/L).
DVT prophylaxis	Use a mechanical prophylactic device if low molecular or unfractionated heparin is contraindicated.
Stress ulcer prophylaxis	A proton pump inhibitor may be preferable to an H_2 blocker in thrombophilia.

DVT: deep vein thrombosis

obtaining a meaningful result, although this should not be allowed to delay antibiotic administration unduly. Cross-sectional imaging may be required when the source of sepsis is not obvious.

Antibiotics are a vital and time critical intervention that must be a priority for the managing clinicians; mortality in patients with septic shock increases by 7% per hour that administration of appropriate antibiotics is delayed.[24] The choice of antibiotic should be protocol driven, but will initially be broad spectrum with the aim of deescalating to more targeted therapy once culture results become available. The choice of antimicrobial agents will be determined by local epidemiology of disease and microbial resistance patterns, and by the availability of drugs in the deployed formulary.

The resuscitation targets are derived from a study examining early goal-directed resuscitation protocols, which showed a reduction in 28-day mortality (30.5% vs 46.5%) and less organ dysfunction in the treatment group.[25] In ventilated patients or in those with intraabdominal hypertension, the CVP target should be revised upward to at least 12 mm Hg. Invasive cardiac output monitoring is not currently available in most deployed settings; transthoracic echo may be available and can provide useful information about volume status and cardiac performance.

The guidelines support the use of crystalloid and colloid equally, and a mixture of a balanced salt solution and synthetic colloid is often used. If despite perceived adequate fluid resuscitation the central venous oxygen saturation remains below 65%, then oxygen delivery should be augmented through transfusion of packed red blood cells to reach a hematocrit of 30% or alternatively by starting a dobutamine infusion (3–20 μg/kg/min). An epinephrine infusion (0.04–0.4 μg/kg/min) is an acceptable alternative to adding dobutamine to a preexisting norepinephrine infusion. The guidelines support the use of dopamine (1–20 μg/kg/min) as an alternative to norepinephrine, although the increased incidence of arrhythmias should be noted.

The Sepsis Management Bundle

Noncompliance with the sepsis management bundle of hemodynamic support and adjunctive therapy results in a significant increase in mortality.

Fluid challenges targeting central venous pressure and lactate should continue while the patient's central venous pressure remains fluid responsive, and the mean arterial pressure should be maintained at 65 mm Hg or above. Intravenous hydrocortisone (50 mg, every 6 hours) should be added in the presence of an increasing vasopressor requirement. An adrenocorticotropic hormone test is no longer recommended and not practical in the deployed environment.

Vasopressin (an antidiuretic hormone) causes vasoconstriction via activation of V1 receptors on vascular smooth muscle while also mediating coronary, renal, pulmonary, and cerebral vasodilatation in low doses. Plasma vasopressin levels fall rapidly in septic shock. Vasopressin administration improves blood pressure and renal function, but the only large randomized control trial completed so far found no difference in mortality when compared to norepinephrine.[26] It did, however, demonstrate a synergistic effect when vasopressin and steroids are combined, which resulted in both a statistically and clinically significant reduction in mortality. At doses exceeding 0.04 units per minute, vasopressin decreases cardiac output and causes myocardial ischemia and renal vasoconstriction.

Other Measures

Although not in the Surviving Sepsis guidelines, high-dose intravenous immunoglobulin and methylene blue are occasionally used in some facilities in cases of severe sepsis. Polyclonal immunoglobulin may suppress the inflammatory response to infection, although it may remain impractical until the casualty reaches a homeland facility. Dose is preparation dependent and outlined in Table 37-1. Methylene blue (2 mg/kg bolus followed by an infusion of 1 mg/kg/h for 12 hours) inhibits nitric oxide-mediated vasodilation and may have a vasopressor-sparing action. The evidence base for its use is currently scanty.

TABLE 37-1

DOSES OF INTRAVENOUS IMMUNOGLOBULIN

Preparation	Dose
Intraglobin	250 mg/kg over 2 days
Sandoglobin	400 mg/kg/day for 3 days
Endobulin	1 gm/kg on day 1, then 500 mg/kg on days 2 and 3
Pentaglobin (IgM enriched)	1,300 mL within 72 h

Septic patients occasionally demonstrate tachyphylaxis to vasopressor agents, requiring alternative use of agents and the addition of agents not normally used in this context such as phenylephrine (0.5–5.0 μg/kg/min).

TAILORING THERAPY

It is possible to develop generic guidelines for all aspects of clinical management of patients in deployed medical facilities, and for most aspects of care, including antimicrobial therapy, this is entirely appropriate.[27] However, geography, environment, and operational context have significant impact on the range of potential pathogens, and in most cases theater-specific guidance is required that reflects local microbial resistance patterns and disease epidemiology. In turn, this mandates that microbiological laboratory support now forms an integral element of deployed medical care, both for early insertion and enduring military operations.

Laboratory Support

Early insertion operations or those with a small medical footprint are generally planned to deploy without discrete laboratory facilities; the medical plan requires immediate evacuation of casualties once stabilized. Any laboratory support required is provided by point-of-care testing and undertaken by either laboratory scientists or medical personnel. In a forward environment the primary role is provision of blood and blood products, and the requirement for microbiological support is limited; patients are not held at the location pending investigations, and those investigations deemed critical are provided by point-of-care testing technology (eg, rapid malaria diagnostics).

For mature and enduring operations the situation is different. Although critically ill casualties will still be evacuated as soon as possible, there is now the requirement to provide extended care. This care might be for Allied forces, local police and military personnel, homeland civilian contract personnel, and local civilians. Intensive care support is most likely to be sited with this level of medical provision. The laboratory requirement is significantly different compared to the light role, with the need for both infectious disease diagnostic capability and appropriate bacteriological support. It is clear from recent operational experiences that "appropriate bacteriological support" is at a much

higher level than previously considered, reflecting the high quality of care now delivered, the increasingly complex resistance patterns of endemic bacteria, and the need to accurately direct antimicrobial therapy.

Antibiotic Guidelines

For deployed medical facilities, antibiotic guidelines are generally divided into trauma-related and non-trauma-related components. For trauma, guidelines recommend the use of particular antimicrobials depending on the area of the body injured, type of injury, level of care (ie, prehospital or hospital), and the time since injury. These are evidence based when possible and kept under regular review.[28] For non-trauma-related infections, guidelines give recommendations based on the differential diagnosis and likely pathogens involved.[27]

Several factors must be considered when developing local guidelines suitable for use in a deployed ICU. In civilian settings ICU guidelines can be tailored to local resistance patterns based on data gathered over many years and adjusted as required by the microbiology or infectious diseases senior consultants. In contrast, no local data will initially be available to inform military guidelines for the deployed hospital, and the guidelines must be adaptable to a wide range of environments and a wide variety of microbial resistance patterns. Deployed hospitals will often lack a deployed specialist microbiologist or infectious diseases specialist to advise on appropriate alternatives.

There are also other constraints not encountered in civilian practice. For example, therapeutic monitoring of drug levels is often unavailable, and there may be difficulties with supply chains for pharmaceuticals. Classes of antibiotics used daily in civilian ICUs include aminoglycosides (such as gentamicin) and glycopeptides (such as vancomycin) that have a narrow therapeutic index, with dose-related side effects. In the absence of monitoring and given the potential for drug toxicity, an alternative antimicrobial with similar cover should be considered (eg, teicoplanin rather than vancomycin). Logistic restraints limit the variety of antibiotics on the formulary. It is easier to maintain stock levels and supply lines for a small number of drugs that are regularly used, sometimes requiring that a suitable rather than ideal option is chosen.

A further requirement is that clinicians must find the guidelines easy to adopt on joining a deployed unit. The drugs in the UK and US guidelines are commonly used in civilian departments and will be familiar to all intensivists. Antibiotic guidelines and prescriptions for operational theaters must also allow for the variation in antibiotic preference and licensing seen between different coalition partners. Different national guidelines will recommend different antibiotics at different points in a patient's treatment. Licensing differences are a common occurrence; eg, the UK Medicines and Healthcare Products Regulatory Agency may not have approved a drug that has been approved by the US Food and Drug Administration (Center for Drug Evaluation and Research). In Afghanistan the International Security Assistance Force contains more than 45 different nations' troops, with two to three nations' medical staff in the deployed hospital at Bastion at any one time. As an example of the potential complexities, a 2006 review of antimalarial chemoprophylaxis of North Atlantic Treaty Organization forces in Afghanistan indicated that every nation had a different policy.[29]

It is now widely accepted that in the face of dwindling numbers of new antibiotics and increasing resistance, antibiotic use must be monitored and controlled. The term often used for this control is antibiotic stewardship, and its aims are to reduce the unintended consequences of antimicrobial use such as toxicity and emergence of resistance.[30] In the deployed setting the use of broad-spectrum antibiotics, and the selection pressure caused by this practice, has been implicated in the increased isolation of MDR organisms from patients. One military study showed that reducing the surgical antibiotic prophylaxis given in an Air Force theater hospital in Iraq to that recommended in US military guidelines reduced the number of VAP cases with MDR organisms.[12] This reflects findings from civilian practice, with studies showing an increased mortality associated with inappropriate antibiotic prescription in patients on a civilian intensive care unit[31] and increased mortality in patients with severe sepsis and shock-complicating gram-negative bacteremia who had recent antibiotic exposure.[32]

The emergence of novel MDR strains of bacteria from central Asia remains a cause for concern, and is subject to active surveillance.[33] One such strain may be actively selected for by widespread use of carbapenem antimicrobials in North Atlantic Treaty Organization forces.[34]

A number of antibiotic stewardship strategies can be used to control antibiotic use and prevent the development of resistance. Although clinician education is important, the main method of stewardship in a deployed setting with rotating personnel is the strict use of antibiotic guidelines. Military guidelines are designed to restrict the use of broad-spectrum antibiotics to situations when they are required, and adherence to the guidelines is essential to prevent the selection pressure that leads to the colonization of patients with resistant organisms. Other options such as telephone approval, antibiotic cycling, heterogeneity of antimi-

crobial use, prior-approval programs, and automatic stop orders either currently lack supporting evidence or would not be practical in a deployed setting because of logistic and communication constraints.[30]

SUMMARY

Infection control in deployed medical facilities is perhaps more important today than ever before because the consequences of failure can be significant and readily visible to a wide audience. The effects may include operational impact, with loss of one or more medical facilities as a result of an outbreak or the control measures employed,[35] and exportation of MDRs to civilian medical facilities in the homeland.[36] The subject is discussed in more detail in Chapter 40, Multidrug-Resistant Organisms and Infection Control Practice in the US Military Medical System. Revised guidance has been recently produced by a joint US-UK group.[37]

In the ICU setting, infection control is important at all times and in the operational setting may be subject to additional pressures. Different patient populations may be managed alongside each other, with some patients rapidly transferring to home countries on evacuation after initial care, while others remain for extended periods.

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Chapter 38

AIR TRANSPORT OF THE CRITICAL CARE PATIENT

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INTRODUCTION

Aeromedical evacuation (AE) has been undertaken in some form or other for well over a century. Currently, US Air Force (USAF) Critical Care Air Transport Teams (CCATTs) and Royal Air Force (RAF) Critical Care Air Support Teams (CCASTs) assist in carrying out the AE mission, providing the AE system's core critical care capability by delivering optimal care to severely injured or ill patients during air transport. The addition of an intensive care capability on mobility airlift aircraft has added a new dimension to the AE mission. This capability ensures that the level of medical care for critically ill and injured patients remains constant throughout the entire en route care transport system in military aircraft.

CCATTs and CCASTs are a limited, highly mobile, rapidly deployable resource available in certain situations. They are typically utilized after the patient has received essential stabilizing care by ground medical units. The teams provide continuous monitoring, ongoing resuscitation, and continuing stabilization of critical care patients during evacuation within theater or to higher roles of care.

CCATTs are utilized for a wide spectrum of operations, including humanitarian relief, disaster response, small-scale contingencies, homeland security, and major theater war.[1] CCATTs function independently of an AE team, but are required to have an AE team on the aircraft while transporting CCATT patients. CCASTs are able to operate independently of the routine AE system although the two tend to be used in parallel. CCASTs are used to transport service members and entitled civilians (diplomatic staff and defense contractors with whom a specific arrangement has been made). CCASTs are not used in the humanitarian role unless specifically tasked.[1,2]

HISTORY

AE was initially performed ad hoc, with nonmedically modified aircraft being used. The first documented episode of AE occurred during the Franco-Prussian War of 1870–1871, when observation balloons were flown out of Paris (under siege by the Germans) containing mail, high-ranking officials, and 160 casualties.[3] Fixed wing (FW), powered aircraft were first used to evacuate wounded in 1915, during the retreat of the Serbian army from Albania, and by 1919 the RAF was using modified De Havilland DH9 aircraft in Somaliland (part of modern-day Somalia). AE in a form recognizable today (using dedicated aircraft over long distances) was used in the Spanish Civil War by the German Luftwaffe.

World War I efforts led to the organization of an integrated AE system by the US Army Air Corps during World War II. This system included nurses with specific AE training serving on cargo aircraft returning from the theater of battle. In 1942 the US Army Medical Service formed the 38th Medical Air Ambulance Squadron at Fort Benning, Georgia. More than a million patients were evacuated during World War II using AE. Subsequent advances in technology have seen rotary wing (RW) assets being used, first in Burma in 1944 and more famously in Korea with "Dustoff" helicopters in the 1950s. During the Vietnam conflict, concepts of operation developed further with a more formalized system of in-flight care.[4]

By the 1990s the AE system included command and control functions, trained crews, mobile facilities for staging patients preflight, and extensive logistical support. The system could rapidly deploy, set up, and evacuate large numbers of stable casualties, but it lacked the intrinsic capability to manage critically ill casualties, instead relying on medical attendants, supplies, and equipment provided by the sending medical facility. The requirement to provide these resources was a particular challenge for small field hospitals with limited personnel, which cannot lose personnel without seriously degrading their capability.

Following Operation Desert Storm in 1990, calls were made for the addition of AE physicians and equipment capable of managing unstable patients in flight. However, the problem remained, becoming most evident in Somalia when the surge of combat casualties on October 3–4, 1993, overwhelmed the medical response capabilities, casualties accumulated, and the most critical patients could not be immediately evacuated.

The USAF is responsible for the intra- and intertheater transport of injured US military both within the theater of operations and from the theater of operation to the continental United States (CONUS). This requires the ability to provide ongoing care during long-distance flights. The system relies on available USAF aircraft that are temporarily converted into AE-capable platforms as the need arises. USAF teams involved in patient transport include the aircraft crew, AE medics, and CCATTs. Until the mid-1990s, most if not all injured patients requiring AE transport had to be relatively stable for transport. Limited medical care could be performed during transport byAE

teams; for example, if a patient in Germany had an uncomplicated exploratory laparotomy, he or she would have to stay at the hospital where the surgery was performed until considered stable for transport, anywhere from 3 to 5 days postoperation. If patients required any special care or pain medicine other than oral or intermittent intravenous (IV) bolus, a medical attendant had to travel with the patients to manage their care during transport.

Early AE teams typically consisted of a mix of registered nurses and medical technicians specifically trained for air transport. A typical AE team included two nurses and three technicians; an expanded team consisted of three nurses and four technicians. These personnel varied from outpatient clinic staff to critical care staff, and the patient care abilities and comfort levels of AE team members ranged vastly. Anything other than basic care was limited by the makeup of the AE team. The typical AE transport had a patient load of anywhere from 1 to over 50 patients, depending on the types of patients, whether they were ambulatory, and the aircraft available. To support this method of AE, the holding capabilities of medical facilities in and out of theater had to be robust, which was logistically difficult to support and often not in the best interest of the patient.

During the 1980s and early 1990s, Dr Paul K Carlton Jr, a surgeon and later the USAF surgeon general, developed capability for the rapid effective stabilization and transport of significantly injured or traumatized casualties. Carlton based his method on his experience at Wiesbaden, Germany, receiving casualties from the embassy bombing in Beirut, Lebanon. In 1994 Carlton and Dr Joseph C Farmer, a medical intensivist, launched the CCATT program, consisting of teams with a critical care physician, critical care nurse, and respiratory therapist, accompanied by the supplies and equipment necessary to create a critical care environment that would move with the patient during evacuation.

Team members were specifically trained to provide specialized care in the high-altitude, extreme aircraft environment. The concept of CCATT is to manage stabilizing casualties—those who have undergone initial resuscitation but remain critically ill. A physician was included on the team to provide continuous medical decision-making, so that therapies could be titrated to the patient's condition, with new therapies started if required, and patients could continue progressing toward stability without interruption or setback for transport. Having a CCATT physician available during an AE mission also allowed better medical care for the non-CCATT patients, including pain management.

The timing of CCATT development allowed the US military healthcare system to adjust its doctrine in response to changing military strategy. During the Cold War, US forces prepared for large battles in predictable locations supported by established hospitals with the capacity to hold large numbers of casualties until they had completed convalescence and were returned to duty. After the Cold War ended, the US military became engaged in a large number of activities ranging from humanitarian and peacekeeping operations to combat. These operations often arose quickly, took place in unpredictable locations, and in some cases changed locations rapidly; establishing large-capacity hospitals whenever and wherever needed became impossible. Instead, the military needed to deploy small, high-capability, limited-holding-capacity facilities that could stabilize and evacuate casualties with far less logistic support. To accomplish this objective, medical personnel needed to be able to evacuate even unstable casualties safely, and CCATT offered that capability.

PATIENT MOVEMENT CONCEPTS

Patient evacuation from point of injury to initial role of care is the responsibility of each service component.[2,5] Casualties are evacuated through five roles of care with increasing capability, from self- and buddy-care with initial management at aid stations close to the point of injury, through advanced rehabilitative care at military and Veterans Administration medical centers in the United States.

Casualty evacuation (CASEVAC) is the movement of unregulated casualties by non-medical units aboard non-medical vehicles or aircraft, without en-route care by medical professionals. The casualty is taken from the point of injury to the most appropriate medical facility; typically Role 1 or Role 2 facilities. The CASEVAC mission may involve care under fire, and speed and security are more important than advanced en-route care. In the US military CASEVAC is overwhelmingly an Army, Marine Corps, or Navy mission.

Medical evacuation (MEDEVAC) refers to patient movement using predesignated tactical or logistics vehicles or aircraft equipped and staffed for en-route care. MEDEVAC has generally implied the use of US Army RW aircraft with specially trained medical attendants. In MEDEVAC, casualties are transported onboard medical helicopters under the care of combat medics with advanced flight training. Constituting a paramedic level of care, this capability can be used from the point of injury to a medical facility, or between facilities.

Patient movement of US military and DoD personnel through the AE system is the responsibility of the USAF.[2] USAF AE implies patient movement on fixed-wing aircraft. Deployed CCATTs are assigned to a deployed AE squadron and are responsible for patients transported via AE who require intensive care or monitoring during flight. The AE function can be categorized as tactical evacuation (TACEVAC) within a military theater of operations or strategic evacuation (STRATEVAC) between theaters of operation. STRATEVAC is primarily the domain of the USAF. The AE system refers to the regulated movement of casualties from Role 2 or Role 3 through Role 4 facilities by fixed-wing USAF aircraft.

For STRATEVAC missions, the USAF has two types of staging facilities: contingency aeromedical staging facilities (CASFs) and mobile aeromedical staging facilities (MASFs). The main difference between the two is size and the number of personnel. The MASF is smaller and designed to be highly mobile and rapidly deployable; the CASF is a more fixed facility for long-term operations. Both are positioned at major air-hubs of the AE system, serving as buffers that allow non-critical casualties to be housed, fed, and prepared for flight at a location where they can be rapidly loaded as aircraft become available. At the staging facility (CASF or MASF), basic medical care and wound care, as well as basic pain control (oral and IV bolus), are provided. Patients waiting at the air-hubs typically have minor injuries preventing them from immediately returning to duty. The CCATT (or critically injured) patients stay in the medical facility, typically in the intensive care unit, until time for transport. Aboard the aircraft, an AE crew consisting of flight nurses and medical technicians who have undergone specialized training manage the AE patients. The care given by an AE crew is limited by the large number of patients they are tasked to manage and their level of medical training. If a patient requires more care than this basic level, the sending facility has historically been responsible for providing a medical attendant during evacuation. Today, for casualties who are critically ill or injured, the AE system is augmented with a CCATT.

For United Kingdom (UK) forces, Air Publication 3394, *The Royal Air Force Aeromedical Evacuation Service,*[6] provides the details of all aspects of AE. RAF Medical Emergency Response Teams evacuate patients from point of injury to Role 2 or 3 facilities in theater. After resuscitation and damage control surgery has been performed, patients who require in-flight critical care are returned to the UK by CCAST. Those less severely injured are transported in the routine AE chain.

OPERATIONS

Team Composition

An individual CCATT consists of a physician with experience in managing critical care patients (board certified in critical care medicine, anesthesiology, cardiology, emergency medicine, or pulmonary medicine); a critical care nurse; and a cardio-pulmonary respiratory therapist. One team (physician, nurse, and respiratory therapist) can provide care for a maximum patient load of three high acuity (ie, ventilated) patients, or up to six lower-acuity (ie, nonventilated) stabilized patients.[1] A CCAST is comprised of a flight nurse trained in intensive care, a medical devices technician, a consultant anesthesiologist (and usually a trainee anesthesiologist), and a flight nursing assistant. This basic team is able to provide care for one patient requiring intensive care. Equipment is modularized and can therefore be augmented (as can the staffing) to provide care for further patients if necessary. A second intensive care nurse usually augments the CCAST, and additional equipment is routinely included to provide care for a second patient, who may be identified while the team is en route. While each team member has specific primary duties, the flexibility of individuals to perform the duties of other team members is essential for efficient team functioning.

Capabilities and Responsibilities

CCATTs and CCASTs provide capability to evacuate critical care patients requiring ongoing stabilization or advanced care during transport to the next role of care. Prior to transport, the team is responsible for preparing the critically ill patient for movement, as well as ensuring that the patient is stable enough for transport and no other major interventions are required (Exhibit 38-1). The team will then accompany the patient from the originating medical staging facility onto the aircraft and to its destination. The team continuously monitors and intervenes during flight operations as required. CCATTs and CCASTs do not routinely provide primary stabilization at point of injury and do not replace forward surgical or ground medical support team capabilities.

CCATTs function as components of the AE system and are not trained or equipped to operate as an autonomous capability; however, they have operated independently at various times. As an AE asset,

EXHIBIT 38-1

PREFLIGHT PATIENT CONSIDERATIONS

- General preferred clinical parameters prior to intra-theater trauma patient transport:
  - heart rate < 120 beats/minute
  - systolic blood pressure > 90 mm Hg
  - hematocrit > 24%
  - platelet count > 50/mm^3
  - INR < 2.0
  - pH > 7.3
  - base deficit > 5 mEq/L
  - temperature > 35°C
- Air Publication 3394 provides guidance on specific conditions for CCASTs.
- It may be in the patient's best interest to be transported to a higher role of care even if the above parameters have not all been met, based on specific patient and mission situations.
- Medical evacuation requests should include required transport equipment and provider requirements.
- The patient must be sufficiently stabilized for the anticipated duration of travel.
- The airway should be appropriately secured in ventilator-dependent patients.
- All patients should have their pain adequately managed prior to transfer with sufficient provision for likely additional requirements during transfer. In ventilator-dependent cases, this is achieved with balanced sedation and analgesia. Conscious patients may well have neuraxial blocks (either central or individual nerves) or will require appropriate alternative pain management options, eg, patient-controlled analgesia.
- IV lines, drainage devices and tubes should be fully secured and patent.
- Fasciotomies and escharotomies should be used appropriately.
- Casts must be bivalved.
- Cabin altitude restriction is required for transport of patients with decompression sickness.
- Consider a cabin altitude restriction for the following:
  - penetrating eye injuries with intraocular air
  - free air in any body cavity
  - severe pulmonary disease
  - arterial gas embolism
- A chest tube is required for pneumothorax, even if it is small and asymptomatic.
- Personal effects and all medical records (including digital copies of any radiology) should accompany the patient.
- Three litter straps should be used to secure the CCATT patient to the litter. CCASTs use a five-point harness system which is fixed to the litter.
- For critically ill patients, the CCATT or CCAST at the originating MTF should assess the patient's clinical status for flight whenever feasible.
- Patients should be transitioned to CCATT/CCAST equipment and assessed for stability in an MTF environment when feasible. Any interventions required to enhance the patient's stability for transport should be performed prior to transport.
- Determination of continuing preflight care requirements must be ongoing because a change in clinical status may require postponement or cancellation of the scheduled transport.

CCAST: Critical Care Air Support Team
CCATT: Critical Care Air Transport Team
INR: international normalized ratio
IV: intravenous
MTF: medical treatment facility

Data sources: (1) US Army Institute of Surgical Research website. Intratheater Transfer and Transport of Level II and III Critical Care Trauma Patients. Joint Theater Trauma System Clinical Practice Guideline. 2008. http://www.usaisr.amedd.army.mil/assets/cpgs/Intratheater_Transfer_and_Transport_19_Nov_2008.pdf. Accessed February 3, 2014. (2) Defence Council of the Ministry of Defence. *The Royal Air Force Aeromedical Evacuation Service.* 4th ed. London, United Kingdom; 2012. Air Publication 3394. (3) *Emergency War Surgery, 3rd United States Revision.* Washington, DC: Borden Institute; 2004:4.1–4.9.

CCATTs are involved in the full spectrum of operations to move critically injured and ill patients to the next role of care. A full AE team is always required to travel with a CCATT, even when only CCATT patients are being transported, which can at times result in an overall poor utilization of assets. CCASTs can either operate within the routine AE system or independent of it.[3,6] Missions to established operations requiring a CCAST capability usually involve combined routine and CCAST components, but if the only requirement for the mission is a CCAST capability, the team can deploy on its own. This is more frequently the case when a patient is in a location outside established operational areas (eg, a service member who has become critically ill while on a Navy ship and was evacuated to the nearest medical facility, and its capabilities are insufficient to meet the patient's ongoing needs). In enduring operations (eg, Afghanistan), a tactical CCAST may be deployed with the field hospital component to provide in-theater transport capability where, for reasons of capability or capacity, patients may need to be transferred from one hospital to another.

While the maximum patient load for the basic CCATT three-member team is three ventilator patients or six lower-acuity patients, CCATT extender teams can increase the capability to five ventilator patients or ten lower-acuity patients, based on the total patient acuity level for the mission.[1] Additional CCATTs may be required on a given mission depending on the acuity and number of patients transported. A specialized lung team based at Landstühl Regional Medical Center (Germany) is capable of transporting patients with severe lung disease requiring advanced ventilatory support, and a specialized burn team based at San Antonio Military Medical Center (Texas) is available for transporting high-percentage burn patients. However, the vast majority of the critical injured are transported via CCATT alone; both the lung and burn teams are utilized only for the most extreme cases.

There is no equivalent of the lung team in UK practice, but the standard team is capable of managing all but the most complicated patients. The equipment in one "575 (CCAST)" module is sufficient for the transfer of one patient, independent of aircraft power and oxygen, for a 24-hour period. Because the CCAST, augmented with a second flight nurse or flight nursing officer, can provide care for a maximum of two patients with a standard module of equipment, additional patients require the deployment of further augmentations to the equipment and nursing personnel, with the aim of providing one-to-one nursing care and to mitigate staff fatigue. One anesthesiologist can provide care to up to four patients.

Tasking

The patient movement requirement center (PMRC) validates the requirement for CCATT patient movement (Exhibit 38-2). CCATTs may be tasked to supplement TACEVAC or STRATEVAC. Requirements for support are based on expected casualties, location, available medical capability, and en-route care requirements. For CCASTs from the UK, the tasking authority is the Aeromedical Evacuation Co-ordination Centre (AECC) at RAF Brize Norton in Oxfordshire.[6] Tactical teams deployed in the theater of operations will be tasked locally according to need.

CCATTs may be deployed with an AE unit based on operational requirements. The request for CCATT transport should come through coordination between the originating physician, the PMRC validating flight surgeon, the CCATT theater director, and the destination accepting physician. The PMRC validating flight surgeon and CCATT theater director work with the sending, accepting, and transporting CCATT physician when planning and coordinating the patient's transfer. The transporting CCATT physician, in coordination with the CCATT theater director, makes the final determination to transport a patient after a thorough assessment that includes the patient's ability to tolerate transport and other flight logistics considerations. The CCATT theater director determines the number of CCATTs on each mission.

A similar system exists for CCAST tasking,[6] with coordination between the team referring the patient and the tasking authority (either AECC or the local medical chain of command, depending on the type of transfer)

EXHIBIT 38-2

PATIENT MOVEMENT REQUIREMENT CENTERS (AS OF JANUARY 1, 2014)

- Joint Patient Movement Requirement Center, Al Udeid Air Base, Qatar
- Global Patient Movement Requirement Center, Scott Air Force Base, Illinois
- European Command, Ramstein Air Force Base, Germany
- Pacific Command, Hickam Air Force Base, Hawaii
- Royal Air Force Aeromedical Evacuation Control Center, Brize Norton, Oxfordshire, United Kingdom

provided by an AE liaison officer. An AE coordinating officer is deployed to provide clinical direction. These two officers are supported by an AE operations officer in larger operations. Four signals are generated. Signal 1 provides only administrative details of the patient to be transferred. Signal 2 provides all the details of the care received by the patient up to the point of referral and any planned interventions. These data are covered by the legal principles of medical confidentiality. Signal 1 has a larger distribution (for administrative purposes) than Signal 2 (limited to those who need clinical information). Signal 3 is the authority (from AECC) to enplane the patient. Once the CCAST has assessed the patient, the team leader decides whether the patient is to be enplaned or not and whether any restrictions to flight (for example cabin altitude and pressurization) are necessary. Signal 4 informs AECC that the aircraft has left its point of departure and details any further clinical changes the patient has undergone, as well as any specific requirements for the onward transport of the patient from the airhead to the hospital, particularly if a police escort is required for a multivehicle convoy or a high-lift device is needed to assist egress from the aircraft. If an AE liaison officer is not deployed in that location, coordination may be directly with the referring clinician or via diplomatic staffs to AECC. For STRATEVAC missions, AECC coordinates with the receiving facility.

Rotary Wing Operations

CCATT and CCAST personnel may transport critically ill or injured patients on RW aircraft when patient requirements so dictate, and it is necessary to save life or limb. Utilization of CCATT/CCASTs on RW aircraft must be approved through the command and control agency governing the team (usually the execution arm of the air mobility division). Because of space and weight limitations on some RW aircraft, it may be necessary to pare personnel and equipment.

Fixed Wing Operations

Most AE transfers are done using FW assets. Both CCATT and CCAST are well practiced using the C-17 Globemaster and C-130 Hercules airframes due to their size and capabilities. The C-130 is the most commonly used aircraft for TACEVAC. This aircraft is capable of operating from unimproved airfields and in hostile locations. It flies at 318 knots at 20,000 feet, with a maximum ceiling of 23,000 feet. The C-130 has the capacity for up to 74 litter patients, but does not have onboard oxygen systems, mandating that oxygen to be carried onboard as a portable liquid oxygen system or a compressed gas. The electrical system provides 400 Hz AC power through specially configured outlets, limiting its direct utility for medical devices. Therefore, CCATT/CCAST missions must rely on battery power, or power provided through an electrical converter, which limits the total amperage output for medical equipment use. Lighting and environmental control systems are minimal, requiring additional measures for patient warming and visualization for patient care. Lastly, access to patients is limited to 180 degrees.

The C-17 Globemaster III has the unique quality of being an excellent aircraft for both TACEVAC and STRATEVAC. It has a speed of 450 knots at an altitude of 28,000 feet, with an unrefueled range of 2,400 nautical miles and unlimited range with aerial refueling. This range makes it useful for transoceanic missions. The C-17 can also utilize small, unimproved airfields with runways as small as 3,500 feet long and 90 feet wide. The aircraft's interior is well lit and the system of litter stanchions provides 360-degree access to critical patients. The aircraft contains built-in systems that provide medical oxygen at 50 psi and 60 Hz AC electric power through standard US outlets. Currently the workhorse in patient transport, the C-17 can be rapidly configured from use as a cargo aircraft to accommodate 36 litter patients.

Other airframes are frequently used (particularly the KC-135 by CCATTs), depending on availability and service needs. CCASTs are able to use civilian airframes for certain missions. These aircraft may be specifically designed and modified for the purpose or may be adapted to accommodate CCAST patients.

FW operations provide the advantage of greater speed and comfort than RW assets, in addition to the vastly increased range of these aircraft. Although physiological considerations (particularly the effects of ascent to altitude) must be considered when using any form of air asset, these effects are seen more with FW than with RW aircraft due to their higher operating altitude. However, FW assets, unlike RW aircraft, can be pressurized, so some of these effects can be mitigated with appropriate planning and briefing of the aircrew during the mission planning phase.

Documentation

USAF Form 3899, *Aeromedical Evacuation Patient Record*, used to direct and record en-route care, should accompany each patient to ensure that care is appropriately documented during transport. The form also serves as the legal record of patient care throughout the AE system. Copies of patient medical documentation including operative reports should be provided to the CCATT team chief.

RAF Form 7526[6] is the equivalent form for STRATEVAC, and RAF Form 7527 is used for TACEVAC. Both types of CCAST use the Medical Form 152 prescription chart. All relevant documentation is provided to the CCAST team leader. Of particular importance is an electronic copy (usually on CD-ROM) of any patient imaging.

Resupply and Patient Movement Items

Patient movement items (PMIs) should either be carried by CCATTs or made available at the next AE staging point via the PMI system. Ideally, the originating facility will provide the medications and supplies the patient requires at time of transport. To ensure the provision of necessary care in case of a diversion to an area without medical assets, a 3-day supply should be provided for intra-theater movement and a 5-day supply for inter-theater movement. However, during wartime, these medication supply levels may be adjusted based on command directives. CCASTS require a 24-hours supply of medication for STRATEVAC unless a longer flight without opportunity for resupply is anticipated. TACEVAC requirements are dictated on a patient-by-patient basis according to anticipated duration of flight.

TRAINING AND RESEARCH

Air Publication 3394[6] documents the training requirements for all CCAST personnel, including professional qualifications, AE-specific qualifications, and equipment training requirements, which differ depending on each team member's profession. All CCAST personnel are expected to take an active part in audits and research, whether designing studies, collecting data, or analyzing data. Examples of areas of active research include studies of body areas at risk of pressure sores and endotracheal microaspiration (and the safety of feeding intubated patients in flight). Other recent studies include the change in the arterial concentration of oxygen compared with the inspired concentration (the P:F ratio) during flight. Laboratory research has been done on the effects of pressure changes due to altitude on the function of ventilators.

CCATT training consists of a 12-day initial course at Wright-Patterson Air Force Base and a 12-day advanced course at the Center for Sustainment of Trauma and Readiness Skills at the University of Cincinnati. Team members must revalidate with the advanced course every 2 years.

SUMMARY

The CCATT capability was developed to provide US Air Force AE with the intrinsic capability to transport stabilizing critically ill and injured casualties. This capability permits surgical teams to remain small and mobile enough to keep pace with the military operations they support and still provide advanced resuscitation. CCATT has allowed the resource-intensive burden of postresuscitation care to remain at more fixed facilities. Since the program's inception in 1994, CCATT has performed superbly in support of peacekeeping operations and sustained this performance in support of sustained combat operations lasting for more than 13 years and producing over 10,000 critical casualties.

This capability has also performed well in manmade and natural disaster response operations, helping to remove casualties who are both most vulnerable and consume the greatest quantity of resources from the disaster area. Similarly, CCAST provides a highly trained, mobile intensive care capability for the British armed forces and entitled civilians wherever they may be serving overseas. Both services can be rapidly mobilized and deployed anywhere in the world, using the latest advances in both equipment and air transport resources.

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Chapter 39

BASICS OF PEDIATRIC TRAUMA CRITICAL CARE MANAGEMENT

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INTRODUCTION

Children represent a particularly vulnerable segment of the population and have increasingly become more affected by armed conflict during the last century. As modern warfare technologies advance and armed conflict spreads, the lines between civilians and combatants have blurred such that children often become the victims of displacement, malnourishment, and sexual assault or are the direct targets of violence.[1] It is estimated that more than 2 million children worldwide died as a result of armed conflict during the past decade, and more than 6 million were permanently injured.[2] Review of recent military conflicts suggests that in Operations Enduring Freedom and Iraqi Freedom, more than 6,000 children presented to combat support hospitals by late 2009. Of these, more than 75% were admitted with traumatic injuries, primarily gunshot wounds and explosive injuries.[3] This chapter will highlight basic observations, understandings, and implications of pediatric critical care to better inform anesthesia providers of the principles of initial trauma resuscitation and stabilization of pediatric casualties.

RECEIVING THE PEDIATRIC CRITICAL CARE PATIENT

Recognizing the many anatomic, physiologic, and developmental differences between adult and pediatric trauma patients is fundamental to caring for children (Table 39-1). These unique characteristics factor significantly into the patterns and pathophysiology of injury and in a patient's response and recovery after trauma and illness. Compensatory mechanisms often obscure the true degree of illness, although both hypotension and bradycardia should be noted as late and ominous findings. Because of the wide variation in size and physiology across the spectrum of pediatric patients, clearly identifying the proper equipment is essential. Vital sign norms and preferred equipment sizes are often best interpreted by age (Table 39-2).

Developmentally appropriate behavior and curiosity make children more apt to explore their environments than adults. When combined with the inability to recognize threats, this curiosity may lead to disastrous outcomes. Psychological response to trauma and illness differ by age and developmental stage, which may require modification of often-used trauma assessment tools such as the Glasgow coma scale that are based on age and developmental stage (Table 39-3).

The handoff of care from the operative setting to the intensive care unit (ICU) represents a vulnerable period during which key information is exchanged that dictates future care needs (Exhibit 39-1). Teamwork and protocol-driven handoff techniques can minimize communication errors, errors of omission, technical errors, and duration of handoffs.[4,5] During the transition from the operative to postoperative environment, special attention should be given to preventing undue artificial airway manipulation and unintentional dislodgment of catheters or drainage devices. Once the patient is situated in the ICU and handoff has been completed, the intensivist should repeat a primary and limited secondary survey.[6] A chest radiograph should be obtained in intubated patients to establish location of the endotracheal tube as well as other indwelling devices. Selective repetition of blood gas tests, complete blood counts, coagulation studies, and chemistries should be considered, particularly if preoperative or intraoperative blood loss was estimated to be greater than complete blood volume. Nearly all medications are weight based; quickly estimating a pediatric patient's dosing weight is necessary to appropriately dose medications. In these instances, a length-based dosing tape, such as the Broselow Pediatric Emergency Tape (Armstrong Medical Industries, Lincolnshire, IL), can help provide a quick estimate of a patient's weight and absolute doses of resuscitation medications as well as estimates of appropriately sized resuscitation equipment.

PULMONARY SUPPORT AND MECHANICAL VENTILATION IN PEDIATRIC TRAUMA PATIENTS

Children may require mechanical ventilation and pulmonary support for a variety of reasons (Exhibit 39-2). Polytrauma, including intrathoracic injury, is also a common precipitant of respiratory distress and failure in pediatric patients; therefore, understanding basic therapeutic pediatric pulmonary strategies is crucial to minimizing morbidity and mortality.

Chest Trauma

Intrathoracic injury, primarily as a result of blunt injury, occurs in 4% to 6% of pediatric traumas.[3,7] Children tend to tolerate such severe intrathoracic injury poorly because of low functional residual capacity, greater oxygen consumption, and decreased pulmo-

TABLE 39-1

KEY ANATOMIC AND PHYSIOLOGIC DIFFERENCES BETWEEN INFANTS/CHILDREN AND ADULTS

Differences	Importance
General	
Vital signs age- and size-based	Normative values may vary drastically by age and size
Smaller total body mass but increased body surface area compared to mass	Less subcutaneous tissue and fat increases force per unit body area; vital organs are closer together; increased caloric, glycemic, and fluid needs; increased risk of environmental exposure and hypothermia
Proportionally larger heads	Increased risk of head trauma
Fontanelle closure delayed	Anterior fontanelle remains patent until 7–19 months and may assist with volume assessment
Skeletal calcification incomplete	Increased risk of solid organ injury from blunt trauma
Blood volume relatively greater per unit body mass	Increased risk of hypovolemia from seemingly small hemorrhages
Immature renal function	Impaired fluid and electrolyte regulation in infants
Respiratory	
Short neck and chin and larger tongues	Increased risk of upper airway obstruction
Anterior and cephalad larynx (C3/4); airway narrowest at cricoid cartilage	Infants require padding under torso to maintain airway and cervical spine in neutral ("sniffing") positioning
Shorter trachea (4–9 cm)	Increased predilection for mainstem intubation
Smaller diameter of conducting airways	Disproportionately increased peripheral airway resistance and airway obstruction with minimal edema
Increased alveolar minute ventilation; small thorax in relation to abdomen	Similar tidal volume per kilogram results in increased respiratory rates
Increased chest wall compliance; protuberant abdomen with weak musculature	Inefficient lung expansion during distress evidenced by subcostal retractions/abdominal breathing in infants; increased risk of functional residual capacity loss when sedated because of unopposed elastic recoil; potential for respiratory compromise secondary to abdominal distention
Immature central respiratory drive	Immature respiratory control predisposes neonates to apnea in response to hypoxia
Cardiovascular	
Neonatal myocardium relatively stiff	Relatively fixed cardiac stroke volume requires heart-rate elevation to increase cardiac output
Cardiac index increased 30%–60%	Required to meet high oxygen consumption
Sympathetic nervous system maturation delayed until 4–6 months; parasympathetic system mature at birth	High vagal tone and potent laryngeal reflex with apnea, bradycardia, and laryngospasm

nary but increased chest wall compliance. Pulmonary contusion is relatively common in pediatric thoracic trauma (48%) and it is generally well tolerated.[8] However, up to a fifth of pediatric patients with pulmonary contusions develop secondary complications, including aspiration, infection, and acute respiratory distress syndrome.[9] Bronchospasm and acute asthma exacerbations may occur subsequent to or independent of chest trauma and require unique diagnostic and therapeutic considerations (Figure 39-1).

TABLE 39–2

PEDIATRIC PARAMETERS AND EQUIPMENT

Age	Neonate	3 mo	6 mo	1 y	2 y	3 y	4 y	6 y	8 y	12 y	14 y
Wt (kg)	3.5	6	8	10	12	14	16	20	25	40	50
~ BSA (m^2)	0.24	0.34	0.42	0.49	0.56	0.62	0.68	0.79	0.92	1.3	1.5
HR	80–190	80–160	80–160	80–160	80–130	80–130	80–120	75–115	70–110	65–110	60–105
RR	30–50	24–38	24–38	22–30	22–30	22–30	20–24	20–24	18–24	16–22	14–20
SBP*	60–90	70–110	70–110	70–110	74–110	76–110	78–115	82–115	86–120	94–125	98–130
DBP	35–60	40–60	40–60	40–60	45–60	50–65	50–70	55–75	60–80	60–80	65–85
BP cuff	Neonate	Infant	Small child	Small child	Child	Child	Child	Small adult	Small adult	Adult	Adult
BVM	Infant	Infant	Child	Child	Child	Child	Child	Child	Child/ adult	Adult	Adult
Oral airway	Infant 50 mm	Small 60 mm	Small 60 mm	Small 60 mm	Child 70 mm	Child 70 mm	Med 80 mm	Med 90 mm	Med 90 mm	Large 100 mm	Large 100 mm
ETT blade	#0–1	#1	#1	#1	#2	#2	#2	#2	#2–3	#3	#3
ETT size†	2.5–3.5	3.5–4.0	3.5–4.0	4.0–4.5	4.0–4.5	4.5–5.0	4.5–5.0	5.0–5.5	5.5–6.5	6.0–7.0	7.0–8.0
Suction cath	6 Fr	8–10 Fr	8–10 Fr	8–10 Fr	10 Fr	10 Fr	10 Fr	10 Fr	10 Fr	12 Fr	14 Fr
NGT	5–8 Fr	5–8 Fr	8–10 Fr	8–10 Fr	10 Fr	10 Fr	10–12 Fr	12–14 Fr	14 Fr	14–18 Fr	14–18 Fr
Foley	6 Fr	8 Fr	8 Fr	8 Fr	8 Fr	8 Fr	8 Fr	10 Fr	12 Fr	14 Fr	14 Fr
IV access	22–24 g	22–24 g	20–24 g	20–24 g	18–22 g	18–22 g	18–22 g	18–20 g	18–20 g	16–20 g	16–20 g
Central line	4 Fr 8 cm	4 Fr 9 cm	4 Fr 12 cm	5 Fr 8 cm	5 Fr 8 cm	5 Fr 12 cm	5 Fr 12 cm	5 Fr 15 cm	5 Fr 15 cm	7 Fr 15 cm	7 Fr 15 cm

*Hypotension = systolic BP ≤ 70 + (2 × age in years over 1 year); < 1 mo SBP ≤ 60; 1 mo – 1 y SBP ≤ 70
†ETT size = [age (years) + 16]/4; use cuffed tube for ≥ 6.0; ETT depth = 3 × ETT internal diameter or (age in years/2) + 12
BP: blood pressure
BSA: body surface area
BVM: bag-valve mask
cath: catheter
DBP: diastolic blood pressure
ETT: endotracheal tube
Fr: French
HR: heart rate
IV: intravenous
NGT: nasogastric tube
RR: respiratory rate
SBP: systolic blood pressure
Wt: weight

Airway Equipment

Pediatric intubation should follow a routine stepwise algorithm (Figure 39-2). Equipment sized for pediatric patients is necessary to safely deliver respiratory support. Appropriately sized bag-valve masks (BVMs) are necessary to administer the proper tidal volumes of positive-pressure ventilation. BVMs with inappropriately small bags pose the risk of insufficient ventilation, whereas use of excessively large BVMs risks gastric distension and barotrauma. If only adult-sized bags are available, the operator must closely observe chest wall motion to gauge appropriate ventilation. Endotracheal tube internal diameter (ID) can be quickly sized with the following formula:

$$\text{endotracheal size (mm)} \frac{16 + \text{age (years)}}{4}$$

This size roughly correlates to the child's fifth finger (the "rule of pinky"). Depth of insertion in centimeters

TABLE 39-3

MODIFIED GLASGOW COMA SCALE

Activity	Infant	Child/Adult	Score
Eye opening	Spontaneous	Spontaneous	4
	To speech	To speech	3
	To pain only	To pain only	2
	No response	No response	1
Verbal response	Coos and babbles	Oriented, appropriate	5
	Irritable cries	Confused	4
	Cries to pain	Inappropriate words	3
	Moans to pain	Incomprehensible sounds	2
	No response	No response	1
Motor response	Moves spontaneously and purposefully	Obeys commands	6
	Withdraws to touch	Localizes painful stimulus	5
	Withdraws to pain	Withdraws in response to pain	4
	Abnormal flexion posture to pain	Flexion in response to pain	3
	Abnormal extension posture to pain	Extension in response to pain	2
	No response	No response	1

EXHIBIT 39-1

ANESTHESIA HANDOFF CHECKLIST

Patient Details
- Name
- Age
- Weight (kg)
- Preoperative diagnosis
- Allergies

Operative Course
- Operation performed
- Anesthesia technique
- Airway classification
- Endotracheal tube and laryngoscope sizes
- Access (type, location, size, placed by)
- Tubes/drains (type, location)
- Problems in OR
- Bleeding issues (preoperative hemoglobin, estimated blood loss [total and mL/kg])
- Blood products given (totals, types, last hemoglobin)
- Crystalloid given
- Urine output

Present Status
- Hemodynamics (rhythm, HR, BP, MAP, CVP, NIRS)
- Ventilation (settings, difficulties, iNO, blood gases)
- Infusions (vasopressors)
- Antibiotics (total dose, last dose)
- Opiates (total dose, last dose)
- Neuromuscular blockade (last dose, reversal)

BP: blood pressure; CVP: central venous pressure; HR: heart rate; iNO: inhaled nitrous oxide; MAP: mean arterial pressure NIRS: near-infrared spectroscopy; OR: operating room

Data sources: (1) Joy BF, Elliott E, Hardy C, Sullivan C, Backer CL, Kane JM. Standardized multidisciplinary protocol improves handover of cardiac surgery patients to the intensive care unit. *Pediatr Crit Care Med*. 2011;12(3):304–308. (2) Catchpole KR, de Leval MR, McEwan A, et al. Patient handover from surgery to intensive care: using Formula 1 pit-stop and aviation models to improve safety and quality. *Paediatr Anaesth*. 2007;17(5):470–478.

when placed orally is estimated by either of the following two rules:

$$\text{endotracheal tube depth (cm)} = (3 \times \text{ID})$$

$$\text{endotracheal tube depth (cm)} = \frac{\text{age (years)}}{2} + 12$$

Placement should be verified by auscultation, end tidal carbon dioxide detection, and radiograph. Given their smaller anatomy, children are at greater risk of mainstem intubation. Cuffed endotracheal tubes are typically half a size lower than that calculated. Cuffed endotracheal tubes are safe and preferred over uncuffed endotracheal tubes in children with significant lung disease; when used appropriately, they can minimize ventilator leak and aid in achieving goal tidal volumes. Cuff pressures should be monitored and kept below 20 cm H_2O to avoid mucosal damage, scarring, and subglottic stenosis.

Many adult-type ventilators can be adapted for use in children, provided appropriately sized circuits are used; however, the inconsistent delivery of appropriate tidal volumes at variable peak end expiratory pressures, coupled with inadequate safety alarms, warrants careful review of individual ventilator capabilities and capacities prior to use.[10] For example, the simplified automated ventilator (SAVe) has been used successfully in adult trauma patients; however, the preset factory

EXHIBIT 39-2

INITIAL INTUBATION INDICATIONS FOR MECHANICAL VENTILATION

Cardiorespiratory failure
- Cardiopulmonary benefit (shock, cardiopulmonary resuscitation)
- Inability to oxygenate in the absence of cyanotic heart disease
- Inability to ventilate, acute and unresponsive to intervention
- Neuromuscular weakness (negative inspiratory force > - 20 cm H_2O)

Compromised airway, actual or anticipated
- Absent airway protective reflexes (cough and gag)
- Aspiration of oral secretions
- Complete airway obstruction
- Glasgow coma score ≤ 8

Additional considerations
- Diagnostic or therapeutic intervention (ie, intracranial hypertension)
- Emergency drug administration
- Pulmonary toilet
- Residual anesthetic effect
- Transport stability

Data source: Thompson AE. Pediatric airway management. In: Fuhrman BP, Zimmerman J, eds. *Pediatric Critical Care*. 3rd ed. Philadelphia, PA: Mosby; 2006.

settings do not deliver peak end expiratory pressure and are not adjustable.[11] Therefore, the SAVe can result in significant ventilator-induced injury in children.

Ventilatory Management Techniques

Once the decision has been made to provide invasive ventilation, regardless of cause, ongoing mechanical ventilation strategies vary based on the severity of lung disease and coexisting conditions such as intracranial hypertension or cardiac dysfunction. Pressure ventilation is typically selected in patients with lung disease to achieve goal tidal volumes at lower pressures. Synchronized intermittent mandatory ventilation mode with pressure control and pressure support is well tolerated by pediatric patients. Typical starting settings will vary based on individual pathology, but basic initial settings are given in Table 39-4. In certain circumstances, such as cardiac disease and significant restrictive lung disease, a lower peak end expiratory pressure of 3 to 4 cm H_2O may be optimal. General criteria for the extubation of a pediatric patient following surgery are presented in Exhibit 39-3.

As with any mode of ventilation, a number of monitoring parameters are recommended to safely deliver mechanical ventilation and minimize barotrauma, atelectrauma, and volutrauma. These include inline capnography, chest radiography, blood-gas sampling (via arterial line), and peak and mean airway pressures. In the absence of acute lung injury, goals of normocapnia and partial pressure of oxygen in arterial blood are 70 to 80 mm Hg (SpO_2 90%–100%). In the presence of acute lung injury or acute respiratory distress syndrome, management strategies are similar to adult ARDSNet (www.ardsnet.org) strategies targeting permissive hypercapnia and low-tidal volumes[12] (Exhibit 39-4). However, pediatric data are insufficient to completely recommend all-adult protocols. The calculation of an oxygenation index (OI) and PaO_2 to FiO_2 (P/F) ratio may also be helpful, where the OI is defined as:

$$OI = \frac{FiO_2 \times 100 \times MAP}{PaO_2}$$

where FiO_2 is the fraction inspired oxygen, MAP is the mean airway pressure, and PaO_2 is the partial pressure of oxygen. Generally, an OI greater than or equal to 40 and a P/F ratio under 100 are suggestive of failed conventional ventilation.

Figure 39-1 (facing page). Acute management of asthma exacerbation algorithm.
Data source: Gorelick MH, Stevens MW, Schultz TR, Scribano PV. Performance of a novel clinical score, the Pediatric Asthma Severity Score (PASS), in the evaluation of acute asthma. *Acad Emerg Med*. 2004;11(1):10–18.

Airway - Breathing – Circulation
Continuously monitor vital signs and pulse oximetry
Administer supplemental oxygen to maintain $SpO_2 \geq 92\%$
Obtain brief history (prior admissions, ICU admits, intubations?)
Conduct focused physical exam (respiratory rate, work of breathing, auscultation, PEF)

↓

Acute Asthma Category:			
Clinical Finding	**Mild**	**Moderate**	**Severe**
Wheezing	None or mild	Moderate	Severe or absent
Air Entry	Good	Fair	Poor or absent
Work of Breathing	Mild	Moderate	Severe
Expiratory Prolongation	Normal or mild	Moderate	Severe
Tachypnea (above mean)	30 %	30 – 50%	> 50% or slow
Mental Status	Normal	Agitated	Drowsy
PEF	> 70%	40 – 69 %	< 40%

Moderate to Severe? ↓ **Yes**

Albuterol 2.5 – 5 mg INH q 20 minutes x 3
AND
Methylprednisolone 2 mg/kg/dose (load) IV/IM x 1 (max dose 80 mg)
AND
Ipratroprium 0.25 – 0.5 mg INH q 6 hours

↓

Continuous nebulized albuterol: 0.5 mg/kg/hour (usual max 20 mg/hr)
(< 7.5 kg: 2.5 mg/hour INH; 7.5 – 14.9 kg: 5 mg/hour INH;
15 – 29.9 kg: 10 mg/hour INH; > 30 kg: 20 mg/hour INH)

↓

Magnesium sulfate 75 mg/kg IV x 1 over 20 minutes (max 2000 mg), monitor for hypotension

↓

Terbutaline 10 mcg/kg/dose (load) IV x 1 over 30 minutes,
followed by and infusion at 0.4 – 6 mcg/kg/min IV
→ Monitor EKG q 24 hours and cardiac enzymes q 6 – 12 hours

↓

Heliox 70/30, monitor for hypoxia

↓

Consider **CPAP/BiPAP**, may use ketamine

↓

Consider **intubation, mechanical ventilation and inhalational anesthetics** as a last resort;
Initial settings: TV 8-12 mL/kg (peak pressure < 45 cmH_2O), low PEEP, RR 6 – 12, I_{time} 1-1.5s
(I:E 1:4 – 1:6); Allow permissive hypercapnia; Goal: spontaneous / pressure supported mode

BiPAP: bilevel positive airway pressure
CPAP: continuous positive airway pressure
EKG: electrocardiogram
INH: inhaled
IV: intravenous
PEEP: positive end-expiratory pressure
PEF: peak expiratory flow
PR: per rectum
TV: tidal volume

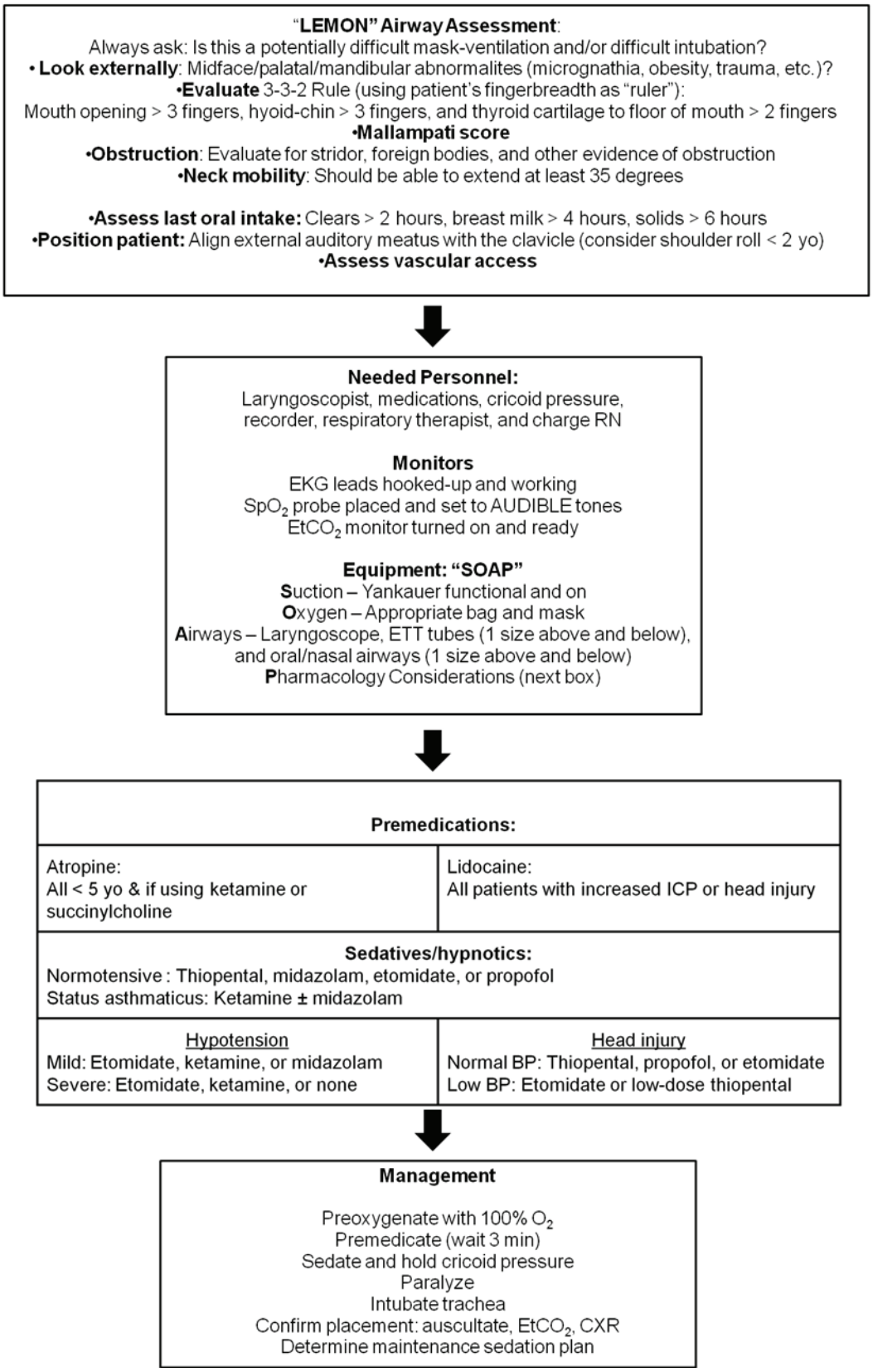


Figure 39-2. Intubation management algorithm. BP: blood pressure. CXR: chest x-ray; EKG: electrocardiogram; ETT: endotracheal tube; ICP: intracranial pressure; RN: registered nurse

TABLE 39-4

INITIAL PEDIATRIC VENTILATOR SETTINGS*

Parameter	Setting
PIP	20 cm H_2O
TV	6–10 mL/kg†
PEEP	5 cm H_2O
I_t	0.3–1.2 sec‡
Rate	Age appropriate§
PS	10 cm H_2O
FiO_2	Begin at 1.0; rapidly wean to < 0.6

*Preferred mode is synchronized intermittent mandatory ventilation using either pressure or volume control.
†Decrease TV if measured PIPs > 30–35 or if there is excessive chest rise.
‡Inspiratory time should be no less down than 0.3 seconds for infants and up to 1.2 seconds for adolescents.
§Begin with a rate of 30 for infants to 15 for adult-sized adolescents.
FiO_2: fraction of inspired oxygen
I_t: inspiratory time
PEEP: peak end expiratory pressure
PIP: peak inspiratory pressure
PS: pressure support
TV: tidal volume

EXHIBIT 39-3

RECOMMENDED EXTUBATION CRITERIA*

- Resolution of etiology of respiratory failure
- Oxygen saturation adequate with $FiO_2 \leq 0.4$
- PEEP ≤ 5 cm H_2O
- PIP ≤ 25 cm H_2O
- pH > 7.35
- $PaCO_2$ < 60 mm Hg
- Stable hemodynamics (heart rate and blood pressure normal for age)
- Adequate hemostasis
- Spontaneously breathing
- Airway protective reflexes intact
- Easily arousable to verbal stimuli/light touch

Consider spontaneous breathing trial with minimal pressure support for 30 minutes to 2 hours prior to extubation. Minimal pressure support is determined to overcome the resistance of the endotracheal tube (eg, for endotracheal tube size 3.0 to 3.5 mm, use 10 cm H_2O pressure support; for 4.0 to 4.5 mm, 8 cm H_2O; for > 5.0 mm, use 6 cm H_2O).
FiO_2: fraction of inspired oxygen
$PaCO_2$: partial pressure of carbon dioxide in blood
PEEP: positive end-expiratory pressure
PIP: positive inspiratory pressure
pH: percent of hydrogen

If peak pressures cannot be maintained below 30 to 35 cm H_2O, alternative ventilation strategies should be considered, namely high-frequency oscillatory ventilation, to avoid the risk of further barotrauma. Extracorporeal membrane oxygenation is typically used as rescue therapy for failed conventional and high-frequency ventilation at some medical facilities. The use of this modality as rescue therapy in field military medicine is still in its infancy and, thus, unlikely to be a modality offered to children in theater.

Pediatric ventilator-associated pneumonia has been independently linked with longer duration of ventilation, use of gastric tubes, and sedation or analgesia, and is the second most common nosocomial infection and most common cause for antibiotics in the pediatric ICU (PICU).[13,14] A ventilator-associated pneumonia prevention bundle has been shown in children and adults to decrease the likelihood of ventilator-associated pneumonia (Exhibit 39-5).[15,16] Appropriate sedation and analgesia with daily wake-up tests, as hemodynamics allow, are also useful in decreasing the number of days a patient remains on a ventilator and reducing the risk of ventilator-associated pneumonia.

PEDIATRIC HEMODYNAMIC PRINCIPLES IN TRAUMA

Shock in a patient of any age is characterized by compromised tissue oxygenation, substrate delivery, and metabolite removal. Primary etiologies can be hypovolemic, distributive, and cardiogenic, although hypovolemia from hemorrhage is more likely in early trauma critical care, including during the postoperative period. Shock is a life-threatening emergency that must be quickly recognized and treated. Shock may be differentiated into compensated, decompensated, and irreversible. Early compensated shock is clinically recognizable and characterized by tachypnea, tachycardia, altered mental status, hypothermia or hyperthermia, cool extremities (cold shock), or peripheral vasodilation (warm shock) and decreased urine output in conjunction with progressive metabolic acidosis and rising serum lactate levels. This phase of shock is most optimal for response to rescue therapy. Hypotension is the defining characteristic of decompensated shock and is a late finding in shock, evident only once 15% to 20% blood loss has occurred. In pediatric patients, hypotension is defined as a systolic blood pressure of less than the fifth percentile for age. Blood pressure for

EXHIBIT 39-4

RECOMMENDATIONS FOR THE MANAGEMENT OF PEDIATRIC ALI AND ARDS

Recommended

- Avoid hypoglycemia and hyperglycemia
- Avoid tidal volumes ≥ 10 mL/kg
- Hemoglobin target ≥ 10 g/dL, if unstable
- Hemoglobin target ≥ 7 g/dL, if stable
- Keep plateau pressures < 30 cm H_2O
- PaO_2 goal 60–80 mm Hg (SpO_2 ≥ 90%)
- pH goal 7.3–7.45
- Sedation and analgesia
- Include stress-ulcer prophylaxis

Consider

- 4–6 mL/kg tidal volume protocol
- Corticosteroids for lung inflammation
- Endotracheal surfactant
- Extubation readiness testing
- Noninvasive lung ventilation (ie, BiPAP)
- Restrictive fluid management

Not Recommended

- High-flow nasal cannula
- Inhaled bronchodilators
- Inhaled nitric oxide
- Prone positioning
- Tight glycemic control

ALI: account lung injury
ARDS: acute respiratory distress syndrome
PaO_2: partial pressure of oxygen in blood
SpO_2: saturation of peripheral oxygen
Data source: Randolph AG. Management of acute lung injury and acute respiratory distress syndrome in children. *Crit Care Med*. 2009;37(8):2448–2454.

EXHIBIT 39-5

KEY ELEMENTS OF A RECOMMENDED PEDIATRIC VENTILATOR-ASSOCIATED PNEUMONIA BUNDLE

- Perform routine oral care every 4 hours
- Rinse oral suction devices following use
- Store oral suction devices in non-sealed plastic bags at bedside when not in use
- Wash hands before and after contact with ventilator circuits
- Drain condensate from ventilator circuit at least every 2–4 hours
- Change ventilator circuits and inline suction catheters only when visibly soiled
- Elevate head of bed to 30°–45° unless contraindicated
- Always drain ventilator circuit prior to patient repositioning
- For patients > 12 years of age, use a cuffed endotracheal tube with dorsal lumen above the cuff to help keep secretions off of the cuff
- Always wear a gown before providing patient care when soiling from respiratory secretions is expected

Data source: Bigham MT, Amato R, Bondurrant P, et al. Ventilator-associated pneumonia in the pediatric intensive care unit: characterizing the problem and implementing a sustainable solution. *J Pediatr.* 2009;154(4):582–587.e2. Epub 2008 Dec 3.

children 1 to 10 years of age is calculated as:

systolic blood pressure (mm Hg) = 70 + (2 × age in years)

Hypotension is blood pressure lower than this calculated value. Early goal-directed therapy, similar to that practiced with adults, is the preferred therapeutic management strategy for shock and has been associated with improved outcomes among pediatric patients.[17–19]

Vascular Access

Vascular access is essential for rapidly correcting shock states. Large-bore, peripheral intravenous (IV) access may not be feasible if significant volume loss has occurred. As a general rule, if IV placement is unsuccessful after three attempts or 90 seconds, intraosseus (IO) needle placement is recommended for rapid fluid resuscitation. The preferred insertion site for IO access is the anteromedial aspect of the tibia 1 to 2 finger breadths below the tibial tuberosity, taking care to avoid the physeal growth plate (Figure 39-3). This site may generally be used up to 6 to 8 years of age. Alternate sites include the distal femur, sternum, lateral and medial malleoli, iliac crest, and proximal humerus. IO needles should not be placed distal to an injury site. Subsequent attempts at IO needle placement may be made in the same limb, provided each attempt is made proximal to the last attempt. Styleted bone marrow biopsy needles (16 and 18 gauge) may be placed manually; however, the EZ-IO (Vidacare, Shavano Park, TX) and the Bone Injection Gun (WaisMed, Houston, TX) offer automated alternatives with options for pediatric-appropriate needle size and depths (Figures 39-4 and 39-5). The commonly available FAST1 IO (Pyng Medical, Vancouver, Canada) should not to be used in children younger than 12 years

Figure 39-3. Intraosseous insertion site in children.
Reproduced with permission from: Vidacare, Shavano Park, TX. Copyright 2012 Vidacare.

of age because the length of the catheter could traverse the sternal cartilage and enter the mediastinum. If marrow is available by aspiration, blood laboratory evaluation may be done. Like a central venous catheter, an IO needle can be used to deliver medications and fluids. Once placed, an IO needle should be removed after 24 hours to avoid infection. After a period of

Figure 39-4. Intraosseous insertion device for pediatric patients: The EZ–IO.
Reproduced with permission from: Vidacare, Shavano Park, TX. Copyright 2012 Vidacare.

Figure 39-5. Intraosseous insertion device for pediatric patients: the pediatric bone injection gun (BIG). Reproduced with permission from: WaisMed, Houston, TX.

TABLE 39-5

VASOACTIVE AGENTS USED IN PEDIATRICS

Drug	Dose
Dobutamine	2–20 μg/kg/min IV/IO
Dopamine	2–20 μg/kg/min IV/IO; begin 5 μg/kg/min
Epinephrine	0.03–1 μg/kg/min IV/IO
Milrinone	Infusion: 0.25–1 μg/kg/min IV/IO
Norepinephrine	0.05–1 μg/kg/min IV/IO
Phenylephrine	0.1–4 μg/kg/min IV/IO
Vasopressin	0.3–2 mU/kg/min (18–120 mU/kg/h) IV/IO

IO: intraosseous; IV: intravenous

fluid resuscitation and relative hemodynamic stability, longer-term access methods, such as placement of a central venous catheter, can be used.

Resuscitation

Resuscitation fluids of choice are isotonic crystalloids, either lactated Ringer or 0.9% normal saline (NS). Both are administered in 20 mL/kg rapid IV boluses. In small infants, this may amount to administering prepackaged 10-mL NS IV flushes for ease of delivery. If there is ongoing blood loss, packed red blood cells in a similar amount may be substituted. If no physiologic response has been observed after a total of 60 mL/kg (three 20-mL/kg boluses) have been administered over 15 to 20 minutes, a vasopressor is indicated for additional hemodynamic support (Table 39-5). Typical vasopressors include dopamine, norepinephrine for warm shock, and epinephrine for cold shock. Vasopressor support via a peripheral IV is generally safe prior to obtaining central venous access, provided the site is monitored for distal perfusion.

Beyond heart rate and blood pressure, central venous pressure, differential skin temperature, and capillary refill (normally less than 2 seconds) are useful clinical measures for evaluating therapy response. Serial laboratory assessment monitoring, including serum lactate levels, blood gas determination, and mixed central venous oxygen saturation measurement further augment characterization of the resuscitation response. Hydrocortisone therapy should be considered for patients with catecholamine-resistant shock at risk of absolute adrenal insufficiency. Broad-spectrum antibiotics should be administered during the first hour of resuscitation for all patients presenting with septic shock, though the routine use of antibiotics in burn patients presenting in shock is discouraged because it promotes antimicrobial resistance. Ideally, blood cultures should be obtained and sent to a laboratory before antibiotics are administered.

POSTOPERATIVE FLUID MANAGEMENT AND NUTRITION

Maintenance Fluids and Electrolytes

The classic teaching in pediatric fluid management emphasizes meticulous attention to balancing inputs and outputs. A child's postoperative hourly fluid requirements are based on weight (Table 39-6). Fluid selection is based on the dextrose and sodium needs of the child. Children have fewer glycogen stores than adults and typically need additional dextrose to maintain a supply to their glucose-dependent organs. This is usually achieved by providing 10% dextrose to infants less than 1 year of age and 5% dextrose to children older than 1 year, in addition to the requisite electrolytes, at a maintenance rate. Typical daily sodium requirements in children range from 2 to 5 mEq/kg/day, but may increase due to ongoing losses. Though either lactated Ringer's or 0.9% NS alone are used as isotonic crystalloid for fluid resuscitation, 0.45% NS is usually preferred as the postoperative maintenance fluid in conjunction with either 5% or 10% dextrose for pediatric patients. Potassium chloride is typically added to saline-containing IV fluids, provided there is evidence of adequate renal function (ie, urine output). To meet the daily need of 2 to 3 mEq/kg/day, a standard 20 to 40 mEq/L is generally added, though caution must be taken in severely burned or crushed children.

TABLE 39-6

MAINTENANCE FLUID REQUIREMENTS FOR INFANTS AND CHILDREN

Based on Holliday-Segar Formula	
Weight	**Daily Total Volume**
0–10 kg	100 mL/kg
11–20 kg	1,000 mL plus 50 mL for each kg over 10 kg
≥ 20 kg	1,500 mL plus 20 mL for each kg over 20 kg
Examples: for 15 kg, 1,250 mL/24 h; for 25 kg, 1,600 mL/24 h	
Based on "4-2-1" Rule	
Weight	**Hourly Rate**
0–10 kg	4 mL/kg/h
11–20 kg	40 mL/h plus 2 mL/h for each kg over 10 kg
≥ 20 kg	60 mL/h plus 1 mL/h for each kg over 20 kg
Examples: for 15 kg, 50 mL/h; for 25 kg, 65 mL/h	

Sodium and Fluid Disturbances

Typically, adequate urine output is considered to be more than 1 mL/kg/h, though in fluid-restricted states, smaller output can be expected. An indwelling urinary catheter during the acute phase of illness can be instrumental in accurately measuring urine output. Weighing diapers is another method of estimating urine output in children, though this can be compli-

cated by the addition of stool in the diaper. Given a significant amount of stress, one should expect an antidiuretic hormone surge and a state of relative antidiuresis during the first 24 to 48 hours following trauma, operation, or onset of a critical illness. Decreased urine output must be considered in the context of the child's hemodynamics and physical examination to avoid unnecessary fluid overload. Early clues to compromised preload include decreased perfusion, altered mental status, and elevated heart rate. Laboratory data, such as serum lactate and bicarbonate, may help as well. Sustained urine output in excess of 4 mL/kg/h with rising serum sodium may indicate diabetes insipidus. Elevated urine output with low or normal serum sodium may also be indicative of cerebral salt wasting in children with traumatic brain injury. Brisk urine output can also be seen in healthy patients after receiving significant amounts of fluid resuscitation or after the period of antidiuresis has resolved.

Nutrition

Appropriate nutrition is necessary to prevent catabolism and encourage wound healing. Critical illness results in a neuroendocrine stress response. In general, a critically ill child receiving mechanical ventilation will need fewer calories than an active child, though the hypermetabolic state may lead to increased protein need (Table 39-7).

Early enteral nutrition may decrease ICU length of stay and promote healing while addressing nutritional energy deficit.[20–22] Trophic feeds (1–5 mL/h) may also assist in the preservation of gut function, preferably postpyloric if intubated versus nasogastric feeds.[23,24]

TABLE 39-7

MACRONUTRIENT REQUIREMENTS AND DISTRIBUTIONS*

		Fat							
Age	Total Water (L/day)	Total (g/day)	n-6 Polyunsaturated Fatty Acids Linoleic acid (g/day)	n-3 Polyunsaturated Fatty Acids α–linolenic acid (g/day)	Protein (g/day)	Carbohydrates (g/day)	Carbohydrates (% total kcal)	Protein (% total kcal)	Fat (% total kcal)
0–6 mo	0.7	31	4.4	0.5	9.1	60	ND	ND	ND
7–12 mo	0.8	30	4.6	0.5	11	95	ND	ND	ND
1–3 y	1.3	ND	7	0.7	13	130	45–65	5–20	30–40
4–8 y	1.7	ND	10	0.9	19	130	45–65	10–30	25–35
9–13 y	2.1 (female), 2.4 (male)	ND	10 (female), 12 (male)	1 (female), 1.2 (male)	34	130	45–65	10–35	20–35
14–18 y	2.3 (female), 3.3 (male)	ND	11 (female), 16 (male)	1.1 (female), 1.6 (male)	46 (female), 52 (male)	130	45–65	10–35	20–35
Adults	2.7 (female), 3.7 (male)	ND	12 (female), 17 (male)	1.1 (female), 1.6 (male)	46 (female), 56 (male	130	45–65	10–35	20–35

*Estimated energy requirement for critically ill patients is unlikely to be significantly more than the basal energy expenditure (BEE). As such, the following equations can be utilized to estimate energy requirement:
For boys: BEE (kcal/d) = 68 – [43.3 × age (yr)] + [712 × height (m)] + [19.2 ×weight (kg)]
For girls: BEE (kcal/d) = 189 – [17.6 × age (yr)] + [625 × height (m)] + [7.9 x weight (kg)]
When insufficient data is available to develop recommended daily allowance (RDA), both RDAs and estimated adequate intake are used for individual intake goals.
ND: not determined
Data sources: (1) Institute of Medicine (US) Panel on Macronutrients; Institute of Medicine (US) Standing Committee on the Scientific Evaluation of Dietary Reference Intakes. *Dietary Reference Intakes for Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein, and Amino Acids*. Washington, DC: National Academies Press; 2005. (2) Otten JJ, Hellwig JP, Meyers LD, eds. *Dietary Reference Intakes: The Essential Guide to Nutrient Requirements*. Washington, DC: National Academies Press; 2006.

Radiographic verification of feeding-tube placement prior to use is generally recommended given the known risk of malpositioning and complication.[25] Goal rate for continuous enteral feeds depends upon the desired caloric need and fluid volume (Table 39-8). Although postpyloric feeds must be administered on a continuous basis, for infants and young children gastric feeds may be condensed to six to eight feeds per day as tolerated. For older children, feeds may be further condensed to three to six per day. Overfeeding, particularly in the form of excess carbohydrates, can create additional carbon dioxide and pose ventilatory difficulties.

Most commercially available infant formulas were developed to mimic human breast milk. In infants with lactating mothers, human breast milk (20 kcal/oz) is the preferred option, provided the infant is able to latch appropriately onto the breast or a breast pump is available so the mother can express breast milk. Other enteral formulas vary in composition depending on the age of the patient. Full-term infant formulas typically consist of 20 kcal/oz and are acceptable for use in children up to 1 year of age. Premature infant formulas consist of 22 to 24 kcal/oz and are also used in children up to 1 year of age. Pediatric formulas are indicated for children 1 to 10 years of age and typically have 30 kcal/oz. Adult-type formulas (eg, Boost [Nestle, Vevey, Switzerland]; Ensure, Promote, Osmolite [all made by Abbott Nutrition, Columbus, OH]) typically have between 30 and 60 kcal/oz. Standard adult formulations may be used in children older than 1 year to meet minimum caloric intake, though renal function must be closely monitored because the protein load is 1.5 to 2 times that of pediatric products. If a powder formulation is available, ensure the formula is prepared according to published guidelines with potable water.

TABLE 39-8

GUIDELINES FOR INITIATING AND ADVANCING CONTINUOUS ENTERAL FEEDING*

Age (y)	Initial Infusion	Incremental Advances
0–1	1–2 mL/kg/h	10–20 mL/kg/day
1–6	1 mL/kg/h	1 mL/kg q 2–8 h
> 7	10–25 mL/h	20–25 mL q 2–8 h

*Hourly infusion increases incrementally until goal calories are achieved.

Reproduced from: Fuenfer MM, Creamer KM, eds. *Pediatric Surgery and Medicine for Hostile Environments*. Washington, DC: Borden Institute; 2011.

Parenteral nutrition may or may not be available (based on resource availability) and requires staff skilled in the sterile and accurate preparation, administration, and monitoring of total parenteral nutrition. Depending on patient acuity, parenteral nutrition may be the only method possible to deliver nutrition (Tables 39-9 and 39-10). Depending on ongoing losses from illness or injury, daily requirements may be higher. The addition of multivitamins and trace elements, such as zinc, copper, manganese, chromium, and selenium, is also essential for wound healing. In the setting of cholestasis, decrease the amount of copper by 50% and discontinue manganese. Patients with renal insufficiency should also be given limited amounts of chromium and selenium. Consider daily electrolyte monitoring to adjust electrolytes.

TABLE 39-9

INITIATION AND ADVANCEMENT OF PARENTERAL NUTRITION*

Dose	Glucose infusion rate (mg/kg/min)	Dextrose (%)	Protein (g/kg/day)	Fat† (g/kg/day)
Initial	5–8 (neonate–child), 3–5 (adolescent)	5 (neonate), 10 (infant and older)	2.5 (neonate), 1.5 (infant and older)	1
Advance	1–3	2.5 (neonate), 5–10 (infant and older)	0.5 (neonate), 1 (infant and older)	1
Maximum	11–12 (neonate–child), 5–8 (adolescent)	12.5% peripheral, 25% central	3.5 (neonate), 2 (infant and older)	3–4 (neonate), 2–4 (infant and older)

*Recommended osmolarity should not exceed 900–1,050 Osm/L.

†Parenteral lipid emulsion should be run over 24 hours; 20% concentration is preferred.

Data source: Freeman BK, Hampsey J. Nutrition and Growth. In: Tschudy MM, Arcara KM, eds. *The Harriet Lane Handbook: A Manual for Pediatric House Officers*. 19th ed. Philadelphia, PA: Mosby; 2012.

TABLE 39-10

RECOMMENDATIONS FOR PARENTERAL NUTRITION COMPONENTS*

Component	Infants & Toddler	Children	Adolescents
Sodium	2–4 mEq/kg/day	2–4 mEq/kg/day	60–150 mEq/day
Potassium	2–4 mEq/kg/day	2–4 mEq/kg/day	70–180 mEq/day
Calcium (20 mg/mEq)	0.45–4 mEq/kg/day	0.45–3.15 mEq/kg/day	10–40 mEq/day
Phosphorous (31 mg/mmol)	0.5–2 mmol/kg/day	0.5–2 mmol/kg/day	9–30 mmol/day
Magnesium (125 mg/mEq)	0.25–1 mEq/kg/day	0.25–1 mEq/kg/day	8–32 mEq/day
Multivitamin	5 mL/day, pediatric	5 mL/day, pediatric	10 mL/day, adult
Trace elements†	0.2 mL/kg/day	0.2 mL/kg/day	5 mL/day

*Consider adding acetate (1–2 mEq/kg/day) if serum bicarbonate is less than 20 or chloride is greater than 115. For parenteral nutrition given via central venous catheter, use heparinization (0.25–0.5 units/mL).
†Trace elements include zinc, copper, manganese, and chromium.
Data source: Parenteral nutrition. In: Kleinman, RE, ed. *Pediatric Nutrition Handbook*. 6th ed. Elk Grove Village, IL: American Academy of Pediatrics; 2009.

Refeeding Syndrome

In chronically malnourished children, rapid initiation of full feeds (either total parenteral nutrition or enteral nutrition) can result in a life-threatening depletion of phosphorous, magnesium, and potassium, leading to cardiac arrhythmias, cardiac arrest, and even death. Rapid glucose initiation can also up-regulate insulin secretion, precipitating hypoglycemia. To initiate nutrition in chronically malnourished children, administer 25% to 50% of estimated caloric needs on days 1 and 2 and increase by 20% each day until the patient has attained goal feeds by day 4 or 5. Monitor serum electrolytes and glucose and replace accordingly. Daily weight checks and weekly serum albumin, prealbumin, and triglycerides are useful in monitoring progress.

PEDIATRIC PAIN AND SEDATION MANAGEMENT

Critically ill children who experience pain often have real and underestimated psychological and physiological responses. Therefore, pain is rightfully recognized as the fifth vital sign, and pain relief is a basic right of all patients. The aims of sedation and analgesia, particularly in a mechanically ventilated child, are manifold: to compassionately provide comfort to injured and ill children, facilitate compliance and tolerance of routine care that can be noxious at times, and preserve calories for the healing process. Three primary approaches have been used to measure pain in pediatric patients: (1) self-report, (2) observational, and (3) physiological.[26] The Faces Pain Scale (revised) is an easy-to-use visual identification scale for self-reporting pain in patients over the age of 4 years.[27,28] In the case of comatose or preverbal children, self-reported pain intensity measures have limited utility. The CRIES neonatal pain score or FLACC score may be of greater utility for these patients.[29] When monitoring and titrating sedation, the State Behavioral Scale, COMFORT-Behavioral Scale, or Ramsay Score are also commonly used.[30–33]

Many agents used for sedation and analgesia in adults can be used safely in children; however, most have never been studied directly in pediatric patients. Propofol is a classic example of how altered patient-drug interactions may differ in children. Although relatively safe for short-term procedural use, prolonged use (greater than 12 hours) of propofol in children has been associated with propofol infusion syndrome, which is characterized by intractable metabolic acidosis, rhabdomyolysis, renal failure, cardiac failure, and death.

Adequate analgesia is often best offered through strategies focused on prevention, with the integration of preemptive and multimodal therapies.[29] Non-opioid and opioid-derived analgesics remain an integral component of any strategy (Table 39-11); however, weight-based dosing is necessary, and known toxicities can be expected if weight-based maximum total and daily doses are exceeded. Among opioids, the μ-receptor agonists, particularly fentanyl and morphine, remain the most common drugs used in the PICU. Repeated exposure to an agent predisposes a child to tolerance

TABLE 39-11

COMMONLY USED ANALGESICS IN PEDIATRICS

Nonopioid	
Acetaminophen	10–15 mg/kg/dose (max 1,000 mg) PO/PR q 4–6 h PRN; PO/PR max 90 mg/kg/day up to 4,000 mg/day; 15 mg/kg IV q 6 h or 12.5 mg/kg q 4 h PRN; IV max 75 mg/kg/day
Ibuprofen	10 mg/kg/dose PO q 4–6 h PRN (max dose 40 mg/kg/day)
Ketorolac	0.5 mg/kg/dose IV/IM (max 30 mg) q 6 h × 72 h (do not exceed 5 days)
Trisalicylate	7.5–15 mg/kg/dose (max 1.5 g) PO q 6–8 h PRN
Opioid*	
Fentanyl	0.5–2 μg/kg/dose IV/IO q 1–2 h PRN
Hydromorphone	0.015 mg/kg/dose IV/IO q 4–6 h PRN
Morphine	0.05–0.1 mg/kg/dose IV/IO q 2 h PRN
Oxycodone	0.05–0.15 mg/kg/dose (max 5 mg) PO q 4–6 h PRN

*morphine 0.1 mg = methadone 0.1 mg = hydromorphone 0.02 mg = fentanyl 0.001 mg
PO: per os (by mouth); PR: per rectum; PRN: pro re nata (as needed); max: maximum; IM: intramuscular; IO: intraosseous; IV: intravenous; q: quaque (every)

at that particular dose, as occurs in adults. Titration in amounts of 20% to 50% of the baseline dose is usually well tolerated and, depending on the duration of sedation, the absolute administered dose may reach high levels. When used in the neonate, caution must be taken because hepatic biotransformation and clearance may be altered and significantly delayed due to the immaturity of the P450 enzyme system. Also, it should be noted that despite historical anecdotes, oral sucrose is likely ineffective and wholly inadequate as an analgesic strategy.[34] Both patient-controlled analgesia and local anesthesia are viable and important parts of the analgesic strategy as well (Table 39-12). When opioids are used as sedatives in the PICU, a second class of drugs, such as benzodiazepines or ketamine, should also be considered to provide amnesia as needed, which may also potentiate the opiate effect, resulting in a lower total opiate dose.

Even though children have sleep-wake cycles that differ in structure from adults, disrupted sleep architecture in conjunction with anxiety and fear also predispose children to ICU psychosis.[35,36] Commonly used sedatives in the PICU include benzodiazepines, barbiturates, dexmedetomidine, ketamine, and propofol, with the latter used largely for procedural sedation (Table 39-13). The utility of intermittent versus continuous infusion is provider dependent, with the benefit of hemodynamic stability offered via continuous infusion weighed against accelerated tolerance. With repeated use of lorazepam, progressive metabolic acidosis and osmolar gap secondary to polyethylene glycol may develop. Given its catechol-dependent metabolism, ketamine is particularly useful for sedating children with congenital heart disease or asthma.

The use of neuromuscular blockade agents in the PICU is driven by clinical necessity, potential adverse effects, and provider preference. The most commonly used neuromuscular blockade agents in the PICU are pancuronium and vecuronium, though other nondepolarizing agents such as cisatracurium are also often used safely.[37] The key considerations are limiting paralysis to the shortest period possible at the lowest possible dosing and selection of an agent based on renal and hepatic function.

TABLE 39-12

PEDIATRIC PATIENT-CONTROLLED ANALGESIA*

Drug	Bolus	Basal	Max Dose†
Fentanyl	0.25–1 μg/kg/dose	0.25–1 μg/kg/h	3 doses/h; lock out every 10 min
Hydromorphone	0.003–0.006 mg/kg/dose	0.003–0.006 mg/kg/h	5 doses/h; lock out every 7–15 min
Morphine	0.01–0.03 mg/kg/dose	0.01–0.03 mg/kg/h	5 doses/h; lock out every 7–15 min

*Child should be 5 years or older and able to understand the PCA concept. Start low and titrate to effect. Use of basal may improve overall analgesia steady state, including sleep pattern. Consider low-dose nalaxone infusion (1–2.5 μg/kg/h) for side-effect alleviation.
†Recommended bolus max dose: 0–5 doses/h
max: maximum

TABLE 39-13

COMMONLY USED PEDIATRIC SEDATIVES

Drug	Load/PRN	Infusion
Chloral hydrate	25–100 mg/kg/dose PO/PR; max 1 g/dose	N/A
Dexmedetomidine	Load: 0.5 μg/kg/dose IV × 1	0.2–2 μg/kg/h IV
Ketamine	0.5–2 mg/kg/dose IV every 1–2 h	0.5–2 mg/kg/h IV
Midazolam	0.05–0.1 mg/kg/dose IV every 1–2 h	0.05–0.1 mg/kg/h IV
Pentobarbital	1–3 mg/kg/dose IV or 2–6 mg/kg/dose PO/PR/IM every 2–4 h (max 150 mg)	1–2 mg/kg/h IV
Propofol	1–3 mg/kg/dose IV for induction*	75–300 μg/kg/min IV
Opioid	**Infusion†**	
Fentanyl	1–6 μg/kg/h IV	
Hydromorphone	0.010–0.015 mg/kg/h IV	
Morphine	0.05–0.2 mg/kg/hour IV	
Remifentanyl	Load: 0.5–1 μg/kg/dose IV × 1; Infusion: 0.05–0.5 μg/kg/min IV	
Adjuncts	**Dose**	
Clonidine	5 μg/kg/day topical patch (in 50 μg intervals up to 300 μg patch); consider enteral load: 2.5 μg/kg/dose PO every 12 h × 4 doses	
Diphenhydramine	0.5–1 mg/kg/dose (max 50 mg) IV/PO every 6 h	
Lorazepam	0.05–0.1 mg/kg/dose IV/PO every 4–8 h PRN	
Methadone	0.1 mg/kg/dose IV/PO every 4 h × 3 doses, then every 6–12 h (max dose 10 mg)	

*Limit infusion to less than 12 h in children under 18 y because of the association with propofol infusion syndrome.
†To alleviate side effects of continuous opiate infusions, consider antipruritic dosing of naloxone (0.25–1 μg/kg/h IV).
IV: intravenous; max: maximum; N/A: not applicable; PO: per os (by mouth); PRN: pro re nata (as needed)

HEAD AND SPINAL CORD TRAUMA IN CHILDREN

Neurological system failure and brain injury are the most common proximate causes of death in the PICU.[38] For those who survive to discharge, long-term sequelae such as motor deficits, visual deficits, speech and language abnormalities, seizures, and behavioral problems are common.[39] Thus, although pediatric traumatic brain injury management varies with injury severity and elements of it are common to adult algorithms, understanding and instituting appropriate neuroprotective management strategies is fundamental to the PICU plan of care (Figure 39-6).[40] In patients who cannot protect their airways, typically those with Glasgow coma scale scores less than or equal to 8, a secure airway must be established. Induction agents should be selected with a consideration toward minimizing spikes in intracranial pressure, particularly through the use of adjuncts such as lidocaine and induction with agents such as thiopental or alternatives such as etomidate (Table 39-14). Careful cervical spine precautions must be taken to maintain neutral positioning, even in the presence of apparently normal cervical spine radiographs, given the frequent occurrence of spinal cord injury without radiographic abnormality among children. Once intubated, the patient should be adequately sedated with continuous infusions and placed on mechanical ventilation to avoid hypoxia and hypercapnia and minimize intracranial hypertension. Goal cerebral perfusion pressures (CPPs) vary by age and are defined as:

$$CPP = MAP - ICP$$

where MAP is the mean arterial pressure and ICP is the intracranial pressure. In the absence of an ICP measurement, MAP is used as a proxy for CPP.

Regardless of the patient's age, a target intracranial pressure of less than 20 mm Hg is generally accepted. Serum osmolarity, sodium, and glucose should be monitored frequently. Osmolarity between 290 and

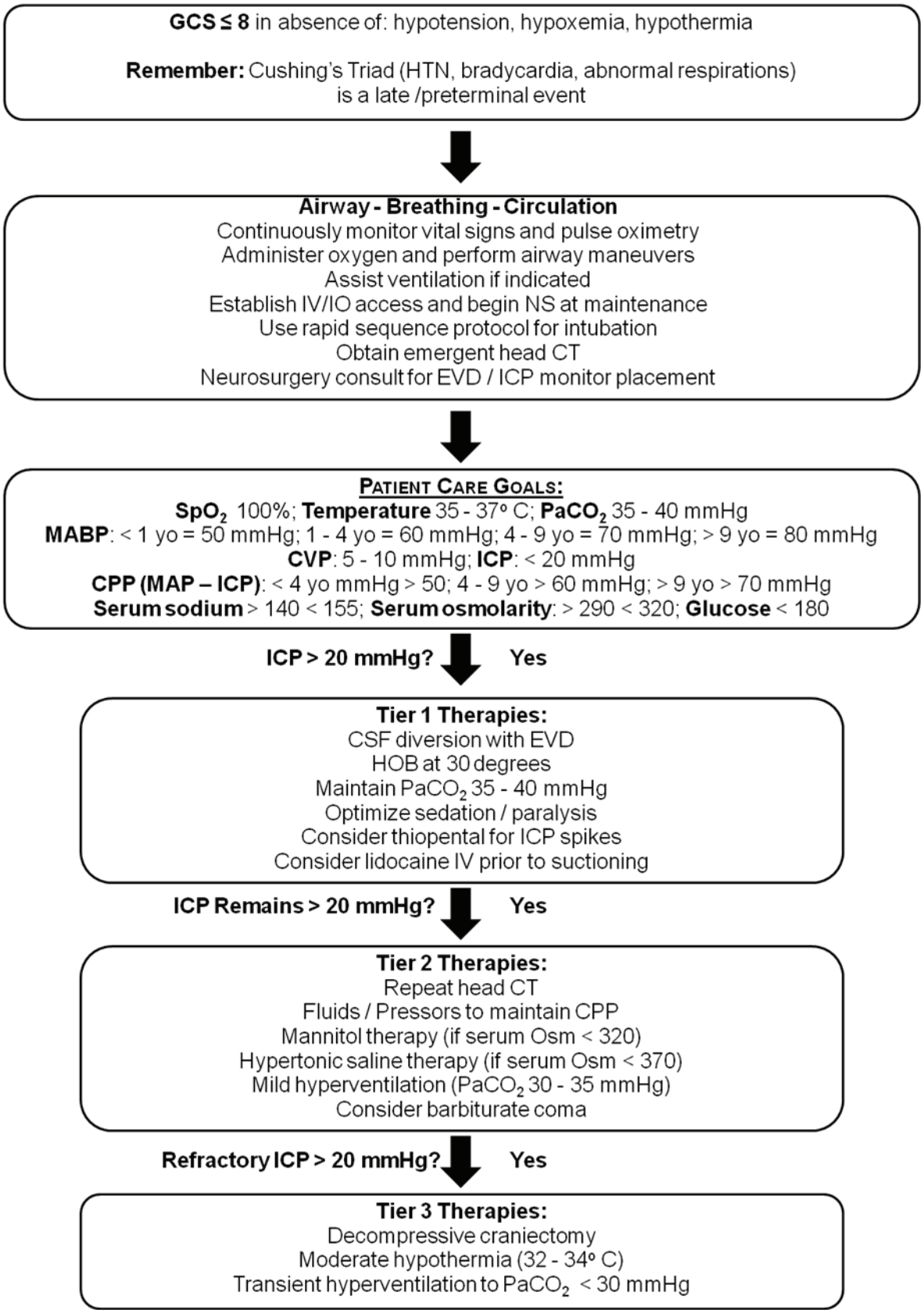

GCS ≤ 8 in absence of: hypotension, hypoxemia, hypothermia
Remember: Cushing's Triad (HTN, bradycardia, abnormal respirations) is a late /preterminal event
Airway - Breathing - Circulation
Continuously monitor vital signs and pulse oximetry
Administer oxygen and perform airway maneuvers
Assist ventilation if indicated
Establish IV/IO access and begin NS at maintenance
Use rapid sequence protocol for intubation
Obtain emergent head CT
Neurosurgery consult for EVD / ICP monitor placement
PATIENT CARE GOALS:
SpO_2 100%; **Temperature** 35 - 37° C; **$PaCO_2$** 35 - 40 mmHg
MABP: < 1 yo = 50 mmHg; 1 - 4 yo = 60 mmHg; 4 - 9 yo = 70 mmHg; > 9 yo = 80 mmHg
CVP: 5 - 10 mmHg; **ICP**: < 20 mmHg
CPP (MAP – ICP): < 4 yo mmHg > 50; 4 - 9 yo > 60 mmHg; > 9 yo > 70 mmHg
Serum sodium > 140 < 155; **Serum osmolarity**: > 290 < 320; **Glucose** < 180
ICP > 20 mmHg? **Yes**
Tier 1 Therapies:
CSF diversion with EVD
HOB at 30 degrees
Maintain $PaCO_2$ 35 - 40 mmHg
Optimize sedation / paralysis
Consider thiopental for ICP spikes
Consider lidocaine IV prior to suctioning
ICP Remains > 20 mmHg? **Yes**
Tier 2 Therapies:
Repeat head CT
Fluids / Pressors to maintain CPP
Mannitol therapy (if serum Osm < 320)
Hypertonic saline therapy (if serum Osm < 370)
Mild hyperventilation ($PaCO_2$ 30 - 35 mmHg)
Consider barbiturate coma
Refractory ICP > 20 mmHg? **Yes**
Tier 3 Therapies:
Decompressive craniectomy
Moderate hypothermia (32 - 34° C)
Transient hyperventilation to $PaCO_2$ < 30 mmHg

Figure 39-6 (facing page). Acute management of increased intracranial pressure algorithm.
CPP: cerebral perfusion pressure; CSF: cerebrospinal fluid; CT: computed tomography; CVP: central venous pressure; GCS: Glasgow coma scale; EVD: estimated blood volume; HOB: head of bet; HTN: hypertension; ICP: intracranial pressure: IO: introssious; IV: intravenous; MAP: mean arterial pressure
Data source: Adelson PD, Bratton SL, Carney NA, et al. Guidelines for the acute medical management of severe traumatic brain injury in infants, children and adolescents. *Pediatr Crit Care Med*. 2003;4(3 suppl).

320 mOsm/L, serum sodium between 140 and 155 mEq/L, and normoglycemia are the optimum goals. During periods of increased intracranial hypertension, hypertonic saline can be administered in a 1- to 2-mL/kg dose of 3% saline, ideally via central line, although it can be administered peripherally with proper site monitoring. Mannitol has also been recommended, although its use has become less common as experience with hypertonic saline increases. As a diuretic, mannitol use risks depleting intravascular volume and decreasing mean arterial pressure and cerebral perfusion pressure. If the blood-brain barrier has been disrupted, mannitol may further contribute to cerebral edema. Mannitol is dosed at 0.25 to 1 g/kg IV. Inducing hyperventilation during the acute period may be an option to reduce cerebral perfusion, although usually only until end tidal carbon dioxide reaches 30 mm Hg. Sustained hyperventilation has been associated with increased morbidity and should be avoided. If an intraventricular catheter has been placed, it may be possible to directly drain cerebrospinal fluid. Data are currently inconclusive regarding the use of therapeutic hypothermia in pediatric brain injury; however, the importance of avoiding hyperthermia is well established.[41,42] Steroids are not routinely recommended for treating pediatric traumatic brain injury.

FEVER AND INFECTION

Fever in a child, particularly a neonate, can be an ominous sign of occult bacteremia and impending sepsis. In neonates up to 30 days old, fever is defined as a core temperature of 38°C or higher (100.4°F). Temperature is most reliable when obtained via the rectal route. Given their immature immune systems, neonates require full sepsis evaluation for neonatal fever, including blood, urine, and cerebrospinal fluid cultures, and initiation of empiric, broad-spectrum antibiotics pending results.[43] Empiric antibiotics in this age range are selected to target perinatally acquired infections such as *Listeria*, Group B streptococcus, *Klebsiella*, *Enterobacter*, and *Pneumococcus* species. Such antibiotics include the combination of ampicillin (200 mg/kg/day IV divided every 6 hours) and cefotaxime (200 mg/kg/day IV divided every 6 hours). Ceftriaxone is not routinely used in this age group because of concerns about cholestasis. If risk factors are significant for herpes simplex virus infection (ie, maternal disease), consider treatment with acyclovir 20 mg/kg/dose every 8 hours IV for 21 days or until a herpes simplex virus polymerase chain reaction test, if available, is negative. In older infants and children, cerebrospinal fluid studies are recommended based on clinical examination and index of suspicion for meningitis. Because occult urinary tract infection remains the most common cause of serious bacterial infection in infants and young children, blood and urine studies and cultures and empiric antibiotics should still be initiated if the level of clinical suspicion is high. Empiric antibiotic selection for older infants is the same as for neonates. For older children, ceftriaxone (100 mg/kg IV every 24 hours) is the recommended initial therapy. If clinical history indicates concern for methicillin-resistant *Staphylococcus aureus*, the addition of vancomycin (20 mg/kg IV every 8 hours) may be appropriate, with appropriate drug-level monitoring at steady state (prior to the fourth dose).

In the first 48 hours following operative intervention, fever may be common in pediatric patients, as it is in adults, and is a poor predictor of serious infection.[44] In this setting, blood-culture yield in particular may be low.[45] In addition to bacteremia and urinary tract infections, wound infection and pneumonia are common causes of serious infection in postoperative patients and should be considered and evaluated.

HEMATOLOGIC ISSUES

Children with significant traumatic injury may require massive transfusion as part of their resuscitation, increasing the risk for metabolic and coagulation derangement. In adults, massive transfusion is defined as greater than one blood volume loss in 24 hours, 50% in 3 hours, or 150 mL/h. In pediatrics, all blood volume loss and replacement estimates are calculated based on weight (Tables 39-15 and 39-16). To prevent dilutional

TABLE 39-14

MEDICATIONS COMMONLY USED FOR PEDIATRIC RAPID SEQUENCE INTUBATION

Drug	Dose
Adjuncts	
Atropine	0.01–0.02 mg/kg/dose IV/IO for < 5 y to blunt vagal reflex; min dose 0.1 mg, max dose child 0.5 mg, max dose adolescent 1 mg
Lidocaine	1 mg/kg/dose IV/IO for patients at risk for increased ICP
Induction	
Etomidate	0.3 mg/kg/dose IV/IO
Fentanyl	2–5 μg/kg/dose IV/IO/IM
Ketamine	1–2 mg/kg/dose IV/IO; 2–4 mg/kg/dose IM
Midazolam	0.1–0.3 mg/kg/dose IV/IO (max 4 mg)
Propofol	2 mg/kg/dose IV/IO
Thiopental	4–7 mg/kg/dose IV/IO if normotensive; 2–4 mg/kg/dose IV/IO if hypotensive
Paralytics–Intubation	
Rocuronium	0.6–1.2 mg/kg/dose IV/IO
Succinylcholine	1–2 mg/kg/dose IV/IO; 2–4 mg/kg/dose IM (premedicate with atropine for < 5 y)
Vecuronium	0.1–0.2 mg/kg/dose IV/IO
Paralytics–Maintenance	
Cisatracurium	0.1–0.2 mg/kg/h IV/IO
Pancuronium	0.1 mg/kg/h IV/IO
Vecuronium	0.1 mg/kg/h IV/IO

ICP: intracranial pressure; IM: intramuscular; IO: intraosseous; IV: intravenous; max: maximum; min: minimum

TABLE 39-15

PEDIATRIC ESTIMATED BLOOD VOLUMES

Age	EBV (mL/kg)
Preterm newborn	100
Term newborn	90
1–12 months	75
≥ 12 months	70–75

EBV: estimated blood volume

TABLE 39-16

PEDIATRIC BLOOD PRODUCT DOSING GUIDELINES

Blood product	Dose	Notes
PRBCs	10–15 mL/kg	Transfusion of 10 mL/kg will increase Hgb by ~ 3 gm/dL and Hct by ~ 10%. Give over 4 hours, no faster than 3–5 mL/kg/h*
Platelets	1 unit/10 kg	Increases platelet count by 30,000–50,000/μL; transfuse over 15–30 min
FFP	10 mL/kg	Increases clotting factors by 10%–20%
Cryoprecipitate	1 bag/5 kg	Raises levels by 40% with greater amount of fibrinogen, factor VIII, vWF, and factor XIII

*In severe compensated anemia (Hgb ≤ 5), transfuse X mL/kg (where X = the patient's Hgb) and transfuse over 4 hours, no faster than 1 to 2 mL/kg/h.
FFP: fresh frozen plasma; Hct: hematocrit; Hgb: hemoglobin
PRBC: packed red blood cell
vWF: von Willebrand factor
Data source: Children's Hospital Boston. *The Medical-Surgical Intensive Care Unit Handbook.* Boston, MA: 2007.

coagulopathy during massive transfusion, consider a transfusion ratio of 1:1:1 for packed red blood cells to fresh frozen plasma to platelets for children weighing more than 30 kg, and a 30:20:20 milliliter-to-kilogram ratio for children weighing less than 30 kg until hemostasis has been achieved.[46] Activated factor VIIa (90 μg/kg IV) has also been used successfully to help restore hemostasis in trauma patients.

To conserve such limited resources, when transfusing products in small children, it is useful to have the blood bank split products into aliquots and save them for future use. If possible, a warmer should be used prior to administering blood products to prevent hypothermia, although chemical blankets and other blankets may also be used. Hypocalcemia is well recognized in children who have received large amounts of blood products because of the citrate binding of ionized calcium. Because calcium is a key inotrope, particularly in the developing heart, transfusion-related hypocalcemia can go unrecognized and result in cardiac dysfunction and arrest. Typical replacement doses of calcium include calcium chloride 20 mg/kg or calcium gluconate 50 to 100 mg/kg, ideally administered via slow IV push into a central line or

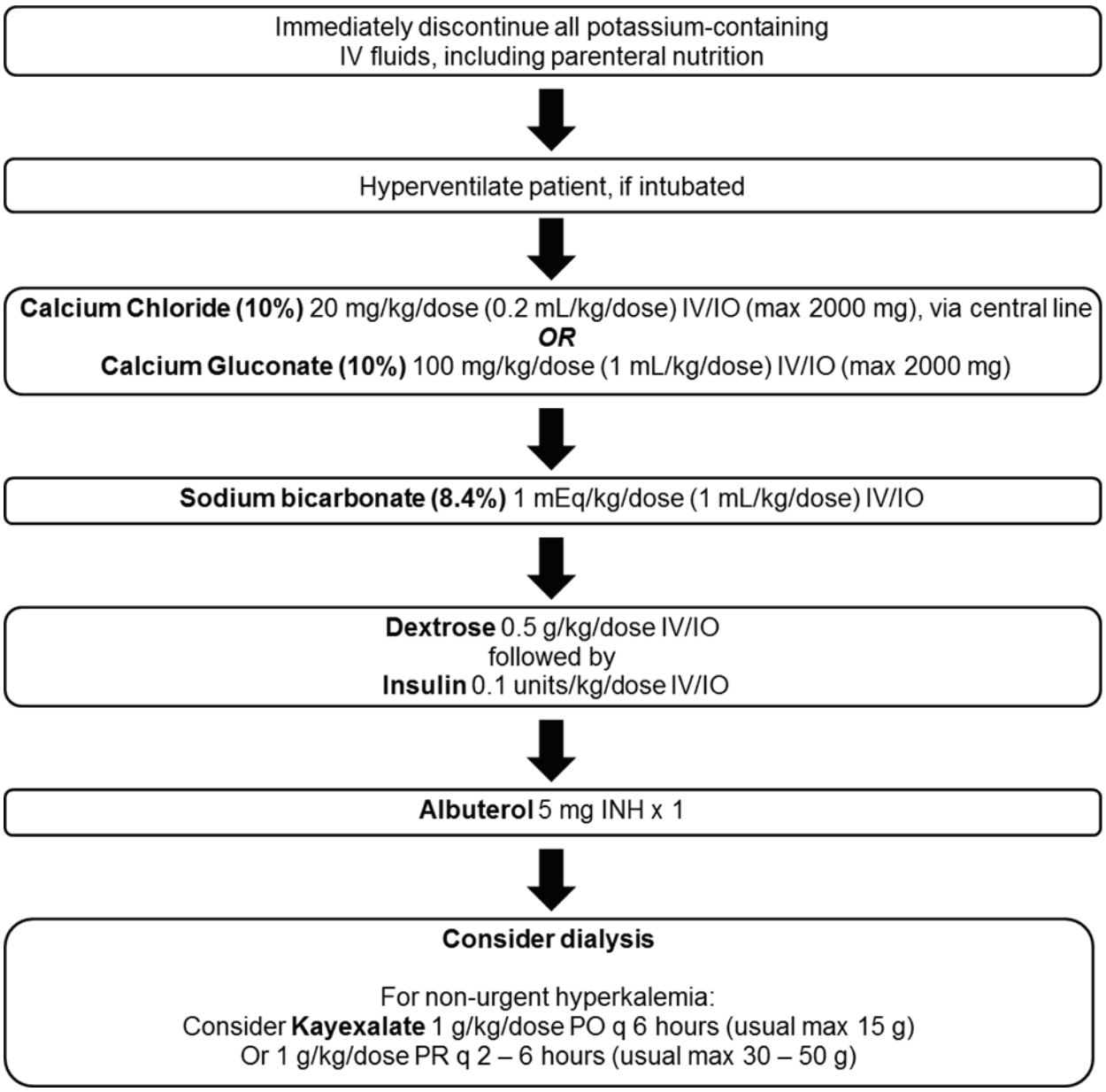


Figure 39-7. Acute management of hyperkalemia algorithm. INH: inhaled; IO: intraosseous: IV; intravenous; PR: per rectum

large-bore peripheral IV. Potassium is released as red blood cells lyse. Older units of packed red cells typically have increased amounts of potassium relative to fresher units. In children with particularly high serum potassium levels due to crush injuries, burns, or renal failure, the addition of potassium could increase serum potassium to clinically significant levels that require treatment (Figure 39-7).

PEDIATRIC BURN CARE

Retrospective review of military operations from the early 2000s suggests that pediatric burn patients comprised nearly 15% of all pediatric injuries cared for by military physicians in Afghanistan.[3] Most burned children who present to a local combat support hospital must receive their medical care in place. The initial approach and treatment of pediatric burn patients does not differ from that of adults; stopping

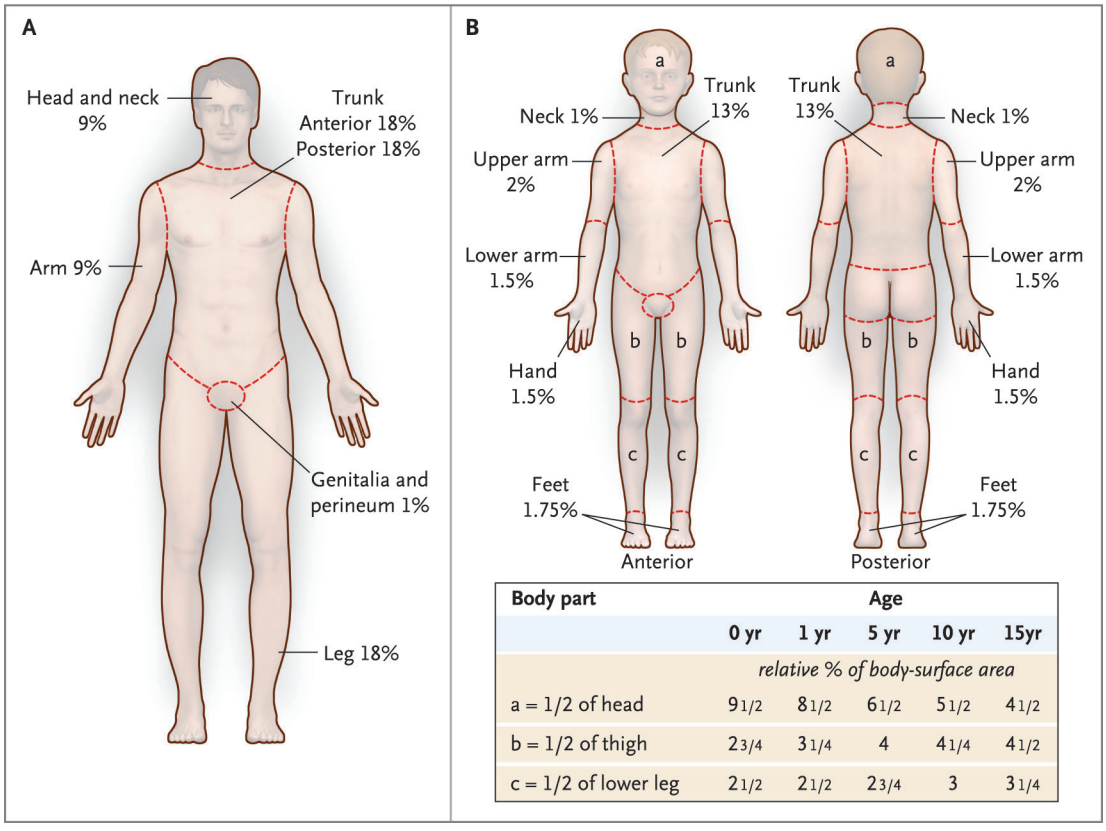
the burning process and avoiding extension of primary and subsequent secondary injuries is paramount.[47] Determining the extent of injury as a proportion of total body surface area varies based on the age of the child (Figure 39-8).

Airway

In an already-at-baseline, smaller-than-adult airway, inhalational exposure to heat and smoke can quickly lead to significant amounts of supraglottic airway edema and obstruction, exacerbated by required fluid resuscitation. A definitive airway should be placed early, particularly if the child presents with facial burns, has noticeable soot in the nares or oropharynx, or presents with stridor. A nasotracheal tube is often easier to secure in the long run, facilitates mouth care, and, during the rehabilitation phase, allows the child to communicate by mouthing words more effectively. Alar necrosis is a concern in a child who is nasally intubated. If significant portions of the oropharynx and nares are affected by burns, early tracheostomy may be the preferred option.

Breathing

Because children have increased minute ventilation relative to adults, airway exposure to toxic byproducts of combustion and higher temperatures is increased manifold. Tissue injury can lead to denuded tissue sloughing and endotracheal tube clogging. This can be avoided with frequent pulmonary toilet and administration of humidified air and mucolytics. Depending on the severity of the injuries, both inhalational injury and extrapulmonary burns may result in acute lung injury and acute respiratory distress syndrome. Carbon monoxide exposure during a burn or inhalation injury is particularly concerning.



Body part	Age				
	0 yr	1 yr	5 yr	10 yr	15yr
	relative % of body-surface area				
a = 1/2 of head	9 1/2	8 1/2	6 1/2	5 1/2	4 1/2
b = 1/2 of thigh	2 3/4	3 1/4	4	4 1/4	4 1/2
c = 1/2 of lower leg	2 1/2	2 1/2	2 3/4	3	3 1/4

Figure 39-8. The rule of nines and Lund-Browder charts. The rule of nines is often used to determine the extent of total body surface area (TBSA) burn injury in adults. Though less exact, this rule may be adjusted for pediatric patients by considering the head and neck as 18% and the lower extremities as 14%. The Lund- Browder chart allows for more formalized and accurate burn estimation in pediatric patients. In the absence of formal estimation tools, a quick estimate may also be performed by using the patient's palm to represent approximately 1% TBSA.
Reproduced with permission from: Orgill DP. Excision and skin grafting of thermal burns. *N Engl J Med.* 2009;360:893–901.

Carbon monoxide has an affinity for the hemoglobin molecule that is over 200 times higher than that of oxygen, which results in a shift of the oxyhemoglobin curve to the left and to decreased oxygen delivery, leading to cellular hypoxia and tissue acidosis. Direct measurements of carbon monoxide hemoglobin (HbCO) and oxyhemoglobin are necessary to determine injury severity. Administering 100% oxygen at high flows displaces carbon monoxide from HbCO and decreases the half-life of HbCO at room air from 4 to 6 hours to 40 to 60 minutes. This is important because hyperbaric oxygen therapy is unlikely to be available in the field.

Circulation

Reliable IV access is necessary for fluid resuscitation and to administer sedation and analgesia in extensively burned patients. Placing an IO needle for immediate resuscitation can be lifesaving, provided it can be placed in a non-burned site. Placing a urinary catheter is also necessary to monitor ongoing fluid balance. Initial fluid resuscitation aims at restoring burn-related fluid losses in addition to providing ongoing fluid requirements. Exclusive focus on urine outputs as an indication of overall fluid status during this time can result in significant fluid overload, although generally 1 mL/kg/h is adequate. The modified Parkland formula is one common formula for burn resuscitation. In addition to calculated maintenance fluid, the amount of resuscitation fluid over the first 24 hours is determined by the following:

Parkland formula: 4 mL × weight (kg) × % total body surface area

The first half of this fluid is given in the first 8 hours after burn injury and the remaining half is given over the following 16 hours. Lactated Ringer solution is an appropriate resuscitation fluid, although in infants and young children, dextrose should be used in maintenance fluids (dextrose 5% in lactated Ringer or dextrose 5% in NS). Because burned tissue releases potassium into the extracellular space, no potassium is necessary during the first 24 hours. Following initial injury, potassium can be added to fluids, but serum potassium must be closely monitored.

Continued electrolyte and blood-count monitoring is prudent, particularly given that approximately 3% of a child's blood volume is lost with every 1% of body surface area excised. With grafting, approximately 2% is lost for every 1% of body surface area grafted.[48] Prophylactic antimicrobial therapy is not indicated in burned children because it predisposes the patient to developing multidrug-resistant organisms. Topical antimicrobial therapy in the form of silver-containing products is often necessary for ongoing wound care. The antimicrobial spectrum of silver-containing substances extends to *Staphylococcus aureus*, *Enterobacteriaceae*, *Escherichia coli*, and *Candida albicans*. Such agents include 1% silver sulfadiazine mesh gauze. Ongoing care also requires particular attention to nutritional needs given a patient's hypermetabolic state following burn injury. As with most critical illnesses, initiation of early enteral nutrition is associated with improved outcomes.

PEDIATRIC TRANSPORT PRINCIPLES

Pediatric and neonatal transport to higher levels of care can be lifesaving. Subspecialty pediatric critical care transport teams have been shown to improve outcomes.[49] In the absence of such a team in theater, proper planning and team selection is essential to ensure safe and timely transport. Personnel skilled in providing critical care, including airway and vascular access, should accompany the patient and be provided with the appropriate equipment and medication to address any contingencies enroute. All catheters, wires, and tubes need to be properly secured prior to movement. Nasogastric tubes, ostomy bags, and chest tubes should remain vented and monitored frequently during transport. Any air-filled device (eg, cuffed endotracheal tube, gastrostomy tube balloon) must be filled with sterile water or pressure monitored, particularly during takeoff and landing. Limbs with circumferential casts at risk of developing compartment syndrome should be bivalved. Given the significant amounts of noise and vibration stressors enroute, adequate sedation and analgesia is necessary to ensure smooth transport with minimal complications. Hearing protection should be considered for patients. If the patient is small enough (ie, less than 10 kg) a flight-approved neonatal isolette provides a quieter environment that preserves ambient heat and humidity. Joint family movement should also be considered, particularly for pediatric patients.

SUMMARY

As a result of unique anatomical, physiological, and psychological characteristics, children represent a particularly vulnerable segment of the population affected by armed conflict. This chapter has highlighted

basic observations, understandings, and implications surrounding the care of pediatric trauma patients, reinforcing the axiom that "children are not just small adults." It has provided military anesthesia providers with the necessary knowledge and resources to be better equipped during the initial trauma resuscitation and subsequent stabilization of a critically ill pediatric patient.

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ATTACHMENT 1: COMMON PEDIATRIC EMERGENCY RESUSCITATION DOSING

Drug	Dose
Adenosine	0.1 mg/kg/dose (max 6 mg) rapid bolus IV/IO; if no effect, repeat 0.2 mg/kg/dose (max 12 mg) rapid IV/IO
Amiodarone	5 mg/kg/dose IV/IO bolus (max 300 mg) if pulseless arrest (VF/pulseless VT); if pulse present, give over 20–60 min (max 300 mg); may repeat to daily max 15 mg/kg (or 2.2 g)
Atropine	0.02 mg/kg/dose IV/IO or 0.04–0.06 mg/kg/dose ETT; min dose 0.1 mg, max dose child 0.5 mg, max dose adolescent 1 mg; repeat every 5 min to max total dose 1 mg child, 2 mg adolescent
Calcium chloride (10%)	20 mg/kg/dose (0.2 mL/kg/dose) IV/IO slow push during arrest (max 2,000 mg); central line preferred
Calcium gluconate (10%)	100 mg/kg/dose (1 mL/kg/dose) IV/IO slow push during arrest (max 2,000 mg)
Dextrose	0.5 to 1 g/kg/dose IV/IO; D_{10} 5–10 mL/kg for < 2 mo; D_{25} 2–4 mL/kg for 2 mo–2 y; D_{50} 1–2 mL/kg for > 2 y
Epinephrine	Pulseless arrest, bradycardia (symptomatic): • 0.01 mg/kg/dose (0.1 mL/kg/dose) 1:10,000 IV/IO every 3–5 min (max 1 mg; 10 mL) • 0.1 mg/kg/dose (0.1 mL/kg/dose) 1:1,000 ETT every 3–5 min Anaphylaxis: • 0.01 mg/kg/dose (0.01 mL/kg/dose) 1:1,000 IM (max 0.5 mg) • autoinjector 0.3 mg/dose (wt ≥ 30 kg) or Autoinjector Junior 0.15 mg/dose (wt 10–30 kg) IM
Insulin (hyperkalemia)	0.1 units/kg/dose IV/IO following 0.5 g/kg/dose of dextrose
Lidocaine (1%)	1 mg/kg/dose IV/IO, 2–3 mg/kg/dose ETT
Magnesium sulfate	25–50 mg/kg/dose IV/IO bolus (pulseless VT) or over 10–20 minutes (VT with pulses)
Sodium bicarbonate (8.4%)	1 mEq/kg/dose (1 mL/kg/dose) IV/IO; dilute 1:1 with sterile water for neonates
Vasopressin	0.5 units/kg/dose (max 40 units) IV/IO push for pulseless arrest
Cardioversion/Defibrillation	
SVT or VT w/ pulse	Cardiovert: 0.5–1 joules/kg synchronized × 1; if no response, 1–2 joules/kg synchronized
VF or Pulseless VT	Defibrillate: 2 joules/kg × 1, 4 joules/kg × 2; adult: monophasic 360 joules, biphasic 200 joules
Reversal	
Naloxone	Respiratory depression: 0.001 mg/kg/dose IV/IO/IM/SQ every 1–2 minutes until adequate respirations; respiratory arrest / full reversal: 0.1 mg/kg/dose IV/IO/IM/SQ (max 2 mg/dose)
Flumazenil	0.01 mg/kg/dose (max dose 0.2 mg) IV/IO; repeat every 1 min to max total dose 0.05 mg/kg/dose or 1 mg as necessary

D_{10}: dextrose 10%
D_{25}: dextrose 25%
D_{50}: dextrose 50%
ETT: endotracheal tube
IM: intramuscular
IO: intraosseous
IV: intravenous
max: maximum
SQ: subcutaneous
SVT: supraventricular tachycardia
VF: ventricular fibrillation
VT: ventricular tachycardia

ATTACHMENT 2. PEDIATRIC DOSING FOR SELECTED CATEGORIES OF COMMONLY USED MEDICATIONS

Antihypertensives	
Amlodipine	0.1 mg/kg/dose (usual max 10 mg) PO daily to BID
Esmolol	Load: 500 μg/kg IV × 1; infusion: 25–300 μg/kg/min IV, repeat load as needed
Hydralazine	0.1–0.5 mg/kg/dose (max 20 mg) IV/IM every 4–6 hours PRN
Labetolol	0.25–1 mg/kg/dose (usual max 20 mg) IV every 10 min PRN; infusion: 0.25–1 mg/kg/hour IV
Nicardipine	0.5–5 μg/kg/min IV
Nitroglycerin	0.5–5 μg/kg/min IV
Nitroprusside	0.5–10 μg/kg/min IV; monitor cyanide and thiocyante for > 4 μg/kg/min
Diuretics	
Bumetanide	≤ 6 mo: 0.01–0.05 mg/kg/dose (max 1 mg) IV/PO daily; > 6 mo: 0.02–0.1 mg/kg/dose (max 10 mg) IV/PO daily; Adult: 2 mg IV/PO daily to BID
Chlorothiazide	10–20 mg/kg/dose IV/PO every 12 h (max IV 500 mg/dose; max PO 188 mg/dose for < 2 yo; max PO 1,000 mg/dose for > 2 yo)
Furosemide	1–2 mg/kg/dose IV/PO every 6–24 h (usual starting max 20 mg); Infusion: 0.05–0.3 mg/kg/h
Spironolactone	1 mg/kg/dose (max 100 mg) PO every 12 h
Endocrine / Metabolic*	
Dexamethasone	Airway edema: 0.1–0.6 mg/kg/dose (max dose 10 mg) IV every 6 h × 4–6 doses; croup: 0.6 mg/kg IM/PO × 1
Hydrocortisone	Stress dose: 50 mg/m^2/dose (usual max 100 mg) IV × 1, then 25 mg/m^2/dose (usual max 75 mg) IV every 6 h; maintenance dose: 5 mg/m^2/dose (usual max 10 mg) IV every 8 h
Methylprednisolone	Loading dose for asthma: 2 mg/kg/dose IV × 1; maintenance: 0.5–1 mg/kg/dose (usual max 60 mg) IV every 6–12 h
Vasopressin	0.5–3 milliunits/kg/h; titrate to maintain UOP < 2 mL/kg/h
Neurologic/Seizure/Cerebral Edema	
Diazepam	0.1–0.2 mg/kg/dose IV/IO every 15–30 min PRN; < 5 yo: 0.5 mg/kg/dose PR every 2 h PRN; 6–11 yo: 0.3 mg/kg/dose PR every 2 h PRN; ≥ 12 yo: 0.2 mg/kd/dose PR every 2 h PRN
Hypertonic saline (2% or 3% NaCl)	3 mL/kg IV over 30 min. Note: 1 mL/kg of 3% NaCl will increase serum sodium ~1 mEq/L
Fosphenytoin	Load: 20 mg PE/kg/dose IV × 1; maintenance: 2 mg PE/kg/dose IV every 8 h; max infusion rate 3 mg PE/kg/min up to 150 mg PE/min
Lorazepam	0.05–0.1 mg/kg/dose (usual max 4 mg) every 15 min PRN
Mannitol	0.25 g/kg/dose IV over 20–30 min PRN × 1
Phenobarbital	Load: 20 mg/kg/dose IV × 1; maintenance: 2.5 mg/kg/dose IV/PO every 12 h

Respiratory	
Albuterol	2.5 mg/dose in 3 mL NS nebulized; may repeat every 20 minutes × 3 or continuous; continuous: 0.5 mg/kg/h (usual max 20 mg/h); < 7.5 kg: 2.5 mg/h INH; 7.5– 4.9 kg: 5 mg/h INH; 15–29.9 kg: 10 mg/h INH; > 30 kg: 20 mg/h INH
Epinephrine	0.01 mg/kg (0.01 mL/kg) 1:1,000 SQ/IM (max 0.5 mg)
Ipratroprium	0.25–0.5 mg/dose INH every 4-6 h
Magnesium sulfate	75 mg/kg/dose IV × 1 over 15–20 min (max 2000 mg); monitor for hypotension
Terbutaline	Load: 10 μg/kg/dose IV × 1 over 30 min; infusion: 0.4–6 μg/kg/min IV
Miscellaneous	
Albumin	0.5 g/kg/dose (5% = 10 mL/kg; 25% = 2 mL/kg)
Heparin (DVT treatment)	Load: 75 units/kg IV × 1; infusion: for < 1yo: 28 units/kg/h; for ≥ 1 yo: 20 U/kg/h; check coagulation panel 4–6 h after change. Adjust dose to give PTT 1.5–2.5 × control

*Glucocorticoid effect (ratio of antiinflammatory potency of hydrocortisone per mg vs the antiinflammatory potency of the other steroid preparations): hydrocortisone, 1:1; prednisone, 4:1; methylprednisone, 5:1; dexamethasone, 30:1.
Mineralocorticoid effect (ratio of potency of hydrocortisone per mg vs the potency of the other steroid preparations): hydrocortisone, 1:1; prednisone, 0.25:1; methylprednisone. 0.4:1; dexamethasone, 0:1.

BID: twice a day (bis in die)
DVT: deep vein thrombosis
h: hour
IM: intramuscular
INH: inhaled
IV: intravenous
max: maximum
min: minute
mo: month
NaCl: sodium chloride
NS: normal saline
PE: phenytoin sodium equivalents
PO: per os (by mouth)
PR: per rectum
PRN: pro re nata (as needed)
PTT: partial thromboplastin time
SQ: subcutaneous
UOP: urine output
yo: years old

Chapter 40

MULTIDRUG-RESISTANT ORGANISMS AND INFECTION CONTROL PRACTICE IN THE US MILITARY MEDICAL SYSTEM

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INTRODUCTION

The pervasive use of improvised explosive devices, mortars, and rocket-propelled grenades by opposition forces, as well as the helmets and body armor worn by US military personnel, make extremity blast trauma the most common type of injury among soldiers and marines wounded in Iraq and Afghanistan.[1] The mechanism of injury (penetrating trauma) and the nature of the wounds (with devitalized tissue and retained foreign bodies) create a milieu conducive to infection, either by organisms inoculated at the time of injury or subsequently by contaminating bacteria and fungi. Although wound microbiology at the time of injury tends to consist of antibiotic-susceptible, skin-commensal, gram-positive cocci,[2] subsequent infections with multidrug-resistant (MDR) bacteria are common.[3]

There is no standardized definition of multidrug resistance, and criteria vary among organisms and institutions. At Walter Reed National Military Medical Center, a gram-negative bacterium is considered MDR if it is resistant to at least three classes of antibiotics among aminoglycosides, carbapenems, cephalosporins, penicillins, and quinolones. Methicillin-resistant *Staphylococcus aureus* (MRSA), vancomycin-resistant *Escherichia coli* (VRE), and the extended spectrum β-lactamase (ESBL) producing gram-negative bacteria are generally recognized as MDR organisms. Additionally, MDR *Pseudomonas aeruginosa*, *Klebsiella pneumoniae*, and species belonging to the *Acinetobacter baumannii-calcoaceticus* complex (ABC) are frequently isolated from infected wounds sustained in Iraq and Afghanistan.[4,5] The discordance in wound microbiology at the time of injury versus the nature of the organisms subsequently cultured from infected wounds suggests nosocomial colonization, which has been documented in at least one investigation.[6]

Because wounded military personnel are evacuated by various means (ambulance, rotary and fixed-wing aircraft, and ship) to one of a number of destinations for transient and definitive care (typically combat support hospitals and military medical centers, respectively), it is unlikely that any single location or method of conveyance can be implicated as the sole source of colonization with MDR organisms. The emergence of MDR pathogens, while not a uniquely military phenomenon, poses challenges in the treatment of hospitalized service members with infected war wounds. As organisms become increasingly resistant, the number of available effective antibiotics diminishes, and use of antibiotics to which MDR isolates are susceptible may be limited by untoward side effects or patient allergies. For example, colistin (colistimethate sodium), a polymyxin antibiotic to which many MDR ABC strains are solely susceptible, is nephrotoxic, causing acute renal failure in almost half of patients and severe enough effects in 21% of patients to warrant discontinuation of the drug.[7] Moreover, drug resistance is emerging at a faster pace than drug development, a trend that is unlikely to be reversed in the foreseeable future.[8]

CHEMOPROPHYLAXIS AND THE COMBAT CASUALTY

Two factors contributing to the emergence of MDR organisms are the selective pressure for resistance by prolonged exposure to antibiotics and the tremendous adaptive capability of bacteria owing to high mutation rates and short generation times. While the emergence of antimicrobial resistance is inevitable,[9] the process is accelerated by the injudicious use of antibiotics. Consequently, antibiotic use in the combat casualty should target the likeliest pathogens in the case of prophylaxis or empiric therapy, and should be culture-driven when the pathogen and its susceptibilities are known. In 2008, guidelines for the prevention of infection among combat wounded were published by a panel of experts, convened by the US Army Medical Command (MEDCOM), in various medical and surgical subspecialties.[10] The panel reconvened in January 2011 and published revisions to its guidelines.[11] This chapter is not intended to supplant these guidelines but rather to provide succinct compatible recommendations. The reader is encouraged to review the guidelines in their entirety at http://journals.lww.com/jtrauma/toc/2011/08002.

It has been estimated that the number of bacteria inhabiting the human body outnumber human cells by 10:1,[12] and that 500 to 1,000 different species comprise the human microbiome.[13] Each of these species occupies a niche, and the nature of the microbial flora varies by anatomic location. Strict anaerobes, for example, are much more prevalent in the gut than on skin, where *Propionibacterium* (an aerotolerant anaerobe), *Corynebacterium*, *Streptococcus*, and *Staphylococcus* are the predominant bacterial genera and *Malassezia* is the predominant fungal genus.[14] It makes sense, therefore, that the choice of an initial empiric antibiotic should be driven, in part, by the anatomic location of the wound (Table 40-1). For wounds of skin, soft tissue, and bone (such as extremity wounds), as well as maxillofacial fractures and penetrating chest wounds, an antibiotic

TABLE 40-1

RECOMMENDED EMPIRIC ANTIMICROBIAL THERAPY FOR INFECTION PROPHYLAXIS IN THE COMBAT CASUALTY

Injury	Recommended Agent	Alternate Agent	Duration
Skin, soft tissue, or bone injury	Cefazolin 1 g IV every 8 h	Clindamycin 900 mg IV every 8 h	72 hours
Penetrating chest wound	Cefazolin 1 g IV every 8 h	Clindamycin 900 mg IV every 8 h	24 hours
Maxillofacial fracture	Cefazolin 2 g IV every 8 h (note higher dose)	Clindamycin 900 mg IV every 8 h	24 hours
Penetrating abdominal wound	Cefoxitin 1–2 g IV every 6–8 h or piperacillin-tazobactam 4.5 IV every 6 h	Levofloxacin 750 mg IV daily or ciprofloxacin 400 mg IV every 8–12 h and metronidazole 500 mg IV every 6 h, or moxifloxacin 400 mg IV daily	24 hours after definitive washout
Central nervous system injury	Cefazolin 1 g IV every 8 h. Add cefazolin, penicillin, and gentamicin for gross contamination or metronidazole 500 mg IV every 6–8 h if also an abdominal wound.	Ceftriaxone 2 g IV daily. Add cefazolin, penicillin, and gentamicin for gross contamination. For penicillin allergic patients: vancomycin 1 g IV daily and ciprofloxacin 400 mg IV every 8–12 h	5 days
Eye injury	Erythromycin or Bacitracin ophthalmic ointment 4 times daily for non-penetrating injuries, burns or abrasions	Fluoroquinolone 1 drop 4 times a day	Until epithelium healed
Burns	Mafenide acetate topical each morning and silver sulfadiazine topical each afternoon	Either mafenide acetate or silver sulfadiazine twice daily. Bioprane may be used for partial thickness burns and silver impregnated dressings for limited, clean, full thickness burns.	Until healed or grafted

IV: intravenous

Adapted with permission from: Hospenthal DR, Murray CK, Andersen RC, et al. Guidelines for the prevention of infections associated with combat-related injuries: 2011 update. *J Trauma*. 2011;71(2 Suppl):S214–S215.

targeting susceptible gram-positive cocci is sufficient (eg, cefazolin, clindamycin). Conversely, a broad-spectrum antibiotic such as piperacillin-tazobactam would be indicated for a penetrating abdominal injury with a perforated viscus and fecal spillage. For penetrating injuries of the brain and spinal cord, cefazolin is the preferred agent, with extended coverage (eg, the addition of penicillin and gentamicin) for gross contamination or an antibiotic with anaerobic coverage (eg, metronidazole) if the abdominal cavity is involved. Systemic antibiotics are not recommended for eye injuries; however, a topical antibiotic such as erythromycin or bacitracin is appropriate for ocular burns or abrasions (but not for penetration). Similarly, systemic antibiotics are not recommended for bodily burns.

With respect to the timing of antibiotics, there is sufficient data to suggest that at least for some wounds (eg, extremity wounds) early debridement and administration of antibiotics are associated with lower infection rates, whereas delays in antibiotic administration beyond 2 to 6 hours are associated with higher infection rates.[15] Therefore, when evacuation from the battlefield is expected to be delayed beyond 3 hours, the recommendation for open extremity wounds is a single early dose of a fluoroquinolone (moxifloxacin 400 mg per os [PO], levofloxacin 500 mg PO, or gatifloxacin 400 mg PO); patients with penetrating abdominal injuries, shock , or for those unable to take medication orally should be given ertapenem 1 g IV/IM, cefoxitin 2 g IV/IM, or cefotetan 2 g IV.[16] These antibiotics were chosen by the MEDCOM panel for their spectrum of activity, ease of dosing, and stability during storage.

It is worth noting that antibiotics are one of several interventions recommended for the prevention

of infection in the combat casualty; the others include prompt wound irrigation, debridement, and coverage, as well as stabilization of fractures. As soon as tactically feasible, wounds should be copiously irrigated (1–3 L) with normal saline or sterile water (or less ideally, potable water) under low pressure to remove gross contaminants. However, removal of deeper foreign bodies such as fragments is best left to a surgeon. Once the wounds have been irrigated, a sterile bandage should be applied and, in the case of extremity wounds, bony fractures should be splinted. In addition to protecting the limb, splinting reduces the risk of infection by reducing the risk of vascular injury, which may lead to limb ischemia and tissue necrosis.

MULTIDRUG-RESISTANT ORGANISM SURVEILLANCE

Historically, surveillance of microbial pathogens was limited to only those organisms with epidemic potential such as *Mycobacterium tuberculosis* or *Salmonella typhi*. Over time, standardization and accreditation of clinical laboratories, as well as the ease of data acquisition and analysis afforded by the computer, has led to more widespread monitoring. However, most surveillance data is still collected and shared only locally (eg, to determine local rates of antimicrobial resistance), and only a minuscule fraction of data is accessible to researchers or clinicians outside the immediate environs of a particular hospital.[17] In a summary statement to the US Congress, the Infectious Diseases Society of America recognized antimicrobial resistance as "one of the greatest threats to human health worldwide" and called for a federally funded network of sentinel sites "to evaluate rapidly emerging resistance in a variety of clinically important organisms and infections, and to develop, implement, and evaluate prevention strategies."[18]

In response to the proliferation of infections caused by MDR gram-negative bacteria among hospitalized military personnel, the Walter Reed Army Institute of Research (WRAIR) stood up the Multidrug Resistant Organism Repository and Surveillance Network (MRSN) in July 2009.[19] Conceived as a performance improvement mandate from MEDCOM, the purpose of the MRSN is to create a repository of targeted MDR organisms accompanied by relevant clinical and demographic information, and to perform antibiotic susceptibility analysis and other testing. Under the mandate, Army hospitals are required to submit specimens, and hospitals from other services are invited to do so. The MRSN submits reports to hospital commanders and consultants to the surgeon general; this data will eventually be available for clinicians as well on the MRSN website.[20] For the deployed provider with access to little or no microbiology assets, the advantages conferred from submitting bacterial isolates to the MRSN include generation of a facility-specific or regional antibiogram and, in the case of a suspected outbreak, comparison of isolates by molecular biology techniques. Information on submitting specimens to the MRSN is available on its website. Other ways in which the military is collecting data on MDR organism infections among war wounded service members include the addition of an infectious disease module to the Joint Theater Trauma Registry,[21] and collaborating with the National Institutes of Health Infectious Disease Clinical Research Program on the Trauma Infectious Diseases Outcome Study.[22]

INFECTION CONTROL FOR THE DEPLOYED PROVIDER

Infection control in a combat environment can be challenging for the deployed provider. However, certain rudimentary practices can be readily implemented which, if adhered to, are proven effective in reducing the risk of nosocomial pathogen transmission. These practices can broadly be categorized as either individual or institutional interventions. Included among the former are standard precautions and isolation precautions (ie, contact, droplet, and airborne precautions). The latter include patient cohorting, disinfection protocols, and antibiotic stewardship.

Individual Interventions

Perhaps the simplest yet most effective infection control practice is handwashing.[23] In fact, the greatest limitation of handwashing's impact is noncompliance or improper technique rather than the use of an ineffective soap.[24] The World Health Organization (WHO) recommends handwashing prior to patient contact, prior to a procedure, and after body fluid exposure, patient contact, or contact with a patient's surroundings.[25] Alcohol-containing sanitizers are an effective alternative to soap and water, especially in the deployed setting where running water may not be available. However, limitations of these products include their lack of activity against spore-forming bacteria (such as *Clostridium difficile*) and decreased effectiveness when the hands are grossly dirty.[26] Of note, while glove use is an important component of infection control, wearing them does not obviate the need for handwashing because gloves often have small invis-

ible tears, and hands routinely become contaminated while removing them.[27,28]

Isolation precautions are categorized according to the three major routes by which nosocomial pathogens are transmitted: contact, droplet, and airborne spread. Contact precautions are indicated for patients colonized or infected with MDR bacteria (such as MRSA and VRE) or *Clostridium difficile*, and for patients with various other conditions easily transmitted by person–person or person–fomite contact (eg, scabies). Contact precautions consist of donning gloves and gowns and, when possible, the use of dedicated medical equipment for individual patients. Although strict contact isolation may be difficult to implement in an austere setting, adaptations include separating patients by empty beds or clearly delineating a designated area for those patients on contact isolation.[29]

Droplet precautions consist of wearing a face mask and should be implemented for patients with confirmed or suspected infections caused by *Neisseria meningitidis* (bacterial meningitis), *Bordetella pertussis* (whooping cough), *Haemophilus influenzae* or *Mycoplasma pneumoniae* (atypical pneumonia), *Corynebacterium diphtheriae* (diphtheria), *Yersinia pestis* (plague), group A streptococcus, or various droplet-borne viruses (rhinovirus, influenza, rubella, mumps, adenovirus, respiratory syncitial virus, and parvovirus B19). Because droplets remain suspended only briefly, it is probably unnecessary to wear a mask beyond 3 to 6 feet from the patient.[30]

Airborne precautions are indicated for infectious agents that remain suspended in the air for a long time. Examples included rubeola virus (measles), varicella virus (chickenpox), and *Mycobacterium tuberculosis*. Ideally, a patient on airborne precautions would be placed in a negative pressure isolation room with special air handling and ventilation capability, and healthcare workers entering the room would wear an N95 mask or respirator. Because such resources are unlikely in the deployed setting, the transmission risk can be mitigated by masking the patient, placing the patient in a private room with the door closed, and providing healthcare workers N95 masks or respirators before entering the room.

Institutional Interventions

At the facility level, a number of interventions can be implemented to reduce the risk of nosocomial pathogen transmission. One such intervention is patient cohorting, which entails grouping people colonized or infected with the same drug-resistant bacterium. Because patients with recent prior hospitalizations or those who have been hospitalized for more than 72 hours are more likely than newly admitted patients to be colonized with nosocomial pathogens, separating these two cohorts can also reduce transmission risks.[31]

Environmental cleaning, disinfection, and sterilization are other simple methods proven to reduce the transmission of nosocomial pathogens.[32] Cleaning refers to the removal of foreign material from objects and is typically accomplished with water and detergents. Disinfection and sterilization both refer to the elimination of microbes from inanimate objects. However, the former (usually accomplished with chemicals) does not eliminate bacterial spores, while the latter (by means of steam, heat, pressure, or gas) does. Sterilization is usually accomplished by autoclaving or by using ethylene oxide gas or chemical sterilants (eg, 2% glutaraldehyde-based products, 6% stabilized hydrogen peroxide, peracetic acid). Some examples of chemical disinfectants include sodium hypochlorite, ethyl or isopropyl alcohol, phenolic and iodophor solutions, and quarternary ammonium germicidal detergents. Although maintaining a clean environment and sterile medical devices may be challenging for deployed providers (especially those in more austere environments), the importance of doing so cannot be overstated.

The perseverance and ubiquity of microbes are testimony to their tremendous adaptive capability. This adaptability means that prolonged exposure to any antibiotic will almost inevitably culminate in resistance to that drug. Hence, the injudicious use of broad spectrum antibiotics has the unintended consequence of selecting for organisms with multidrug resistance. This risk can be mitigated by selecting an empiric antibiotic that targets the likeliest pathogens and then tailoring and narrowing coverage based upon culture data and local antibiograms. Preprinted admission or preoperative orders prescribing broad spectrum antibiotics for all patients should be avoided. Additionally, the duration of antibiotic use should be limited to the shortest effective length of time.[33]

After reviews in 2008 and 2009, a number of measures were put in place addressing infection control practices and challenges in theater hospitals. Among these was a mandate that all deploying combat support hospitals should have a designated infection control officer (ICO), and also the creation of a formal infection control course taught at the Army Medical Department Center and School (AMEDD C&S) at Fort Sam Houston, Texas. The purpose of this 5-day instruction is to provide training for military personnel who will be performing the duties of an ICO or overseeing an infection control program within Role 3 hospitals.[34] The course consists of a pre-test to assess attendee baseline knowledge, 18.5 hours of didactics, and a post-test. Topics include combat theater infection control overview and principles; clinical micro-

biology; preventing transmission of infectious agents; hand hygiene; principles of cleaning, disinfection, and sterilization; special patient populations (surgical, burn); healthcare acquired infections; blood and body fluid exposure management; program management; and infectious disease threats. Additional information about this course can be found on the AMEDD C&S website.[35]

INFECTIONS RELEVANT TO THE DEPLOYED PROVIDER

Just as extremity blast trauma and traumatic brain injury are the signature injuries of the combat in Iraq and Afghanistan, certain infections have also come to define the medical experience in these theaters. In addition to those caused by MDR bacteria, common infections are caused by protozoa belonging to the genera Leishmania and Plasmodium, causative agents of leishmaniasis and malaria, respectively.

Malaria

Although malaria, typically caused by chloroquine-sensitive *P vivax*, occurs with a very low prevalence in Iraq, both *P vivax* and, to a lesser extent, *P falciparum* are highly endemic in Afghanistan, with one case report describing 38 cases of *P vivax* among a 725-soldier Ranger Task Force that deployed to eastern Afghanistan between June and September 2002.[36] The US Army Central Command has articulated a policy on malaria chemoprophylaxis for deploying units.[37] Among its key features is the requirement for mandatory glucose-6-phosphate dehydrogenase (G6PD) deficiency screening prior to deployment with results annotated either in Defense Department form 2766 or the service-specific immunization database. The policy also dictates that doxycycline be used as the primary malaria chemoprophylactic agent, with mefloquine and atovaquone/proguanil (Malarone [GlaxoSmithKline, London, UK]) as second and third alternatives, respectively, for those with a contraindication to doxycycline. Personnel should be cautioned that doxycycline should be taken with food or water and not within an hour of lying down to mitigate potential side effects. They should also be advised to avoid taking the drug with milk or antacids, which may impair absorption. When chemoprophylaxis with mefloquine is being considered, it is important to exclude any history of depression, anxiety disorders, psychosis, or other psychiatric disorders as well as cardiac conduction defects.

Service members should deploy with sufficient malaria chemoprophylaxis in hand to cover the preexposure period (2 days for doxycycline and Malarone, 2 weeks for mefloquine); the period of exposure; and the terminal prophylaxis period (4 weeks for doxycycline and mefloquine, 1 week for Malarone). Deploying personnel are not required to hand carry primaquine because terminal chemoprophylaxis with primaquine will occur after redeployment (return to garrison). Providers should be aware that primaquine dosing recommendations often refer to the base ingredient (primaquine phosphate) and that 26.3 mg tablets contain 15 mg of primaquine base. According to the policy, malaria prophylaxis is indicated year round for Afghanistan, Pakistan, and Yemen and from May through October in Tajikistan. An exception applies to individuals whose deployment is restricted exclusively to the months of January and February.

Leishmaniasis

In contrast to malaria, leishmaniasis is endemic to both Iraq and Afghanistan; unlike malaria, which is transmitted by mosquitoes, this protozoan infection is typically transmitted by the bite of an infected sand fly. The nature of the infection depends upon the particular Leishmania species. In Iraq, where *L major* predominates, most infections are restricted to the skin, with occasional lymphadenitis. Lesions are typically painless, dry, and ulcerated, and may have an overlying eschar (Figure 40-1). A purulent discharge is not typical and may represent a secondary bacterial infection. In Afghanistan, where other Leishmania species are also found (eg, *L tropica* and *L infantum-donovani*), visceral

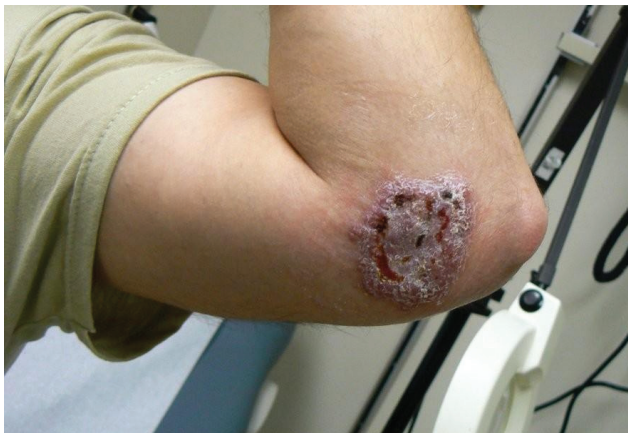


Figure 40-1. Typical lesion caused by Leishmania major. Note the lack of erythema and pus.

disease may rarely occur and has been documented among deployed soldiers.[38] The clinical presentation of visceral leishmaniasis varies but classically consists of fever, pancytopenia, hepatosplenomegaly, and cachexia.

The diagnosis of cutaneous leishmaniasis is made by confirming the presence of the amastigote in a skin biopsy or scraping. The diagnosis of visceral leishmaniasis is made by demonstration of amastigotes in biopsy specimens of bone marrow, lymph node, liver, or spleen. Additionally, serologic assays such as the rK39 immunochromatographic assay (Inbios International, Seattle, WA) and the *Leishmania* immunofluorescence assay (Centers for Disease Control and Prevention) are sensitive for systemic infection with Leishmania. Testing, by means of culture, histopathology or polymerase chain reaction (PCR) amplification is done at the WRAIR Leishmania Diagnostic Laboratory in Silver Spring, Maryland. With prior arrangement, samples can be sent for testing via commercial delivery services. The laboratory can be contacted via e-mail or 24 hours a day by telephone (301-573-3763), and additional instructions can be found at the WRAIR website.[39] Supporting documentation including a patient information sheet and specimen collection procedures are provided as Exhibits 40-1 and 40-2, respectively. On June 6, 2011, the US Food and Drug Administration approved a rapid diagnostic (SMART Leish PCR) for the diagnosis of cutaneous leishmaniasis.[40] The assay, developed in partnership among WRAIR, the Army Medical Materiel Development Activity, and a commercial partner (Cepheid, Inc, Sunnyvale, CA), utilizes real-time PCR to amplify *Leishmania major*-specific DNA sequences from skin scrapings. It is anticipated that this assay will be used at the Leishmania Diagnostic Laboratory at WRAIR and perhaps eventually by deployed medical assets.

With respect to treatment, patients with leishmaniasis are managed differently depending on whether they have cutaneous or visceral disease. Infection with *L major* is usually self-limiting, and watchful waiting is reasonable in many cases. For patients who need more immediate treatment (eg, those with large facial lesions), options include cryo- or thermo-therapy, topical paromomycin, azoles, pentavalent antimonies, and a lipid formulation amphotericin. The Army surgeon general holds the investigational new drug approval for pentavalent antimony, and this drug, as well as topical paromomycin, are solely given at Walter Reed National Military Medical Center. Because visceral leishmaniasis is life threatening, systemic therapy is always indicated and should be done under the direction of or in consultation with an infectious diseases specialist.

Other Common Infectious Diseases in Theater

In addition to those already discussed, the deployed provider should be aware of other infectious diseases endemic to the Middle East and Southwest Asia. A detailed discussion of each of these is beyond the scope of this paper, and ample reviews have been published[41,42]; however, a few relevant comments are appropriate here . According to the WHO, Afghanistan has a high burden of tuberculosis, with an incidence in 2009 of 187 cases per 100,000 persons. During the same year, the WHO reported an incidence of 67 cases per 100,000 persons in Iraq.[43] In line with Department of the Army Personnel Policy Guidance for Overseas Contingency Operations,[44] personnel deploying to either country require tuberculin skin testing (TST) within 12 months prior to deployment and again upon redeployment for soldiers considered to have been at high risk for exposure. High risk exposure is defined as indoor exposure to local people or third country nationals of greater than 1 hour per week in a region with greater than 25 cases per 100,000 persons annually (for the purpose of this policy, both Iraq and Afghanistan are considered to be high risk tuberculosis incidence areas). Individuals with previous positive tuberculin skin tests do not require TST. Interferon-gamma release assays such as the QuantiFERON-TB Gold (Cellestis,Inc, Valencia, CA) may be considered for those individuals with indeterminate TST results or for foreign-born individuals vaccinated with the Bacillus Calmette–Guérin (BCG) vaccine. Although the results of interferon-gamma release assays appear to decline after treatment for latent tuberculosis infection, and more significantly after treatment for active tuberculosis, there is insufficient data to support using these assays to monitor response after treatment for latent tuberculosis infection.

Q fever, a zoonotic disease caused by the Rickettsia-like bacterium *Coxiella burnetii*, is another infectious disease threat to deployed troops. Reservoirs include ruminants (as well as other mammals, birds, and arthropods), and humans become infected after inhalation of aerosolized bacteria or consumption of unpasteurized dairy products. Acutely infected individuals classically present with fever, atypical pneumonia, and hepatitis, and some will develop chronic disease including culture-negative endocarditis. The true risk to deployed personnel is unknown, but a recent study of banked sera from soldiers deployed to Iraq showed a 10% seroconversion rate, indicating exposure to the bacterium.[45] Because *Coxiella burnetii* is both fastidious and highly infectious, the diagnosis of Q fever is usually made by serology from acute and convalescent sera. This testing is cur-

EXHIBIT 40-1

WALTER REED ARMY INSTITUTE OF RESEARCH LEISHMANIASIS PATIENT INFORMATION SHEET

Leishmaniasis Patient Information Sheet

Soldier completes Part A; Clinical provider completes Part B

PART A – SOLDIER

Patient Name: _____ **SSN:** _____ **Rank/ Service:** _____

Blood type_____ **Weight** _____ **Med Allergies** _____ **Age** _____ **DOB:** _____

Unit: Company _____ **BN** _____ **BDE/BCT** _____ **DIV** _____

Date soldier arrived in Theater: _____ **in Iraq:** _____

Places/dates lived in Iraq: (e.g., *FOB Murphy, 10 Jun – 15 Jul 03*) _____

_____

Were rodents present around bivouac area? Y / N **Were dogs in the area? Y / N**

Places You Slept	# Weeks or N/A	Screens Or Windows? (Y/N)	A/C (Y/N)	Use Bednet (Always/ Sometimes/Never)	Use Repellent (Always/ Sometimes/Never)	Insect Bites Per Night? *(<5, 5-20, >20)*
Vehicle or Ground						
Tent						
Building						

Your Use of Insect Repellents	Product Was Not Available to Soldier	Product was Available to Soldier			
		Did Not Use	Used Only After Insect Bites – After how many bites? *(<5, 5-20, >20)*	Used Every Night	Used Other Times *Describe When*
Bed Net, Treated w/ Permethrin					
Bed Net w/o Permethrin					
Permethrin Treated DCUs					
DEET (green tube) on Skin					
Commercial Insect Repellent *If Yes, List in Box*					

PART B – CLINICAL PROVIDER (*Send form with slides and biopsy*)

Lesion Location & #: _____ Duration? _____

Antibiotic Treatment (type/dose/length): _____

Photos Taken? N / Y If Yes, sent to WRAIR? N / Y

Procedures Done: Scrape Biopsy: N / Y Punch Biopsy: N / Y Touch Prep: N / Y

Culture: N / Y Preserved Tissue: N / Y PCR: N / Y

Date Eval: _____ MTF: _____ POC: _____ Phone: _____

E-mail(POC): _____

Clinician Name E-mail (Provider): _____

(stamp) ═══════════════════════════

Results: (POS / NEG) _____

Notes: _____

For questions regarding Leishmaniasis, contact the Leish Diagnostic Lab (peter.weina@us.army.mil)

version 12Apr04

EXHIBIT 40-2

WALTER REED ARMY INSTITUTE OF RESEARCH LEISHMANIASIS SCRAPING AND BIOPSY PROCEDURES

DEPARTMENT OF THE ARMY
WALTER REED ARMY INSTITUTE OF RESEARCH
503 ROBERT GRANT AVENUE, ROOM 2S04
SILVER SPRING MARYLAND 20910-7500

Leishmania Scraping & Biopsy Procedures

1) **Criteria for scraping or biopsy**:
   - Any patient who has had a non-healing lesion (does not have to be an open, weeping ulcer) for greater than 3 to 4 weeks needs to be suspected of having leishmania.
   - These patients need to be placed on a course of antibiotic therapy for 7 to 10 days with an antibiotic, which has proven activity in Iraq (recommendation is Augmentin 875mg BID for 7 to 10 days).
   - At the conclusion of therapy, the patient should be seen by the same practitioner and a decision needs to be made if there was any efficacy to the course of antibiotics. If the lesion has persisted or worsened, a scraping or biopsy should be performed.
   - Photos of the lesion prior to scraping or biopsy being done should be accomplished if the practitioner has the capability. E-mail these photos to WRAIR since this may help in the diagnosis (peter.weina@us.army.mil).

2) **Scraping procedure**:
   - Clean area with alcohol pads and allow to dry.
   - Anesthetize with lidocaine 1% or 2% with epinephrine 1:100,000 (unless the epinephrine is contraindicated due to anatomic site).
   - 2 tissue smears are performed by horizontally scraping (lightly enough to elicit an exudates, but not vigorously enough to cause bleeding) the base of the underlying ulceration with a #15 blade (this often requires removal of the overlying crusted debris). The dermal tissue is then thinly applied in a circular fashion to a dime to nickel sized area in the center of the slide. Minimize blood, epithelium (keratinocytes), and purulence on the specimen.
   - Additionally, material from the scrapings (and even the overlying crusted debris) should be inserted into a small vial of 95-100% ethanol for PCR analysis.
   - Ensure slides are labeled per the format of your affiliated pathology department and submit per their protocol. If pathology services unavailable locally, ship per address below. Work closely with pathologists to verify adequacy of tissue smear samples.

2) **Biopsy/touch prep-impression smear procedure**:
   - An area of the lesion needs to be cleaned thoroughly with alcohol pads and dried.
   - The anticipated area of biopsy should be anesthetized as described above.
   - A 4 mm sterile disposable punch or sterile scalpel (#15, #11, or #10) should to be used to remove a piece of tissue approximately 3 to 4 mm in circumference and approximately 1 mm deep from the edge of the lesion (see photo for preferred area of biopsy). Lesions on the face, anterior of the neck, and near larger vessels and/or nerves need to be biopsied with extreme caution and a simple surface scraping (described above) may be preferred to a true biopsy.
   - The biopsy should be placed on a sterile, clean, dry gauze 2X2 briefly to absorb excess blood on the tissue that may interfere with the reading of the touch preparations.

(**Exhibit 43-2** *continues*)

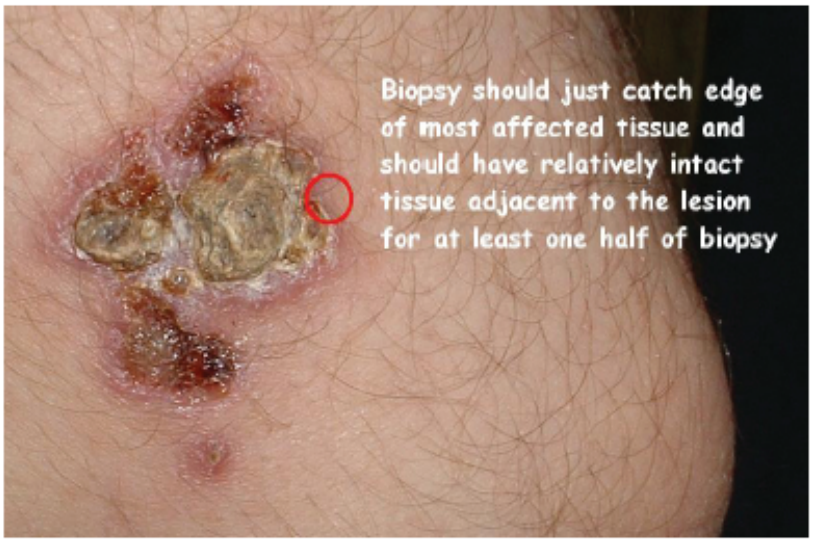
Exhibit 40-2 *continued*

- The tissue should be grasped with forceps and impression smears made on clean slides (4 for each biopsy) by rubbing the tissue gently across the surface of the slide in a circular motion.
- Dry thoroughly. Fix with methanol if available.
- The tissue biopsy (after the impression smears are made) should then be placed in a very small amount of ethyl alcohol (just enough to cover the specimen) in a leakproof vial (such as a "nunc" transport tube).
- The slides and the vial with the tissue should be shipped per your local pathology section protocol or via DHL or Federal Express to the address below. The container should be labeled as diagnostic specimens and no shipping permit is required (all MTFs have personnel and resources to ship diagnostic specimens correctly).
- **PLEASE LABEL WITH PATIENT NAME, SPECIMEN SOURCE, DATE, AND MATERIAL TRANSPORTED IN** (formalin, ETOH, etc.).
- Complete the patient information sheet (attachment #2 below) and include with the specimen for each patient biopsied.
- Procedural inquiries should be made to COL Peter Weina at (301) 319-9956.

SHIPPING ADDRESS

Colonel Peter J. Weina, PhD, MD
Director, Leishmania Diagnostic Laboratory
Division of Experimental Therapeutics
503 Robert Grant Avenue
Walter Reed Army Institute of Research
Silver Spring, Maryland 20910-7500

Preferred biopsy area:



rently unavailable in the deployed setting. However, a diagnostic utilizing real-time PCR with a rugged deployable platform is being developed.[46] Guidelines promulgated by the Armed Forces Infectious Diseases Society TriService Q Fever Working Group recommend empiric therapy with doxycycline, 100 mg twice a day for 21 days, for patients suspected of having acute Q fever.[47] The working group also recommends sending serum for testing to the US Air Force School of Aerospace Medicine at the time of presentation and again in 2 weeks. If a patient has positive serologic testing for acute Q fever, a transthoracic echocardiogram (TTE) should be performed to document any baseline cardiac valvular abnormalities, and infectious diseases consultation should be obtained. However, the guidelines do not recommended medical evacuation exclusively for the purpose of obtaining a TTE unless there are clinical signs suggesting more urgent evaluation is indicated.

Like *Coxiella burnetii*, species belonging to the genus Brucella (in particular, *B melitensis*) cause acute and chronic infections in humans, have a reservoir in ungulates, and are transmitted via aerosols or contaminated dairy products. Brucellosis is a common cause of fever of unknown origin, and the clinical presentation may be nonspecific. However, well-described complications include sacroiliitis, epididymo-orchitis, meningitis, endocarditis, and hepatic abscess. The diagnosis is made by culture (*B melitensis* is a potential hazard to laboratory workers, and the laboratory should be notified when brucellosis is suspected), serology, or PCR. Although *B melitensis* is endemic worldwide, including the Middle East, only a few cases have been reported among redeploying troops.[48,49] However, brucellosis should be considered in an individual with chronic fever and past exposure to ungulate animals or consumption of raw dairy products. Treatment involves a prolonged course of multiple antibiotics and should be done under the direction of or in consultation with an infectious diseases specialist.

SUMMARY

The deployed provider can expect to encounter patients with infectious and noninfectious conditions. Orthopedic injuries are common among the latter, while among the former, gastroenteritis and upper respiratory infections predominate. While the preponderance of infections are the same as those encountered stateside, deployments present special challenges for the healthcare provider. In addition to the commonplace infections are those endemic to the region. Moreover, prevention of infection, both combat associated and nosocomial, can be difficult even under ideal conditions. A useful asset available to any military provider with Internet access is the remote consultation service offered by infectious diseases specialists assigned to stateside military medical centers. Providers can submit case presentations and solicit advice via e-mail (id.consult@us.army.mil) and expect thoughtful, comprehensive replies typically within several hours.

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Chapter 41

HUMANITARIAN OPERATIONS AND AID AGENCY ANESTHESIA

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INTRODUCTION

Humanitarian Assistance as an Additional Mission

Traditionally, the focus of military medicine has been care of the wounded during combat operations. While this continues to be the primary focus, military personnel are increasingly involved in secondary humanitarian assistance (HA) efforts as well. The distinction between a primary and secondary mission is important. For example, in deployments such as the United Nations Mission to Haiti, 1994–1996, and Operation Iraqi Freedom, 2003–2011, the focus of medical care is in support of deployed troops and authorized civilians. HA is a secondary mission. Both Role 2 (the US Army's forward surgical teams and the Navy's Forward Resuscitative Surgical System) and Role 3 (the Army's combat support hospitals, the Navy's fleet hospitals, and the Air Force's expeditionary hospitals) medical units are outfitted to meet expected combat trauma, their primary mission. Typically, these units do not have instruments and equipment sets for specialty surgery and are extremely limited in their ability to care for pediatric patients.

Additionally, it is vital that HA efforts initiated in theater be cleared through command surgeon channels up to the health affairs attaché in the US embassy. Also, there must be host nation approval for these projects consistent with the existing Status of Forces Agreement between the two involved countries and their allies. When approval is obtained for HA projects and with security permitting, the initial approach should be to assist local medical authorities in local hospitals. If required care is beyond the capability of the local hospital, the next step is to arrange for evacuation to a suitable civilian institution in theater. Another option is to treat HA patients at the Role 2 or 3 medical facility. It must be clear that in these circumstances, military trauma takes priority over HA patients and local civilians will be discharged after postoperative recovery. In a few rare cases, arrangements may be made to evacuate a patient with a treatable medical or surgical condition outside of theater, possibly to a medical center in the United States. This option requires close coordination at all levels and support of a nongovernment agency willing to sponsor the patient.

Humanitarian Assistance as a Primary Mission

Outside of combat operations, military personnel are also involved in primary HA missions. During these missions assistance is provided to a local populace by predominantly US forces in conjunction with military operations and exercises.[1] This assistance is specifically authorized by Title 10, United States Code, Section 401, and funded under separate authorities. Under these provisions, the assistance must fulfill unit training requirements and is limited to (*a*) medical, dental, and veterinary care provided in rural areas of a country; (*b*) construction of rudimentary surface transportation systems; (*c*) well drilling and construction of basic sanitation facilities; and (*d*) rudimentary construction and repair of public facilities.[2] Since 2006, primary HA missions have been conducted annually in an effort to promote goodwill and the national interests of the United States. These missions alternate between countries in the Pacific region and Southeast Asia and Central and South America.

Humanitarian Assistance Supporting Disaster Relief

In addition to planned HA missions, the US military is also called upon to assist civilian authorities in disaster relief (DR) operations both within the United States and in foreign countries. Interventions in the past decade include relief efforts in Southeast Asia following the tsunami in 2004, assistance following the Hurricane Katrina disaster in 2005, and humanitarian aid to victims of the Haitian earthquake in 2010 and the victims of the Japanese earthquake and nuclear disaster in 2011.

Today, the US military provides HA in a variety of situations. These missions differ in size, scope, and platform. They vary from targeted interventions that support a larger combat operation to large-scale missions with a global outreach objective. In each of these situations, as with combat operations, anesthesia personnel perform a critical role in assisting with the coordination and delivery of surgical and pain management services. Understanding that role and the circumstances unique to the HA mission will better prepare the anesthesia provider deployed on such a mission.

HUMANITARIAN ASSISTANCE MISSIONS: A GENERAL OVERVIEW

Hospital Ships

Large-scale humanitarian operations are generally carried out aboard the military's two hospital ships, USNS *Mercy* (T-AH 19) and USNS *Comfort* (T-AH 20). Built in 1976 as oil tankers, both vessels were later acquired by the US Navy and converted to hospital ships. USNS *Mercy* was put into service in 1986 and USNS

Comfort in 1987. The primary mission of these ships is to provide an afloat, mobile, acute surgical medical facility to the US military that is flexible, capable, and uniquely adaptable to support expeditionary warfare. Their secondary mission is to provide full hospital services to support US DR and humanitarian operations worldwide.[3,4]

Both ships are capable of providing Role 3 care including triage, resuscitation, transfusion, laboratory testing, radiology services (including computed tomography and interventional procedures), dental care, initial and definitive surgery, postoperative care, intensive care, and patient holding capacity. Each ship can provide care to patients of all ages, with a total patient capacity of 1,000 hospital beds (including 80 intensive care, 20 postoperative recovery care, 280 intermediate care, and 120 light care beds), twelve operating rooms, and ancillary services similar to shore-based facilities.

Mission Overview and Process

HA annual missions (Pacific Partnership 2008 and 2010 in the Pacific region and Southeast Asia, and Partnership for the Americas 2007 and Continuing Promise 2009 and 2011 in Latin America) range in length from 4 to 6 months and occur between April and October. The number of countries visited, the time spent in each country, and the services provided depend on the overall strategic plan as well as other factors such as budget, staffing, and mission duration. Once the budget, staffing, and schedule are determined for a mission, an initial planning conference is held. Members from the involved military command (Pacific Command [PACCOM] or Southern Command [SOUTHCOM]), the US Public Health Service, nongovernmental organizations (NGOs), and host nation ministries of health meet to discuss and set the objectives for the mission. Next, several weeks prior to the start of the mission, predeployment site survey teams visit each country to meet with US embassy and ministry of health personnel, distribute a surgical capability list, and initiate country-specific plans. Then an advance coordinating element team will review and finalize plans with each country several days prior to the arrival of the mission crew. During these visits, each host nation compiles a manifest identifying the diagnoses of the patients to be evaluated. This preidentification of surgical patients facilitates the preoperative process and simplifies operating room scheduling.[5]

Upon arrival at each site, teams consisting of surgeons, anesthesia providers, and support personnel screen patients ashore. Basic laboratory and radiology tests are performed and the patients are assigned a surgery date. On the day before surgery, the patients are transported by either boat or helicopter to the hospital ship. This allows for a review of information and compliance with preoperative fasting. Typically, surgeries are scheduled until 1 to 2 days prior to departure to allow for appropriate postoperative care and discharge of all patients.[6]

Disaster Relief

In contrast to the HA mission, extensive advance planning is rarely possible when responding to a disaster. Consequently, maintaining a state of readiness and utilizing personnel trained for such missions is critically important in maximizing efficiency and decreasing response time.

Other key differences with a DR mission include the level of urgency and types of injuries treated. The care provided during an HA mission focuses on improving quality of life, whereas the goals of DR are saving life and restoring basic services to the affected population. Medical treatment involves emergent care of trauma-related injuries, and surgical procedures are aimed at saving life, sight, or limb. Additionally, unlike the HA mission, the emergent and chaotic nature of the disaster situation leaves little time for patient evaluation. This, in particular, makes the process of patient triage an essential component in the overall DR operation. Triage quickly prioritizes patients based on extent of injury to ensure maximum chances of survival and the most efficient use of resources.

During Operation Unified Response (2010) in Haiti, little information was available in advance about the number of people injured or the extent of their injuries. To address this problem, patient triage was quickly implemented on shore to rapidly evaluate earthquake victims and facilitate an orderly process for patient movement. Each day a member of the surgery team, usually a trauma surgeon, evaluated patients on shore and communicated pertinent information to staff on the ship. This allowed for the proper allocation of personnel and resources in treating the more critical patients first.

Host Nations and Nongovernmental Organizations

The number of host nations visited varies according to overall mission objectives and length. Host nations are often revisited, though on-site locations may change. One example is Haiti. In 2007 patients were screened at a general hospital in the capital city, Port-au-Prince. For the Continuing Promise mission in 2009, the screening site was a coast location in the city of Killick. Site locations change for a number of reasons including host nation needs, available resources,

logistics and transportation capabilities, and security concerns.

During an HA mission, host nations and NGOs participate extensively throughout the operation. Representatives from these groups are involved in the initial planning process and other personnel assist with implementation and delivery of aid on site. Two such NGOs are Operation Smile and Project HOPE. These organizations have provided assistance in both the Pacific and Latin American missions that remain ongoing today. In 2007, during the Partnership for the Americas mission, Project HOPE augmented the surgical staff with a full-time surgeon, an operating room nurse, and anesthesiologists in six countries.[5]

In a disaster situation, support from NGOs is especially vital to achieving an effective response and outcome. The level of urgency and limited preparation time makes mobilization of resources and personnel with the necessary skills a challenge. NGOs can provide staff with specific skills and capabilities appropriate for the situation.

Medical Personnel

The number and mix of medical personnel varies depending upon the scope of the mission and the identified needs of host nations. For example, a majority of personnel deployed on an HA mission are adult and pediatric primary care providers, whereas a greater number of critical care and trauma specialists are involved in the DR situation. Additional specialties (infectious disease, cardiology, pathology, radiology) are included to support the overall mission.

Active duty anesthesiologists and certified registered nurse anesthetists constitute the core group of anesthesia providers. Fellowship-trained pediatric anesthesiologists are included in this group, as well as those with skills in pain management procedures. The size of this core group typically ranges from six to seven providers for an HA mission and is increased during a disaster or combat situation. During HA and DR missions, additional personnel from volunteer organizations and, occasionally, the military reserve complement this core group. Civilian volunteers are not usually involved in combat operations.[5,7,8]

Due to the elective nature of the cases selected during an HA mission, surgery is rarely performed at night. However, providers are scheduled to be available if a need arises. In contrast, during a combat or disaster situation, operating rooms function continuously. During the DR mission in Haiti, two of the ten operating rooms were used around-the-clock daily for approximately 6 weeks. To maintain this level of productivity, appropriate assignment of staff is critically important.

Surgical Services

The schedule followed during Continuing Promise 2009 (Table 41-1) is typical for the delivery of surgical services during an HA mission. At each site, during Continuing Promise 2009, the first 2 days were reserved for preoperative screening. Surgical cases were scheduled over the next 5 to 6 days, leaving the last 2 days for patient discharge. Average surgical caseload was 25 cases per day. Although typical, this schedule can vary due to factors such as length of site visits, number of patients treated, and complexity of cases.

The surgical services provided during an HA mission are diverse, with general surgery and ophthalmology accounting for the greatest number of cases. Procedures most commonly performed include inguinal hernia repair, umbilical hernia repair, cataract removal, and soft tissue mass excision. Cleft lip and palate repair procedures are also common.

In a disaster situation, the array of surgical services expands to include trauma, burn, and neurosurgery specialties. In Haiti, during Operation Unified Response, approximately 840 surgeries were performed in all, with nearly 700 procedures completed in the first 3 weeks. The majority of these cases were orthopedic trauma involving extremity and pelvic injuries. These included repairs of 33 pelvic fractures, more than 100 femur fractures, 32 primary amputations, and numerous amputation revision procedures. In addition to the orthopedic cases, 16 burn debridement procedures, 16 craniotomies, 44 spine surgeries, and 75 head and neck procedures were also performed.

TABLE 41-1

SURGERY SCHEDULE, USNS *COMFORT* (T-AH 20), CONTINUING PROMISE 2009

Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7	Day 8	Day 9	Day 10
Prescreen	Prescreen	OR	OR	OR	OR	OR	OR	Discharge	Discharge

OR: operating room

PRACTICAL CONSIDERATIONS FOR THE ANESTHESIA PROVIDER

HA missions can present unique and challenging situations for the anesthesia provider. Patient populations often have little or no previous medical care and poor access to healthcare services. Disease states not typically seen in developed countries are frequently encountered. Also, time constraints do not always allow for the optimization of disease processes that would otherwise occur prior to the administration of anesthesia. To assist the anesthesia provider in managing these situations, several factors that affect the delivery of anesthesia are discussed below. Strategies used on previous missions are also presented.

Preoperative Assessment

Patient Evaluation and Selection Criteria

Prior to arrival, host nations will have compiled a manifest of surgical candidates. These lists, however, are often incomplete and not very reliable. Additionally, the patient prescreening process (Figures 41-1 and 41-2) occurs over a 1- to 2-day period, leaving very little time for lengthy preoperative evaluations and optimization of comorbid conditions. Consequently, to facilitate efficiency and patient safety, patient selection criteria should be discussed and agreed upon by the medical providers prior to arrival and throughout the site visits. The goal is to select cases that provide the best possible outcome while minimizing surgical risk. Patients that are declined are referred, when possible, to other organizations for care. The screening criteria (Table 41-2) and overall process (Figure 41-3) followed during the Pacific Partnership 2008 mission provides good examples.[7]

Patient Demographics

Data from previous missions provides some information regarding demographic characteristics of patients selected for surgery. Though reporting is not uniform, the data shows a majority of patients were assigned to an American Society of Anesthesiologists physical status category 1 or 2. This is consistent with and reflects the goal of selecting surgical candidates with minimal risk.

Another important characteristic is patient age. Approximately one-third of cases were performed on patients 18 years old or younger.[9]

Uncommon Disease

HA missions are conducted in areas where access to medical care is very limited. As a result, patient populations often present with extremes of common disease states, such as malignant hypertension and profound anemia, as well as diseases not typically seen in developed countries. An example is tuberculosis. Rates range from 50 per 100,000 in Latin America to more than 200 per 100,000 in some parts of Southeast Asia.[10] Tuberculosis spreads by droplet contamination. Precautions using special protective gear and

Figure 41-1. Anesthesia prescreening station, Colon, Panama, USNS *Comfort*, Partnership for the Americas, 2007.

Figure 41-2. Surgical screening area, Central America, USNS *Comfort*, Partnership for the Americas, 2007.

TABLE 41-2

ANESTHESIA SCREENING CRITERIA BY SYSTEMS, USNS *MERCY*, PACIFIC PARTNERSHIP 2008

System, Condition, or Age	Criteria for Cancellation of Cases
Cardiovascular	HTN: SBP > 160 or DBP > 90 or PP > 80 mm Hg CAD: MI within past 6 months or remote MI not revascularized CHF: evidence of uncompensated CHF Arrhythmia: frequent symptomatic palpitations Valvular disease: type III/VI or diastolic murmurs; aortic stenosis; mitral regurgitation
Respiratory	Asthma: active wheezing or decreased breath sounds Chronic obstructive pulmonary disease: symptomatic shortness of breath Obstructive sleep apnea: snoring, daytime somnolence, witnessed apneic events Difficult airway: recognized difficult airways
Endocrine	Diabetes mellitus: fasting blood glucose > 300 mg/dL or evidence of end organ damage Thyroid: goiters evaluated by computed tomography and surgeon before surgery; complicated lesions (ie, intrathoracic involvement) Obesity: body mass index > 35
Neurologic	Cerebrovascular events: any residual deficit or frequent transient ischemic attacks Gait disturbance: cancel all Seizures: new onset or history of epilepsy
Obstetrical/ gynecologic	Pregnant: cancel all Breast, ovarian, or cervical cancer: cancel all Postpartum: if < 2 months postpartum; counsel re: breastfeeding
Oncology	Any current cancer: cancel all
Pediatric	Age: < 6 months Syndromic appearance: cancel all CHD: known lesions/any cyanotic history; refer all murmurs to cardiologist for evaluation Upper respiratory infections: symptoms within past 4 weeks

CAD: coronary artery disease
CHD: congenital heart disease
CHF: congestive heart failure
DBP: diastolic blood pressure
HTN: hypertension
MI: myocardial infarction
PP: pulse pressure
SBP: systolic blood pressure

Data source: King HC, Baker W. Pacific Partnership 2008: the surgical mission, surgical screening process, and the anesthetic management of uncontrolled, untreated hypertensive patients. *Mil Med*. 2010;175(1):33–40.

negative-pressure ventilation are required to prevent the spread of the disease to others. Though isolation is available on the hospital ships, it is limited to the intensive care unit, making it difficult to treat these patients. Because of these limitations, patients with active tuberculosis infection are generally not candidates for elective surgery aboard. All patients and escorts are evaluated for tuberculosis and receive a chest radiograph during the prescreening process. The presence of active disease generally results in case cancellation. If the patient is accepted, additional preparation is required.

Intraoperative Anesthesia Management

Anesthetic Techniques

As Role 3 facilities, both USNS *Mercy* and USNS *Comfort* are capable of providing general and regional anesthesia to adult and pediatric patients in a manner similar to that of shore-based hospitals. In addition to standard equipment, both ships have devices for difficult airway management, including fiberoptic bronchoscopes and GlideScopes (Verathon Inc, Bothell, WA), and portable ultrasound units for use with

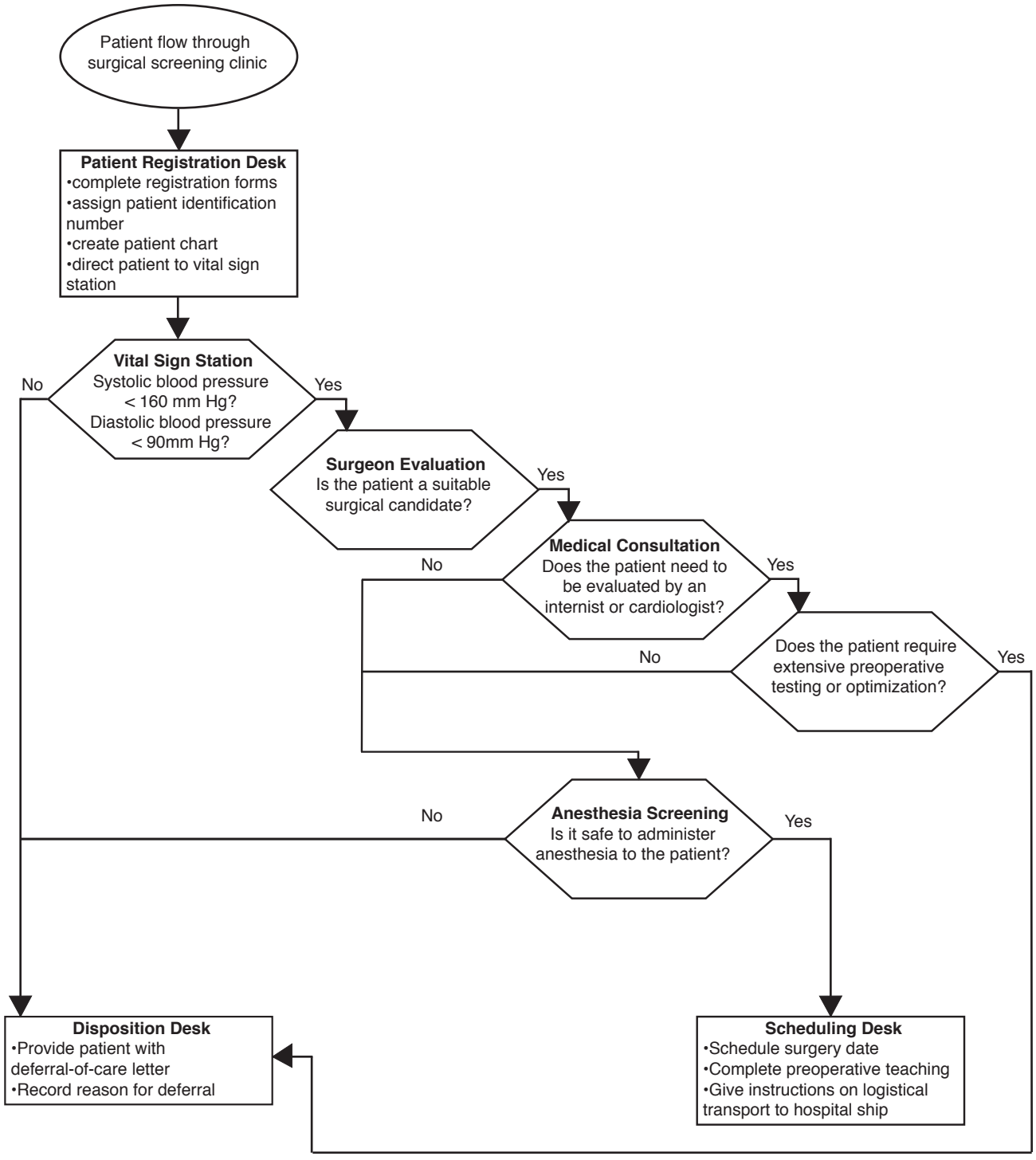

Figure 41-3. Surgical screening patient flowchart, USNS Mercy (T-AH 19), Pacific Partnership 2008.
Reproduced from: King HC, Baker W. Pacific Partnership 2008: the surgical mission, surgical screening process, and the anesthetic management of uncontrolled, untreated hypertensive patients. *Mil Med*. 2010;175(1):34.

regional anesthesia procedures and insertion of central vascular catheters.

Reported data shows the majority of cases (approximately 83%) are performed under general anesthesia. The use of local anesthesia with and without sedation accounts for the rest. The majority of the local anesthesia cases are retrobulbar blocks administered for ophthalmology procedures. With the exception of a few cases, neuraxial techniques and peripheral nerve blocks are largely performed as adjuncts to general anesthesia and management of postoperative pain. During past missions, such as Partnership for the Americas and Continuing Promise 2009, epidural and caudal procedures were performed for postoperative pain management in patients who underwent abdominal hysterectomy, open cholecystectomy, and pediatric inguinal hernia repair.[5]

Overall, the use of regional anesthesia is limited during an HA mission for several reasons. Language barriers make communication and patient cooperation more challenging. Interpreters are helpful but not always available. The majority of surgical procedures are low-risk cases that do not require regional anesthesia for postoperative pain management. These patients are typically discharged within 24 to 48 hours following surgery. Additionally, the limited time at each site makes it nearly impossible to evaluate patients for late complications, and host nations often lack the resources to address these situations.

In contrast to the HA mission, the use of regional anesthesia is beneficial during a DR operation despite the aforementioned challenges. The number of patients requiring acute care can be significant. Trauma patients undergo more extensive surgery and require a more intense level of pain management. The use of regional anesthesia addresses the needs of these patients and helps decrease the burden on staff, who can quickly become overwhelmed. The number of injured from the 2010 earthquake in Haiti was staggering, and the USNS *Comfort* became the busiest orthopedic trauma hospital in the region. Using nerve stimulators and ultrasound guidance, peripheral nerve blocks (Table 41-3) were performed in children and adults both awake and under general anesthesia without any known complications. The benefit of regional anesthesia in caring for these patients was tremendous, demonstrating the utility of regional anesthesia in the disaster response situation.

TABLE 41-3

PERIPHERAL NERVE BLOCKS ADMINISTERED IN OPERATION UNIFIED RESPONSE, USNS *COMFORT*

Block Type	Number
Femoral	60
Sciatic	30
Lumbar plexus	1
Popliteal/saphenous	4
Interscalene	1
Supraclavicular	6
Radial/median/ulnar	4
Transabdominal plane	1

Blood Products

The availability, storage, and utilization of blood products are significant concerns aboard the US military's hospital ships and other vessels with medical capabilities. In contrast to land-based facilities, greater challenges exist with storage capacity and resupply issues at sea. To address these issues, the US military has the ability to utilize options not available in civilian practice. One of these is the use of frozen packed red blood cells. Though approved for use by the Food and Drug Administration, this product may have quality degradations (class D blood > 10 years old) and needs special reagents for processing before use. During Operation Unified Response in 2010, a total of 348 units of packed red blood cells were used, and of these, 46 units were frozen. No adverse events from the use of frozen blood were reported. Another option available to the military is instituting the "walking blood bank," a program in which military members are typed and cross-matched to serve as a readily available pool for emergency whole blood donations.

In contrast to the combat or disaster situation in which blood transfusion is frequently necessary, the goal during an HA mission is to minimize the use of blood products, so elective surgeries with the potential for significant transfusion requirements are not routinely performed.

Postoperative Care Considerations

Management

Cases that require a prolonged and intensive postoperative recovery are typically not performed during an HA mission. Though medical and surgical capabilities on both hospital ships are extensive, host nation resources are often very limited, making transfer of care difficult. The majority of cases performed usu-

ally require a level of care equivalent to ambulatory surgical procedures, and these patients are admitted to ward beds. Patients who require any greater degree of care and monitoring (eg, abdominal hysterectomy, open cholecystectomy, thyroidectomy) are sent to the intensive care unit. In the disaster situation, the treatment and care of trauma patients is more complex, involving ongoing resuscitation and pain management. Many patients require intensive care monitoring and postoperative mechanical ventilation.

Patient Transport

The transport of patients (and staff) is a complex process and must be considered during mission planning. It is affected by whether the hospital ship is anchored in port or off shore. If the hospital ship is anchored pier-side, transport time for patients (and staff) is greatly decreased. However, due to the size and excessive draft of these ships, access to many ports is limited. Consequently, the hospital ships are frequently anchored off shore and patients are transported by boat or helicopter. Small boats can only transport a limited number of people and operations are subject to sea conditions. Regulations permit the landing of only one helicopter at a time on the ship's flight deck. These limitations significantly increase the number of trips required to transport patients to and from shore. If transport time is excessive, it can affect the ability to deliver care.

AUSTERE AND RESOURCE-LIMITED ENVIRONMENTS

The previous sections describe the approach undertaken by the US military operating from well-equipped and resourced ships. The situation can be very different in austere, resource-limited environments. These situations are well described by Robin Coupland, a Red Cross surgeon, writing in the *British Medical Journal*[11] about experiences in Afghanistan in 1992. He describes how the surgical team could only work for a few hours each day (due to the proximity of the battle), so they gave priority to patients needing operations for abdominal wounds. Coupland notes that patients rushed into the operating room with severe injury usually died due to insufficient preoperative resuscitation, and others died postoperatively due to lack of postoperative care. This is a clear contrast to the current situation in NATO hospitals in Afghanistan. A key lesson from Coupland's paper is that the severely injured die unless adequate people and infrastructure are there to care for them, and that triage must be done with this in mind or precious resources will be wasted. The extensive resuscitation and surgery that current improvised explosive device casualties receive at US and UK hospitals in Afghanistan could not be undertaken in a resource-limited environment such as most NGO hospitals.

The Red Cross publication *Hospitals for War Wounded* describes three triage categories[12(p91)]: Category I, priority for surgery, are those needing urgent surgery and with a good chance of recovery. Category II, no surgery, are those with either minor injury or nonsurvivable injury. Category III are those who need surgery but can wait. The book also gives guidance for managing a limited supply of blood, including setting limits of four to six units for patients with a hemoglobin concentration less than 8.0 g/dL, or calculating that every 100 war-wounded casualties admitted to the hospital will need about 45 units of blood.

The current edition of the Red Cross's *War Surgery*[13] contains basic guidance on anesthesia and analgesia. The following are key areas to note:

- In austere environments equipment such as suction apparatus will be manually operated. Intraoperative ventilation will also be by hand.
- Oxygen concentrators will be used to preserve gas cylinder supplies.
- The type of surgery and resuscitation that can be carried out in an austere situation will be dictated by availability of equipment, consumables, and utilities (water, power, and gases).
- The security of the environment (location of fighting, timings of curfews, travel constraints) will also affect what a surgical team can and cannot deliver.

All of the above conditions influence the choice of anesthesia that can be delivered, and the choice of anesthesia in turn influences the conduct of surgery.

Anesthesia Techniques

Anesthesia techniques common in austere settings include the following:

- Infiltration of local anesthetic, single shot nerve blocks, and single shot spinal or epidural techniques. These may or may not be supplemented by systemic analgesics and anesthetics.
- Ketamine administered orally, intramuscularly, or intravenously. Intramuscular ketamine

can be (and is) used as the sole agent in many resource-limited environments. Intravenous ketamine may be given as intermittent bolus or continuous infusion from a drip. Benzodiazepines may be used along with ketamine to minimize hallucinations and dreams associated with ketamine, although they reduce the airway-protective reflexes that ketamine usually leaves intact. Atropine may also be used with ketamine to dry oral secretions, but this effect must be balanced against the associated tachycardia.

- Anesthesia produced by inhalational agents and delivered by draw over apparatus with or without supplement by the techniques described above.

In turn the patient may be allowed to breathe spontaneously or undergo muscle relaxation, endotracheal intubation, and manual ventilation depending on the surgery planned and the resources available. Examples of intramuscular and intravenous regimes used by the authors are given in Table 41-4**.** Many others can be found in the published literature.

To give a practical example of these techniques in use, one of the authors (PFM) has observed the following approaches to emergency cesarean section while working with aid agencies in Africa and Asia:

- Intramuscular ketamine followed by local infiltration of skin and muscle layers.
- Single shot spinal anesthesia
- Intravenous ketamine followed by spontaneous ventilation of air, oxygen, and halothane.
- Rapid sequence induction with thiopentone and suxamethonium, and endotracheal intubation followed by manual ventilation with air, oxygen, and halothane.

TABLE 41-4

ANESTHESIA REGIMES FOR AUSTERE ENVIRONMENTS

Delivery (Based on Required Duration)	Regime Examples
Induction and bolus maintenance for short procedures	• Midazolam 5 mg or diazepam 2–5 mg IV with a small dose of morphine IV, followed by ketamine 80–100 mg IV over 20 seconds. Intermittent boluses of ketamine IV, one quarter of the induction dose, every 15 minutes. Doses of benzodiazepines or opioids as necessary added in response to increasing vocalization or purposeful movements with surgical stimuli.[1] • Midazolam 0.07mg/kg IV, followed 2 minutes later by ketamine 1 mg/kg IV. In mass casualty events, ketamine 7 mg/kg IM may be used.[2] • Atropine, diazepam, or midazolam followed 3 minutes later with ketamine 1–4 mg/kg IV, with intermittent boluses.[3] • 1–2 mg/kg ketamine IV over 60 seconds providing anesthesia for approximately 10 minutes.[4] • 10 mg/kg ketamine IM given 5–10 minutes before surgery providing anesthesia for approximately 12–25 minutes.[4]
IV infusions bags	• Ketamine infusion (0.5 mg/mL ketamine in a liter of normal saline) titrated to effect following IV ketamine bolus induction.[5] • 40 mL of 1% propofol, 250 μg fentanyl (5 mL of 50 μg/mL) and 250 mg ketamine (5 mL of 50 mg/mL) added to 50 mL saline. This gives an infusion of 4 mg propofol, 2.5 mg ketamine, and 2.5 μg fentanyl per milliliter of solution. Using a 20 drop/mL giving set (tubing allows 20 drops per mL), use one drop per second to deliver 150 μg/kg/min propofol infusion (assuming patient weight of 80 kg).[6]

IM: intramuscular
IV: intravenous

(1) Paix BR, Capps R, Neumeister G, Semple T. Anaesthesia in a disaster zone: a report on the experience of an Australian medical team in Banda Aceh following the "Boxing Day tsunami." *Anaesthes Intensive Care*. 2005; 33:629–634. (2) Grande CM, Baskett PJ, Donchin Y, Wiener M, Bernhard WN. Trauma anesthesia for disasters: Anything, anytime, anywhere. *Crit Care Clin*. 1991;7:339–361. (3) Dalenius E, Asaker BM. Training for wartime anesthesia in Sweden. *AANA J*. 1989;57:250–256. (4) Wood P, Mahoney PF. Anaesthesia and analgesia. In: Ryan J, Mahoney PF, Greaves I, Bowyer G. Conflict and Catastrophe Medicine: A Practical Guide. London, England: Springer; 2002:341–351. (5) Giannou C, Baldan M. *War Surgery: Working With Limited Resources in Armed Conflict and Other Situations of Violence*. Vol 1. Geneva, Switzerland: International Committee of the Red Cross; 2009; 305–317. (6) Mahoney PF, McFarland CC. Field anaesthesia and military injury. In: Smith CE, ed. *Trauma Anesthesia*. 1st ed. Cambridge, England: Cambridge University Press; 2008: 343–359.

Similar approaches have been successfully used for managing anesthesia for other procedures such as limb amputation.

Postoperative Care

The central role of postoperative care is illustrated by Coupland.[11] Postoperative care can be considered as immediate and longer term. Immediate care includes fluids, analgesics, antibiotics, and oxygen, and the people to deliver them. Longer term care in the current US and UK military systems means strategic air evacuation to Role 4 and Role 5 hospitals Evacuation may not be possible in austere environments and future conflicts, so the likely postoperative and critical care requirements of a severely ill or injured patient must form part of the triage process and decision on whether they should or should not have initial surgery.

Immediate postoperative care may have to be delegated to people with minimal experience or limited reading and mathematical skills. A number of approaches can be used to help these personnel, such as drawing simple clock pictures on intravenous fluid bags (to show when different volumes need to have completed) or sun and moon pictures to illustrate day and night timings for care or medication delivery. Surgical ward routines are described further by the Red Cross.[12]

Longer term postoperative care may include prolonged critical care and the complex rehabilitation pathway needed by the current generation of allied war wounded. If a health care system cannot provide this level of care, providers must carefully consider whether to begin surgery for highly complex injuries.

CONCLUSION

Today, medical personnel in the military are deployed in a variety of situations: combat operations, HA missions, and disaster relief efforts all around the world. As they do in combat operations, anesthesia providers play a key role in the HA mission, assisting in the coordination and delivery of surgical services. When working in an austere or resource-limited environment, anesthesia providers must adapt their techniques accordingly and understand the constraints that may be placed on patient care. Although the focus of this chapter is the large-scale HA missions that are conducted aboard the US military's hospital ships, much of this information can also be applied to other missions with similar objectives.

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Chapter 42

ETHICAL CHALLENGES OF DEPLOYED MILITARY CRITICAL CARE

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INTRODUCTION

Ethics is a branch of philosophy that deals with the study and practice of moral choices and values, and the judgments underpinning these choices and values. Medical ethics is the application of this discipline to moral choices in medicine. This chapter discusses medical ethics in deployed critical care. Of the many approaches to medical ethics, the most widely recognized are deontology, utilitarianism, and the "four principles" approach. These will be described in turn. While ethics proposes what *should* be done, the law enforces what *must* and *must not* be done. Hence, legal sources of medical ethics will also be discussed. The chapter will then broadly consider ethical problems encountered in critical care in the deployed setting, provide examples of potential ethical conflicts, and propose some possible mechanisms to resolve these conflicts.

ETHICAL MODELS

Deontology

Deontology was espoused by 18th century philosopher Immanuel Kant. The term's etymology is from the Greek "deon," meaning duty. Often called "duty ethics," deontology holds that actions are right or wrong depending upon their conformity with moral principles and regardless of their practical consequences. For example, a deontologist might argue that it is always wrong to lie, even if the lie results in a positive outcome. The most commonly cited principle that might be regarded as deontological in medicine is the principle of the sanctity of life, which states that the value of life exceeds all other values and that all lives are of equal value. This position is rarely adopted by medical practitioners, though none would be likely to deny that all lives have value.

Utilitarianism

In contrast to deontology, utilitarianism holds that the right course of action is the one that maximizes the overall "good" consequences of the action. It is thus a form of consequentialism, meaning that the moral worth of an action is determined by its results. Utilitarian philosophy may be traced to the 18th and 19th century British thinkers John Stuart Mill and Jeremy Bentham. Utilitarian principles are classically seen in the concept of triage, as proposed by Baron Dominique-Jean Larrey (1766–1842), surgeon to Napoleon.[1] Contemporary utilitarian bioethics theory continues to recommend directing medical resources where they will have most effect for good, and is used in healthcare planning, including the use of quality-adjusted life years, but the concept is controversial in many other areas.

The "Four Principles"

The "four principles" approach to medical ethics, developed in the United States by Beauchamp and Childress,[2] offers a universal approach to ethical decision-making in healthcare that can be applied by everyone, regardless of personal politics, religion, or philosophy. The four principles are as follows:

(1) **Autonomy.** The principle of autonomy requires that the medical practitioner respect the decision-making capacities of autonomous persons.
(2) **Beneficence.** The principle of beneficence requires that a practitioner take action for the good of patients.
(3) **Non-maleficence ("primum non nocere").** Non-maleficence requires that a practitioner avoid causing harm. All treatment, even if minimal, has the potential for harm, and the harm should not outweigh the benefits of treatment.
(4) **Justice.** The principle of justice requires that the benefits, risks, and costs of healthcare be distributed fairly. This principle is often considered to be about resource allocation—the notion that because resources are not infinite, they must be allocated in a fair and equitable manner.

The difficulty with the "four principles" approach is that it is not clear how to resolve situations in which the principles are in conflict, as shown in the examples given below.

Despite this variety of approaches, a broad consensus about what constitutes ethical behavior in medicine has existed since the 5th century BCE, when the code of Hippocrates was written.

MEDICAL ETHICS IN TIMES OF WAR

German physicians famously violated Hippocratic principles during the National Socialist period, participating inter alia in horrific medical experimentation during the Holocaust. Hippocratic principles were

therefore reinvigorated after the Second World War in the World Medical Association's "Regulations in Times of Armed Conflict," a list of ethical guidelines for doctors practicing in war zones, first produced in 1956.[3] The World Medical Association is an international organization of which the American and British medical associations are constituent members. The regulations state that "the primary task of the medical profession is to preserve health and save life," and that "physicians have a clear duty to the sick and injured." Medical attention should be given based on clinical need, not on any other criterion. Furthermore, regulation 1 states that "medical ethics in times of armed conflict is identical to medical ethics in times of peace. . . . If in performing their professional duty, physicians have conflicting loyalties, their primary obligation is to their patients." This last sentence involves a major source of ethical conflict for military physicians: the so-called "dual loyalty" problem, in which the physician has potentially conflicting duties to the patient and to the chain of command. This conflict will be discussed in further detail below.

Other responses to the unethical medical experimentation in World War II came with the Nuremberg Code (1947) and subsequently the Declaration of Helsinki (1964), both of which set out principles for the ethical conduct of medical research on humans. Central to all these documents is an inviolable respect for the unique value of human life. That medical ethics not be subordinated to political imperative is the responsibility of both doctors and civil society.

The Law of Armed Conflict

The law of armed conflict is a body of international law based on treaties and customs whose main purpose is to protect combatants and noncombatants from unnecessary suffering, and to safeguard the fundamental human rights of persons who are not, or are no longer, taking part in the conflict, such as prisoners of war; the wounded, sick, and shipwrecked; and civilians. It is traditionally divided into two parts, each named after the city where the law was devised. Hague law (based on the Hague Declaration of 1899 and Hague Convention of 1907) is concerned with how military operations are conducted, for example, prohibiting the use of expanding bullets. Geneva law, which is more relevant to military medicine, sets out requirements for the humanitarian treatment of victims of war. The first Geneva Convention of 1864 was updated in 1906 and 1929. The Conventions were revised completely in 1949, with four new Conventions dealing with the protection of (1) the wounded and sick (replacing the Conventions of 1864, 1906, and 1929); (2) the wounded, sick, and shipwrecked at sea (replacing the Hague Convention of 1907); (3) prisoners of war (replacing the Convention of 1929); and (4) civilians. Two further protocols were added in 1977 to give greater protection to victims of international and internal armed conflicts, and another in 2005 allowing emblems other than the Red Cross to be used as symbols of humanitarian relief and protection.

The Hague and Geneva Conventions and other relevant international law have been summarized by the United Kingdom (UK) Ministry of Defence in the *Joint Service Manual of the Law of Armed Conflict*[4] which provides definitions of the wounded and sick, outlines the duty of care to the wounded and sick based on clinical need, discusses permitted medical treatments, and emphasizes the importance of consent (Exhibit 42-2). The US Department of Defense Law of War Program provides an equivalent US source.[5]

While the ethical and legal frameworks described above may seem clear and consistent, in reality they do not provide answers to some of the practical difficulties the deployed intensivist routinely faces. Furthermore, very little academic literature deals with these particular ethical problems. In recent years, the literature has focused on ethical dilemmas faced by the military physician who is witness to torture or maltreatment of detainees.[6,7] An additional difficulty is that no clear mechanism exists for dealing with ethical difficulties that cannot be resolved in the field or referred up the military chain of command, leaving the deployed military physician somewhat isolated in terms of ethical and legal oversight. Examples of such issues are provided later in the chapter.

Standards of Deployed Medical Care

NATO now requires military medical services, wherever possible, to provide standards of medical care for military casualties that are equivalent to, or surpass, those delivered in the home nation, despite the austere environment.[8] This standard could potentially be offered to a large population of both coalition military and local national casualties. A previous consideration of standards of medical care for civilians argued that for broader reasons of medical infrastructure development, the standard of military medical care for local nationals should be that of the host nation.[9] These authors assert that host nation medical development may be undermined by the presence of a high quality foreign military service. This argument is not usually applied to emergency care and is difficult to justify when life is at risk. The case can therefore be made that critical care should be provided to the same standard as critical care in the developed world for all eligible civilians. However, in the military context, allowing the field hospital to

EXHIBIT 42-1

LAW OF ARMED CONFLICT

Definition of the Wounded and Sick

"The wounded and sick are 'persons, whether military or civilian, who, because of trauma, disease or other physical or mental disorder or disability, are in need of medical assistance or care and who refrain from any act of hostility' [Geneva Conventions and Protocols, Additional Protocol I, 1977, Article 8(a)]. The definition goes beyond persons wounded on the battlefield to encompass anybody in need of medical treatment. That includes 'maternity cases, new-born babies and other persons who may be in need of immediate medical assistance or care, such as the infirm or expectant mothers' who refrain from any act of hostility [Geneva Conventions and Protocols, Additional Protocol I, 1977, Article 8(a)]. Those who carry on fighting despite their wounds are not included in the wounded and sick category."

Protection and Care of the Wounded and Sick

"The wounded and sick are to be protected and respected. They may not be attacked. They must be treated humanely. They must be provided with medical care. They may not willfully be left without medical assistance nor exposed to contagious diseases or infection. Priority of treatment is dictated by medical need only [Geneva Convention I and II, Article 12; Geneva Convention III, Article 13; Geneva Convention IV, Article 27; Additional Protocol I, 1977, Articles 9, 10 and 11]. Violence and biological experiments are forbidden. Women must be treated with special respect [Geneva Conventions and Protocols, Additional Protocol I, 1977, Article 76(1)] and no less favourably than men [Geneva Convention I and II, Article 12; Additional Protocol I, 1977, Article 10.6]." However, "There is no absolute obligation on the part of the military medical services to accept civilian wounded and sick—that is to be done only so far as it is practicable to do so. For example, the commander of a field hospital placed to deal with casualties from an impending battle would be entitled to refer non-urgent cases elsewhere, even if the hospital had the capacity to treat them at the time. Once the treatment of a civilian patient has commenced, however, discrimination against him on other than medical grounds is not permissible."

Permitted Medical Treatment

"Any medical procedure which is not indicated by the state of health of the person concerned and which is not consistent with generally accepted medical standards which would be applied under similar medical circumstances to persons who are nationals of the Party conducting the procedure and who are in no way deprived of liberty' is prohibited [Additional Protocol I, 1977, Article 11(1)]."

Right to Refuse Consent

"Persons protected have the right to refuse any surgical operation. In cases of refusal, medical personnel must try to obtain 'a written statement to that effect, signed or acknowledged by the patient' [Additional Protocol I, 1977, Article 11(5)]. The right still exists to carry out surgery necessary to save life in an emergency without obtaining the consent of the patient in accordance with medical ethics and on the same basis as for the general population under domestic law."

Quotations reproduced from: United Kingdom Ministry of Defence. *The Joint Service Manual of the Law of Armed Conflict*. Shrivenham, UK: Ministry of Defence; 2004. Joint Service Publication 383.

focus too much on civilian casualties may render it incapable of fulfilling its primary mission of treating service member casualties. Balancing these different priorities can be very difficult.[10]

POTENTIAL ETHICAL CONFLICT IN MILITARY CRITICAL CARE

Resource Allocation and Dual Loyalties

Triage

The problem of critical care prioritization and allocation of scarce resources is common to both the civilian and military intensive care unit (ICU). However, the deployed context involves particular constraints that do not arise in the civilian environment. The deployed field hospital is small and configured to support military operations. It may need to be mobile and usually must be capable of operation independent of other

secondary care facilities. The ICU may be quite small, with as few as two beds.[11] The unit's function is to provide critical care support to entitled persons, according to an established eligibility matrix, specific to the military operation. For casualties with the potential for rearward evacuation while still critically ill, a holding policy of up to 48 hours is common. Local nationals are likely to have a significantly longer length of stay, and the capacity of the ICU may be overwhelmed. In civilian practice, capacity constraints may be mitigated either by expansion of local capacity or by inter-hospital transfer. Both of these possibilities may be more difficult to achieve in the military environment.

These constraints may make triage at the point of admission to the ICU necessary. In civilian hospitals triage is used to prioritize the care of patients according to an equitable and responsible allocation of resources. The goal is to attend first to those most in need of medical attention, placing individual well-being above any broader concern. Such an approach requires the hospital to be well resourced. In most circumstances military triage follows the same general philosophy; however, critical care triage in the resource-limited deployed environment involves a choice among patients who may all benefit from emergency treatment. Patients who are not admitted for critical care are likely to have a higher mortality rate,[12] some of which may have been prevented by admission. The UK General Medical Council guidance on critical care triage states that if there are constraints on resources, the doctor must "provide as good a standard of care as you can for the patient, while balancing sometimes competing duties towards the wider population, funding bodies and employers."[13] Guidance is explicit about withdrawing or withholding treatments because of resource constraints: the doctor "should not withdraw or decide not to start treatment if doing so would involve significant risk for the patient and the only justification is resource constraints. If you have good reason to think that patient safety is being compromised by inadequate resources, and it is not within your power to put the matter right, you should draw the situation to the attention of the appropriate individual or organisation." In the deployed ICU, this guidance often translates into triage decisions being made by a group of senior clinicians, involving the intensivist, the medical director, and the referring surgeon or physician.

Reverse Triage

In the extreme circumstances of battle, military physicians may reverse the triage procedure to focus care on those who are lightly injured or most likely to need the fewest resources to survive, allowing the most severely injured to die.[14] Such an approach allows the lightly injured to return to duty rapidly and avoids using valuable resources to treat patients who need intensive care. This approach was most famously used by the British in North Africa in 1943, when combat effectiveness of the field army was severely hampered by an epidemic of gonorrhoea. A decision was made to use penicillin, then very scarce, to treat soldiers with gonorrhoea rather than those with wound infections. The purpose was to return as many soldiers to health as quickly as possible to prepare for the invasion of Italy.[15] In the future, a decision to treat lightly wounded soldiers in preference to the more severely injured might be taken, for example, in circumstances when the field army is about to be overrun, in order to maintain fighting strength as efficiently as possible. Such decisions are made against the best interests of the severely wounded soldiers, prioritizing the interests of the nation or the group above the individual. It is here that the "dual loyalty" problem is at its most stark, forcing the military physician to balance duty to the individual soldier against duty to the chain of command.[16,17] Fortunately such circumstances have not occurred in coalition forces' field hospitals in recent conflicts.

Best Interests

The population of patients admitted to the deployed ICU may be drawn from several different demographic groups: coalition service personnel; local combatants; UK and foreign civilian contractors; UK and foreign civilian journalists and other noncontracted persons; and local civilians including the elderly, pregnant women, and children. These groups have differing healthcare needs, and on leaving the field ICU will have differing access to further medical interventions, with varying quality and clinical governance in receiving facilities. Coalition casualties will be evacuated within 48 hours to state-of-the-art tertiary referral centers in their home countries. Foreign nationals and local civilians may not have such facilities available. In many cases this will mean that either noncoalition casualties must remain in the ICU beyond 48 hours, or that transfer is effected to local facilities with attendant uncertainty about the quality of ongoing clinical care in the receiving unit. The quality and nature of rehabilitation after critical care will also differ. UK and US service members will be offered advanced rehabilitation medicine and prosthetics.[18] For noncoalition casualties, rehabilitation may be of lower quality or nonexistent.

These factors raise another ethical dilemma for the deployed critical care physician: how to judge whether treatment is in the patient's best interests.

This principle is particularly important in the emergency treatment of casualties with impaired capacity at the time of presentation, for whom treatment "is immediately necessary in order save their life or to prevent a serious deterioration"[19] and is justified on the grounds of best interests. However, for the benefits of treatment to outweigh its costs, casualties must have a chance of being restored to a state of health that is acceptable to them. This goal may depend upon later rehabilitation, which is not in the control of the critical care physician. Where rehabilitation is not available, the preservation of life by surgical and critical care interventions may permit a casualty to survive the critical phase with unacceptable burdens of ill health. The logical consequence of this problem is that in the case of two casualties with identical injuries and no comorbidities, it may be appropriate to resuscitate only the casualty who has access to rehabilitation. Such a decision might be considered unjust or in conflict with triage guidance.[3] It might be easier to accept if surgery and critical care are not seen in isolation, but rather as part of a healthcare continuum beginning with injury and ending with reentry into the community. Although all aspects of this process may be provided for coalition casualties with coalition resources, not all may be available for noncoalition casualties.

Culture and Autonomy

Ethical medical practice requires doctors to understand the values and beliefs of the people they treat, and to be aware of cultural differences. This requirement is particularly important in the case of unconscious or emergency casualties who are temporarily or permanently without capacity, or those who have capacity but are unable to communicate their preferences due to language barriers. However, nearly all religions and cultures agree that taking all necessary measures to maintain life in an unconscious casualty is appropriate, as long as medical treatment is in the patient's best interests. This is the position of the UK General Medical Council, English law in the Mental Capacity Act 2005, and the code of ethics endorsed by the First International Conference on Islamic Medicine held in Kuwait in January 1981. The Conference's code also states that it is permissible for a non-Muslim doctor to treat a Muslim when the patient's condition and skills of the doctor necessitate it. Furthermore "it is permissible for the purpose of treatment to look at hidden and private parts of the body" as "necessities override prohibitions."[20] Despite this declaration, in some Islamic societies women routinely refuse to be examined by a male doctor, even if the consequences are potentially life threatening.

In most cultures doctors will respect a refusal of treatment medically considered to be in the patient's best interests as long as the patient is competent to make the decision. For example, in the UK, doctors will not administer blood to a Jehovah's Witness who has given a competent refusal, even if this means that the patient will die a preventable death. To give such treatment (or indeed any treatment) without consent ignores the patient's autonomy and could make the doctor legally liable to a charge of battery or even assault. In a deployed environment, however, it is often difficult to be certain of the patient's capacity when doctor and patient do not speak the same language or when consciousness is impaired by injuries or illness. In recent conflicts field hospitals have had interpreters for speaking with local nationals; however, gaining consent via an interpreter is difficult, and assessing the validity of a decision to refuse treatment is even more difficult.

Hypothetical Scenarios

Resource Allocation

The intensive care consultant is managing a four-bed facility with all beds occupied. All are ventilated, and there is no possibility of rearward evacuation to a critical care facility. Three casualties are local nationals, including one child. The nearest local medical facility is open to admissions, but can provide only ward-level care. The fourth casualty is a coalition service member, who has multiple cavity injuries and is receiving regular blood product transfusion and ventilator support. He is currently judged too unstable to transfer and may require further surgery within the next 6 hours, depending upon progress. No other coalition medical facilities are within the theater of military operations. The intensive care consultant is informed that a coalition soldier has been wounded at a location close to the medical facility and is inbound by road ambulance. The injuries are severe and critical care will be required.

The practical possibilities are:

- Temporarily expand the critical care resources, either inside or outside the ICU. This may be possible if advanced planning has allowed for expansion beyond four beds for a limited period. However, the same scenario might arise when an expansion has already occurred and no additional nurses are available.
- Transfer one of the other critical care casualties to a local facility. In all cases this is likely to involve deterioration in the person's condition. Failure to provide critical care to the

incoming casualty, however, will also cause deterioration. If this option is chosen, the difficult decision of which patient to transfer must be made.

- Principles in conflict:
  - Beneficence (the obligation to provide life saving treatment to all those who need it).
  - Non-maleficence (the duty to prevent any patient from coming to harm). This is particularly relevant to the local national who may be transferred to a lower level of care local facility and could die as a consequence, and perhaps even more relevant to the child, because pediatric critical care expertise is likely to be less developed than adult critical care expertise in the local facility. It is also relevant to the inbound casualty to whom a duty of care is owed.
  - Justice (the need to allocate resources fairly and equitably). Defining "fair" and "equitable" in such extreme circumstances is difficult. Even if it is possible to keep all the casualties in the deployed field hospital, overworking staff for a period of time to avoid a nonclinical transfer to a local facility may result in poorer care for the next group of casualties.

Best Interests

The intensive care consultant is managing a four-bed facility with one bed occupied. The casualty is a local national combatant with bilateral lower limb traumatic amputations and acute respiratory distress syndrome. He is ventilated and recovering. The intensive care consultant is informed that an improvised explosive device has been inadvertently triggered nearby, causing injury to a UK service member and a local civilian adult male. On arrival at the emergency department, the two casualties are assessed. The service member has suffered traumatic amputation of three limbs, genital injuries, and significant facial, including eye, injuries. The patient is deeply unconscious but brainstem reflexes and movement have been noted. The decision is taken to attempt resuscitation, followed by preparation for either a computed tomography (CT) scan or emergency surgery, depending upon the response to resuscitation. The local civilian casualty has suffered similar injuries and a decision is taken to attempt stabilization in the same way. Both casualties are stabilized sufficiently to permit a trauma series CT scan examination. Both have cerebral contusions judged potentially survivable, but with significant risk of functional impairment. Both may be offered surgery and critical care with a reasonable possibility of survival and independence from organ support, but they would be left with a heavy burden of chronic ill health. Local medical services do not have a well developed rehabilitation capability. All attending clinicians agree that it is appropriate to undertake surgery and critical care for the UK service member, whose rehabilitation will be extensively supported in the UK. One of the attending clinicians asks whether surgery and critical care are appropriate for the local civilian, given that he is likely to have a significant burden of chronic ill health or die later from complications of his injuries without the benefit of sophisticated rehabilitation.

The practical possibilities for the civilian are:

- Aggressive resuscitation, surgery, and critical care. This course of action would be in accordance with the presumption that life should be sustained whenever possible. It may, however, commit this casualty to a protracted period of suffering, followed by a delayed death from the complications of his injuries.
- Palliative care. This requires an assumption that the state of health realistically achievable at discharge from medical care would be unacceptable to the patient. This assumption must of course be made without the opportunity to consult the patient, and quite possibly with no opportunity to consult a relative.
- Principles in conflict:
  - Beneficence (the duty to provide medical care to any patient according to clinical need).
  - Non-maleficence (the duty to do no harm and prevent patients from coming to harm).
  - This scenario presents a classical "best interests" assessment problem: how to assess the benefits and burdens of treatment to the local national, making sure his medical, psychological, and social best interests have been taken into account. Such an assessment might be considered impossible in the acute situation, and therefore treatment to maintain life should continue.

Culture and Autonomy

A female Muslim patient is admitted to the emergency department with an abdominal gunshot wound. She has significant blood loss but is currently conscious, although unable to communicate in a comprehensible manner. She is continuing to deteriorate and needs urgent resuscitation and surgery. A male family member is present, and the interpreter says he is beg-

ging for the patient not to be touched by male staff. It is obvious from the man's demeanor he feels strongly about this, and stories have been heard of women being badly treated on returning to home after receiving medical care from male doctors. The only female medical staff available are an emergency department consultant (attending) and an anesthetic specialist registrar (resident). However, there are enough female nursing staff to care for the patient.

The practical possibilities are:

- Ask all male staff to leave the trauma bay. Manage the patient with the two available female doctors, who should have the ability to assess and resuscitate her (although not as efficiently as if more doctors were available). This still leaves the problem of how to proceed in the operating room, where male staff will need to treat the patient under general anesthesia if she is to survive.
- Ask all male relatives to leave the trauma bay and continue treatment as normal, with both male and female staff present. Afterwards, use the interpreter to discuss the Islamic Code of Military Ethics with the male family member (and patient if possible) and explain that it was necessary for male non-Muslim doctors to treat the patient due to the severity of her condition and the skills of the doctors present. Principles in conflict:
  - **Autonomy.** The refusal of treatment by a male relative is not a competent refusal on behalf of the patient and would not be respected in a developed nation.
  - **Beneficence.** It is clearly in the patient's medical best interests to be treated by the normal number of staff in the normal fashion. Any other arrangement may result in compromised care.
  - **Non-maleficence.** At the same time, it is better for the patient not to inflame sensitivities or put her at risk of ostracism after hospital discharge because she has been touched by non-Muslim males.

DEVELOPING AN ETHICS OF MILITARY CRITICAL CARE

The ethical issues raised in this chapter are not theoretical; they are practical matters of concern to military critical care providers. As military critical care matures, an ethical framework must be built to resolve problems such as those outlined above. The principles of medical ethics are well established. Military critical care providers should not attempt to redefine these principles, but rather should use them to illuminate the proper route to moral choices. A number of mechanisms may help achieve this ethical framework.

In the Field Hospital

If an ethical dilemma arises in civilian practice, the usual mechanism for resolution involves second medical opinions from within the hospital, second medical opinions from another institution, discussion of the case at the local clinical ethics committee, and finally, if all else fails, a referral to the courts. Similarly, in the deployed field hospital, physicians should seek second opinions from their colleagues and the deployed medical director through the normal chain of command. Issues that cannot be resolved locally should be referred back to the military medical chain in the home country if necessary. When legal oversight is felt to be necessary, the matter should be discussed with the military legal service. It is likely that any such case would need to be judged on its particular facts, with resolution of any jurisdictional issues arising from the patient's demographic group before the ethical dilemma is approached.

Before Deployment

Empirical studies may allow critical care providers to determine what is currently considered ethically acceptable. For example, Delphi methodology could be used to determine consensus views[21] in the context of military medicine. This methodology could be applied to those who undertake critical care practice in deployed military facilities to develop a framework for decision-making. Consensus may also be developed from clinical conferences devoted to ethics in military critical care, and incorporation of hypothetical scenarios into training for military deployment. All these approaches are being explored within uniformed medical services.

The formation of a flying ethics tribunal has been proposed to evaluate and make decisions on difficult ethical problems.[22,23] Such a tribunal would be independent from the military command structure and might contain representatives from the legal professions, university ethics departments, religious communities, and medicine. How such a body could provide advice rapidly in an emergent scenario is, however, unclear.

However the framework is developed, the decision-making process must be legal, practical, accountable,

and clear. The choices made must be open to external scrutiny. Various groups have an interest in decision-making in this context, including clinicians, patients, regulatory bodies, uniformed service members, and the public at large. The ethical framework must ultimately be acceptable to all these groups.

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Chapter 43

MILITARY PEDIATRIC ANESTHESIA

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INTRODUCTION

Pediatric ballistic cases represent a significant proportion of the cases experienced at both Role 3 military hospitals in southwest Afghanistan, consistently providing 15% of the surgical workload. The patients' ages vary, the median being between 6 and 8 years of age; however, babies aged 6 months or less are not uncommon. This experience suggests that military anesthesiologists must be prepared to deal with this workload by updating their pediatric skills prior to deployment.

This chapter is a guide for those anesthetists without a normal pediatric practice. It gives a general description of managing the anesthetic components of pediatric trauma care. The preparation section provides some overall considerations, and the following sections are based on the primary survey as taught in the United Kingdom Battlefield Advanced Trauma Life Support course. No detailed consideration is offered regarding maintenance of anesthesia; neither is a particular anesthetic machine described in detail, because maintenance procedures and equipment change with the theater of operations and location. However, the clinical management of the pediatric trauma patient provided here is mostly based on recent experience in Afghanistan.

PREPARATION

Key Considerations

Injury Patterns

Children are smaller than adults, and their injury patterns are different. Expect injuries in a greater number of anatomical areas. In particular a child's head is proportionally larger than an adult's and more prone to injury.

Equipment

Calculate the drug dosages and equipment sizes and work out normal physiological values beforehand. This will help alleviate stress for the team. Weight prediction in Afghanistan is problematic (Tables 43-1, 43-2, 43-3, and 43-4; Figures 43-1 and 43-2; and Exhibit 43-1).

TABLE 43-1

NORMAL PHYSIOLOGICAL VALUES FOR CHILDREN

Age (years)	Heart Rate (beats/min)	Respiratory Rate (breaths/min)	Systolic Blood Pressure (mm Hg)
Newborn	130–170	40–60	60–70
< 1	110–160	30–40	70–90
2–5	95–140	25–30	80–100
6–12	80–120	20–25	90–110
> 12	60–100	15–20	100–120

Analgesia

Give children analgesia promptly. Be aware of the different administrative methods including intranasal (Tables 43-5 and 43-6).

TABLE 43-2

PEDIATRIC AIRWAY EQUIPMENT SIZES ACCORDING TO AGE

	Equipment						
Age	Facemask	Guedel Airway	Resuscitator	Laryngoscope	Tracheal Tube	Suction Catheter (French Gauge)	Laryngeal Mask
0–6 mo (1–6 kg)	0	000/00	Infant	Miller size 1	2.5–3.5	6	1
6–12 mo (4–9 kg)	1	0/1	Infant	Macintosh size 1	3.5–4.0	8	1.5
1–3 y (10–15 kg)	2	0/1	Infant	Macintosh size 2	4.0–5.0	10–12	2
4–7 y (16–20 kg)	3	1/2	Adult	Macintosh size 2	5.0–6.0	14	2
8–11 y (22–33 kg)	4	2	Adult	Macintosh size 3	5.5–7	14	2.5

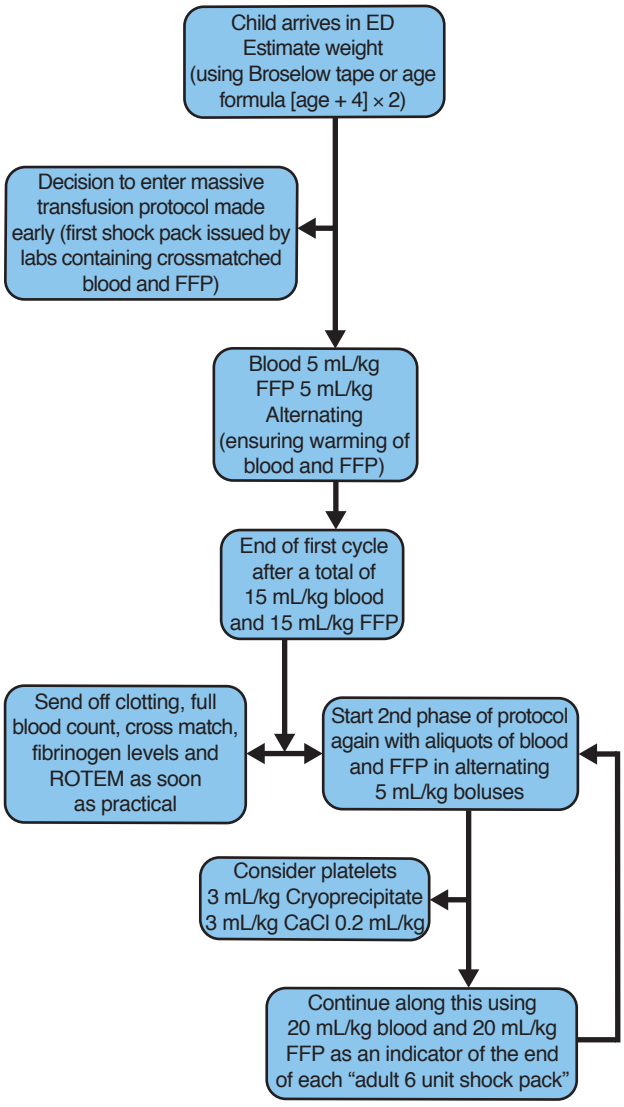


Figure 43-1. Pediatric massive hemorrhage resuscitation protocol.
Reproduced from: Bree S, Wood K, Nordmann GR, McNicholas J. The paediatric transfusion challenge on deployed operations. J *R Army Med Corps*. 2011;156(4 Suppl 1):S361–364.
ED: emergency department; FFP: fresh frozen plasma; ROTEM: rotational thromboelastometry (TEM Innovations GmbH, Munich, Germany)

TABLE 43-3

ESTIMATION OF ENDOTRACHEAL TUBE SIZE AND LENGTH IN CHILDREN*

Age	Size	Length (cm)
6 months	3.5 tube	11
Term infant	3.0 tube	9
Premature	2.5 tube	7–8

* Size (internal diameter) = (age/4) + 4
Length (oral) = (age/2) + 12

Parents

The language barrier and the strange environment of a Western military hospital and its staff will cause children and their parents stress and anxiety in addition to that already caused by the child's injuries. Do not add separation anxiety to this. Involve the parents early to help alleviate this stress.

EXHIBIT 43-1

WEIGHT ESTIMATION FOR AFGHANISTAN LOCAL NATIONAL CHILDREN

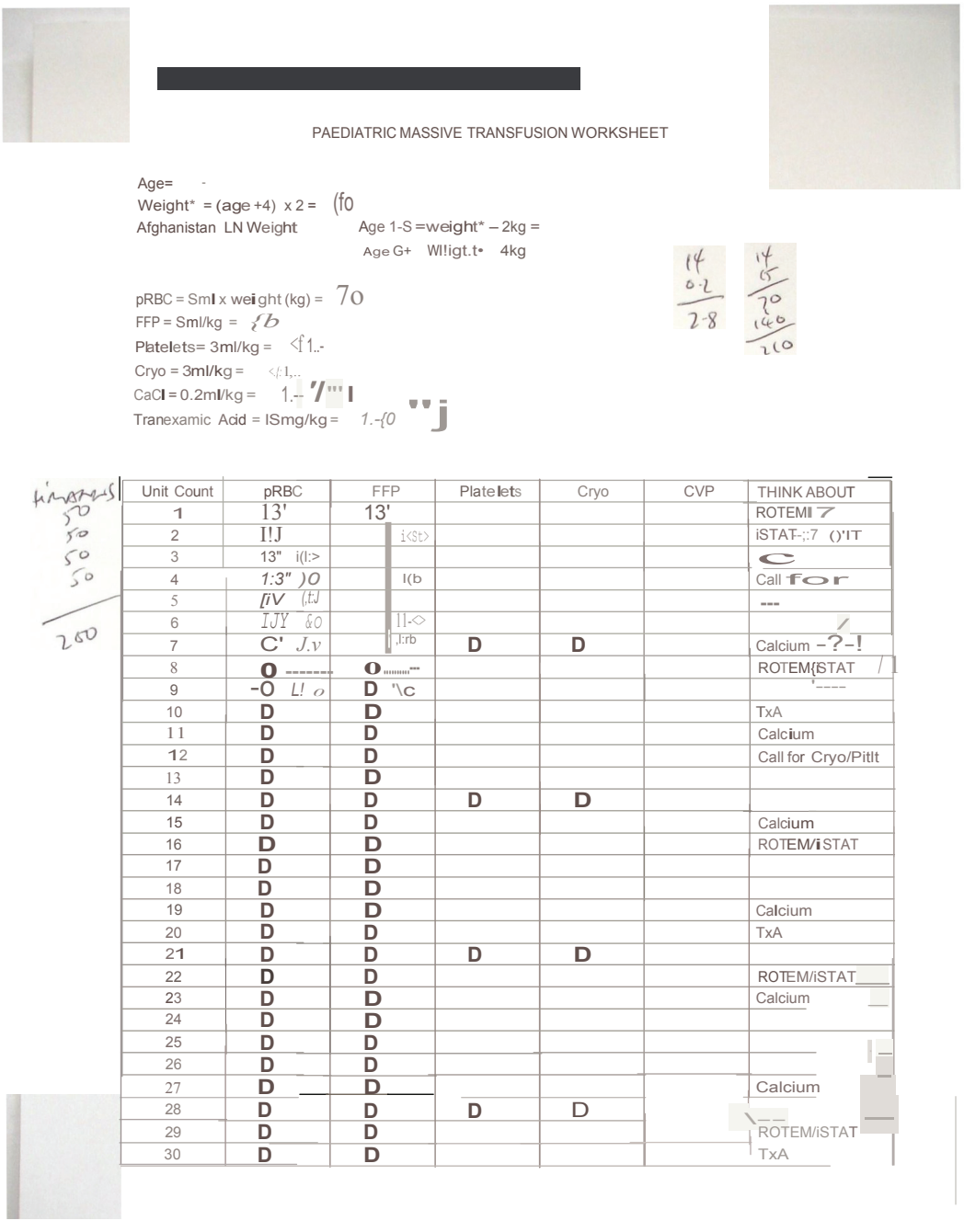
Age = a
(a + 4) x 2 = b

Age: 1–5 y
Weight (kg) = b – 2 kg

Age: 6+ y
Weight (kg) = b – 4 kg

PEDIATRIC CONSIDERATIONS IN TRAUMA

- Trauma predominates. Although children do present with normal medical problems, in the military hospital in Afghanistan trauma predominates (94% of admissions due to trauma in one recent case series[1]).
- Interaction is different. Responses to strangers and strange situations differ by age. Be aware of quiet children who will let you examine them uncomplainingly; they are potentially very sick. Children less than 6 months old will tolerate separation easily; those aged 6 months to 4 years usually have a fear of strange environments and prominent separation anxiety; those in the 4- to 6-year age group are similar to those younger but communication is easier, which helps alleviate stress; and those 6 years and above tend to be increasingly more tolerant of changing situations.



PAEDIATRIC MASSIVE TRANSFUSION WORKSHEET

Age=
Weight* = (age +4) x 2 = (f0
Afghanistan LN Weight
Age 1-S =weight* – 2kg =
Age G+ Wl!igt.t• 4kg

pRBC = Sml x weight (kg) = 70
FFP = Sml/kg = {b
Platelets= 3ml/kg = <f1..-
Cryo = 3ml/kg = </.1,..
CaCl = 0.2ml/kg = 1.-- '/"I
Tranexamic Acid = ISmg/kg = 1.-{0 "j

Unit Count	pRBC	FFP	Platelets	Cryo	CVP	THINK ABOUT
1	13'	13'				ROTEMI 7
2	I!J	i<St>				iSTAT-;:7 ()'IT
3	13" i(l:>					C
4	1:3")0	I(b				Call for
5	[iV (.tJ					---
6	IJY {0	11-<>				
7	C' J.v	].rb	D	D		Calcium –?–!
8	0 -------	0				ROTEM(iSTAT
9	-O L! o	D '\c				
10	D	D				TxA
11	D	D				Calcium
12	D	D				Call for Cryo/Pitlt
13	D	D				
14	D	D	D	D		
15	D	D				Calcium
16	D	D				ROTEM/iSTAT
17	D	D				
18	D	D				
19	D	D				Calcium
20	D	D				TxA
21	D	D	D	D		
22	D	D				ROTEM/iSTAT
23	D	D				Calcium
24	D	D				
25	D	D				
26	D	D				
27	D	D				Calcium
28	D	D	D	D		
29	D	D				ROTEM/iSTAT
30	D	D				TxA

Figure 43-2. Pediatric massive transfusion worksheet.

- Normal physiological values differ with age (see Table 43-1), but bradycardia and slow respiratory rate are always ominous.
- The primary and secondary surveys are no different for children (though be aware of the potentially different injury patterns).
- Children have a larger surface area for a given weight compared to adults, which leads to heat loss and hypothermia in addition to increased fluid loss. Keep them warm. Keep an eye on fluid balance.
- Neonates are more sensitive to drugs, more

TABLE 43-4

PEDIATRIC DRUG DOSES AND DATA FOR MASSIVE TRANSFUSION

	5kg	10kg	15kg	20kg	30kg	40kg	50kg	80kg
Ketamine iv/IM	10 / 50	20/ 100	30/ 150	40/ 200	60/ 300	80/ 400	100/ 500	160/ 800
Suxamethonium	10	20	30	40	60	80	100	160
Fent iv/in mcg	5/ 10	10/ 20	15/ 30	20/ 40	30/ 60	40/ 80	50/100	50/100
Vecuronium	0.5	1	1.5	2	3	4	5	10
Ket iv/ in Analgesia	1.5/ 15	2.5/ 30	4.0/ 45	5.0/ 60	7.5/ 90	10/ 120	12.5/ 150	20/ 240
M & M	0.5	1	1.5	2	3	4	5	8
Atropine (mcg)	100	200	300	400	600	600	600	600
Tube ETT	3.5	4	4.5	5.5	6.5	7	7	8
Blood/ FFP(5ml/kg)	25	50	75	100	150	200	250	1 unit
Platelets (3ml/kg)	15	30	45	60	90	120	150	1 bag
Cryo (3ml/kg)	15	30	45	60	90	120	150	1 bag
TXA (15mg/kg)	75	150	225	300	450	600	750	1.5g
CaCl 10% (ml)	1	2	3	4	6	8	10	10

Vital Signs					
	< 1yr	1-2yr	2 -5 yrs	5- 12yrs	>12 yrs
HR (beats/min)	110-160	100-150	95-140	80- 120	60-100
Systolic (mmHg)	70-90	80-95	80-100	90-110	100-120

Cryo: cryoprecipitate; ETT: endotracheal tube; Fent: fentanyl; HR: heart rate; IM: intramuscular; IV: intravenous; Ket: ketamine; M & M: morphine and midazolam (mg); TXA: Tranexamic acid

prone to hypoglycaemia, and desaturate more quickly due to a smaller functional residual capacity. They are more prone to apnoea and bradycardia, particularly if hypoxic and hypercapnic.

ANATOMY AND PHYSIOLOGY

Specific differences between pediatric and adult anatomy and physiology affect the management of trauma in children.

Airway

Children have a large head relative to body size, which tends to flex the neck and exacerbate airway obstruction, potentially aggravating any cervical spine injury. Their small oral cavity with a relatively large tongue and tonsils predisposes them to airway obstruction, particularly if their consciousness level is reduced. The larynx is more cephalad (the glottis is at C3 in infants, C5-6 in adults), and the epiglottis is floppy and U-shaped, making visualization of larynx during intubation more difficult. The narrowest part of the airway is at the cricoid ring rather than the vocal cords, so trauma can be caused at this level if an inappropriately large endotracheal tube (ETT) is used. This may result in edema and increased airway resistance on extubation in the short term and possible subglottic stenosis in the long term. The trachea is also short (4–5 cm in newborns, 7–8 cm at 18 months), making endobronchial intubation and tube displacement with movement more likely. Finally, it is important to remember that infants up to 6 months of age are obligate nasal breathers, which means the work of breathing can be significantly increased by nasal blood, secretions, or prongs.

Breathing

Children have increased oxygen consumption (6–8 mL/kg/min in neonates vs 4–6 mL/kg/min in adults) and thus require proportionately higher minute ventilation. Their functional residual capacity (FRC) is proportional to that in adults by size, but a high minute ventilation to FRC ratio results in more rapid desaturation in cases of apnea or airway obstruction. The FRC will be reduced by induction of anesthesia with barbiturates or inhalational agents but maintained or increased with ketamine. Children are more prone to

TABLE 43-5

PEDIATRIC ANALGESIC DRUG DOSES

Drug	Class of Analgesic	Routes of Administration	Doses
Paracetamol	Antipyretic analgesic	IV	5 mg/kg 6 hourly (< 50 kg) 10 mg/kg 6 hourly (< 6 mo)
		PO	115–20 mg/kg 6 hourly 10–15 mg/kg 6 hourly (< 6 mo)
Ibuprofen	NSAID	PO	10 mg/kg TDS (> 6 months) 5 mg/kg QDS (< 6 months)
Diclofenac	NSAID	PO/PR	1 mg/kg TDS (max dose 50 mg) Not suitable for infants < 6 mo
Ketorolac	NSAID	IV	0.5 mg/kg QDS (max dose 10 mg)
Codeine	Opioid	PO	1 mg/kg QDS
Tramadol	Opioid/5-HT antagonist	PO/IV	1 mg/kg QDS
Morphine	Opioid	PO	0.2–0.4 mg/kg 4 hourly (> 1 y) 0.1mg/kg 4-6 hourly (< 1 y)

IV: intravenous; NSAID: nonsteroidal antiinflammatory drug; PO: per os (by mouth); PR: per rectum; QDS: four times a day; TDS: three times a day

TABLE 43-6

SUGGESTED DOSES OF KETAMINE AND FENTANYL IN ACUTE SEVERE PEDIATRIC TRAUMA

Drug	Route of Administration	Dose
Fentanyl	IV/IO	0.5 μg/kg initially then titrate
Ketamine	IV/IO	0.5 mg/kg initially then titrate
Fentanyl	Nasal	2 μg/kg
Ketamine	Nasal	1–3 mg/kg

IO: intraosseous; IV: intravenous

atelectasis, so continuous positive airway pressure (CPAP) is useful in maintaining alveolar recruitment in the neonate.

Ventilation in children is mainly diaphragmatic. For this reason, any abdominal distension from trauma or insufflation of air into stomach may cause respiratory difficulties. Pliable ribs also mean that fractures are less likely than in adults, but pulmonary contusions are more likely due to transmitted energy in blunt trauma. The increased mobility of mediastinal contents make tension pneumothoraces more likely than in adults.

Circulation

Children have higher resting cardiac outputs (350 mL/kg/min in neonates, 150 mL/kg/min at 2 months, falling gradually to adult levels of 75 mL/kg/min), with higher resting heart rates but lower blood pressures due to lower systemic vascular resistance (see Table 43-1). Their circulating blood volume is relatively higher than adults (90 mL/kg in neonates, 85 mL/kg in infants, 80 mL/kg in children). Because of the higher blood volume, combined with a vasoconstrictive response to blood loss, hypotension does not develop until over 35% of the blood volume is lost. Significant blood loss should therefore be suspected if the child has cold clammy skin, prolonged capillary refill time, and altered consciousness level. Finally, it should be remembered that small children have a bradycardic response to hypoxemia, which should be managed with oxygenation in the first instance rather than atropine.

Thermoregulation

Children have a higher surface area to body weight ratio, making them more susceptible to hypothermia than adults. Impaired temperature regulation under anesthesia and high thermal losses due to surgery make careful attention to temperature control essential.

MASSIVE HEMORRHAGE

Resuscitation of the circulation is a high priority in polytrauma. In children, signs must be compared with normal physiological values. As part of coordinated trauma resuscitation, the anesthetist's first task is to

secure appropriate vascular access (discussed below). In the presence of significant hypovolemia, the best access in small children is intraosseus (IO), followed by supradiaphragmatic central venous access.

Hypovolemia

Poor perfusion secondary to hypovolemia can be indicated by central capillary refill time of over 2 seconds and differences between central and peripheral pulses and skin temperature. A change in mental status, specifically reduced reactivity and responsiveness, is a good indicator of cerebral perfusion and hence overall perfusion. Importantly, low blood pressure is a late sign and shows decompensation. Bradycardia is a preterminal sign.

Access

Vascular

Peripheral venous access. The best sites for peripheral access are the dorsum of the hand, radial aspect of the wrist, antecubital fossa, dorsolateral aspect of the foot, and saphenous vein. A 24-gauge catheter is appropriate for neonates and a 22-gauge catheter for young children. If access is difficult in young children, the palmar aspect of the wrist may provide a useful site, as may the scalp in neonates. In hypovolemic children, intravenous access may well be difficult and it may be necessary to resuscitate them through the IO route in the first instance.

Central venous access. If the massive hemorrhage protocol is being used, a large-gauge catheter in a central vein above the diaphragm is preferred. Using a single large-bore line (4–6 F depending on age), if available, will maximize filling rates. If multilumen central lines are the only available option, appropriate sizes are 3 F, 5-cm lines for infants; 4 or 5 F, 8-cm lines for toddlers; and 5 F, 10- to 12-cm lines for older children.

In hypovolemic children, the vessels collapse more easily than in adults. The use of ultrasound is strongly recommended, and anesthetists should become sufficiently skilled in its use before deploying. Direct vision of the needle and then wire in the correct vessel, extending into the superior vena cava, will minimize complications. The subclavian vein remains open for a longer time in hypovolemia; however, the internal jugular vein is the alternative and is more easily visualized with ultrasound. These vessels will completely occlude with the respiratory pattern in extremis, so consider IO filling before placing a central venous catheter.

Arterial

Arterial lines are used for continuous monitoring of blood pressure and for successive arterial blood gas measurement. The radial or femoral artery are the most common insertion sites. Because of the size of the vessels, complications become more frequent with radial cannulation in children under 5 years of age. Insertion of the catheter into the femoral artery has greater success in neonates and infants. Recommended catheter sizes are 24 gauge in neonates, 22 gauge in infants, and 20 gauge in older children. A catheter-over-needle technique is appropriate for the radial artery. A preferred technique is transfixion of the vessel: After observing blood in the clear plastic needle hub (or flashback), advance the cannula through the posterior wall of the artery. Then remove the needle and attach a syringe. Slowly withdraw the cannula while aspirating. Once aspiration without resistance is achieved, gradually advance the cannula within the artery. Inserting the catheter over the needle is also appropriate when approaching the femoral artery in infants, although the Seldinger technique can be used in older children.

Intraosseus Vascular

Use IO access for resuscitation when intravenous access is difficult; it is particularly useful as an early route for initial resuscitation of the hypovolemic child. For most children a 22-guage, 15-mm needle is appropriate, but after inserting the needle into the bone, ensure there is 5 mm of needle visible above the skin on contact. If less than this is visible, use the next size up. Needles can be placed in either the tibia or humerus. Successful insertion is suggested by the loss of resistance as the needle passes from the cortex to the medulla. The needle will then remain upright unsupported. In the tibia, the IO needle should be inserted in the anterior surface of the proximal tibia, approximately 2 cm distal to the tuberosity and 2 cm medially. Insertion of the needle into the humeral head is slightly more difficult. With the arm adducted and hand across the groin, the greater tubercle of the humerus is found at the apex of an equilateral triangle, the base of which is a line between the coracoid and acromion of the scapula.

Blood samples can be taken at the time of insertion but not thereafter. All fluids and drugs given intravenously can be given via this route. A three-way tap and syringe will be needed to ensure fluids given are done at a sufficient rate.

Resuscitation

Most pediatric patients with ballistic trauma will

need blood products, and the majority will need a massive transfusion to ensure the patient is adequately resuscitated. Management of major hemorrhage occurs predominantly in the emergency department and continues into the operating room, but it may need to happen elsewhere. Because of their small circulating volumes, the threshold for initiating a massive transfusion protocol is lower in children than in adults (Exhibit 43-2; see Figure 43-1).

It is essential to deliver the fluids warmed and keep a close record of what has been given. This is particularly so in smaller patients. The worksheet in Figure 43-2 will aid this process. For pediatric cases, the anesthetist should give the fluid in a 50-mL syringe added to the setup illustrated in Figure 43-3. At the proximal

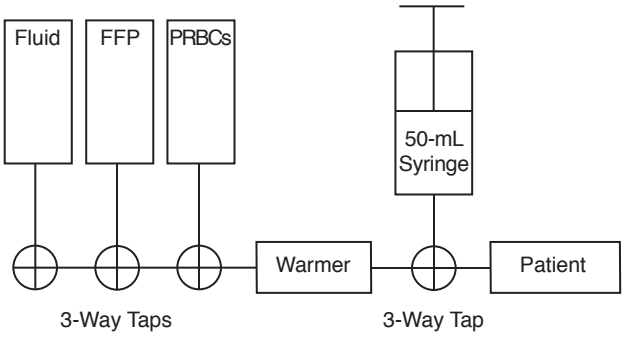


Figure 43-3. Massive transfusion equipment setup.
PRBCs: packed red blood cells
FFP: fresh frozen plasma

EXHIBIT 43-2

KEY POINTS OF THE UK PEDIATRIC MASSIVE TRANSFUSION PROTOCOL

- As with any pediatric case, calculate the patient's weight. This can be difficult; one method is shown in Exhibit 43-1.
- Initially order 1 U of PRBC and 1 U of FFP for every 20 kg of the child's weight. Eg, a 23-kg child will need 2 U blood and 2 U FFP initially. This will ensure a minimum of approximately 15 mL of blood per kg of weight.
- Give boluses of PRBC and FFP, alternating between the two, in volumes of 5 mL/kg each.
- Be aware that rapid transfusion of blood products through a large-bore central line has the accompanying risk of significant hyperkalemia.
- Keep track of blood and other products given by using the Pediatric Massive Transfusion Calculation Sheet (Figure 43-2).
- 15 mL/kg (3 boluses) of each product for the child equates to the first phase of transfusion for an adult (4 U of PRBC and 4 U of FFP).
- Use all clinical signs to help guide resuscitation, including arterial waveform, central venous pressure, blood pressure, and urine output. The variance of the arterial waveform with respiration in conjunction with its overall shape is a key guide.
- UK military research has shown that novel hybrid resuscitation is the best resuscitation model for blast injuries.[1] It entails resuscitating patients for the first hour after injury to achieve a palpable radial pulse in older children and a brachial pulse in children under 2 years of age. Thereafter aim to achieve a blood pressure that is normal for that age of child (Table 43-1). The exception to this is a child with head injury (see chapter text).
- Laboratory results are particularly useful:
  - Arterial blood gases (degree of acidosis) help guide whether resuscitation is adequate.
  - Rotational thromboelastometry (ROTEM [TEM Innovations GmBH, Munich, Germany]) will indicate degree of coagulopathy and need to vary blood product usage; variance from the 1:1 ratio of blood and FFP is usually not needed, however the ROTEM will guide need for platelets, cryoprecipitate, or antifibrinolytics.
- Platelets should be given initially at 3 mL/kg; however, ROTEM may indicate a requirement for more platelet transfusion. It takes up to 2 hours for transfused platelets to function adequately, and this may be reflected in a ROTEM suggesting further platelets despite an adequate platelet count.
- Hypocalcemia is common; the dose for calcium chloride 10% is 0.2 mL/kg.
- The treatment of fibrinolysis is tranexamic acid, 15 mg/kg. Current data suggests all patients qualifying for massive transfusion should receive this dose even without evidence of fibrinolysis.

FFP: fresh frozen plasma
PRBC: packed red blood cells
U: unit

1. Kirkman E, Watts S, Cooper G. Blast injury research models. *Philos Trans R Soc Lond B Biol Sci*. 2011;366:144–159.

end of the fluid warmer four 3-way taps should be attached in series to three to four bags of fluid and their giving sets. The three or four bags may contain any of the following; crystalloid, colloid, blood, or fresh frozen plasma. At the distal end of the fluid warmer, a further two 3-way taps should be attached in series: one for a 50 mL syringe (to use as the main "pump" for each fluid bolus) and the other for the addition of drugs. Distal to this should be a short connector to the central line. The connector should be secured in some manner to the bed so it takes the weight of the 50-mL syringe and lines. Platelets should be given through a different giving set to a different line. The dose of 3 mL/kg is conservative and use of the whole bag should be considered. Appropriate monitoring is essential and invasive arterial and central venous pressure monitoring should both be used, but monitoring should not excessively delay necessary surgical intervention.

AIRWAY

The trauma anesthetist may need to manage a pediatric airway due to low consciousness level, as part of anesthesia for surgical intervention, or due to airway trauma.

Management of Airway Obstruction

Children may present with a reduced consciousness level and airway obstruction due either to head injury or hypovolemic shock. In all cases secretions, blood, vomit, and foreign bodies in the airway should be removed and oxygen delivered. To open the airway, the head should be placed into slight extension and the mandible pulled forward, taking care not to put pressure in the submental triangle, which will lead to posterior displacement of the tongue and worsening airway obstruction. When cervical spine injury is suspected, a suitably sized cervical collar or sandbags with head tape should be applied in the neutral position, and airway opening maneuvers should be restricted to pulling the mandible forward. If consciousness level is sufficiently depressed that a gag reflex is no longer present and the above maneuvers do not provide a satisfactory airway, a Guedel airway should be inserted (see Table 43-2 for sizing) and chin lift maintained. In small children, airways should be inserted with care and not inverted to prevent damage to the tissues of the oropharynx or tonsils.

Endotracheal Intubation

When endotracheal intubation is required, it should be performed once any hypoxia has been reversed by airway opening maneuvers and, if needed, mask ventilation. In children under 6 months of age, a straight-bladed laryngoscope should be used, lifting the epiglottis and withdrawing the laryngoscope slowly to reveal the laryngeal inlet. If the child is older than 6 months, a Macintosh blade should be used, placing the blade tip into the vallecula and lifting the laryngoscope to elevate the epiglottis and expose the vocal cords. Traditionally, uncuffed tubes are used in children up to the age of 8; however, recently interest in using cuffed tubes in children has been revived, and research is ongoing.

At present military modules contain uncuffed ETTs for pediatric use in sizes 3.5 to 6.0. The ETT may be sized against the child's nostril or fifth finger, or if age is known size may be calculated from the formulae in Table 43-3. Appropriate ETT length at the lips can be calculated from the formula: (age/2) + 12 cm. See Table 43-3 for children under 12 months of age. Because there is increased potential for inadvertent endobronchial intubation in children, it is essential that auscultation of both axillae is performed to ensure bilateral breath sounds. Tubes should be fixed securely to prevent movement. A common strategy is to use two "trouser-shaped" lengths of strong adhesive tape with a Guedel airway alongside the ETT to prevent lateral movement.

In all cases the ETT should pass through the glottis and cricoid without resistance, and there should be a small audible leak at inflating pressures of 20 cm H_2O. If there is no leak the ETT should be downsized to prevent damage to the airway mucosa; if there is a large leak the ETT should be upsized to allow satisfactory ventilation. If required a moistened throat pack can be used to reduce a leak or increase protection to the lower airways from ongoing hemorrhage. In children a nasogastric tube should be passed after the ETT is sited and secured unless contraindicated. If there is any question of damage to the cribriform plate an orogastric tube should be passed instead. The nasogastric or orogastric tubes reduce the gastric distension associated with acute trauma in children, which is exacerbated by swallowing large quantities of air when stressed and by facemask ventilation. Decompression reduces the risk of vomiting and aspiration, diaphragmatic splinting, and compression of the inferior vena cava. Caval compression may diminish venous return and cause hypotension, particularly where there is concurrent hypovolemia.

Rapid Sequence Intubation

The process of a rapid sequence intubation is de-

EXHIBIT 43-3

RAPID SEQUENCE INTUBATION

- Preoxygenate with 100% oxygen using a reservoir mask or anesthetic circuit for at least 2 minutes.
- Consider administering atropine (20 μg/kg IV) to dry oral secretions and prevent bradycardia from suxamethonium for children under 6 months of age.
- Administer an induction agent. In hypovolemia secondary to trauma the preferred agent is ketamine.
- Apply cricoid pressure using the thumb and index finger.
- Administer suxamethonium (2 mg/kg for children < 10 kg; 1 mg/kg for children > 10 kg) or rocuronium (0.6–1 mg/kg).
- Intubate.
- Confirm correct endotracheal tube placement with capnography followed by auscultation and observation of appropriate chest movement.
- Release cricoid pressure.
- Secure endotracheal tube.
- Obtain secondary confirmation of proper placement with chest radiograph or CT.

CT: computed tomography
IV: intravenous

EXHIBIT 43-4

CANNULA CRICOTHYROIDOTOMY IN CHILDREN

- Extend neck with roll under shoulders.
- Stabilize cricothyroid/trachea with nondominant hand.
- Aim in caudad direction.
- Confirm position by aspiration of air.
- Connect to 4-bar oxygen source with flowmeter (match flow L/min to child's age) and Y connector.
- Set inflation: expiration = 1:4 seconds.
- Cautiously increase inflation pressures or oxygen flow rate by 1 L/min increments to achieve adequate chest expansion.
- Maintain upper airway patency, eg, with a Laryngeal Mask Airway (Teleflex, San Diego, CA).

scribed in Exhibit 43-3. Preparation is key: calculate weight, normal physiological values, doses of drug needed, and size of equipment. Table 43-4 is a calculation sheet currently used in Camp Bastion, Afghanistan, to aid this process.

Cricothyroidotomy and Tracheostomy

Both needle and surgical cricothyroidotomy and tracheostomy are difficult in children due to cephalad cricoid position and small cricothyroid membrane. Both procedures can give rise to serious complications and should only be used in life threatening situations. The surgical and anesthetic teams need a skills assessment, and a joint plan should be made for managing this situation in advance. It is generally accepted that cricothyroidotomies should be converted to tracheostomies once a child is stabilized to reduce the risk of subglottic stenosis. Cannula cricothyroidotomy for children is described in Exhibit 43-4.

Facial Injuries and Burns

Facial injuries causing bleeding into the airway require immediate intubation for control of the airway. In all cases blood, secretions and foreign bodies should be removed and airway opening maneuvers performed, as described above, aiming to reverse any hypoxamia if possible before tracheal intubation. Airway burns should be suspected when a burn occurs in a closed space or when there is physical evidence of singed nasal hairs or eyebrows, carbonaceous sputum, wheeze, or change in cry or voice. In these cases early intubation is recommended before significant edema develops. An uncut tube should be used in anticipation of insidious facial swelling.

BREATHING

Assessment

Breathing should be assessed in all children as part of the primary survey and reassessed regularly, particularly after intubation. The chest wall should be examined for bruises and wounds. Due to children's highly compliant chest walls, significant intrathoracic organ damage may occur with minimal external evidence of chest wall injury. Inspect and auscultate to ensure both sides of the chest are being adequately ventilated. The respiratory rate should be noted with reference to the normal values for the child's age (see Table 43-1). Look for central cyanosis and measure oxygen saturations—readings may be poor

EXHIBIT 43-5

CAUSES OF INADEQUATE VENTILATION FOLLOWING PEDIATRIC TRAUMA

Bilateral
- Obstruction of upper respiratory tract
- Esophageal intubation
- Circumferential thoracic burns

Unilateral
- Pneumothorax
- Hemothorax
- Pulmonary contusion or laceration
- Intrapulmonary hemorrhage
- Flail segment
- Bronchial rupture
- Foreign body in bronchus
- Rupture of diaphragm
- Endobronchial intubation

EXHIBIT 43-6

HOURLY FLUID MAINTENANCE REQUIREMENTS IN CHILDREN

- First 10 kg: 4 mL/kg
- Second 10 kg: 2 mL/kg
- Thereafter: 1 mL/kg

Example: for a 34-kg child, use 74 mL/h (40 mL [first 10 kg] + 20 mL [second 10 kg] + 14 mL [last 14 kg])

in a hypovolemic, vascularly constricted child, so use clinical judgment and seek additional perfusion information from capillary, venous, or arterial blood gases. Additional information from imaging such as chest x-ray, focused assessment with sonography for trauma (FAST) and computed tomography (CT) will aid diagnosis but (CT in particular) should not delay prompt initial management. Causes of inadequate ventilation following pediatric trauma are detailed in Exhibit 43-5.

Management

The anesthetist's purpose is to maintain adequate oxygenation and ventilation and to manage hypovolemia or cardiovascular collapse caused by the injuries while permitting surgical management. In the first instance airway opening maneuvers, application of supplemental oxygen, and if required facemask ventilation should occur. Tension pneumothoraces require immediate drainage.

When intubation and ventilation are required, children should be ventilated to a tidal volume of 6 to 10 mL/kg with an age appropriate rate to maintain a normal $Paco_2$ unless otherwise indicated. Peak end expiratory pressure (PEEP) should be started at 4 cm H_2O and increased as required, in addition to adjusting FiO_2 to maintain an adequate Pao_2. It is usually recommended that children are ventilated in a pressure control mode (peak pressures normally 16–20 cm H_2O); however, volume control ventilation may be appropriate in the clinical circumstances. During intrathoracic procedures, particularly in very small children, hand ventilation may be optimal.

CIRCULATION

Fluid Management

When blood products are not initially needed, fluid resuscitation should be initiated with 5 mL/kg boluses of Hartmann solution or Ringer lactate. Reassess the patient after each bolus to determine whether more fluid or a change to blood is required. Children, like adults, have a basic fluid maintenance requirement, which can be calculated using the equations shown in Exhibit 43-6.

Assessment of Circulation

Observation of mental status, heart rate, pulse quality, peripheral pulses, and temperature are the main indicators (see Massive Hemorrhage, above). Remember that a child can be in shock and still have a normal blood pressure; also be aware that inadequate resuscitation is common.

DISABILITY

Neurological Injuries

Assessment

Calculate the Glasgow Coma Score. It is imperative to do this prior to anesthetising (Exhibit 43-7). Consider whether the patient is exhibiting signs of neurological deficit. A brief but comprehensive neurologic examination is very important. Specific concerns that can be quickly answered include: Is there posturing, and if

EXHIBIT 43-7

GLASGOW CHILDREN'S COMA SCORE*

Eye opening	Score
Spontaneous	4
To verbal stimulus	3
To painful stimulus	2
No response to pain	1
Best motor response	
Obeys verbal command	6
Localizes to pain	5
Withdraws from painful stimulus	4
Abnormal flexion to pain	3
Abnormal extension to pain	2
No response to pain	1
Best verbal response	
Alert, babbles, coos; words appropriate for age	5
Spontaneous irritable cry, less than usual words	4
Cries only to pain	3
Moans to pain	2
No response to pain	1

*For children under 4 years of age.

so, what kind? The motor component of the Glasgow Coma Score is important and should be recorded. Address neurogenic shock by assessing for hypotension, warm peripheries, and flushed skin. Spinal shock is evidenced by absent deep tendon reflexes, hypotonia, flaccid sphincters, and priapism. Signs of increased intracranial pressure should be sought. These include headache, vomiting, altered mental status, and pupillary dilation. Evidence of Cushing signs, irregular respiratory pattern, hypertension, and bradycardia should also be noted.

Management

It is important to minimize secondary brain injury with the following measures: elevate the head of the bed to 15 to 30 degrees; avoid hypoxia, hypercarbia, and hypotension; avoid seizures; control fever aggressively; control glucose, avoiding hyperglycemia and hypoglycemia; avoid hyponatremia; control shivering; maintain adequate analgesia; avoid coughing and straining; tape the ETT; and maintain neutral head alignment. In the presence of elevated intracranial pressure, consider administering mannitol (0.25–1.0 g/kg) or hypertonic saline (3% sodium chloride, 2–4 mL/kg) to decrease intracranial pressure and help restore intravascular volume.

Traumatic brain injury with altered consciousness or CT changes should be discussed with a neurosurgeon at an early stage. Relevant guidelines should be followed for CT scanning and referral.

Preoperative Sedation

Preoperative sedation is rarely needed, particularly if the patient is in extremis. Oral midazolam (0.25-0.5 mg/kg to a maximum of 15 mg) in flavored paracetamol solution (20 mg/kg) will work well as a premedication or added with ketamine (2 mg/kg) for short procedures (eg, change of dressing).

Anesthesia

Maintenance of Anesthesia

Military hospital personnel prefer using volatile inhalational anesthesia for maintenance of anesthesia in children due to concerns about potential "propofol infusion syndrome" (after case reports of fatal metabolic acidosis and cardiac failure in children), as well as its advantages in ensuring therapeutic concentrations of anesthetic when there is ongoing blood loss. Intravenous anesthesia, however, is preferred when transferring patients. Propofol is not recommended for anesthesia maintenance in children under 3 years of age, although it can be used for brief periods when needed because propofol infusion syndrome is associated with prolonged infusions. Various infusion regimes are available, although infusion pumps are currently not available in Afghanistan. A useful infusion starting point is 13 mg/kg/h for the first 10 minutes, reducing the dose to 11 mg/kg/h for the next 10 minutes, and then maintaining the dose at 9 mg/kg/h.

Sevoflurane is the most common volatile agent used in children, particularly for inhalational induction, although its use carries a risk of apnea at higher concentrations. The appropriate minimum alveolar concentrations for the most common inhalational agents, isoflurane and sevoflurane, are detailed in Table 43-7.

Regional Anesthesia

Administration of regional anesthesia is as relevant to children as to adults, though with differences in

TABLE 43-7

AGE-APPROPRIATE MINIMUM ALVEOLAR CONCENTRATION

Age	Isoflurane	Sevoflurane
Preterm	1.3	Unknown*
Neonate	1.6	3.3
Infant	1.9	3.2
Child	1.6	2.5
Adult	1.16	2.0

*Commonly used in the United Kingdom, but minimum alveolar concentration has not been determined.

volumes of local anesthetic, depth of needle insertion, and needle sizes. Administration requires expertise but satisfactory analgesia may be achieved with opioids by the nonspecialist. further below.

Equipment

Due to children's physiological differences and smaller sizes, pediatric circuits must have less dead space and reduced resistance. The preferred anesthetic circuits are therefore the Mapleson F and circle systems. The Mapleson F (also called the Jackson-Rees modification to the Ayre's T-piece) is the most common circuit used for children under 20 kg. It is simple and lightweight with low resistance and minimal dead space. Partial occlusion of the open-ended 500-mL bag can enable the application of PEEP and CPAP. Monitor end tidal carbon dioxide continuously and change fresh gas flows appropriately to maintain normocapnia. When using a circle system for children, the circuit must be narrower (15 mm is appropriate) to reduce the compression volume, and the connections (particularly distally) must be smaller to minimize dead space. The narrow-bore circle circuit can be used for older children (>20 kg) providing the reservoir bag is changed from a 500-mL bag to one holding 1 or 2 liters, as appropriate.

Analgesia

Acute Pain

For minor injuries, pediatric pain can be managed according to the World Health Organization pain ladder (see Chapter 18, Multimodal Analgesia for Specific Injury Patterns, Figure 18-1). Table 43-8 contains doses of commonly used formulations in operations. Attention should also be paid to adjunctive measures such as splinting and immobilization, reassurance, and distraction.

For more severe injuries, rapid-acting analgesics, usually ketamine or fentanyl, should be carefully titrated to response. In cases where intravenous access is difficult or not obtained, or in mass casualty situations, intranasal ketamine and fentanyl have been shown to be rapid acting and effective, and may be of particular use in the prehospital care setting (see Table 43-6). For the most severe injuries, it may be more appropriate to move quickly to anesthesia induction to manage pain, maintain the airway, and facilitate resuscitation and damage control surgery.

Postoperative Pain Management

High quality postoperative pain management should be encouraged to support early mobilization and rapid transfer to a facility equipped for definitive care. Rigorous intraoperative and postoperative attention to an analgesic regime based on the World Health Organization pain ladder lays the foundations for good postoperative pain control. For children old enough to use patient-controlled analgesia, the standard field unit can be used with a morphine solution containing 20 μg/kg/mL and standard settings (eg, 1-mL bolus with 5-minute lockout and no

TABLE 43-8

DRUG DOSING FOR PEDIATRIC SINGLE-INJECTION PERIPHERAL NERVE BLOCK*

Block	Dose Range (mL/kg)	Midrange Dose (mL/kg)	Maximum Volume (mL)
Parascalene	0.2–1.0	0.5	20
Infraclavicular	0.2–1.0	0.5	20
Axillary	0.2–0.5	0.3	20
Paravertebral	0.5–1.0	0.7	5
Femoral	0.2–0.6	0.4	17
Proximal sciatic	0.3–1.0	0.5	20
Popliteal	0.2–0.4	0.3	15
Lumbar plexus	0.3–1.0	0.5	20

*Children < 8 y: 0.2% ropivacaine or 0.25% bupivacaine. Children > 8 y: 0.5% ropivacaine or 0.5% bupivacaine. Do not exceed maximum recommended doses of local anesthetic.
Reproduced from: Buckenmaier C, Bleckner L. *Military Advanced Regional Anesthesia and Analgesia Handbook*. Washington, DC: Borden Institute; 2009: Table 30-4.

continuous infusion). A morphine infusion is needed for smaller children. These patients must be cared for in the critical care unit until adequate enteral opioids are tolerated.

When the situation permits, analgesia with local anesthetics (ie, infiltration by a surgeon, nerve blocks/catheters, caudal anesthetics, or epidurals) should be performed to reduce the need for systemic pain medications. As with adult regional anesthesia coagulation screening, platelets count and thromboelastometry (where available) should be normal before placing catheters or performing neuraxial blockade. In the complex trauma patient, siting of epidurals and nerve catheters in the days following initial resuscitation and stabilization may facilitate early extubation and reduce complications from prolonged ventilation and sedation.

Epidural Anesthesia

Epidural anesthesia in infants and neonates is a subspecialist practice and should be undertaken only by those with sufficient experience. In children weighing more than 10 kg, a 19-gauge adult-length Tuohy needle with a 21-gauge catheter is appropriate. A standard aseptic and syringe loss of resistance to saline technique is used. At lumbar levels the epidural space is roughly at a depth of 1 mm per kilogram of body weight for children between the ages of 6 months and 10 years. At least 4 cm of catheter should be left in the extradural space. If used for surgical anesthesia, 0.75 mL/kg of 0.25% plain levobupivicaine should produce an adequate block. Postoperatively, levobupivicaine 0.125% should be administered at a rate of 0.1 to 0.4 mL/kg/h, with a maximum rate of 15 mL/h.

Caudal Anesthesia

Caudal anesthesia is an easy-to-learn technique providing good postoperative analgesia. A dose of 0.5 mL/kg 0.25% levobupivicaine provides sufficient anesthesia to block levels L_5–S_5, and a dose of 1.0 mL/kg 0.25% levobupivicaine will block T_9–S_5 (maximum dose: 20 mL). Duration of action can be prolonged by adding preservative-free ketamine (0.5 mg/kg) or clonidine (1–2 μg/kg), although this dose has possible side effects of hypotension and sedation. See Table 43-8 for pediatric dosing with individual blocks.

EXPOSURE

Injuries

As with adults, a thorough secondary survey is imperative. Remember that children are at greater risk of suffering injuries in more anatomical areas compared to adults.

Hypothermia

Use all methods available to treat hypothermia and maintain normothermia. Heated mattresses, forced air warmers, and fluid warmers should be used in all trauma cases.

Hypoglycemia

Neonates and in particular preterm infants are particularly vulnerable to hypoglycemia during periods of trauma due to low carbohydrate reserves. During the acute trauma phase, blood sugars should be monitored regularly and hypoglycemia treated with 5 to 10 mL/kg of 10% dextrose (500 mg–1 g/kg). Hypoglycemia in the neonate is not clearly defined (in neonatal intensive care units it is usual to delay treating at-risk neonates who are otherwise healthy until their blood glucose falls below 2.0 mmol/L). However it would be pragmatic during an acute trauma episode to treat blood sugars below 3.0 mmol/L (54 mg/dL). Fluid requirements change very rapidly in the first week of life, from 60 mL/kg/day at birth to 150 mL/kg/day. As a rough guide, neonates who are not obtaining nutrition from other sources should be given 90 mL/kg/day of 10% dextrose (6 mg/kg/min) as maintenance carbohydrate intake.

Older infants and children rarely suffer from hypoglycemia, but if they do it should be treated with boluses of 5 to 10 mL/kg of 10% dextrose. Hyperglycemia is frequently present in pediatric trauma patients and is associated with increased morbidity and mortality.

SUMMARY

Children will always be victims in war. In the present theater of operations for the UK and US military, pediatric patients having suffered from ballistic trauma remain a significant proportion of the surgical workload. Military anesthetists should endeavor to update their pediatric experience prior to deploying to war zones so both their skills and knowledge is up to date. This chapter is an adjunct

to this training. It is intended to be a valuable source of information to help nonpediatric anesthetists cope with the challenges of caring for the injured child.

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Chapter 44

ANESTHESIA CONSIDERATIONS IN THE ELDERLY POPULATION

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INTRODUCTION

The focus of deployed military medical facilities is the care of its personnel. However, the realities of medical care in a combat zone make civilian casualties inevitable. Each nation establishes its own criteria for how these patients are to be treated, but the biological contrast between civilian casualties and young, fit soldiers is stark. A significant injury score is far more likely to prove fatal in the elderly than in service members, so those who survive trauma are likely to be less severely injured than their military counterparts. Depending upon the proximity of Role 3 facilities to local population concentrations, some of these patients will present with nontraumatic surgical issues.

Most developed nations define the elderly as aged over 65 years. However, characteristics of older patients as a group vary widely according to the geographical location of the conflict. Patients from developed countries are more likely to suffer comorbidities associated with a higher socio-economic lifestyle, while patients in third world locations are characterized by a high prevalence of poor nutrition and ill health, including infective disease. Currently in Afghanistan some 40% of patients admitted to the surgical facilities at Camp Bastion are Afghan nationals aged over 40 years. Many of these people are physiologically much older than Westerners of the same age by virtue of poor general health, often exacerbated by narcotic abuse.

PHYSIOLOGY OF OLD AGE

Wherever their location, older people share a common physiology. Exhibit 44-1 lists the key features relevant to anesthesia. This list is perhaps best summarized by acknowledging that the elderly have limited physiological reserve and will not tolerate gross disturbances in physiological function. The physiology of the elderly must be respected in the practical conduct of anesthesia.

Preoperative Assessment

As with all patients, preoperative assessment of the elderly begins with a history and physical examination. The aim is to ascertain the extent of known pathology or detect unsuspected conditions. Subsequent radiological and laboratory investigations should be conducted with the objective of identifying problems that should be corrected or improved prior to surgery. The assessment will be limited when the need for surgery is urgent.

Cardiovascular Disease

In the Western world ischemic heart disease is common, and cardiac failure is also not unusual. The potential morbidity associated with cardiac failure and ischemic heart disease is apparent in the relative risks given to various preoperative risk factors in Goldman's landmark study of patients aged over 40 years (Exhibit 44-2).[1]

Patients with arrhythmia (or a history of arrhythmia) require an electrocardiogram, unless the problem is obviously benign. Atrial fibrillation is a common problem, and a rapid uncontrolled ventricular rate needs correction. Note any history of syncope, seizures, or repeated falls. Such patients may have a bradycardia, and any clinical diagnosis must be supported by an electrocardiogram because some uncommon conduction defects require cardiological intervention before

EXHIBIT 44-1

PHYSIOLOGICAL CHARACTERISTICS OF THE ELDERLY RELEVANT TO ANESTHESIA

- Lung volumes decrease with age; lung is less elastic. Consequent reduction in gas exchange.
- Atheroma and reduction in myocardial contractility cause impaired response to hemorrhage; this situation is worsened by arrhythmia or conduction disorders.
- Hypotension has potentially significant implications for cerebral and coronary artery perfusion.
- Renal function: kidney mass, volume, and blood flow are reduced, with a consequent reduction in GFR. Intolerance of water and salt loading
- Temperature control: loss of subcutaneous fat and reduced muscle mass cause susceptibility to hypothermia.
- Basal metabolic rate is lowered, which also contributes to difficulty in maintaining normothermia.

GFR: glomerular filtration rate

EXHIBIT 44-2

GOLDMAN RISK FACTOR AND SCORE

- Third heart sound (11)
- Elevated jugular venous pressure (11)
- Myocardial infarction in past 6 months (10)
- Electrocardiogram shows ventricular ectopic activity or nonsinus rhythm (7)
- Age > 70 years (5)
- Emergency procedure (4)
- Known or suspected significant aortic stenosis (3)
- Intrathoracic, intraabdominal, or aortic surgery (3)
- Poor general status (3)

Patients with scores > 25 had a 56% incidence of death and a 22% incidence of cardiovascular complications.

Data source: Goldman L, Caldera DL, Nussbaum SR, et al. Multifactorial index of cardiac risk in noncardiac surgical procedures. *N Engl J Med*. 1977;297:845-850.

EXHIBIT 44-3

FACTORS INFLUENCING POST-OPERATIVE RESPIRATORY FAILURE

- age > 65 years
- ASA class ≥ II
- cachexia
- cardiac failure
- chronic obstructive pulmonary disease and smoking
- reduced consciousness level
- general anesthesia
- emergency surgery
- upper abdominal surgery, vascular surgery
- raised creatinine
- albumin < 35 g/L

ASA: American Society of Anesthesiologists

surgery. Beware of any new murmur found in patients aged 60 or over, particularly so if the physical signs suggest aortic stenosis. Patients with undiagnosed or poorly controlled hypertension are unlikely to be deferred for treatment. In the acute setting, recognize that pain and anxiety may be a factor.

Respiratory Disease

In certain geographical locations there may be an increased incidence of a specific respiratory pathology such as tuberculosis. In the developed world chronic obstructive pulmonary disease is frequent in the older population. These patients can often be improved by nebulized therapy and treating any coexistent chest infection. Preoperative physiotherapy can also be beneficial in improving physiological function. A chest radiograph can be decisive in excluding a pneumothorax or pleural effusion, and arterial blood gas analysis will measure the effects on gas exchange. Exhibit 44-3 lists preoperative features associated with postoperative respiratory failure in noncardiac surgery. These indices are extracted from a study of US veterans.[2] Like the Goldman index, this list demonstrates the direct influence of biological age and general medical condition on morbidity.

Renal Disease

Chronic renal impairment is frequent in elderly patients. In these cases, prolonged preoperative starvation is potentially deleterious, and preoperative intravenous fluids may be necessary.

Medication

When relevant, the history should include recording the patient's own medication. During the perioperative period a number of pharmacological influences on anesthesia and surgery are possible (Table 44-1).

Preoperative Clinical Investigations

In the United Kingdom, the National Institute for Clinical Excellence has produced exhaustive recommendations for preoperative testing in elective surgery.[3] The recommendations are based upon the fitness of the patient and the complexity of surgery. These are standards expected in mature healthcare systems; in the deployed environment, particularly when faced with a significant number of casualties requiring emergency care, their application must be dictated by sound clinical assessment of the patient's physiology. When managing trauma, investigations should include urinalysis; hemoglobin, urea, and electrolyte measurement; arterial blood gases; and digital radiology or computerized tomography as necessary.

TABLE 44-1

PHARMACOLOGICAL CONSIDERATIONS FOR SURGERY IN THE ELDERLY

Drug	Comments
Steroids	Dose must be increased in perioperative period to ensure adequate stress response.
Warfarin	Warfarin is long acting. In an emergency, reversal can be imitated with IV vitamin K (1 mg) and maintained with fresh frozen plasma (initial dose: 10–15 mL / kg) and/or further doses of vitamin K.
Insulin	Acute illness, surgery, and anesthesia disturb glucose homeostasis. For all major surgery (with patient dependent on intravenous fluids postsurgery), a sliding scale regime is required until normal oral intake is established.
Antihypertensives	The reduced plasma volume of the starved or hypovolemic patient dictates that diuretics and/or ACE inhibitors and angiotensin receptor antagonists be omitted before surgery.
Angina therapy	Maintain nitrates and calcium channel antagonists.
Antiarrhythmia medications	Continue perioperatively to optimize hemodynamics.
Antiplatelet medications	Note the implications for proposed regional anesthesia. Where permissible, preoperative omission may reduce hemorrhagic complications.
Bronchodilators	Patients should use their usual inhalers as usual to minimize the potential hazards of bronchospasm. Nebulized delivery may be required perioperatively.

ACE: angiotensin-converting enzyme; IV: intravenous

ANESTHESIA

There is no one superior anesthetic technique for this age group, but some key practical considerations are listed in Exhibit 44-4. With respect to drug delivery, this list might be summarized as "inject with care." The elderly patient can experience significant hemodynamic and respiratory depression following inappropriate intravenous bolus injections.

Regional anesthesia has traditionally proven very useful in elderly trauma patients, but will have limited utility in the presence of acute trauma associated with hypovolemia. In the normovolemic patient, the strength of regional techniques (even when provided as a single injection) is the ability to provide good perioperative analgesia while reducing exposure to opiates and volatile anesthetics. The relationship between regional anesthesia and thromboprophylaxis must be monitored.[4]

Preoperative fasting is poorly tolerated in the elderly. Accepted general guidelines for the "nil by mouth" period are 6 hours for solids and 2 hours for clear fluids. Sips of water should be allowed closer to the operation, as well as small quantities of water taken with any essential medications. When there is uncertainty over the exact time of surgery, it is sound clinical practice to commence a crystalloid infusion during the waiting period. The usual fluids are 0.9% saline and lactated Ringer (Hartmann) solution.

Preoperative intravenous fluids may also be necessary to correct dehydration or electrolyte imbalance. The formula should vary according to the patient's condition but must address the normal daily requirement (40 mL/kg), plus any additional losses resulting from illness or trauma and electrolyte requirements. Diuretics cause sodium and potassium loss in the urine. Hyponatremia and hypokalemia are easily worsened by using inappropriate fluid replacement therapy; it is an error to routinely prescribe 5% dextrose in large volumes. Anemia is also poorly tolerated by the elderly, particularly in the postoperative phase. The anesthetist should have a low threshold for transfusion.

Exhibit 44-5 describes an anesthetic technique used by the authors that provides an example of how the recommendations listed above can be successfully combined.

OPERATIONAL FACTORS

This chapter discusses patients being managed in a conflict zone alongside military casualties. The deployed military medical system should have a predefined eligibility matrix that prevents resources

EXHIBIT 44-4

ANESTHETIC CONSIDERATIONS IN ELDERLY PATIENTS

- Venous access: thin skin and inelastic vessel walls can make cannulation difficult.
- Induction agents: doses must be titrated to effect to avoid hypotension.
- Opiate intolerance: sensitivity to opiates occurs because of reduced metabolism; doses must be adjusted accordingly.
- Intravenous fluids: balanced salt solutions should be used for maintenance; DO NOT USE 5% dextrose. During surgery fluid overload and cardiac failure is a risk.
- Respiration: hypercapnia must be avoided because carbon dioxide narcosis can result.
- Critical target organs share a blood pressure/flow dependency; maintain normotension.
- Drug metabolism: drugs that rely on renal excretion require dose reduction; competitive muscle relaxants require adequate reversal to ensure full return of neuromuscular function.
- Hypothermia: ambient temperature control necessary as well as active warming of patient and fluids.
- Pressure area care: damage is more likely than in younger patients due to reduced body mass.
- Recovery from anesthesia: in the acute phase supplemental oxygen is necessary to modify diffusion hypoxia and reduce the metabolic consequences of shivering.
- Postoperative confusion is common; it is often drug related (eg, with opiates) but also arises for cognitive reasons.
- Regional anesthesia: attractive as a means of minimizing exposure to opiates but must be applied carefully in the presence of thromboprophylaxis or coagulation disorders.

EXHIBIT 44-5

GENERAL ANESTHESIA FOR INTERNAL FIXATION OF A FRACTURED NECK OF THE FEMUR

- Pre-oxygenate the patient.
- Establish secure intravenous access and check with a saline flush.
- Use slow intravenous induction. The final dose may be as little as one-tenth that used in a young patient.
- Anticipate hypertension during endotracheal intubation, followed by hypotension when a volatile agent is introduced.
- Decide what levels of hypertension and hypotension need intervention and respond with fluids or vasoactive drugs as necessary.
- After induction, perform a femoral nerve block and supplement with intravenous paracetamol.
- During surgery correct anemia or additional hemorrhage with transfusion.
- Encourage wound infiltration with further local anesthetic.
- Actively warm the patient throughout and prescribe supplemental oxygen during recovery.

EXHIBIT 44-6

CONSIDERATIONS FOR DEPLOYED MILITARY TEAMS

- Disposition and extent of local healthcare resources.
- Need to avoid overloading deployed facilities.
- Lack of support specialties for surgical teams, which limits the ability to investigate and optimize patient's medical condition.
- Treatment decision making is influenced by all of the above.

intended for military personnel from being drained inappropriately by treatment of indigenous populations. This system can create ethical difficulties, particularly when prolonged treatment or intensive care might otherwise be indicated. The eligibility matrix will vary from one conflict to another and will always be influenced by the existing local civilian medical facilities. Exhibit 44-6 lists some pertinent considerations for deployed medical directors and other senior commanders that can limit the level of care that might otherwise be offered.

SUMMARY

The accepted principles of anesthesia and perioperative care apply to the elderly population, but their practical applications must always respect and where necessary be modified by the physiology of old age.

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Chapter 45

OBSTETRIC ANESTHESIA

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INTRODUCTION

The management of obstetric cases by United Kingdom (UK) and US defense medical services has been a relatively rare event in recent operations. Practical experience from the deployed environment is therefore scarce. It is entirely plausible, however, that such cases may become a more significant feature of future US and UK operations, particularly those involving the management of refugee populations or relief following natural disasters (Figure 45-1). Consequently, medical personnel will need to maintain knowledge and skills in this area and include the potential for obstetric cases in operational medical planning.

The aims of this chapter are to:

- assess the likely challenges of obstetric care in the deployed environment,
- provide an overview of current civilian best practice, and
- point to some additional resources that may be helpful in preparing the military anesthesiologist for deployed obstetric practice.

CHALLENGES OF OBSTETRIC ANESTHESIA IN THE DEPLOYED ENVIRONMENT

In future operations, the number of obstetric cases presenting to UK and US military surgical facilities will depend on the tempo of operations (and consequent workload from trauma), as well as how the eligibility matrix for civilian personnel is conceived and applied. When humanitarian aims form a significant part of the overall concept of operations, there is likely to be a far greater emphasis on primary care, with a greater likelihood of obstetric referrals. In these situations, the potential obstetric workload is high, with caesarean section being the most likely nontrauma surgical intervention required in a conflict area.[1]

Figure 45-1. Operating theater door, Australia and New Zealand Army Corps Hospital, Banda Aceh, Indonesia, during Operation Sumatra Assist, 2004.
Photograph: Courtesy of the Australian Defence Force.

Preexisting Indigenous Standards of Care

The aim for all deployed medical teams should be to provide the highest possible standard of care. The provision of obstetric care in the absence of midwives or obstetricians will of necessity fall short of the clinical governance standards of the UK or United States. It is important, therefore, that the standard of care during deployment be considered in the context of the care otherwise available in the area of operations. Deployed providers should have an appreciation of the status of maternity care worldwide.

The World Health Organization (WHO) estimates that 1,000 women die daily from preventable causes related to pregnancy and childbirth. The leading causes are hemorrhage, infection, preeclampsia, obstructed labor, and unsafe abortion.[2] Where skilled intervention is not readily available, as in many developing countries, complications of childbirth become the leading cause of death for women of childbearing age.

United Nations (UN) Millennium Development Goal 5 aims to reduce maternal mortality by 75% between 1990 and 2015.[3] A number of national and international initiatives have been started to reach this goal, including "task-shifting": the training of traditional birth attendants and surgical technicians (capable of performing forceps and caesarean deliveries) to alleviate a lack of midwives and obstetricians.[4]

Despite such efforts, the statistics remain sobering. The published maternal mortality ratios (maternal deaths per 100,000 live births) for 2008 in the UK and United States were 8 and 17, respectively, compared with a world average of 251. In conflict zones, where healthcare infrastructure has broken down, this ratio can rise dramatically. In Sierra Leone the figure for 2008 was 1,033; in Afghanistan, 1,575.[5]

It is worth noting that a military force may have a far greater impact on maternal and neonatal mortality

with nonmedical interventions: by providing physical security, thereby minimizing sexual violence and allowing nongovernment organizations (NGOs) to operate freely; by ensuring safe water and sanitation; and by distributing shelter, food, and UN Population Fund and UN Children's Fund clean delivery kits.[6]

Obstetric Experience of Deployed Surgeons

Most deploying anesthesiologists have experienced obstetric anesthesia during their training. Deployed surgeons, however, may have little or no previous experience with obstetrics. Predeployment surgical training is likely to focus on core trauma surgery skills and may do little to advance the knowledge of obstetrics.

When humanitarian operations are planned or likely, individual or collective obstetrics training should be considered. The Australian Defence Force initiated simulation training in obstetrics for general surgeons following Operation Sumatra Assist in 2004, during which obstetric cases were numerous. If such training is not arranged at command level, individual hands-on training for surgeons by obstetric colleagues can prove to be time well spent. Guidance for surgeons can also be found in several field guides.[7–9]

Obstetric and surgical management is beyond the scope of this chapter, which is directed toward anesthesia and critical care. However, in the absence of obstetricians and midwives in the deployed environment, obstetric management will rely on shared anesthetic and surgical knowledge and skills. For these skills to be applied effectively, excellent communication and teamwork are mandatory.

Equipment Considerations

Military anesthesiologists may see pregnant patients in the context of obstetric emergency, trauma, or urgent nonobstetric surgery. The care of the parturient requires the ability to monitor fetal status and manage the unique physiologic changes that occur in pregnancy. These physiologic changes often complicate the treatment of other disease or injury. Pregnant patients may require equipment and drugs over and above what is routinely available in a field surgical unit. Some bulky or expensive items might be judged impractical to deploy where only an occasional obstetric case is expected. Such items might include neonatal resuscitation trolleys and cardiotocographs. Other important items, however, are readily transportable and should be considered as part of the equipment scaling whenever obstetric cases are possible or likely.

The World Federation of Societies of Anaesthesiologists established international guidelines for the practice of anesthesia in 1992, revising them in 2008.[10] These guidelines include recommendations for minimum equipment, medications, and supplies that should be available for the safe administration of anesthetics. The American Society of Anesthesiologists Task Force on Obstetric Anesthesia's 2007 practice guidelines for obstetric anesthesia provide both practice and equipment recommendations for the care of pregnant patients.[11] Table 45-1 provides a suggested equipment list that incorporates the recommendations of both organizations, while omitting those items that are routinely available in the deployed surgical facility.

Environmental Considerations

Normal Delivery

Sterility in the field environment can be difficult to maintain; fortunately, a sterile environment is not essential for a vaginal delivery. The UN clean delivery kits contain all the required supplies.[6]

Surgical Interventions and Nonobstetric Surgery

The same measures that are taken to provide a warm and sterile environment for a trauma case should be implemented to care for the pregnant surgical patient. In addition, steps must be taken to ensure that the operative environment is suitable for a neonate and that the operating room is prepared and equipped for the performance of a possible emergency caesarean section. A caesarean section may be a resuscitative surgery in its own right by serving to decompress the aorta and vena cava.[12] Surgical care of the parturient is therefore resource intensive, requiring additional nursing, anesthesia, and surgical assistance; advance planning is essential for its successful execution.

Preexisting Pathology in the Pregnant Patient

Obstetric Risk Factors

Where medical infrastructure is poor, unidentified maternal risk factors are of great concern when caring for the pregnant patient. Risk factors that should be identified in the obstetric history include quality of prenatal care, multiparity, previous caesarean sections and the techniques used, genital mutilation (see below), frequent or untreated venereal disease, and multiple sexual partners. These risk factors impact on the likelihood of maternal complications such as placenta previa, placenta accreta, ectopic pregnancy, uterine rupture, uterine atony, cervical adhesions, and preeclampsia.

TABLE 45-1

EQUIPMENT RECOMMENDATIONS FOR OBSTETRIC CARE

Durable Equipment	Drugs	Consumable Equipment
Maternal Care	***Spinal/ Epidural***	***Maternal Care***
Uterine displacement device (Cardiff wedge)	Bupivacaine "heavy,"0.5%	Spinal needles (25-g Whitacre or Sprotte [B Braun; Melsungen, Germany])
Fetal heart rate monitoring device (fetal stethoscope, ultrasonic stethoscope, Doppler ultrasound device or ultrasound imaging system with low frequency [<5 MHz] probe)	Lidocaine, 2%	Epidural kits
	Bupivacaine "plain,"0.5% (or levobupivacaine/ropivacaine as preferred)	***Neonatal Care***
Neonatal Care	***Uterotonic***	Face masks (sizes 00, 0, 1)
Pediatric anesthesia circuits (Ayres T-piece)	Oxytocin	LMAs (size 3-4)
Neonatal bag valve masks with PEEP valve (240–500 mL)	Ergometrine/methylergonovine	ETTs (size 2–4.5)
Pediatric skin temperature probes	Carboprost tromethamine	IV cannulae (22–24 g)
Laryngoscopes (Miller, Oxford, and Wisconsin, sizes 0–1)	Misoprostol	Low-pressure suction, tubing and catheters (size 8 Fr)
ETT stylets (size 6 Fr)	***Tocolytic***	Bulb suction
	Nifedipine	
	Nitroglycerine (tab and solution)	
	Antihypertensive	
	Labetalol	
	Hydralazine	
	Magnesium	
	Inotropic/Vasopressor	
	Ephedrine	
	Phenylephrine	
	Epinephrine	
	Antacid/Prokinetic	
	Ranitidine	
	Sodium citrate	
	Metoclopramide	
	Resuscitative	
	Intralipid (KabiVitrum Inc, Alameda, CA)	

ETT: endotracheal tube
IV: intravascular
LMA: laryngeal mask airway
PEEP: positive end-expiratory pressure
Data sources: (1) World Federation of Societies of Anaesthesiologists. The 2008 international standards for a safe practice of anaesthesia. *Anesteziol Reanimatol*. 2009;(6):4–10. (2) Hawkins JL, Arens JF, Bucklin BA, et al. Practice guidelines for obstetric anesthesia. American Society of Anesthesiologists Task Force on Obstetric Anesthesia. *Anesthesiology*. 2007;106(4):843–863..

Cardiac Disease

In the developing world, cardiac disease is high on the list of causes of maternal mortality. The most common presenting pathology is mitral stenosis secondary to rheumatic fever. Other conditions include other valve lesions, cardiomyopathy (sometimes relating to human immunodeficiency virus [HIV]), and ischemic heart disease.

Patients tend to present late with decompensation secondary to the increased cardiovascular demands of late pregnancy or labor. The pathology may be unknown, and unless sufficient transthoracic echocardiography skills are available within the deployed medical team, diagnosis will depend upon a comprehensive cardiovascular examination.

Key management principles include the following:

- Good analgesia should be maintained during labor to minimize sympathetic drive and myocardial oxygen demand.
- Caution should be exercised with fluids to avoid overload.
- Oxytocin should be given slowly and with caution because it may precipitate hypoten-

sion and tachyarrhythmias.
- In the patient showing signs of cardiac failure, invasive cardiovascular monitoring may assist in maintaining optimal fluid balance, cardiac output (with or without inotropic support), and hence placental perfusion. Patients should be monitored for 48 hours postdelivery because decompensation may occur postpartum.
- Both regional and general anesthesia are safe if performed carefully, although general and epidural techniques are generally safer than spinal anesthesia in patients with fixed cardiac output (cardiomyopathy or aortic stenosis). An elective caesarean section under "slow-loading" epidural is perhaps the most stable combination in the high-risk parturient.

Human Immunodeficiency Virus Infection

HIV infection is common in the developing world. In the absence of advanced end-organ sequelae, HIV represents a low risk to the parturient but a significant risk to the medical team (including from concurrent hepatitis B or C infection) and a high risk of vertical infection to the neonate. When a high viral load is known or suspected, planned caesarean section at 38 to 39 weeks is the safest mode of delivery.

Ideally, antiretroviral therapy should be given from the second trimester. If this has not been possible, postexposure prophylaxis should be given to the neonate (nevirapine and zidovudine) for 6 weeks with formula-feeding to reduce transmission risk. However, if formula-feeding cannot be done hygienically in the home setting, breast-feeding will be safer, with antiretrovirals continued until weaning is complete.

Malaria

Malaria is endemic in many parts of the developing world and presents with anemia and low birth weight. Infection rates are higher in primigravidae, and reduce with subsequent pregnancies (this protective effect is reduced with concurrent HIV infection). WHO control strategies include impregnated mosquito netting and intermittent preventive treatment with sulfadoxine-pyrimethamine during pregnancy. Advice on current recommendations for antimicrobial therapy in the pregnant patient should be sought from a microbiologist or infectious diseases specialist. Concurrent anemia may also require treatment (see below).

Anemia

Severe anemia (hemoglobin < 7 g/dL) is associated with increased maternal and perinatal mortality, as well as developmental problems in children. Causes include helminth infestation; HIV; malaria; sickle-cell disease; hemoglobinopathies; and dietary iron, B12, and folate deficiencies. Helminth infestation (most commonly hookworm) can be diagnosed by the presence of eggs in the feces. Where suspected (ie, in endemic areas), helminth infestation can be treated after the first trimester with a single dose of mebendazole 500 mg or albendazole 400 mg.

In acute presentation of anemia, particularly with concurrent hemorrhage, transfusion may be necessary to bring hemoglobin to appropriate levels. In the longer term, a rise of 0.2 g/dL/week can be achieved with ferrous sulfate 200 mg three times a day and folic acid 5 mg once a day.

Sexual Violence

Sexual violence has sadly been a feature of a number of recent conflicts, including those in Africa and the Balkans, with vulnerable subjugated populations and refugees subjected to systematic as well as opportunistic rape. As a consequence, medical staff should be alert to the potential psychological damage parturients may present with. These effects may manifest as increased fear of clinical examination or ambivalence toward the newborn child. Particular sensitivity should be exercised as well as maximum use of interpreters to facilitate good communication. Chaperones including female staff, friends, or relatives should be employed as appropriate.

Female genital mutilation, though internationally condemned, remains a widespread practice in about 28 countries in sub-Saharan Africa and Yemen, with an estimated 92 million women and girls affected.[13] The level of damage is variable, including a 15% to 55% increase in perinatal mortality, as well as urinary tract infections and cyst and abscess formation. Severe, constrictive scarring (following type III female genital mutilation, or infibulation) carries a high risk of failure to deliver vaginally, resulting in a 30% increase in caesarean sections and a 70% increase in postpartum hemorrhage.[14]

CURRENT CIVILIAN BEST PRACTICE

Patient Information

In all contact with pregnant women, efforts should be made to ensure that they have access to information, including an explanation of pain relief options as well as the risks and benefits of any surgical or

anesthetic interventions. All information should be given in appropriate language, either written or via interpreter. In patriarchal cultures, care should be exercised if male relatives are used as interpreters to ensure that the woman retains freedom of choice about her care.

Antenatal Care

A well-managed obstetric anesthesia service can help reduce maternal mortality. The deployed medical team may have limited access to parturients in the antenatal period. However, it may be possible to establish cooperative liaisons with local antenatal or primary care services, whether indigenous or provided by NGOs. Guidelines should be made available to local primary care clinicians on conditions requiring referral.

Management of Normal Labor

Aortocaval Compression

Pregnant women should never lie completely supine because the gravid uterus will cause aortocaval compression, leading to compromised placental perfusion. A tilting table, wedge, or pillows should always be used to create 15 degrees of left lateral tilt.

Pain Relief During Labor

Pain in the first stage of labor is caused by uterine contractions and progressive dilatation of the cervix, and so falls within a T8–L1 nerve root distribution. During the second stage, pain is also caused by stretching of the birth canal and perineum (T8–S4 nerve roots).

Parental Opioids. All parental opioids may cause respiratory depression, so both mother and baby need to be monitored closely in the intrapartum and postpartum period.

- ***Pethidine***. This drug is widely used by midwives in the UK as a first-line drug for labor analgesia. It may sometimes be available in the deployed environment. It is usually administered intramuscularly (IM), 75 mg every 4 hours. Its onset is within 10 minutes and it lasts 2 to 3 hours. It readily crosses the placenta and becomes ionized in the relative acidic fetal circulation leading to accumulation. Peak fetal concentrations occur 2 to 3 hours after administration. It should be used with caution in patients with epilepsy, renal failure, or preeclampsia.
- ***Morphine***. Morphine 5 to 10 mg may be administered IM or intravascularly (IV) as an incremental bolus dose or via a patient-controlled analgesia pump. Its peak analgesic effect is 30 to 60 minutes after IM administration, and it lasts for 3 to 4 hours. It rapidly crosses the placenta but diffuses back into the maternal circulation.
- ***Fentanyl***. IV boluses of 25 to 100 μg can be effective up to 30 to 60 minutes. It is highly lipid soluble and readily crosses the placenta.
- ***Remifentanil***. Because of its short elimination half-life (9.5 min) and context-sensitive half-time (3 min), remifentanil provides high quality analgesia without neonatal depression. It is therefore becoming widely used in UK and US obstetric practice, and is likely to be available in the deployed setting. It is best used in the form of a patient-controlled analgesia regimen. It should be administered by experienced practitioners only, using an established protocol such as a 40-μg bolus with a 2-minute lockout.
- ***Inhalational Methods: Entonox.*** A 50:50 mix of oxygen and nitrous oxide with dissociation and relaxation properties, Entonox (BOC, Guildford, UK) decreases the mother's perception of labor pain. The drug diffuses freely across alveolar membranes to provide rapid effects with minimal accumulation, but 15% of mothers experience nausea. Under anesthetic supervision, isoflurane 0.2% or sevoflurane 0.4% can be added to provide additional anxiolysis and sedation.

Regional Techniques. Indications for regional analgesia in labor (both epidural and combined spinal-epidural techniques) include maternal request, preeclampsia, augmentation or induction of labor, maternal cardiac or respiratory disease, predicted difficult airway or general anesthesia, or occipito-posterior presentation. Regional analgesia should not be used in labor unless an obstetric team is available in the same hospital to treat emergencies. There should be a regional analgesia record and a protocol for prescription and administration of epidural drugs. The provider must adequately inform the patient and obtain her consent. The patient must have wide-bore IV access before a regional technique is performed, and Intralipid (KabiVitrum Inc, Alameda, CA) should be available as a resuscitative drug to treat local anesthetic toxicity.

- ***Epidural***. Once an epidural is inserted for labor, an appropriate block should be estab-

lished. The possibility of intrathecal and IV placement cannot be ruled out until the epidural is tested. Following a test dose (typically 3–4 mL of bupivacaine 0.25%), bolus doses of local anesthetic solutions (eg, bupivacaine 0.1%–0.25% or equivalent ropivacaine) are injected to give sufficient analgesia. In the first stage of labor, the block must extend from T8 to L1; in the second stage, from T8 to S4. Maintenance of analgesia can be achieved with further bolus doses or continuous infusion (a typical infusion in the United Kingdom contain 0.1% bupivacaine with 2 μg/mL fentanyl. In the United States, 0.2% ropivacaine is typical). Epidurals that provide good quality analgesia for labor can also be used for caesarean section, forceps delivery, perineal suturing, and manual removal of placenta.

- ***Combined Spinal-Epidural.*** A combined spinal-epidural can be used to achieve rapid onset of analgesia in labor. Either a needle-through-needle technique or separate insertion sites can be used. The spinal component must use a lower dose than for operative interventions (see below), typically consisting of 1 mL of 0.25% bupivacaine with 25 μg fentanyl.

Management of Operative Interventions

Regional Anesthesia

Regional anesthesia is the method of choice for all operative interventions. A block to T4 is required for caesarean section (or for trial of forceps to enable a swift conversion to caesarean section if required). If an epidural is in place for labor analgesia and is working well, it can be "topped-up," typically using either

- 0.5% bupivacaine (in 5 mL increments up to 30 mL)

or

- fentanyl 100 μg plus 5 mL increments of a mixture containing 2% lidocaine (17 mL), epinephrine (1 mL of 1:10,000) and bicarbonate (2 mL of 8.4%) titrated to the block level.

In the absence of an existing epidural, spinal anesthesia offers a more rapid onset of block with a lower incidence of inadequate block requiring conversion to general anesthesia. This is usually achieved with a 24- or 25-gauge Whitacre or Sprotte (B Braun; Melsungen, Germany) needle at the L2-3 or L3-4 interspace. The block is typically established with 2.5 to 2.6 mL of 0.5% "heavy" bupivacaine, with the addition of 300 to 400 μg diamorphine or 15 μg fentanyl.

General Anesthesia

General anesthesia is necessary when regional anesthesia is contraindicated, refused, unavailable, or unsuccessful. Rapid sequence induction is mandatory, and the anesthesiologist should expect and prepare for difficult intubation. Pregnant women are at particular risk of aspiration because of reduced effectiveness of the lower esophageal sphincter, increased intragastric pressure caused by a large uterus, and delayed gastric emptying if opiates are used during labor. The increased risk of aspiration starts from 16 to 18 weeks' gestation and continues into the first week postpartum. The risk is reduced by avoiding general anesthesia (using regional anesthesia when possible) and by decreasing gastric volume and acidity with the administration of H_2 antagonists (ranitidine 150 mg, orally, every 6 h or 50 mg slow IV together with metoclopramide 10 mg orally or IV when general anesthesia becomes likely. Rapid sequence intubation with cricoid pressure should be performed if control of the airway is required, with nonparticulate antacids (sodium citrate 30 mL orally) given prior to preoxygenation.

Fetal Monitoring

The American Congress of Obstetricians and Gynecologists Committee Opinion 474 holds that if the fetus is considered viable, fetal heart rate and contraction monitoring should be done pre- and postoperatively[15] for surgery in pregnant patients. However, it is unlikely that the equipment necessary for contraction monitoring will be available in the field environment, nor is it likely that individuals trained in the interpretation of such monitors will be at hand. The American Society of Anesthesiologists Practice Guidelines recognizes that "continuous electronic recording of the fetal heart rate may not be necessary in every clinical setting."[11] This guideline agrees with the observation that neonatal outcome is most affected by the course of the maternal illness, rather than any specific monitoring modality, unless the fetal monitoring affects maternal outcome.[16] It would seem appropriate to monitor fetal heart rate at regular intervals (with frequency determined by the clinical course) when managing any parturient to assess fetal well-being and the possible need for emergent delivery. Additional fetal monitoring is likely of no benefit. In the case of nonobstetric surgery, pre- and postoperative monitoring should be sufficient.

Tocolysis

Uterine relaxation may be required to facilitate removal of retained placenta or to aid in the delivery of infants with head entrapment during a caesarean section. Short-duration uterine relaxation can be accomplished with nifedipine, volatile anesthetics, or nitroglycerine.

Procedures

- **Assisted delivery**. Takes place on the ward with an obstetrician in attendance. The patient often requires a good working epidural.
- **Trial of forceps**. Takes place in the operating room because of the significant likelihood of conversion to caesarean section. Regional analgesia needs to be in place with a block up to the level of T4. Oxytocin 5 units IV should be given after cord clamping.
- **Caesarean section**. Should be under regional anesthesia unless contraindicated or there is insufficient time to establish a block due to severe maternal or fetal compromise. A T4 block is required. Oxytocin 5 units IV should be given after cord clamping.
- **Manual removal of placenta**. Performed in the operating room. Close observation of maternal blood loss is required. Tocolysis may be required.
- **Perineal repair**. Can be performed on the ward using a local anesthetic block by the obstetrician. More extensive tears (grade 3–4) are repaired in the operating room.

Management of High-Risk Conditions

Preeclampsia and Eclampsia

Preeclampsia complicates up to 8% of pregnancies and is characterized by hypertension, proteinuria, and edema. If uncontrolled it can lead to death from intracranial hemorrhage, pulmonary edema, or hepatorenal failure with coagulopathy (HELLP syndrome: hemolysis, elevated liver enzymes, and low platelets). Hypertension should be controlled with labetalol or hydralazine. Headache, epigastric pain, and hyperreflexia are markers of severe disease and impending eclamptic seizure and should prompt the use of a magnesium infusion and a plan for early delivery.

In eclamptic seizures, immediate infusion of magnesium (4 g IV over 20 min followed by 1 g/h IV infusion) should be added to supportive treatment. Caesarean section should not be performed until blood pressure has been controlled, due to the high risk of cerebral and operative hemorrhage.

Regional anesthesia is safe and effective provided that platelet numbers are adequate. If general anesthesia is required, a generous induction agent and opioids should be used to reduce the sympathetic response to laryngoscopy, which may provoke intracranial hemorrhage (opioid narcosis in the neonate should be expected and managed with naloxone if required).

Throughout management, fluids should be restricted (typically replacing losses plus 85 mL/h) to avoid pulmonary edema. Fluid boluses may be given to counteract hypotension caused by regional anesthesia (although parturients with preeclampsia are relatively protected from this effect), but oliguria in the first 24 to 48 hours postpartum should not prompt excessive fluid administration. The risk of eclamptic seizures and HELLP syndrome persists for several days postdelivery, so close observation and serial electrolytes, blood counts, and liver function tests are mandatory.

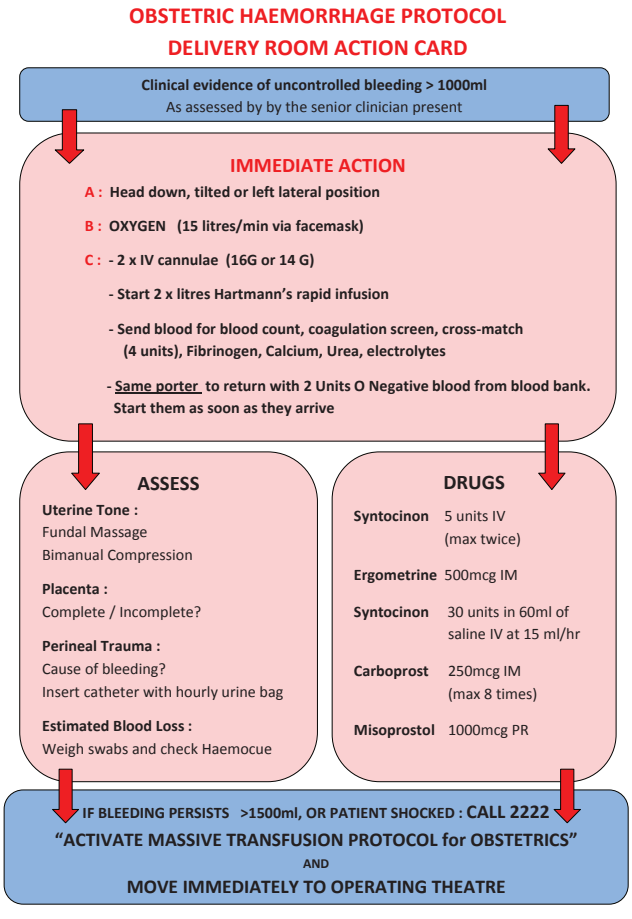

Figure 45-2. Obstetric Haemorrhage Protocol Delivery Room Action Card, printed by Siggers B, Baden-Fuller J, January 2011 (reproduced with permission).

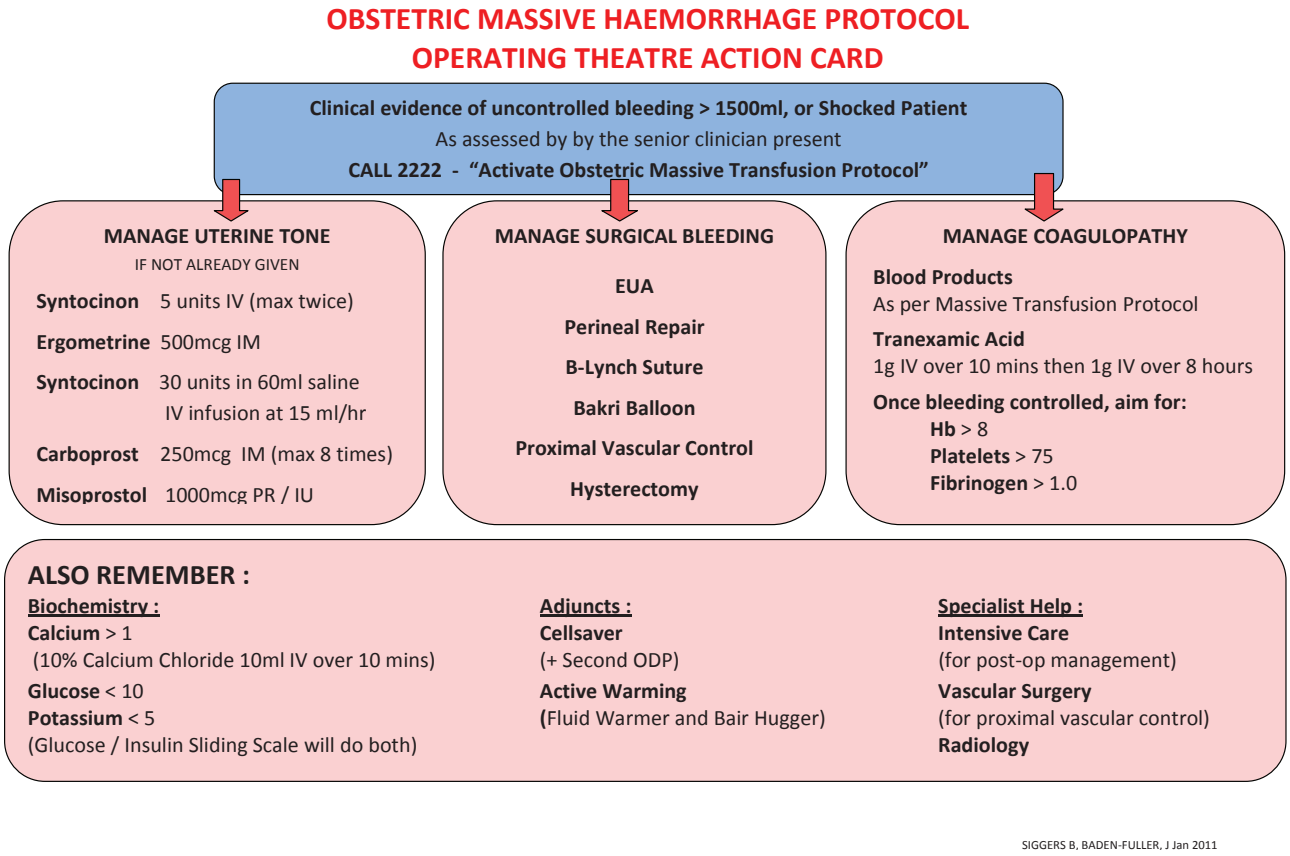

Figure 45-3. Obstetric Massive Haemorrhage Protocol Operating Room Action Card, printed by Siggers B, Baden-Fuller J, January 2011 (reproduced with permission).

Massive Hemorrhage

Antepartum hemorrhage may occur in association with placental abruption or uterine rupture. Postpartum hemorrhage is most commonly associated with uterine atony (risk factors include placenta previa, multiparity, twins, and prolonged labor) as well as placenta accreta or cervical, vaginal, or perineal tears.

Both the hemorrhage and associated coagulopathy may be rapid and catastrophic and should be managed aggressively in line with the massive transfusion protocol in place for traumatic hemorrhage. In addition, measures to stop the bleeding should include mechanical uterine compression, surgical hemostatic maneuvers, and pharmacological means to restore uterine tone. Uterine muscle requires good perfusion to contract effectively. Figures 45-2 and 45-3 provide algorithmic guides to initial and operative management of obstetric hemorrhage. Initial transfusion should occur with type O, Rh-negative blood until type-specific or cross-matched blood is available, to prevent isoimmunization.[12]

Amniotic Fluid Embolism

Contamination of the maternal circulation with amniotic fluid containing fetal skin squamae at the time of delivery may produce a systemic inflammatory response as well as an embolic effect if the bolus of amniotic fluid is large. The effects range from mild to complete cardiovascular collapse and cardiac arrest. Management is wholly supportive.

Trauma

Since most military medical facilities are deployed with the primary objective of managing trauma, many of the pregnant patients who present do so because of traumatic injury. An understanding of the interaction between pregnancy and trauma is fundamental to effective management of the pregnant trauma patient.[16,17]

Pattern of Injury. Trauma to the abdomen or pelvis may cause placental abruption or uterine rupture, resulting in fetal compromise or maternal blood

loss, amniotic fluid embolism, or coagulopathy. The fetus may also be injured directly. The uterus remains within the pelvis up to 12 weeks gestation. From 12 to 20 weeks, the fundus rises from the pelvic rim to the level of the umbilicus, and by 36 weeks the uterine fundus reaches the anterior costal margin. In the first trimester the uterus is thick-walled as well as being protected within the pelvis. In the second trimester, the fetus is relatively cushioned from blunt and penetrating trauma by the uterine wall and surrounding amniotic fluid. However, by the third trimester, the uterine wall is thinner and the fetus larger, so the uterus and fetus are vulnerable to direct trauma, while other organs are relatively protected behind or above the uterus.[18]

Hemodynamics. Physiological changes in pregnancy have evolved to provide for both increased cardiac output during labor and increased ability to survive blood loss during delivery. The important consequence for major trauma in late pregnancy is the increased capacity to compensate for bleeding, which may provide false reassurance by masking signs of blood loss. If a source of bleeding is missed as a consequence, the eventual decompensation can be sudden and catastrophic. A high index of suspicion is mandatory.

Aortocaval compression in the supine position may severely compromise maternal cardiac output. Compromised placental perfusion may be one of the first consequences of maternal hypovolemia, so fetal distress may occur before signs of shock in the mother.

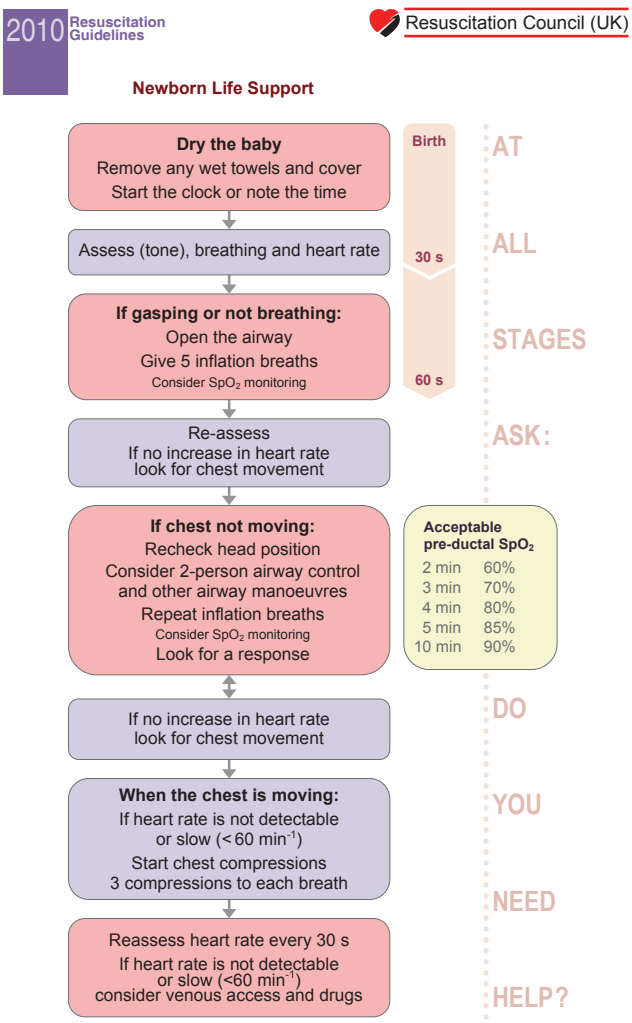

Figure 45-4. Resuscitation Council (UK) 2010 Resuscitation Guidelines, Newborn Life Support algorithm card. Reproduced with the kind permission of the Resuscitation Council (UK).

Trauma Management Principles

- Maintaining maternal physiology is the best way to protect the fetus.
- Manage with left tilt to prevent aortocaval compression.
- Maintain maternal cardiac output aggressively to maintain placental perfusion.
- Actively look for signs of abruption as part of the primary survey. These signs include perivaginal bleeding and uterine tenderness or irritability (frequent contractions or tetany, contractions triggered by palpation)
- Actively look for signs of uterine rupture as part of the primary survey. These signs include easily palpable fetal parts, cramping pain, and tenderness and guarding, especially when associated with maternal shock or fetal distress.
- Focused abdominal ultrasound scan for trauma (FAST scan) may assist in diagnosing both of these conditions, but the presence of the pregnant uterus may make it more difficult to see free fluid elsewhere in the abdomen.
- Indications for caesarean delivery include uterine rupture (including penetrating injury to the uterus), significant placental abruption, or fetal compromise.
- Monitor fetal wellbeing for 48 hours after maternal stabilization.
- Rh-negative mothers with torso trauma should receive rhesus immunoglobulin therapy within 72 hours of injury due to the high risk of fetomaternal hemorrhage.

Postnatal Care

Observation

Basic observations are required for all women postdelivery, including heart rate, blood pressure, respiratory rate, pulse oximetry, sedation scores, and pain scores. Effective postdelivery monitoring reduces the incidence of maternal morbidity and mortality. All women who receive anesthesia or regional analgesia should receive an anesthetic review between 18 and 36 hours postdelivery. The review should include inquiries into effectiveness of analgesia or the anesthetic intervention, maternal satisfaction, and any anesthetic-related morbidity.

Fluid Management

Careful fluid management is required to prevent postoperative renal failure or postoperative fluid overload and pulmonary edema. This is particularly important in the presence of preeclampsia (see above). Preoperative deficit, blood loss during delivery, and daily maintenance should be taken into consideration.

Pain Control

A multimodal approach is required, with a combination of paracetamol (acetaminophen), nonsteroidal antiinflamatory drugs (NSAIDs), and opioids. NSAIDs should not be given in cases of large blood loss or preemclampsia.

Neonatal Care

Most newborn babies require only minimal intervention following birth; however, some will need extra support. Many cases of death and disability can be prevented by early recognition of at-risk pregnancies, timely intervention for complicated labor, and immediate resuscitation of at-risk babies. Following birth, the newborn should be dried and covered to conserve heat. An assessment should be made as to whether further intervention is required. For the compromised newborn, resuscitation is a priority. Figure 45-4 shows the Resuscitation Council (UK) Newborn Life Support algorithm (2010), an internationally recognized best practice standard.

RESOURCES FOR THE DEPLOYING ANESTHESIOLOGIST

Civilian Best Practice

Training with obstetric anesthesia colleagues remains the most effective means of revising and updating skills in preparation for deployment. A variety of organizations also offer courses and seminars aimed at updating civilian obstetric anesthesia practice. These include, in the UK, the Obstetric Anaesthetists Association, and in the United States, the Society for Obstetric Anesthesia and Perinatology. Current literature, in particular the *International Journal of Obstetric Anesthesia*, contains relevant articles from the developing world, as well as US and UK best practice. Textbooks of particular relevance to this subject include *Obstetric Anaesthesia for Developing Countries* by Clyburn, Collis, and Harries.[19]

Military Experience

The Australian Defence Force medical services have encountered numerous obstetric cases in recent years following a series of operations with a humanitarian component. These include operations in East Timor (1999), tsunamis in Papua New Guinea (1998) and Indonesia (2004; see Figure 45-1), and the 2005 Pakistan earthquake. Reports of these deployments provide helpful insights into the role and challenges facing military medical teams in such circumstances.[20]

Specific advice on the management of ballistic trauma in the pregnant patient can be found in the relevant chapter of *Ryan's Ballistic Trauma* (3rd edition).[21]

Deployed Civilian Experience

The burden of obstetric care in conflict and disaster areas in recent years has largely fallen on NGOs. Guiding principles for maternity services were agreed on by the Reproductive Health Response in Conflict Consortium and published as a field-friendly guide in 2005.[6] The WHO also includes practical guidance for obstetric surgical care in the 2003 manual *Surgical Care at the District Hospital*.[7] This excellent resource includes downloadable teaching slides, videos, and self-directed learning aids that can assist in team training prior to or during deployment. The WHO also provides a range of topic-specific clinical guides, available with open access at www.who.int/reproductivehealth/publications/maternal_perinatal_health. A recent editorial by Dyer et al[22] discusses equipment and principles and includes example protocols for anesthetic management.

SUMMARY

This chapter provides a guide for deploying anesthesiologists and surgical teams who may encounter pregnant patients. It offers some context in the form of a summary of obstetric care worldwide and lists likely challenges. It suggests options and resources for equipment and individual and team preparation. Finally, the chapter provides a detailed breakdown of civilian best practice and how it can be best applied to the deployed military setting.

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Chapter 46

ANESTHESIA FOLLOWING CHEMICAL, BIOLOGICAL, RADIOLOGICAL, AND NUCLEAR EXPOSURE

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INTRODUCTION

In the current global political climate, the possibility of a chemical, biological, radiological, or nuclear (CBRN) incident in combination with a mass casualty situation cannot be ignored. Although unsophisticated groups may attempt a chemical, biological, or radiological attack, the nuclear option is likely only available to nation states. The probability of a nuclear incident is significantly lower than a chemical, biological, or radiological event, but the potential impact of any CBRN attack would be catastrophic. Recent natural disasters throughout the world, such as the devastating earthquake and subsequent cholera epidemic in Haiti in 2010 and the earthquake-induced tsunami in Japan in 2011 (which resulted in a radioactive zone bigger than that left by the 1945 bombings of Nagasaki and Hiroshima) demonstrate how sudden and debilitating a CBRN event can be. Emergency preparedness and planning for CBRN threats is imperative in any community.

Chemical, biological, and radiological events are difficult to recognize and are completely transformative incidents. Traditional chemical weapons are rare, but toxic industrial chemicals (TICs) contribute myriad compounds during combustion and other reactions. Many of the treatments for exposure are supportive, but misdiagnosing a toxidrome for which an antidote exists is a serious shortcoming. Biological and radiological releases are rarely recognized at the scene without adequate background intelligence or detectors and only become apparent as illness progresses.

This chapter aims to provide practical guidance and reassurance to critical care specialists faced with casualties exposed to CBRN events, including TIC exposures. It is likely that intoxication and trauma will combine to worsen the prognosis. However, with careful forethought and preparation, deviating from damage-control resuscitation for poisoned patients should be unnecessary, and trauma surgery can be performed in concert with chemical resuscitation.

INCIDENT MANAGEMENT AND EMERGENCY RESPONSE

Incident Management

When faced with the prospect of multiple poisoned casualties, individual safety must be a priority before attention is turned to the scene and the injured. In the United Kingdom, incidents are managed using the standard major incident medical management and support approach, with minor differences (Figure 46-1). Initial priorities include establishing safety, cordons, command, and communication. Command and control personnel, when possible, should don personal protective equipment (PPE) and be located upwind and uphill from the incident. In the United States, disaster management occurs on three distinct levels or tiers: (1) federal, (2) state, and (3) local responses. In most instances, the local community establishes initial and lasting response authority and sets up the incident command center through an incident commander, often a fire chief. The incident commander is responsible for directing and controlling resources. Prior planning in the predisaster period is crucial to preventing widespread panic and a deteriorating mass casualty situation.

Recognizing Chemical, Biological, Radiological, and Nuclear Incidents

In the absence of local intelligence or obvious environmental clues, recognizing that an incident has a CBRN aspect may be difficult, depending on the agent. Scene assessment precedes clinical assessment, and surveyors should pay attention to unusual smoke, smell, liquids, or patterns of dead animals, including insects. The diagnosis of nerve-agent exposure in Tokyo, Japan, in 1995 was made by clinicians noticing that many of the patients in cardiac arrest had miosis, a situation encountered the year before in the Matsumoto sarin attack.[1] The "Safety Triggers for Emergency Personnel 1-2-3" approach is used by many emergency services to aid recognition. For example, a single patient with symptoms indicating CBRN exposure is likely explained by disease processes other than CBRN exposure, but the arrival of three or more patients with similar suspicious symptoms should alert providers to a CBRN attack. A blast in a confined space is likely to produce some incomplete combustion products that may be harmless in low exposure but could trigger PPE use in the presence of simple detection methods (Exhibit 46-1).[2]

The London bombings of July 7, 2005, demonstrated the uncertainties of a terrorist attack at multiple sites.[3] Initial scene and casualty assessment did not suggest a chemical or radiological hazard. Dust masks would have provided adequate protection from a low-level radiological dispersal device.[4] Radiological detectors are unlikely to be distributed in similar mass-casualty situations. Additionally, excessive dust rarely prompts unsuspecting troops to protect them-

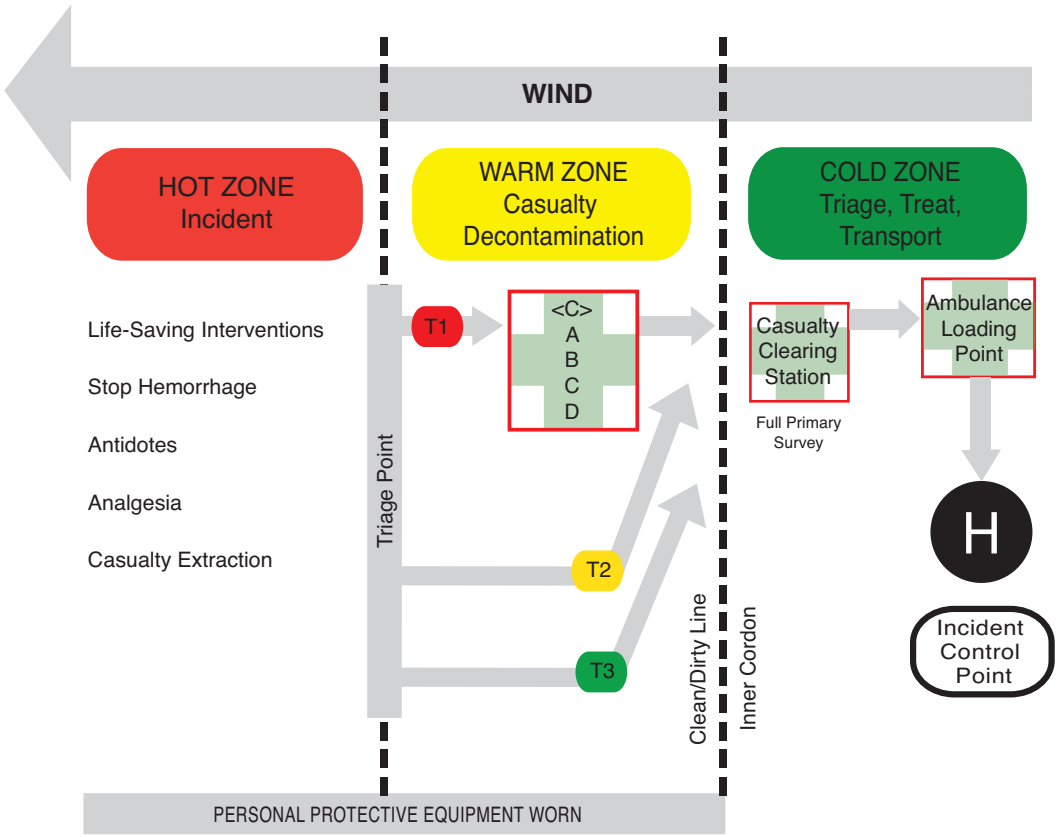


Figure 46-1. Medical hazardous materials site plan. Note the attention to wind direction in assisting decontamination. Care in the "hot zone" is limited to life-saving interventions, including antidotes as dictated by the clinical situation. For tier 1 (T1), serious casualties, decontamination does not detract from the primary assessment and treatment of catastrophic hemorrhage, airway, breathing, circulation, and disability (<C> ABCD). Tier 2 (T2) refers to urgent cases; tier 3 (T3) to delayed case. H: hospital

selves adequately. Given modern weaponry, people worldwide must maintain a high index of suspicion for a CBRN event.

"Quick Look"

"Quick Look" is a rapid method of assessing intoxicated and injured casualties. It is based on the brisk evaluation of a casualty with minimal exposure, to recognize classic toxidromes (Table 46-1).[5]

Personal Protective Equipment

Casualties may be treated within a civilian or field hospital or by an emergency response team at an incident scene. The incident commander should advise medical staff of the type of incident and the appropriate PPE; however, this may not be known at the time, and the correct PPE may not be readily available. Issued respirators will not protect against many TICs and provide no protection from carbon monoxide inhalation. The US Centers for Disease Control and Prevention classifies PPE into four levels (A–D):

- A: self-contained breathing apparatus and gas-tight outer suit.
- B: self-contained breathing apparatus and splash-proof outer suit.
- C: particulate and chemical respiratory filter; this includes the UK military CBRN PPE suit and National Health Service hazardous materials suit.
- D: standard precautions and high-specification particulate filter mask.[6]

Tracheal intubation and potential exposure to bodily fluids and airborne infection dictate that, when possible, minimum PPE should include full-length gown, face mask (high-specification particulate filter mask if airborne infection is expected), eye protection, and

EXHIBIT 46-1

INITIAL INVESTIGATIONS FOR CASUALTIES SEVERELY AFFECTED BY A CHEMICAL, BIOLOGICAL, RADIOLOGICAL, OR NUCLEAR INCIDENT

Conduct the following initial investigations for critical-care patients exposed to chemical agents, where available. Calculation of anion gap may aid diagnosis.

Urea and Electrolytes
- Arterial blood gas analysis with glucose and lactate
- Calcium/phosphate/magnesium
- Liver function
- Amylase/creatine kinase

Full Blood Count
- Store admission blood sample for later specialist analysis.
- Consider clotting studies/group and hold

Urinalysis
- Store admission urine sample for later specialist analysis

Electrocardiogram

Chest Radiograph

single, disposable gloves (level D). If a chemical incident is suspected, a chemical-resistant coverall with integral hood, respirator, chemically resistant boots, and gloves should be worn. If a casualty is not fully decontaminated from a radiological incident, level D standard precautions with double gloves will suffice.[7] In an active CBRN environment, all responders should be wearing individual protection equipment (Figure 46-2). Standard-issue individual protection equipment for UK military includes a CBRN respirator, clothing containing reinforced nylon with charcoal-impregnated felt, butyl rubber gloves, and boot coverings, leaving no skin exposed (level C). For chemical incidents in the field, medical personnel should wear level C PPE with surgical gloves instead of butyl and change them every 10 to 15 minutes.[8]

US CBRN respirators come in several models, including the M9, M17, and M40 series. The UK S10 respirator filter canisters provide good protection against particles greater than 0.3 μm.[9] Military respirators generally provide a level of N95/high-specification particulate filter mask particulate protection (Bland SA, surgeon commander, Royal Navy; personal communication, October 2011). Radiologically contaminated casualties do not generally pose a danger to healthcare workers and first responders; the rare exception to this is when a highly radioactive piece of shrapnel is embedded in a patient presenting for treatment.

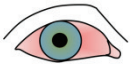
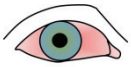
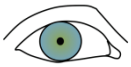
Decontamination

Decontamination after exposure to a CBRN hazard is intended to remove or destroy the contaminant and reduce the risk of harm to the patient, healthcare professionals, and others. Decontamination was the first action before treatment, but this doctrine has recently changed[8] and decontamination is now incorporated into a modified triage and treatment protocol, meaning syndrome recognition and antidote administration take precedence. The most common antidote, which can be administered to oneself or by a buddy, is the nerve agent ComboPen (produced by the UK Ministry of Defence), which contains 2 mg atropine, 500 mg pralidoxime, and 10 mg avizafone (diazepam equivalent 5 mg). Life-saving interventions (LSIs), including tourniquet application for catastrophic hemorrhage, airway maneuvers, and antidotes via the intraosseous (IO) route, can occur in the "hot," or contaminated, zone before formal decontamination occurs (see Figure 46-1).[8,10]

Decontamination may simply involve clothing removal and increased air circulation around the casualty to remove gases and vapors. Physical removal of persistent contaminants should begin as soon as possible by washing with high-flow water with or without detergent.[11] Casualties affected by gases such as hydrogen cyanide or vapor of high volatility do not require decontamination. Destruction of the contami-

TABLE 46-1

"QUICK LOOK" CHART: EFFECTS OF MAJOR CHEMICAL AGENTS AND RELATED SUBSTANCES*

	Symptoms and Signs					
Agent	**Consciousness Level**	**Respiration Rate**	**Eyes**	**Skin**	**Secretions**	**Other/Notes**
Carbon monoxide	Confusion, headache	↑↑	Normal 	Pink	Normal	None
Cyanides	LOC, seizures	↑↑ then ↓	Normal or pupils dilated 	Pink, then cyanotic	Normal	Sudden onset of symptoms
Lung-damaging agents	Agitation	↑↑	Normal or red 	Normal, then cyanotic	Pink frothy sputum	None
Vesicants, acids, alkalis	Normal	Normal or ↑	Normal or red 	Red (delayed)	Normal or ↑	With mustard gas, symptoms and signs may be delayed
Nerve agent	Seizures	↑then ↓	Pinpoint pupils 	Sweaty	↑↑	Fasciculation, bronchospasm
Botulinum	Normal	↓	Dilated pupils 	Dry	↓	Descending paralysis
Opioids	↓	↓↓	Pinpoint pupils 	Normal	Normal	Increased tidal volume
Atropine	Confusion, agitation	↑	Dilated pupils 	Dry	↓↓	None
Methemoglobin	Agitation	↑	Normal 	Cyanotic	Normal	"Chocolate blood," SpO_2: 89%

*Clinicians should be alert to the possibility of a combination of agents and coexistence of trauma. Confusion may be the result of head injury, and a pneumothorax could mimic severe bronchospasm.

↑: increased

↑↑: significantly increased

↓: decreased

↓↓: significantly decreased

LOC: loss of consciousness; SpO_2: oxygen saturation in blood

Data source: Bland SA. Chemical, biological and radiation casualties: critical care considerations. *J R Army Med Corps.* 2009;155:122–174.

Figure 46-2. Chemical, biological, radiological, and nuclear personal or individual protective equipment (Level C) will protect you, the casualty, and your colleagues only if selected, worn, and discarded correctly. Hand hygiene and sharps disposal discipline are paramount.
Reproduced with permission from: United Kingdom Ministry of Defence; ©UK MOD Crown Copyright 2011.

nant using chemical deactivation through hydrolysis, oxidation, or active decontamination specific to the agent is a secondary goal. In the case of radioactive contamination, LSIs should always be instigated before decontamination.

All casualties should ideally be decontaminated at the scene before transfer to the emergency department (ED) or next-level medical facility; however, in a civilian scenario, some contaminated casualties may self-present to the ED, increasing the risk to other patients and staff. In this scenario, the fire service or ED staff may have to decontaminate patients at the ED.[11] In the 1995 Tokyo subway sarin attack, 20 physicians were exposed to the 3,227 patients who were treated at local hospitals. Although many of these physicians experienced varying signs and symptoms of nerve agent exposure (dim vision, miosis, rhinorrhea, and dyspnea), including six who actually received atropine, none was forced to abandon patient-care responsibilities. Recent studies conducted in the United States suggest that hospitals are not prepared for a biological or chemical event in an urban area, including the resultant mass decontamination and medical response.[12] In the UK civilian environment, the Ambulance Service and the Fire and Rescue Service carry out emergency decontamination of the injured.[13] All clothing and foreign bodies (eg, jewelry, hearing aids, and contact lenses) are removed and the casualty is rinsed with warm soapy water, wiped, then rinsed again without heavy abrasion of skin or extremities.[7]

UK forces use Fuller's earth, a highly adsorbent, clay-like powder consisting of hydrated aluminum silicates, and US forces use the M291 carbonaceous resin kit. Both powders are highly adsorbent to chemical agents present on clothes, respirators, and skin, and aid personal decontamination in the field.[14] In 2009, the United States replaced the M291 with Reactive Skin Decontamination Lotion (First Line Technology, LLC, Chantilly, VA) to decontaminate skin but not wounds or eyes. Reactive Skin Decontamination Lotion is a mixture of potassium 2,3-butanedione monoximate and free oxime.[15] For both biological and chemical agents, UK and US forces fully decontaminate casualties by washing exposed areas with dilute warm sodium hypochlorite solution (0.5%), pH 10 to 11, for 10 to 15 minutes and removing and containing contaminated clothing.[9] Sodium hypochlorite is not to be used in eye, exposed brain, spinal cord, or abdominal injuries.[14]

Abdominal and thoracic cavity wounds should be washed out with saline; however, doing so may be hazardous if there is a chemical agent present. Irrigation fluid should be sucked out with a large-bore suction apparatus and disposed of in a hypochlorite solution.[14] Vesicant (blistering agent) decontamination requires water and saline (0.9%) or sodium bicarbonate (1.26%), if available, for the eyes and mucous membranes.[15]

Triage and Hot-Zone Treatment

Triage during a CBRN event involves not only prioritizing patient treatment based on the condition severity but also factoring in responder safety. This makes an already stressful and difficult process resource dependent. Peacetime civilian exercises indicate casualty decontamination time is currently unacceptable.[10] Life-saving first aid, akin to care under fire, can be administered during extraction[8] to save time. The time involved in decontamination may cause casualties to self-select; those able to evacuate the hot zone

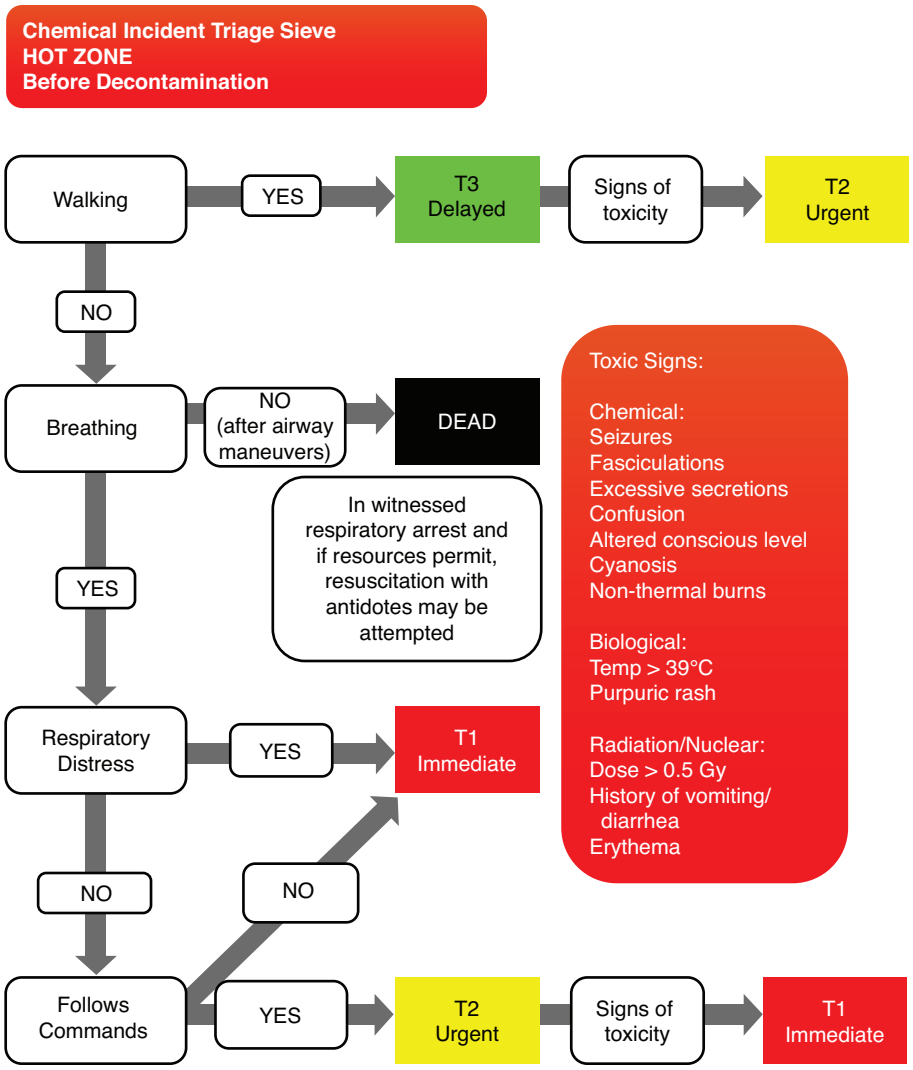


Figure 46-3. Chemical incident triage flowchart, hot zone before decontamination. Extraction to avoid further intoxication is a logical priority; when casualties are numerous, clinical input in the hot zone is useful. While casualties await extraction, the early administration of antidotes and other life-saving interventions may enhance survival. Toxidrome recognition can lead to identification and early supply of possible antidotes from the supply chain. Determining deaths promptly will reduce waste of limited resources.
Data source: UK Ministry of Defence. *Clinical Guidelines for Operations*. London, England: MOD; 2 Feb 2011. Joint Doctrine Publication 4–03.1.

are likely to profit from medical intervention more than those with a combination of trauma and intoxication, who are likely to face a poor prognosis. Despite this grim reality, a small subset of patients requires immediate, life-saving medical treatment prior to decontamination.

Prehospital trauma care still follows the <C>ABCD (catastrophic hemorrhage, airway, breathing, circulation, disability) paradigm, with the addition of antidotes, LSIs, and analgesia where appropriate in the hot zone. LSIs include rolling casualties on their sides or fronts to improve airway patency and tourniquet application. While casualties await evacuation from the hot zone, rapid triage may be performed (Figure 46-3).

Casualty decontamination begins in the "warm" zone (see Figure 46-1). Further LSIs are applied to tier 1 (immediate) casualties at this stage, then casualties are passed over the clean-dirty line and out of the inner cordon to the "cold" zone. Tier 2 (urgent) cases are given antidotes, if available, and decontaminated; tier 3 (delayed) cases are decontaminated and transferred to the cold zone without intervention unless they

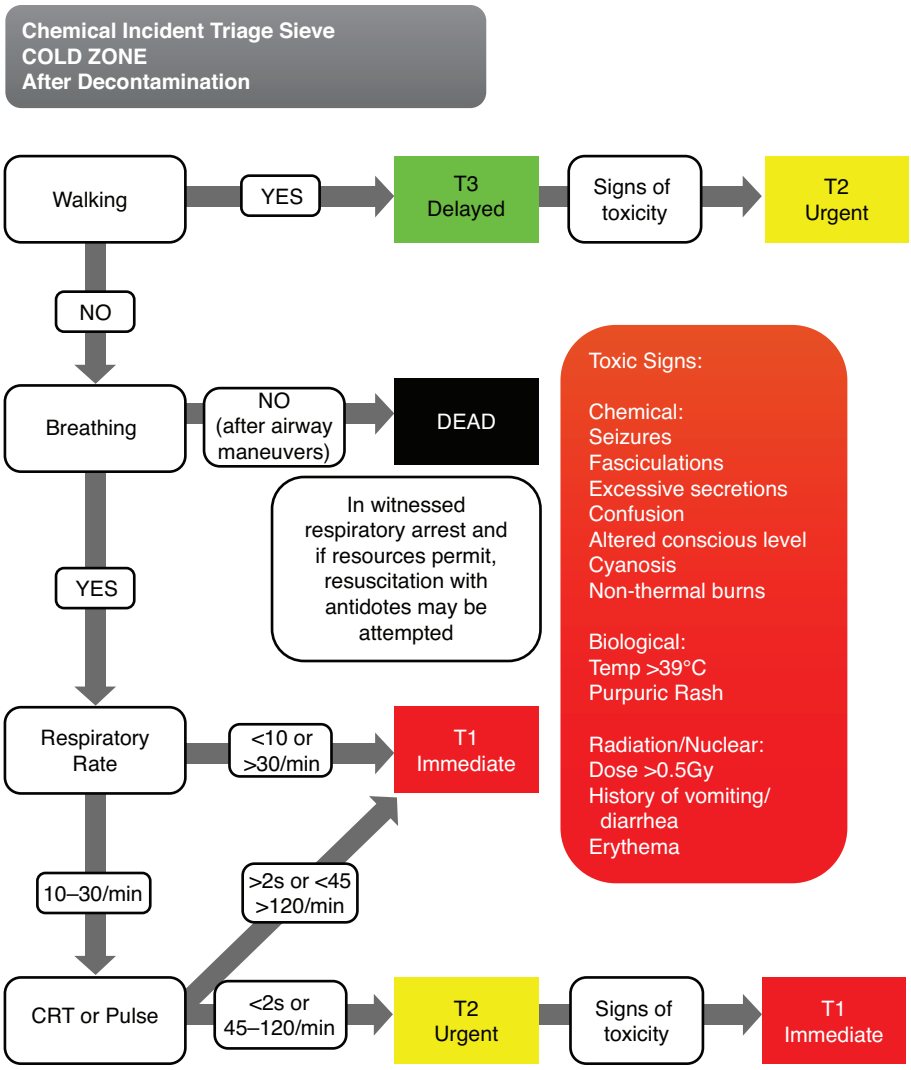


Figure 46-4. Chemical incident triage flowchart, cold zone after decontamination. After decontamination, casualties can be triaged more accurately in the cold zone. If, at any time, a casualty develops signs of toxicity, their triage category becomes a higher priority.
Data source: UK Ministry of Defence. *Clinical Guidelines for Operations*. London, England: MOD; 2 Feb 2011. Joint Doctrine Publication 4–03.1.

deteriorate. Once in the cold zone, triage is repeated and a formal primary survey is performed (Figure 46-4). In conjunction with chemical countermeasures, problems found during the primary survey are treated if imperative for survival to the next echelon of medical care.

AIRWAY ISSUES

Airway Devices

Assessing airway patency in an unconscious casualty while wearing CBRN PPE is difficult, but can be accomplished by placing a surgical glove with one finger cut off over the primary speech module of a UK military respirator (Figure 46-5). This allows assessment of airway patency and respiratory rate, success of airway maneuvers, and ability to triage effectively while avoiding contamination.[16]

Laryngeal mask airway (LMA) insertion by anesthesiologists dressed in CBRN PPE is prolonged but possible,[17] but tracheal intubation with an endotracheal tube carries an increased risk of failed intuba-

Figure 46-5. A glove with one finger removed and placed over the primary speech module can act as a marker for breathing. A valve ensures that gas flows through the primary speech module in one direction only, from the wearer to the atmosphere; therefore, it is a safe and effective means of showing apnea or inspiration (a) or exhalation (b). Photographs courtesy of E Hulse.

tion.[18] Failed intubation rates are also increased when intubating the casualty on the ground rather than a trolley or litter while wearing CBRN PPE.[19] This can be overcome by using the LMA, but casualties with bronchospasm and increased airway secretions will be more prone to aspiration, laryngospasm, and high ventilating airway pressures, making the LMA undesirable. The LMA should be considered a rescue adjunct for casualty evacuation to a more permissive environment. The potential difficulty in securing an airway within a CBRN environment may suggest a role for video-assisted intubation devices, but this is probably unrealistic given the large footprint, financial costs, and limited field durability of these devices.

Chemical Casualties

Nerve agents, cyanides, pulmonary agents, vesicants, burns, vomiting, and riot-control agents can all adversely affect the airway.

Nerve Agents

Sarin, tabun, soman, and methylphosphonothioic acid can be absorbed via many routes, depending on their physical properties, and act swiftly by irreversibly inhibiting the acetylcholinesterase enzyme within the body. This leads to excessive acetylcholine levels at both muscarinic and nicotinic receptors, resulting in cholinergic toxidrome (Figure 46-6).[2] Clinically, excessive acetylcholine levels cause excessive miosis, salivation, rhinorrhea, and bronchoconstriction with copious bronchial secretions, which may make tracheal intubation impossible before atropine administration.[1]

Central cholinergic effects can depress levels of consciousness and the respiratory center, with nicotinic effects causing muscle paralysis and convulsions. Death is from respiratory failure due to airway obstruction by secretions, bronchospasm, paralysis of the respiratory

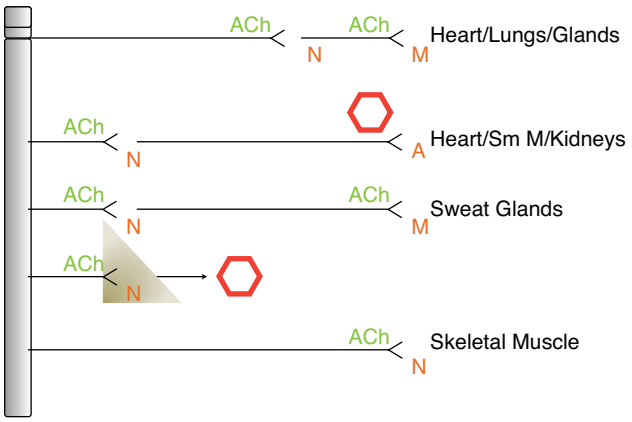


Figure 46-6. Acetylcholine use by the autonomic nervous system and neuromuscular junction. The parasympathetic effects at the top show pre- and postganglionic fibers stimulated by acetylcholine at nicotinic and muscarinic receptors. Shorter preganglionic sympathetic fibers use acetylcholine to stimulate nicotinic receptors. The postganglionic sympathetic fibers release catecholamines (red hexagons) acting viscerally and hormonally from the adrenal gland (brown triangle). The sympathetic control of sweat glands is mediated by acetylcholine acting on muscarinic receptors.

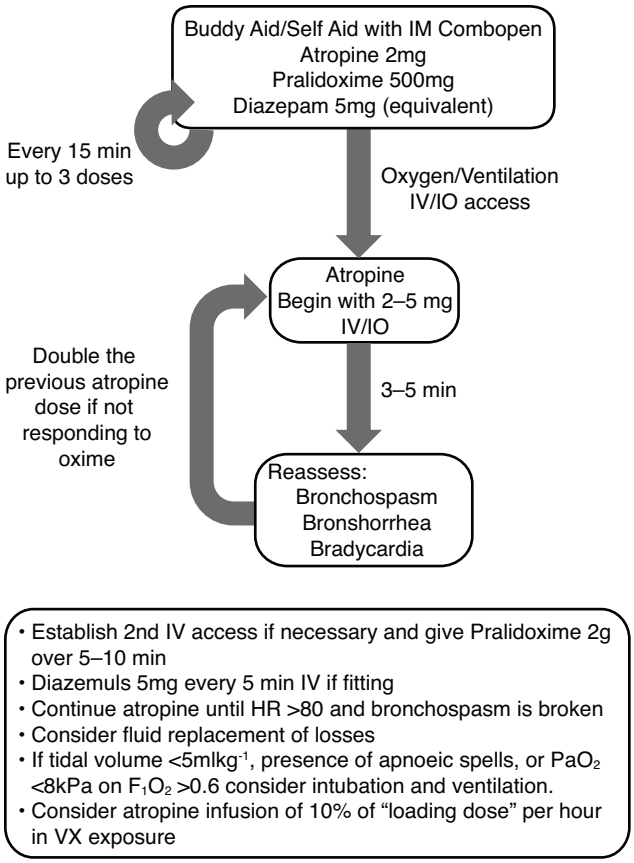


Figure 46-7. Nerve agent treatment algorithm. Atropine dose may need to be doubled in patients who do not respond to oxime. When continued absorption is anticipated, an atropine infusion may be employed. If suxamethonium is used, its action will be greatly prolonged.
Data sources: (1) Eddleston M, Buckley NA, Eyer P, Dawson AH. Management of acute organophosphorus pesticide poisoning. *Lancet*. 2008;371:597–607. (2) Bland SA, surgeon commander, Royal Navy; personal communication, 2010.

muscles, and central respiration depression (Figure 46-7).[20,21]

Vesicants (Blister Agents)

Mustard agents, Lewisite (arsenical), and phosgene oxime can burn and blister the eyes, mucous membranes, lungs, and skin. Onset can be delayed by hours for mustard agents, which can irritate and congest the mucous membranes in the nasal cavity, throat, and trachea. Symptoms include rhinorrhea, burning in throat, hoarseness of voice, and dyspnea. Mustard agents can damage the vocal cords. Additionally, airway secretions and necrotic tissue may obstruct the bronchial tree. Respiratory complications occur in more than 70% of mustard victims (Exhibit 46-2).[22]

EXHIBIT 46-2

TREATMENT AIMS: VESICANTS

- Laryngitis and tracheitis can be treated with humidified oxygen
- Mustard injuries should be treated as thermal burns and can cause bone-marrow suppression and carcinogenesis. Fluid resuscitation is less aggressive than that for thermal burns. Aim for urine output > 0.5 mL/kg/h
- Severe exposure results in pulmonary hemorrhage and edema with respiratory failure requiring intubation and ventilation
- Early antibiotic treatment of suspected bronchopneumonia

Severe pulmonary edema can occur with Lewisite and is treated with intramuscular dimercaprol, 10% British anti-Lewisite 3 mg/kg four times daily, or the oral chelating agent dimercaptosuccinic acid 30 mg/kg/day.

Data sources: (1) Bland SA. Chemical, biological and radiation casualties: critical care considerations. *J R Army Med Corps*. 2009;155:122–174. (2) UK Ministry of Defence. *Clinical Guidelines for Operations*. London, England: MOD; 2 Feb 2011. Joint Doctrine Publication 4–03.03.1.

EXHIBIT 46-3

TREATMENT AIMS: PULMONARY AGENTS

- Emergency treatment of laryngospasm may be required, including rapid sequence induction or application of continuous positive airway pressure.
- Bronchodilators and nebulized steroids for bronchospasm may be beneficial.
- Oxygen, intubation, and ventilation with sufficient positive end-expiratory pressure may be required in moderate to severe cases of pulmonary edema. It is logical to limit the fraction of inspired oxygen only to maintain adequate partial pressure of arterial oxygen.
- Observation of asymptomatic exposed individuals for 24 hours is mandatory.
- There is limited in-vitro evidence for nebulized N-acetylcysteine.

Pulmonary Agents (Choking Agents)

Phosgene, chlorine, oxides of nitrogen, and perfluoroisobutene can cause pronounced irritation of the upper and lower airways. Irritation of the larynx by high concentrations of agent may cause laryngeal spasm and death. More commonly, it causes acute lung injury and pulmonary edema. Airway secretions and pulmonary edema can be severe in phosgene toxicity and are sometimes delayed up to 24 hours, suggesting some inflammatory mechanism and possible pathology related to acute respiratory distress syndrome (ARDS; Exhibit 46-3).

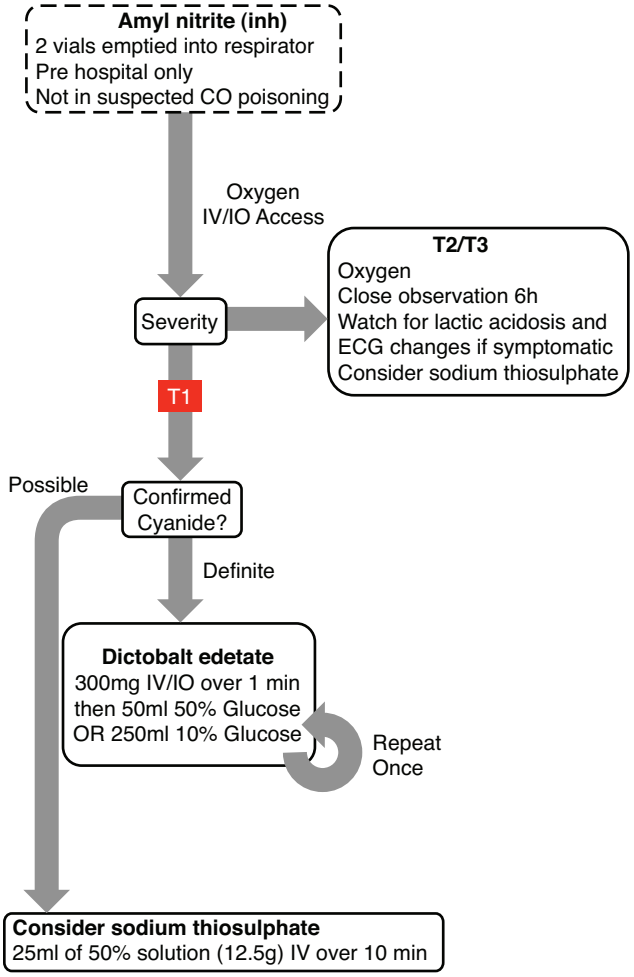


Figure 46-8. Management of cyanide poisoning. Diagnosis is dependent upon an index of suspicion in the absence of a confirmed release. Severe cases will demonstrate a raised central venous oxygen saturation and hyperlactatemia. Oxygen is the mainstay of treatment, with supplementation of sulphur donors with sodium thiosulphate. Sodium nitrite can be considered, inducing a methemoglobinemia to preferentially bind cyanide, but may not be appropriate if oxygen-carrying capacity is to be preserved. Give 10 mL of 3% sodium nitrite solution (300 mg) intravenously over 5 to 20 minutes once only with sodium thiosulphate.

EXHIBIT 46-4

TREATMENT AIMS: BURN AND INHALATIONAL INJURIES

- Senior anesthesiologist should intubate patient early, especially in the presence of stridor or hoarseness.
- In a conscious patient, consider an inhalational induction (this may be painful with a mask on raw tissue).
- Ensure surgical tracheostomy is immediately available if tracheal intubation via laryngoscopy fails.
- If using suxamethonium, use during the first 24 hours to avoid severe hyperkalemia.
- Leave the endotracheal tube uncut to provide room for facial-tissue swelling.
- Consider securing the tube with wire to the upper teeth, or be prepared to adjust tube ties in proportion to swelling (which may be rapid).
- Bronchodilators and a protective ventilatory strategy with good bronchial hygiene will be useful.
- Aim for an adequate urine output (0.5 mL/kg/h) using the Parkland formula (amount of fluid required in 24 hours [ml] = 4 × patient's weight [kg] × body surface area burned [%]).
- Hydrofluoric acid burns can cause hypotension and arrhythmias due to hypocalcemia. Treat with topical calcium gluconate gel and intravenous calcium chloride.
- Early enteral feeding for catabolic state is essential.

Riot Control Agents

Mace (2-chlorobenzalmalononitrile [CS] or 2-chloro-1-phenylethanone [CN]) is commonly used by law-enforcement agencies for riot control and training. CS and CN are solids dispersed as fine particles or in solution exerting their alkylating effect by inhibiting enzymes and increasing bradykinin release.[23] They are irritants affecting mainly the eyes, nose, mouth, airways, and skin. Airway burning and irritation cause coughing, bronchorrhea, and dyspnea, and should cease within 15 to 30 minutes once removed from the source. If the casualty has preexisting lung disease, such as asthma

or chronic obstructive pulmonary disease, bronchospasm and respiratory distress may be triggered by riot-control agents. CN can cause burns and tracheobronchitis and form laryngeal pseudomembranes. CN has also been associated with some deaths; it is not used in the United Kingdom but is permitted in the United States.[24]

Cyanides

Cyanides exist in many forms, including smoke from fires. They are also used extensively in industry. Electron-transport-chain poisoning, specifically of cytochrome c, results in tissue hypoxia and lactic acidosis from forced anaerobic metabolism, which can cause nausea, agitation, hyperventilation, confusion, loss of consciousness, seizures, coma, respiratory arrest, and death (Figure 46-8).

Airway Burns

Airway burns represent a huge spectrum of illness, depending on duration of exposure and the concentration, composition, and temperature of the smoke. House fires may generate toxic chemicals such as carbon monoxide, hydrogen cyanide, inorganic acids, and nitrogen oxides, which have lung-damaging properties. Obvious burns or carbonaceous deposits around the mouth and nose and singed nasal and eyebrow hairs with dyspnea, cough, and wheeze should alert a provider to a possible airway problem. Hoarseness or stridor may indicate impending airway obstruction and require urgent assessment and intervention by a senior anesthesiologist (Exhibit 46-4).

Biological Casualties

Recognizing the characteristically nonspecific clinical features of a biological casualty is both difficult and critical. Therefore, a high index of suspicion must be maintained and steps should be taken to protect medical personnel and first responders physically, chemically, and immunologically. Physical protection takes the form of PPE and protective masks, whereas chemical protection against bacterial agents (eg, brucellosis, plague, tularaemia, and Q fever) comes from antibiotics either before or after exposure. Immunologic protection, typically immunization, helps protect against biological agents such as anthrax and smallpox. This heightened awareness and active protection enables healthcare workers to immediately provide potentially life-saving airway maneuvers prior to decontamination and definitive diagnosis.

Radiological Casualties

Airway management is rarely necessary, even in the most severely exposed radiological casualty. Instead, the medical management of acute radiation sickness focuses dose-dependently on the hematopoietic, gastrointestinal, neurovascular, and integumentary systems. With severe blast injury following a nuclear incident, airway issues, such as pneumothorax and pulmonary failure secondary to barotrauma, may require airway intervention. Additionally, although patients exposed to higher doses of radiation may be triaged into an expectant category, they will likely experience increased episodes of nausea and vomiting, which may impact a provider's decision on how and when to secure the patient's airway.

BREATHING ISSUES

All chemical warfare agents, biological agents, and TICs have a direct or indirect effect on the respiratory system. Radiological casualties rarely exhibit breathing difficulties. Lung injury manifests diversely, with compounds exhibiting effects at different times and sites within the bronchial tree. Preoxygenation and uptake of volatile anesthetic agents is likely to be adversely affected. Currently there are no specific therapies for toxic lung injury, and supportive care remains the cornerstone. Protective lung ventilation strategies may limit further damage; however, in carbon monoxide poisoning, a high fraction of inspired oxygen (FiO_2) should be maintained. Carboxyhemoglobin has a half-life dependent on FiO_2; therefore, severely poisoned patients should breathe an FiO_2 of 1.0 for 5 half lives, or 3.5 hours,[2] before titrating to partial pressure of arterial oxygen.

Direct-Acting Agents

Pulmonary agents such as phosgene and chlorine; biological agents such as staphylococcal enterotoxin type B, plague, tularemia, and inhalational anthrax; and some TICs fall into the category of direct-acting agents. Vesicants such as mustard predominantly affect the upper airway, but in higher or more prolonged exposures can cause lung damage. Phosgene and mustard have delayed effects on the lungs, and a 24-hour period of observation is mandatory for casualties with mild symptoms and a history of exposure. Chlorine may also exhibit delayed effects. Deterioration during anesthesia is a possibility that anesthesiologists must rapidly diagnose and treat. Many direct-acting agents are oxidizing and proinflammatory, prompting studies of antioxidant and

antiinflammatory compounds. There is in-vitro and small-animal-model evidence to suggest that N-acetylcysteine may be effective as oral prophylaxis or in nebulized form following mustard exposure, but less so in phosgene exposure. N-acetylcysteine has also been used topically. The effectiveness of moderate-dose intravenous (IV) steroids is still debated and, when combined with immunosuppression from mustard, may not be appropriate; however, the early use of steroids may have a role in phosgene exposure.[25] There is little evidence for steroid use in chlorine exposure[26]; however, nebulized bronchodilators are recommended for bronchospasm following chlorine exposure. Treatment for exposure to aerosolized staphylococcal enterotoxin type B is primarily supportive and consists of humidified oxygen and steroids. Treatment for direct-acting toxins is escalated from supplementary oxygen to maintain saturation of peripheral oxygen above 94% through noninvasive, continuous positive airway pressure, to invasive ventilation as dictated by the patient's condition. Phosgene and chlorine injured patients should be ventilated using the National Heart, Lung, and Blood Institute's ARDS Network protocol[27] to prevent progression to ARDS. Oxygen toxicity from continued free-radical cycling is minimized by increasing positive end expiratory pressure rather than FiO_2. Large-animal studies of phosgene demonstrate an increased mortality from immediate oxygen therapy and benefit from delay until symptomatic; however, no oxygen therapy carried a higher mortality.[25] Tidal volumes should be limited to 6 mL/kg ideal bodyweight.[25,27]

Inhalation injury is rare because the airway has efficient heat exchange mechanisms, so attention should be directed to the management of airway burn. (see Exhibit 46-4). A protective ventilatory strategy (ARDS Network protocol[27]) should be adopted and other measures considered, such as nebulized bronchodilators, *N*-acetylcysteine, heparin, and bronchoscopic lavage with 1.26% sodium bicarbonate.[2]

Indirect-Acting Agents

Certain incapacitants have opioid- or benzodiazepine-like effects and, in sufficient doses, may result in respiratory failure, but ventilation is usually the most effective treatment until consciousness is regained.

Nerve agents cause bronchoconstriction, bronchorrhea, and ventilatory failure. Bronchoconstriction may be so severe that ventilation is unachievable without an antidote (see Figure 46-7) and tracheal intubation may not be possible because of secretion volume. Atropinization takes priority when resuscitating nerve-agent casualties; once achieved, bag-valve mask ventilation becomes considerably easier. Although there has been concern that atropine given to hypoxemic patients may precipitate tachyarrhythmias, there is no evidence to deny its use in cases of organophosphorus pesticide poisoning.[21] Atropine is titrated until a desired effect on bronchorrhea, bronchospasm, and bradycardia is achieved. After adequate dosing, ventilation is significantly easier and tracheal intubation may be possible. Capnography and airway-pressure monitoring will allow close titration of atropine infusion to effect, especially if supplies are limited. Oximes will reduce the atropine requirement and can be given as a bolus or as an infusion (see Figure 46-7). An infusion is indicated in cases where continued absorption occurs, (eg, dermal absorption of methylphosphonothioic acid, which continues after decontamination).

CIRCULATION ISSUES

Types of Access

Initial treatment of nerve-agent exposure involves self or buddy administration of an intramuscular ComboPen. This may be sufficient in the short term, but casualties with compromised circulation due to trauma or chemical poisoning will ultimately require timely vascular access. In these patients, IO devices may be life saving in the field.

A study using a manikin model found that when medical operators and casualties were dressed in CBRN PPE, they inserted the IO device EZ-IO (Vidacare Corporation, San Antonio, TX) more quickly than trying to obtain peripheral IV access.[28] The casualty may be in CBRN PPE when vascular access is required. After limited decontamination and exposure in the hot zone, access to the sternum may permit use of the sternal IO device FAST1 (First Access for Shock & Trauma, Pyng Medical Corporation, Vancouver, British Columbia; Figure 46-9). A study using advanced patient simulators and the Bone Injection Gun (BIG, Waismed Ltd, Migdal Tefen, Israel), a spring-driven, trigger-operated, IO injection device, enabled nerve-agent antidotes to be administered within 3.5 min. This technique has an insertion success rate of 89% by physicians wearing full CBRN PPE.[29]

Chemical Casualties

Nerve Agents

Nerve-agent casualties can show an early transient

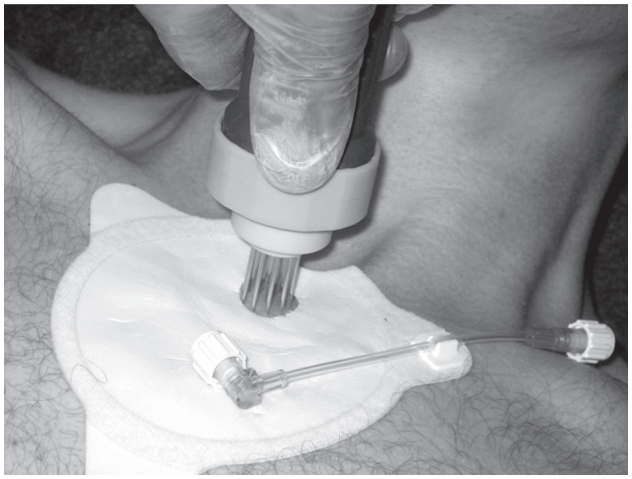


Figure 46-9. Insertion of FAST 1 (First Access for Shock & Trauma, Pyng Medical Corporation, Vancouver, British Columbia) intraosseus, sternal access.
Photograph reproduced with permission from: trauma.org; http://www.trauma.org/images/image_library/11181942171FAST_RSI_photo.JPG

tachycardia or hypertension through stimulation of the adrenal medulla, followed by muscarinic-induced bradycardia and hypotension, which may abolish the compensatory mechanisms necessary to combat hypovolemia. Intense parasympathetic activity may also mask awareness during anesthesia. Other arrhythmias, QT prolongation, and decreased cardiac ventricular contraction occur in severe poisoning.[30] Raised serum creatine kinase was observed in cases from the sarin release in Matsumoto, Japan, in 1994 and is likely due to prolonged muscle contraction.[31] Nerve agents typically require IV fluids to expand circulation, offsetting loss from secretions and atropine to increase heart rate. Inotropes are occasionally required alongside clonidine and magnesium to control cholinergic symptoms (see Figure 46-7).[30]

Chemical Burns

Chemical burns may not need such an aggressive fluid-management strategy as thermal burns, and maintenance of an adequate urine output (0.5–1 mL/kg/h) is an acceptable indicator of fluid levels.[5] Hydrofluoric acid burns can cause intense pain, liquefactive necrosis, hypotension, and fatal arrhythmias due to hypocalcemia, hyperkalemia, and hypomagnesemia. For cutaneous exposure, treatment is frequent application of topical 10% calcium gluconate gel, which may be followed by local infiltration of 10% calcium gluconate until the pain has settled; local and regional anesthesia should be avoided because of this significant clinical endpoint. Calcium and magnesium are aggressively replaced to maintain levels within the normal range, empirically before results are known and especially if symptomatic.[32] An intraarterial infusion, proximal to the burn, of 10 mL 10% calcium gluconate diluted in 40 mL glucose or saline over 4 to 5 hours may be required for severe peripheral burns.[32] Calcium chloride is only given IV because it will otherwise cause tissue necrosis.

Atropine Overdose

Over-atropinization may be observed after inadvertent treatment with the military-issue ComboPen, which is self-administered intramuscularly by military personnel during nerve-agent poisoning scenarios. In the absence of nerve-agent poisoning, the ComboPen would produce antimuscarinic toxicity with the typical features of mydriasis, tachycardia, thirst, absence of sweating, hyperthermia, and confusion. Supportive treatment should suffice and may include the use of benzodiazepines.

Biological Casualties

Victims of biological attacks typically present with nonspecific clinical features, which can be extremely difficult to diagnose and treat. Intravascular volume depletion should be anticipated in the setting of many biological agents, such as those that often present clinically as pneumonia (plague, tularemia, and staphylococcal enterotoxin B), or those that present with febrile illness (Q-fever, Venezuelan equine encephalitis, or brucellosis).

Radiological Casualties

Victims of severe radiation events with acute radiation sickness can exhibit significant circulatory compromise or collapse. Within hours after an event, early symptoms may include severe nausea, vomiting, and watery diarrhea. Severe diarrhea and vomiting may progress clinically to shock, renal failure, and, ultimately, cardiovascular collapse. Other predictable circulatory issues observed in these patients include malabsorption of nutrients, significant fluid and electrolyte shifts, gastrointestinal bleeding, and sepsis.

NEUROLOGICAL ISSUES

Many agents act on the nervous system, resulting in airway, breathing, and circulation issues. Agents that act on the central nervous system include cyanide, opioids, carbon monoxide, and novel incapacitants.

Those that act on the peripheral nervous system include nerve agents, botulinum toxin, and other neurotoxins. The management of each varies in complexity; incapacitants and other novel agents may require airway attention and administration of benzodiazepines for agitation, but nerve-agent poisoning is a multisystem disease.

Carbon Monoxide

Carbon monoxide poisoning usually presents with nonspecific symptoms and signs after inhalation of incomplete combustion products (eg, house fires or poorly maintained heaters or burners). The following are important points to remember about carbon monoxide poisoning:

- Respirators do not filter carbon monoxide.
- Measured carboxyhemoglobin does not correlate with poisoning severity.
- Smokers will have carboxyhemoglobin concentration up to 10%.
- Do not induce methemoglobinemia if concomitant cyanide poisoning is suspected.

Carbon monoxide poisoning is a common cause of death from poisoning in the United Kingdom and can present with neurological signs in severe cases. Injury to watershed areas of the brain, such as basal ganglia, is possible, as is myocardial injury.[2] The mechanisms are complex and involve extreme left shift of the oxyhemoglobin dissociation curve.

Unconscious patients should be maintained on a high FiO_2 (see Breathing Issues in Chemical, Biological, Radiological, and Nuclear Exposure). Attention to raised intracranial pressure must include head-up tilt, maintenance of normotension and normocapnia, care with tracheal tube ties, and regular pupil assessment.[2]

EXHIBIT 46-5

NEUROMUSCULAR EFFECTS OF NERVE AGENTS

After exposure to nerve agents, parasympathetic activity predominates to the extent that tachycardia is unusual, and hypovolemia should be excluded first. The effects on the autonomic nervous system will abolish the compensatory response to hypovolemia and mask a fixed dilated pupil in head injury. Multiple factors combine to cause death from respiratory failure.

Different types of effects are as follows:

Central Nervous System (Nicotinic and Muscarinic Effects)
- Confusion
- Agitation
- Coma
- Respiratory failure

Autonomic Nervous System
- Parasympathetic Nervous System (Muscarinic Effects)
  - Bradycardia, hypotension
  - Bronchospasm, bronchorrhea
  - Salivation, vomiting, diarrhea
  - Miosis, lacrymation, urination
- Sympathetic Nervous System (Nicotinic Effects)
  - Tachycardia, hypertension
  - Sweating
  - Mydriasis

Neuromuscular Junction (Nicotinic Effects)
- Muscle weakness
- Paralysis
- Respiratory failure
- Fasciculation

Data source: Eddleston M, Buckley NA, Eyer P, Dawson AH. Management of acute organophosphorus pesticide poisoning. *Lancet*. 2008;371:597–607.

Nerve Agents

Nerve agents have central and peripheral effects secondary to overwhelming acetylcholine concentrations after inhibition of acetylcholinesterase (Exhibit 46-5). The peripheral nicotinic effects are fasciculation, which may be mistaken for seizures, followed by paralysis. The muscarinic effects are eased by atropine.

The peripheral nicotinic effects are managed supportively until function returns; magnesium and clonidine may decrease presynaptic acetylcholine release.[30] The central effects of seizures are more common in nerve-agent poisoning than in their agricultural organophosphorus cousins and should be managed aggressively with benzodiazepines (see Figure 46-7).

Biological Agents

Numerous biological agents evoke neurologic symptomatology. Inhalational anthrax can present as hemorrhagic meningitis with a dramatically widened mediastinum on chest radiograph. Initial presenting signs and symptoms of inhaled botulinum include progressive ocular, pharyngeal, respiratory, and muscular weakness and paralysis. Other biological agents with neurological symptoms include Ebola hemorrhagic fever, Crimean-Congo hemorrhagic fever, and Venezuelan equine encephalitis. Medical management includes isolation, antibiotics, and supportive care.

Radiation Exposure

Acute neurovascular syndrome, the third of the three subsyndromes (hematopoietic, gastrointestinal, and neurovascular) seen in acute radiation sickness, typically emerges when a victim has experienced extremely high external doses of ionizing radiation. Expected clinical symptoms include an immediate burning sensation, emesis, hyperpyrexia, prostration, hypotension, and neurologic signs (ataxia and delirium). Death is inevitable and frequently occurs within 48 hours. Another condition, early transient incapacitation, is characteristic of very high exposures to radiation that only occur during plutonium and enriched uranium fuel reprocessing accidents. Similar to acute neurovascular syndrome, early transient incapacitation portends a grim prognosis, including a deteriorating level of consciousness, vascular instability, and death.

DRUG INTERACTIONS, CONTRAINDICATIONS, AND HAZARDS

Nerve agents act on a number of different enzymes and receptors. Inhibition of butyrylcholinesterase results in prolonged action of drugs hydrolyzed by it, namely suxamethonium and mivacurium. Metabolism of remifentanil and esmolol by other esterases is unaffected in butyrylcholinesterase deficiency[33]; however, nerve agents affect many esterases and it is unclear whether the metabolism of these drugs would be prolonged.

The interaction between nerve agents and cholinesterases can be disrupted by oximes, which are used to reactivate affected enzyme. A process called "aging" affects the nerve agent bound to cholinesterase, whereby the interaction becomes stronger with time due to loss of a radical. Once aged, oximes are no longer effective. Aging is particularly rapid with soman, which has an aging half-life of 1.3 minutes.[34] To mitigate against this process, pyridostigmine is used as pretreatment, preventing the enzyme from binding to nerve agent and enhancing subsequent treatments (eg, ComboPen). After exposure, pyridostigmine is discontinued, unbound nerve agent undergoes spontaneous hydrolysis, and cholinesterase bound to pyridostigmine dissociates. This unaffected cholinesterase may be sufficient to restore normal neuromuscular function due to redundancy in the mechanism.[35] Where absorption of nerve agent is likely to continue (eg, in cutaneous methylphosphonothioic acid exposure), oxime infusion should be maintained.

If suxamethonium is required for tracheal intubation, expect its action to be prolonged, and nondepolarizing muscle relaxants will require an increased dose in pyridostigmine pretreated casualties. There may even be a role for nondepolarizing muscle relaxants in shielding nicotinic receptors from acetylcholine in severe nerve-agent poisoning.[36] Anesthesia use for nerve-agent-poisoned casualties is limited; clinicians will need to judge neuromuscular function by the standard clinical indicators, including "train-of-four" count. Ultrasound-guided regional anesthesia must also be considered.[2,36] The evidence base for drug interactions in toxicology stems from case reports, limited animal studies, and theoretical interpretations. Providers should be aware of pharmacological concerns when using antidotes and anesthetic agents in the presence of toxic injury (Exhibit 46-6).

EXHIBIT 46-6

COMMON ANESTHETIC DRUGS AND ANTIDOTES

Listed below are some commonly used anesthetic drugs and antidotes, and some of their more important interactions and contraindications. Volatile anesthetic agents probably do not have serious interactions, although titration to an autonomic response in mild to moderate nerve agent poisoning may result in unintended intraoperative awareness.

Cyanide Antidotes

- **Amyl nitrite.** Used to oxidize hemoglobin to methemoglobin, generally used prehospital. Do not induce methemoglobinemia if carbon monoxide poisoning is suspected.
- **Dicobalt edentate.** Use only in confirmed cyanide exposure as a chelator. Administer 300 mg followed by 50 mL of 50% glucose or 250 mL of 10% glucose. Can cause anaphylactoid reaction with pulmonary edema in the absence of cyanide.
- **Sodium thiosulphate.** Use in moderate to severe poisoning. Administer 25 mL of 50% solution (12.5 g) over 10 min. Relatively free of side effects.
- **Alternatives**
  - Sodium nitrite can be used to induce methemoglobinemia in severe cyanide poisoning without carbon monoxide.
  - Cyanokit (hydroxycobalamin 5 g) can be considered if cyanide inhalation from combustion products is suspected.[1]

Induction agents. Familiar induction agents are most suitable. In nerve agent poisoning, atropinization must be achieved prior to induction if anesthesia is required. It is not known whether ketamine will cause excess secretions and brochodilation in this situation, or whether such effects are clinically relevant.

Methylene blue.[2] To reduce methemoglobin in compromised patients (> 30% methemoglobin), give 1–2 mg/kg over 5 min. Can be repeated after 30–60 min. If this treatment fails, consider ascorbic acid and exchange transfusion. Methylene blue should be avoided in patients with glucose-6-phosphate dehydrogenase deficiency.

Nondepolarizing muscle relaxants. May not be required in surgery for nerve-agent–poisoned casualties. Dose increase is required for nerve agent poisoning and in burns after 24 h.

Oximes. Used to reactivate cholinesterase. Do not delay atropine therapy. Give 2 g pralidoxime over 5–10 min.

Suxamethonium

- **Burns.** Use causes severe hyperkalemia >24 h postburn. Can be used acutely.
- **Nerve agent poisoning.** Suxamethonium's action is prolonged in patients with mild to moderate toxicity requiring surgery and possibly in pyridostigmine use. Not required for severe intoxication.

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SUMMARY

The addition of CBRN considerations to the care of trauma casualties completely transforms incident management. The high risk of secondary exposure for first responders must be balanced against the risk of over-decontamination or unnecessary decontamination of casualties. To inform the decontamination process, due attention must be paid to agents' physical properties. Severely poisoned individuals may be minimally exposed to further contamination to enable lifesaving interventions prior to decontamination. Trauma and CBRN exposure conspire synergistically to yield a grave prognosis possibly resulting from delays caused by decontamination and chemical resuscitation. To make the best use of possibly limited resources, expectant casualties should be identified and triage guidelines followed. Adverse outcomes can be minimized with the timely application of specific therapies and antidotes where available, in concert with damage control resuscitation for trauma.

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Chapter 47

CURRENT ANESTHESIA EQUIPMENT

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INTRODUCTION

Military medical equipment technology has advanced significantly over time; however, compromises may still be required, especially when forward surgery is planned. Compact, light, and highly portable medical equipment is ideal for delivering anesthesia in Role 2 medical treatment facilities (MTFs) and for entry operations. This chapter will describe the range of general anesthesia equipment required at Role 2 and Role 3 MTFs to provide modern anesthesia.

DRAW-OVER SYSTEMS

Draw-over anesthesia systems have been part of anesthesia practice since its inception. The key components for any draw-over system are a one-way patient valve and a vaporizer that has a low resistance to breathing. Both UK and US forces have relied on draw-over anesthetic equipment in field medical situations. US forces are increasingly using compact conventional anesthesia machines, and at Role 3 facilities, conventional anesthesia machines have largely replaced draw-over systems. In more recent systems, a mechanical ventilator is usually added along with a means of removing anesthetic gases from the operating room. There are a number of examples of commonly accepted requirements for portable anesthetic apparatus in austere environments (Exhibit 47-1).[1]

The Triservice Anaesthetic Apparatus

The Triservice Anaesthetic Apparatus (TSAA) was developed by Brigadier Ivan Houghton and first described in 1981.[2] It is based on the revised version of the Oxford Miniature Vaporiser (Figure 47-1), the OMV50 (Penlon Ltd, Abingdon, UK). However, the OMV50 has recently ceased production, and the UK Defence Medical Services (DMS) is investigating a replacement for the TSAA.

Revisions from the original OMV50 included an increase in agent quantity to 50 mL and the addition of three "feet" to increase stability. The vaporizer is compact, lightweight, simple to use, and compatible with many modern volatile agents. Although it is not temperature compensated, antifreeze in the base makes it thermally buffered.

Although key components of the TSAA have remained the same, some changes since its inception have been described in a number of articles.[1,3] The one-way patient valve and a self-inflating bag (SIB) come from the Laerdal Resuscitator (Laerdal Medical Ltd, Orpington, UK). For general anesthesia in the spontaneous breathing patient, the one-way patient valve is connected to the SIB by a length of corrugated

EXHIBIT 47-1

ANESTHETIC SYSTEM REQUIREMENTS FOR AUSTERE ENVIRONMENTS

Anesthetic systems in austere environments must be:

- minimally reliant on compressed gases and electrical supplies
- robust
- compact and portable
- simple to operate
- able to withstand climatic extremes
- easily maintained and serviced
- economical
- compatible with various volatile agents
- versatile with regard to patient age/size

Figure 47-1. Oxford Miniature Vaporiser.
Product image used with permission from Penlon Ltd, Abingdon, United Kingdom.

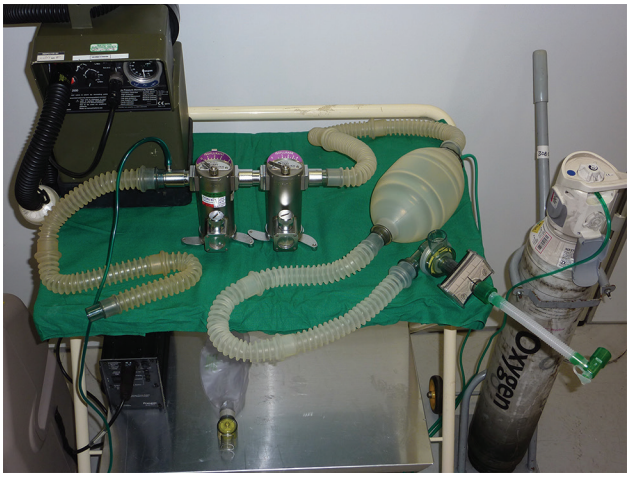

Figure 47-2. Triservice anesthetic apparatus, configured for spontaneous respiration.
Product image used with permission from Smiths Medical, Ashford, United Kingdom.

rubber tubing; a further length of tubing connects the SIB to the OMV50 (there are usually two vaporizers connected in the series because of the small volume of volatile agent each contains). Upstream of the second OMV50 is a Sanders injector (or T-piece), which is used to deliver supplementary oxygen. A further length of corrugated tubing acts as an oxygen reservoir (Figure 47-2).

It should be noted at this stage that all the connectors, from the one-way patient valve through the SIB and the vaporizers, are the old cage-mount standard (23.1 mm). The patient attachment to the one-way patient valve has a conventional International Organization for Standardization (ISO) 22-mm fitting. A ported shroud is usually fitted to the one-way valve to scavenge anesthetic gases, and it is also possible to attach a positive end-expiratory pressure valve at this point. The current UK approach to scavenging, in the absence of an active system, is to use a Cardiff Aldasorber (Shirley Aldred & Co Ltd, Derbyshire, UK). Unlike a conventional anesthetic machine or circuit, there is no bag to provide an indication of ventilation during spontaneous breathing. A modification of the circuit did help in this regard,[4] but a recent change in the SIB design made this modification impossible.

The apparatus may also be modified (Figure 47-3) for pediatric anesthesia.[3,5] A number of volatile agents

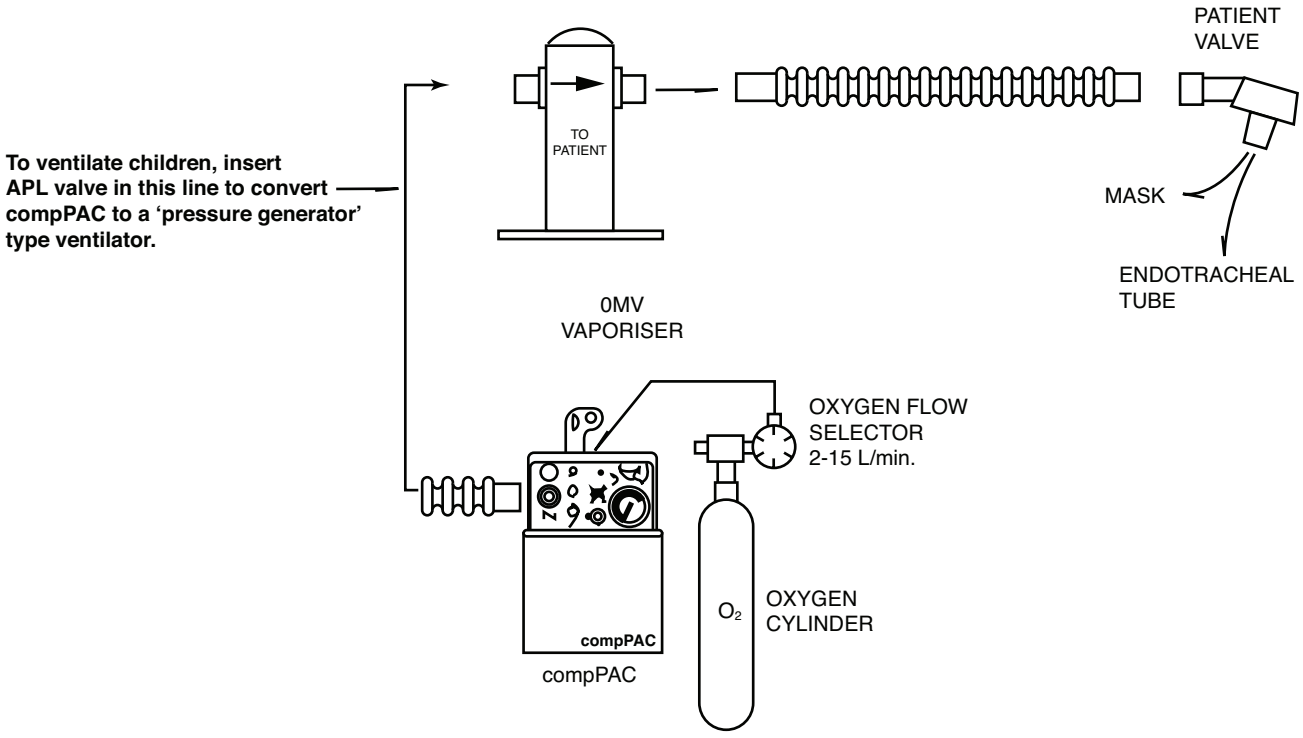

Figure 47-3. Triservice anesthetic apparatus, configured for pediatric anesthesia.
APL: adjustable pressure limit; OMV: Oxford Miniature Vaporiser
Product image used with permission from Smiths Medical, Ashford, United Kingdom.

Figure 47-4. CompPAC 200 ventilator and power supply.
Product image used with permission from Smiths Medical, Ashford, United Kingdom.

may be used, including sevoflurane; however, even with two vaporizers in series, it is difficult to perform an inhalation induction with sevoflurane using the TSAA.

The CompPAC 200 ventilator[6] (Smiths Medical, Ashford, UK) is currently used with the TSAA to provide mechanical ventilation during general anesthesia (Figures 47-4 and 47-5). The CompPAC 200 is a time-cycled, pressure-preset flow generator. The driving gas is produced by an internal air compressor and stored in an internal cylinder, so it remains at the same concentration as room air. The inspired oxygen concentration can be increased by feeding oxygen (maximum 4 L/min) into the rear of the ventilator or by connecting high-pressure oxygen using the supplied Schrader cable to the connector on the front of the ventilator (in this situation the "air mix/no air mix" control is operational). During anesthesia, it is normal to feed the oxygen supply from the concentrator into the T-piece and not use the connection at the rear of the ventilator. The ventilator is versatile; it can be driven from a range of electrical supplies as well as a source of high-pressure oxygen.

A potentially significant patient safety issue may occur when using the ventilator with the TSAA. The original design SIB *must* be removed from the circuit before turning on the CompPAC 200 to ventilate the patient; failing to do so has resulted in separate cases of failure to ventilate (merely ventilating the SIB) and "breath stacking," in which the patient does not exhale during the expiratory phase and continues to receive gas during inspiration. The newer design SIB is located where the ventilator connects to the circuit, making it easier to remember to remove it.

Another potential patient safety issue is the tendency for the circuit to come apart at one of the numerous connections. Users must be aware of this and strive to firmly seat all connections. Because of the unique nature of the TSAA, military anesthetists train to use it in a high-fidelity simulation course.[7]

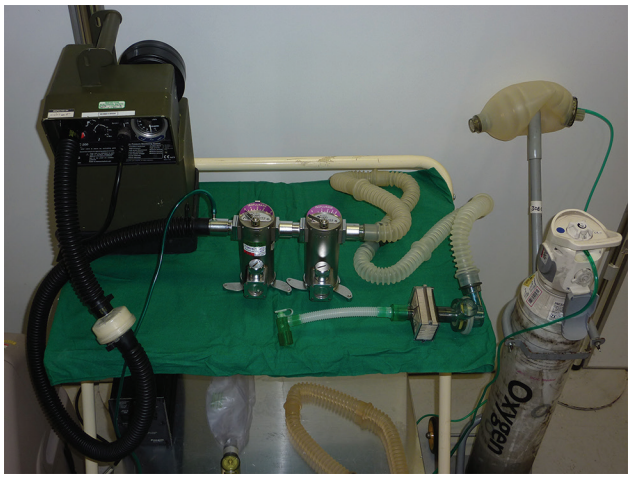

Figure 47-5. Triservice anesthetic apparatus, configured for mechanical ventilation.
Product image used with permission from Smiths Medical, Ashford, United Kingdom.

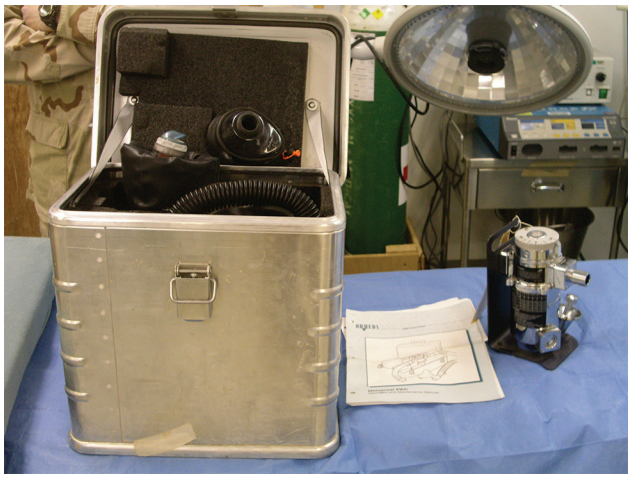

Figure 47-6. Ohmeda Portable Anesthesia Compact vaporizer.
Product image used with permission from General Electric Healthcare, Chalfont St Giles, United Kingdom.

US Draw-Over System

The US draw-over system is based on the Ohmeda Portable Anesthesia Compact (PAC [General Electric Healthcare, Chalfont St Giles, UK]) vaporizer (Figure 47-6). Basic components include an Ambu-E valve (Ambu A/S, Ballerup, Denmark) vaporizer, tubing, and SIB (Figure 47-7). The dial located on the top of the vaporizer can be unscrewed and turned over for use with other anesthetic agents. For example, the settings for sevoflurane are on the reverse of the dial, and a chart stamped on the unit describes settings for other volatile anesthetics. However, the Ohmeda PAC has not been manufactured for some years and alternatives are being investigated.[8]

Although the sevoflurane output has been described,[9] at present the only anesthetic used with this system is isoflurane. Like the OMV50, the vaporizer tends to consume isoflurane (and other volatile agents) more quickly than a conventional anesthesia machine, so it is important to monitor the level of agent remaining in the reservoir. Vapor consumption depends on flow rate as well as the output concentration selected on the dial. Vapor consumption can be calculated using the following formula: $3 \times \%F$, where F is the flow in liters per minute, % is the vapor output setting on the dial, and 1 mL of liquid agent is equivalent to 200 mL of vapor.

The vaporizer unit should be used in a temperature-controlled environment above 12ºC to 15ºC (54ºF–59ºF) and below 35ºC (95ºF). At temperatures above 35ºC (95ºF), potentially hazardous concentrations of agent may be delivered. Experience in Afghanistan when temperatures exceeded 100ºF showed that the dial needed to be set lower than usual to avoid high concentrations of volatile agent. Because the circuit does not lend itself to the attachment of a scavenging system, it should be used in a well-ventilated room.

Figure 47-7. Ohmeda Portable Anesthesia Compact circuit. Product image used with permission from General Electric Healthcare, Chalfont St Giles, United Kingdom.

The Ohmeda PAC can be used in series with an Eagle Univent Ventilator (Progressive Medical International, Vista, CA). It may be used in either a pull-through[10] or a push-through[11] method, depending on where the ventilator is placed in relation to the vaporizer.

Using the Draw-Over With the Univent

This method places the ventilator between the vaporizer and the patient. The ventilator "pulls" the anesthetic gas from the vaporizer, so it is effectively still a draw-over device. It can be difficult to determine the amount of anesthetic gas entering the ventilator. Supplemental oxygen is normally provided via the vaporizer, but caution is advised when using supplemental oxygen via the ventilator. Volatile anesthetic gases enter through the side port into the internal air compressor; by selecting air-oxygen values greater than 21% on the air/oxygen mixer dial, relatively less anesthetic gas will enter the ventilator. At an inspired oxygen concentration of 100%, virtually no anesthetic gas will be provided by the ventilator.

Set up the Univent as follows:

1. Attach the 36-inch, clear plastic hose from the Univent circuit package between the vaporizer output connector and the ventilator's internal air-compressor side port.
2. Attach an 84-inch breathing circuit from the top of the ventilator gas output port to the patient.
3. Connect the clear circuit exhalation valve and green transducer to the circuit and the ventilator.
4. Shorten the Univent connector hose and circuit by cutting the cuffs that are located every 6 inches along the tubing.
5. If a problem occurs and the patient needs to be manually ventilated, disconnect the Univent tubing and attach the vaporizer inhalation limb and breathing bag.

Push-Through Method

The push-through method places the vaporizer between the patient and the ventilator, allowing the ventilator to "push" gas over the vaporizer to the patient. The push-through method can be performed using the following steps:

1. Using the long black tubing of the Ohmeda PAC, connect one end to the "patient gas out" connector on top of the Univent and the other end to the air inlet connector on the Ohmeda vaporizer.

2. Attach the Univent breathing circuit from the Ohmeda vaporizer output port to the patient.
3. Connect the clear circuit exhalation valve and green transducer to the circuit and the ventilator. The Univent ventilator has a hose connector on top of the unit for adding pressurized oxygen to the system. For this feature to work, oxygen pressure must be greater than 40 psi. The air/oxygen mixer dial can then be set for a fraction of inspired oxygen (FiO_2) of 0.21 to 1.00. Low-pressure oxygen may be added to the ventilator via the side port internal compressor. This is done by attaching the clear connector hose from the circuit package to the internal compressor side port of the ventilator. An oxygen tube can then be passed into the hose to provide a reservoir of oxygen. The FiO_2 delivered is a function of both the oxygen flow rate and the length of reservoir hose used, and can be measured with an oxygen sensor (Table 47-1).

CONVENTIONAL GENERAL ANESTHESIA MACHINES

Both UK and US forces now use conventional anesthesia machines. This is becoming a core item for Role 2E (enhanced) and Role 3 MTFs for the United Kingdom; US forces have relied on conventional machines for both Role 2 and Role 3 MTFs for a number of years.

Dräger Fabius Tiro M

UK and US forces have worked with Dräger to develop a militarized version of the Fabius Tiro machine (Dräger AG & Co, Lübeck, Germany). The UK DMS recognized that for enduring operations in a Role 3 facility, a conventional anesthesia machine was more appropriate than a draw-over system, and after reviewing relevant systems available at the time, chose the Fabius Tiro (Table 47-2). The variant adopted was a two-gas machine (oxygen and air) with a conventional three-drawer trolley (Figure 47-8). There are separate control knobs for the gases with light-emitting diode displays for gas flow; a single rotameter indicates total gas flow. The circle system can be fitted with a disposable absorber canister, or a different canister can be fitted to use loose absorbent. Low-volume pediatric tubing is also available. Sevoflurane is the volatile agent used.

The ventilator is an electrically driven (gas-efficient), modern design. There are four modes of ventilation available: (1) volume, (2) pressure, (3) pressure sup-

TABLE 47-1

EXPECTED FIO_2 WITH VARYING OXYGEN FLOW AND RESERVOIR LENGTH

Oxygen Flow (L/min)	Reservoir Hose Length (ft)	Approximate FiO_2 Delivered
2	3	0.45
4	3	0.55
4	6	0.65
6	6	0.85

FiO_2: fraction of inspired oxygen

TABLE 47-2

SPECIFICATIONS FOR THE DRÄGER FABIUS TIRO M*

Characteristic	Values
Tidal volume (VC):	20–1,400 mL (20–1,100 mL, SIMV/PS)
Rate	4–60 breaths/min
PEEP/CPAP	0–20 cmH_2O
Inspiratory flow	10–100 L/min
I:E ratio	4:1 to 1:4
Inspiratory pause	0–50%
SIMV inspiratory time	0.3–4 sec
Inspiratory pressure	(PEEP + 5) to 65 cmH_2O
Inspiratory flow	10–75 L/min (VC, PC), 10–85 L/min (PS)
PS level	PEEP +3 to 20 cmH_2O
Trigger	2–15 L/min
Size, set up on container (H × W × D)	47.2″ × 49.8″ × 31.9″
Weight, set-up on container	91 kg (198 lb)
Power supply	100–240 VAC 50–60 Hz

*Manufactured by Dräger AG, Lübeck, Germany.
CPAP: continuous positive airway pressure
I:E: inspiration to expiration
PC: pressure control
PEEP: positive end expiratory pressure
PS: pressure support
SIMV: synchronized intermittent mechanical ventilation
VAC: volts alternating current
VC: volume control

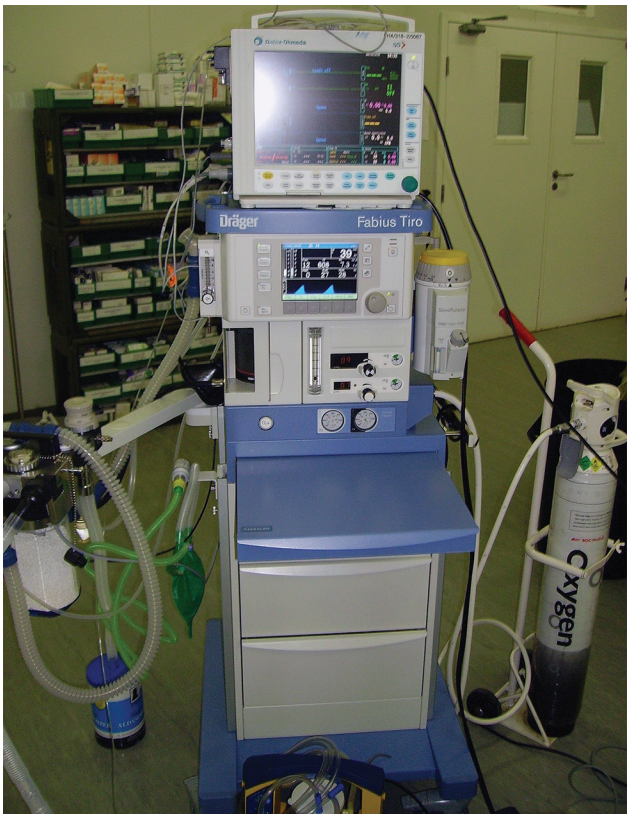

Figure 47-8. Fabius Tiro: UK version.
Product image used with permission from Dräger AG, Lübeck, Germany.

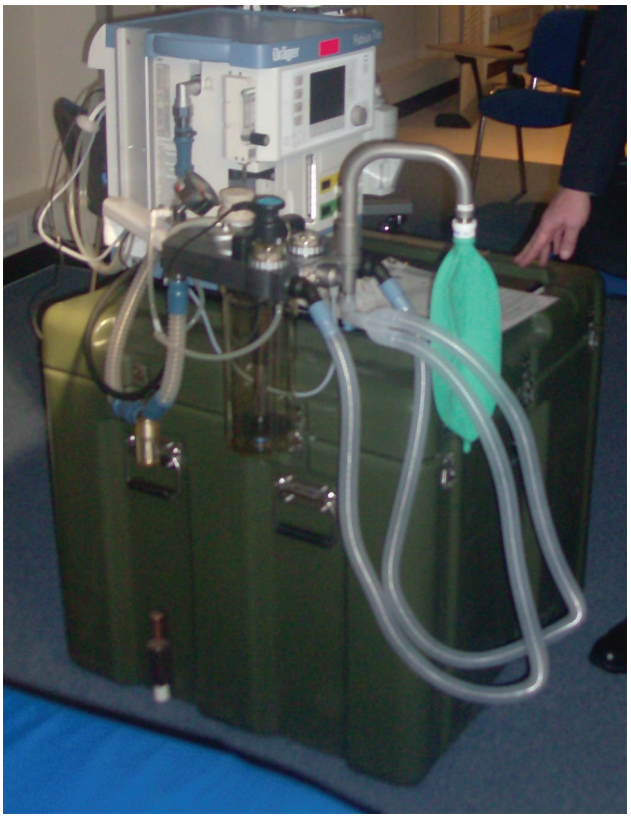

Figure 47-9. Fabius Tiro M: US version.
Product image used with permission from Dräger AG, Lübeck, Germany.

port, and (4) synchronized intermittent mandatory ventilation with pressure support (SIMV/PS). The first two are standard, but the latter have been specified by the UK DMS. The ventilator automatically compensates for the compliance of the tubing to maintain a set tidal volume.

The machine accepts a range of input voltages and has a battery backup, which is quoted to last 45 minutes. Oxygen and air supply requirements are the same as for any modern machine in terms of pipeline and cylinder requirements. In the absence of a high-pressure pipeline supply, the machine works well when connected to a ZX-size cylinder (3,040 L oxygen) instead.

Although US medical services use a similar Fabius Tiro at larger Role 3 facilities, they have worked with Dräger to design a more compact version for Role 2 MTFs in which the three-drawer trolley has been removed and the main working section of the machine is packed in a transport container (Pelican-Hardigg, Torrance, California). This container becomes the working platform for the machine on deployment, which creates a smaller overall package, although the footprint is larger (Figure 47-9). Another US development is a portable oxygen generator system (POGS; On Site Gas Systems Inc, Newington, CT), which can supply a single machine with all of its gas requirements. The POGS-10 only supplies up to 11 L/min of oxygen, so the oxygen flush will not work in the conventional manner because the machine input will always be limited by the POGS-10 output.

Dräger Narkomed M

The Dräger Narkomed M (Dräger AG & Co, Lübeck, Germany) is used at most Role 2 forward operating bases. It is a compact, lightweight, continuous-flow anesthesia system (Figure 47-10). Narkomed M machines are equipped with airway monitoring capabilities and a pneumatically driven ventilator to deliver gases and anesthetic vapor to adult and pediatric patients. The Narkomed M is easily transported in two protective containers.

The machine can deliver oxygen, air, and nitrous oxide, although nitrous oxide does not tend to be used in theater MTFs. There is a single vaporizer, and

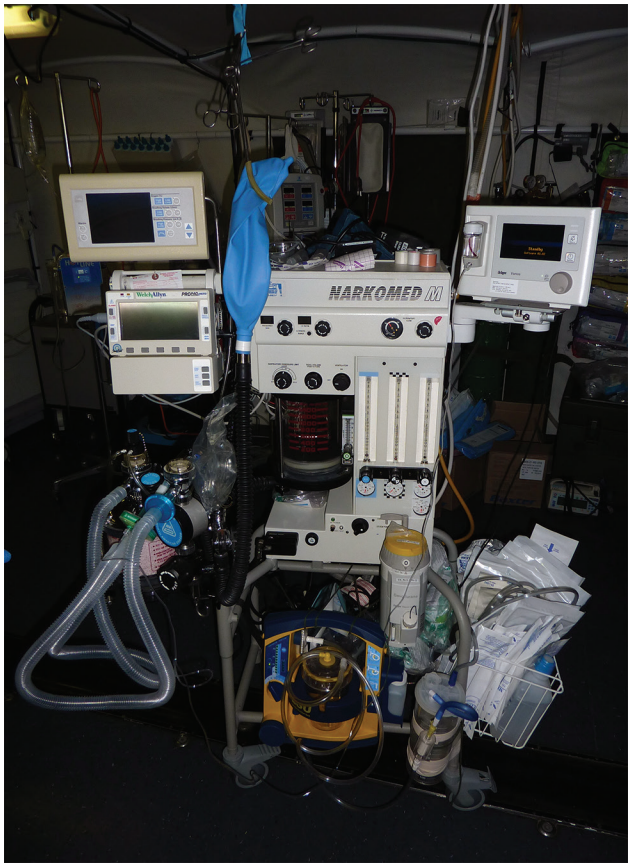

Figure 47-10. Dräger Narkomed M with Vamos and Propaq monitors.
Dräger Narkomed M and Vamos images used with permission from Dräger AG, Lübeck, Germany. Propaq image used with permission from Welch Allyn, Beaverton, Oregon (shown is the Encore 200 model, which has been discontinued and replaced by the Zoll Propaq M and Propaq MD).

pipeline connectors and the oxygen cylinder yoke are located on the back of the machine. However, there is no provision for air cylinders. Individual flow meters for each gas are located above their corresponding flow-control valve. A float indicator is in each flow tube and should be read at the center to determine gas flow rate. A pneumatic interlock system, the oxygen ratio controller, allows independent control of oxygen and nitrous oxide flows and will maintain a fresh gas oxygen concentration of 25% (± 4%) to prevent delivery of a hypoxic mixture. In the event of a loss of oxygen pressure, the Narkomed M is equipped with a pneumatically operated oxygen-failure protection device (Table 47-3).

Isoflurane and sevoflurane are available to US anesthesiologists and are delivered from a Dräger Vapor 2000 vaporizer. The absorber system accommodates sensors to monitor oxygen concentration, tidal volume, respiratory minute volume, and respiratory frequency. The desired mode of operation is selected with the manual/automatic selector valve. Absorbent can be either soda lime or barium hydroxide. A pressure gauge is located on the absorber to monitor breathing-circuit pressure (Figure 47-11).

TABLE 47-3

SPECIFICATIONS FOR THE DRÄGER NARKOMED M*

Characteristic	Values
Tidal volume	50–1,500 mL
Rate	1–99 breaths/minute
PEEP/CPAP	2–15 cmH_2O
Inspiratory flow	10–100 L/min
I:E ratio	4:1 to 1:4.5
Size (H × W × D)	50″ × 21″ × 16″ (128 × 53 × 41 cm)
Weight	47 kg (103 lb)
Power supply	100–240 VAC 50–60 Hz

*The Dräger Narkomed M is manufactured by Dräger **AG & Co,** Lübeck, Germany.
PEEP: positive end expiratory pressure
I:E: inspiration to expiration
CPAP: continuous positive airway pressure
VAC: volt alternating current

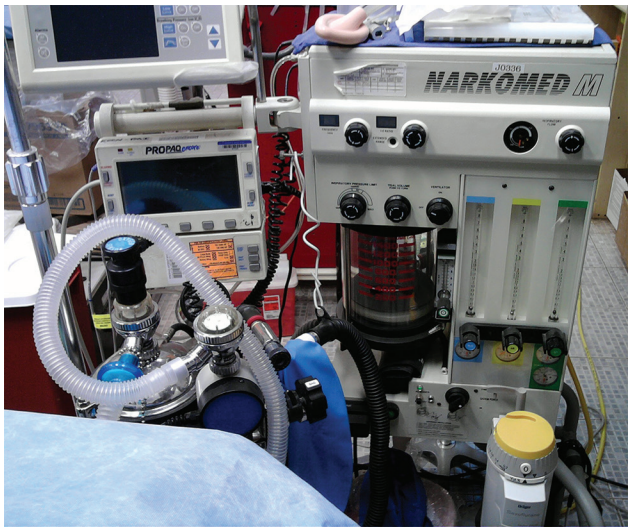

Figure 47-11. Narkomed gas flow and ventilation controls. Product image used with permission from Dräger AG, Lübeck, Germany.

Manual or automatic ventilation can be selected using a selector situated on the circle absorber. Drive gas to the ventilator comes from the main gas supply being used. Oxygen or air can be selected as the drive gas via a selector switch on the right side of the machine. The anesthesia ventilator is a volume-preset, time-cycled, pressure-limited ventilator with electronic timing and pneumatic circuitry and controls for frequency, inspiratory-to-expiratory ratio, inspiratory flow rate, tidal volume, and inspiratory pressure control. The

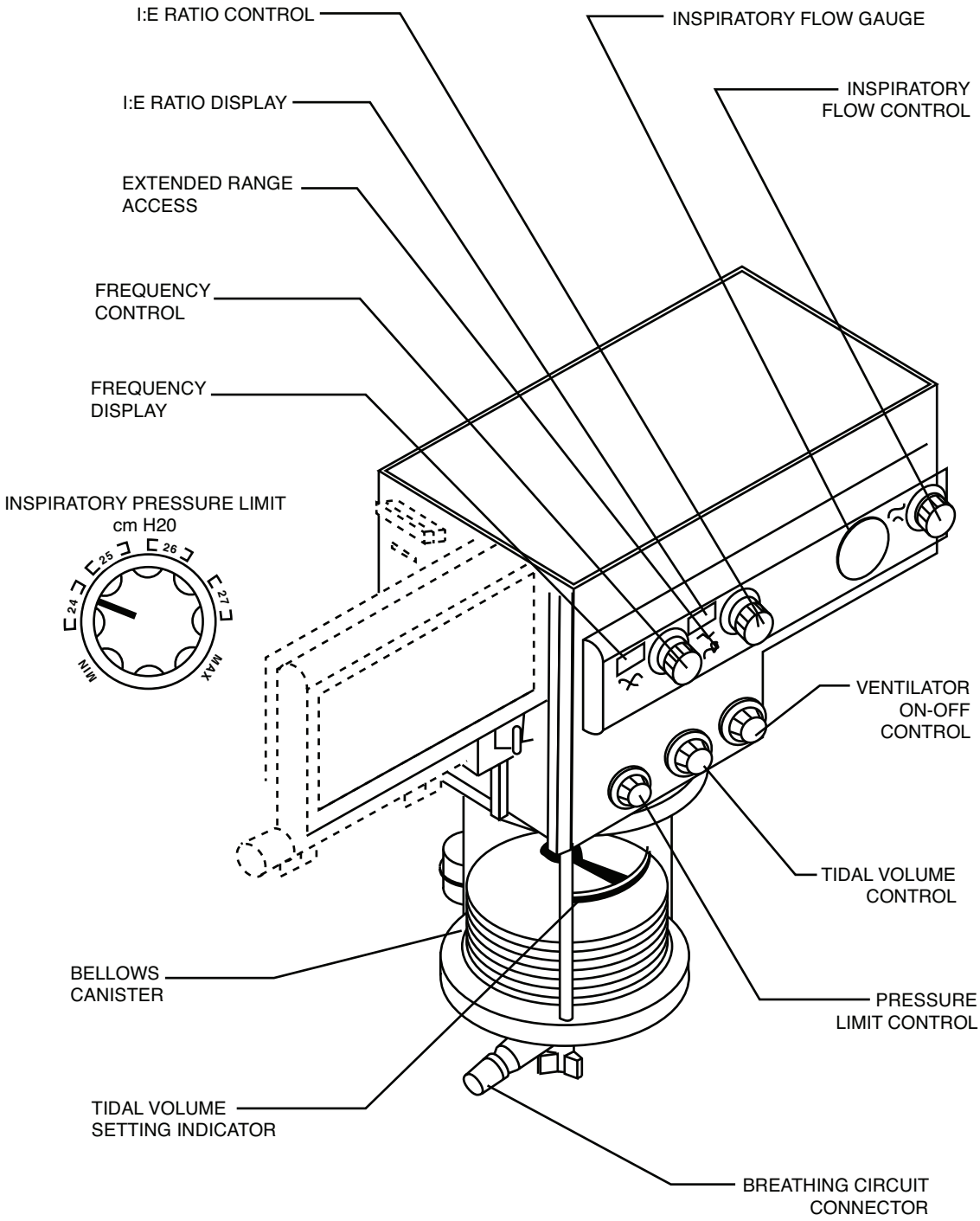

Figure 47-12. Layout of ventilation controls on the Narkomed M. I:E: inspiratory-to-expiratory. Product image used with permission from Dräger AG, Lübeck, Germany.

Narkomed M uses an ascending bellows; the effect of gravity on the bellows causes a positive end-expiratory pressure within the breathing system of about 2 cm H_2O. (Figure 47-12)

The machine is powered from an alternating current (AC) power source, but a backup battery can run the machine for at least 90 minutes. A three-pulse audible alarm will sound every 30 seconds and a caution message will be displayed to signal backup battery activation. When the battery nears depletion, "AC power fail" is displayed and indicates there is approximately 10 minutes of backup battery power remaining. If the battery is depleted and there is no AC power to the system, manual ventilation may be required. The machine cannot provide monitoring or alarm functions until AC power is restored.

OXYGEN SUPPLIES

The logistical burden of supplying oxygen cylinders to a deployed medical facility is significant. UK and US forces have approached these issues in a number of ways.

DeVilbiss Oxygen Concentrator

The DeVilbiss Oxygen Concentrator (DeVilbiss, Somerset, PA) is a compact, portable (16.8 kg) concentrator that uses the zeolite process to concentrate oxygen from room air (Figure 47-13). This particular model has a maximum output of 5 L/min and produces an oxygen concentration of up to 93% (± 3%; the higher the flow, the lower the concentration). It runs on central power (220–240 VAC). It is used by the UK DMS with the TSAA for low-flow oxygen requirements and to feed the intensive care unit (ICU) ventilator (see LTV 1000 Series ICU Ventilator, below). The controls are simple, consisting of a power switch and a flow meter. Warning devices include an orange light that indicates when oxygen output is less than 85%, an audible alert that sounds if oxygen drops below 75%, and a red light that appears if concentration falls below 60%.

Figure 47-13. DeVilbis oxygen concentrator.
Product image used with permission from DeVilbiss Healthcare Ltd, Tipton, West Midlands, United Kingdom.

Expeditionary Deployable Oxygen Concentrator System

The expeditionary deployable oxygen concentrator system (EDOCS) generator (Pacific Consolidates Industries, Riverside, CA) is a large, heavy piece of equipment (42 inches wide, 104 inches long, 67 inches tall, and approximately 1,500 kg) that can be used to fill oxygen cylinders as well as supply pipeline oxygen to a medical facility (Figure 47-14). Initial problems of dust and heat in recent operational environments have led to some design modifications as well as changes in operating procedures. The machine can be used at night to fill cylinders that supply the hospital pipeline. The EDOCS module generates oxygen using vacuum swing adsorption technology. The EDOCS-120B concentrator generates 120 L/min (7.2 m^3/h) at 85 psi. The oxygen (minimum 93% ± 3%) can be delivered directly from the concentrator to the hospital distribution system at 85 psi or supplied to the boost compressor, which compresses the gas to 2,250 psi to fill standard medical oxygen cylinders via a cylinder-filling manifold. A vacuum pump is included with the EDOCS to evacuate cylinders prior to filling. It is used at US Role 3 MTFs as well as at some combat air staging facility units.

Portable Oxygen Generator System

POGS, which was more recently developed, has been used by US forces in Afghanistan (Figure 47-15). Two versions are available. The POGS-10 is used in conjunction with the US Fabius Tiro M. The larger POGS-33 has been used with the Narkomed M but

Figure 47-14. Expeditionary deployable oxygen concentrator system oxygen generator.
Product image used with permission from Pacific Consolidates Industries, Riverside, CA.

Figure 47-15. Portable Oxygen Generator System (POGS-33).
Product image used with permission from On Site Gas Systems Inc, Newington, CT.

can also be used as a stand-alone system for filling oxygen cylinders. POGS-33 produces a total flow of 33 L/min of 93% oxygen or 30 L/min of medical-grade air. POGS consists of three components: (1) a feed air compressor, (2) an oxygen generator, and (3) a high-volume booster that enables the filling of oxygen cylinders. The feed air compressor is typically located outside the medical facility and should be in a clean, sheltered area. This compressed air is then transferred to the POGS. The oxygen generator functions as a molecular sieve and separates the oxygen from the nitrogen and water vapor. The high-volume booster is a compressor system that enables high-pressure oxygen storage. It produces a pressure of 2,250 psig at a flow of 30 to 120 scfh.

MONITORS

General Electric Healthcare Datex-Ohmeda S/5 Compact

The S/5 Compact (General Electric Healthcare, Chalfont St Giles, UK) is a typical modern anesthesia monitor with a color display (Figure 47-16). It has many similarities to the AS/3 model from which it is derived. It is transportable but, at 11 kg, it is heavier than some other portable monitors. However, the weight can be tolerated in favor of its other capabilities, including invasive pressures, volatile agent concentration, and spirometry. The color display is highly configurable, with up to eight wave forms and four digital fields. The left side of the screen can display a 30-minute trend or a flow volume loop with lung compliance measurements (Table 47-4). Trends can be displayed graphically or in tabular form and can be printed out. It can tolerate temperatures from 10°C to 35°C while operating. Recent military conflicts have shown that the S/5 Compact can be used in these temperatures without adverse event. This range of capabilities makes the S/5 Compact suitable for use as an anesthesia and critical care monitor.

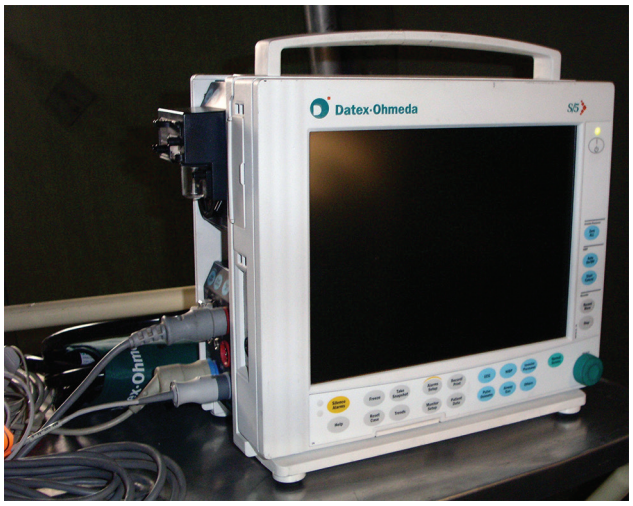

Figure 47-16. S/5 Compact monitor.
Product image used with permission from General Electric Healthcare, Chalfont St Giles, United Kingdom.

TABLE 47-4

SPECIFICATIONS FOR THE S/5 COMPACT MONITOR*

Capabilities	Characteristics
Standard capabilities	ECG, NIBP, SaO_2
Advanced capabilities	Invasive pressure (2 channels), temperature (2 channels)
Gas monitoring	FiO_2, $EtCO_2$, Et volatile, spirometry, airway pressures, RR
Screen	12.1" color display, 800 × 600, 8 waveforms, 4 digital fields
Power supply	100–240 VAC (50–60 Hz)
Battery back up	90 min, NiMH (60 min, NiCd)
Temp range stored	– 10°C to + 50°C
Temp range operational	10°C to 35°C

*The S/5 Compact monitor is manufactured by General Electric Healthcare, Chalfont St Giles, UK.
ECG: electrocardiogram
Et: end-tidal
$EtCO_2$: end-tidal carbon dioxide
FiO_2: fraction of inspired oxygen
NIBP: noninvasive blood pressure
NiCd: nickel cadmium
NiMH: nickel metal hydride
RR: respiratory rate
Temp: temperature
SaO_2: oxygen saturation
VAC: volt alternating current

US Anesthesia Monitoring

US Role 2 and Role 3 facilities use a variety of monitors, including the Datex-Ohmeda AS/3, the Dräeger Vamos, and the Propaq. Because the AS/3 has an almost identical range of capabilities to the S/5, it will not be described further.

Dräeger Vamos

The Dräeger Vamos (Dräger AG & Co, Lübeck, Germany) anesthetic gas monitor is commonly supplied with the Narkomed M (See Figure 47-10) and is not normally seen as a stand-alone monitor. It is a compact module with a 5-inch, electroluminescent, amber display with various viewing angles. The device will monitor anesthetic agents, carbon dioxide, and nitrous oxide. There is graphic display of the carbon dioxide waveform and numeric displays of inspiratory and end-tidal concentrations of carbon dioxide, nitrous oxide, and volatile agent. The gas sensor samples at a flow of 200 mL/min, and the sampled gas can be returned to the anesthesia circuit.

Propaq

A number of Propaq (Welch Allyn, Beaverton, OR) models are used, but the variant with end-tidal carbon dioxide monitoring is preferred (see Figure 47-10). In addition to electrocardiogram, noninvasive blood pressure, oxygen saturation, and two temperature channels, the Encore 206EL can monitor two invasive pressure channels as well as end-tidal carbon dioxide. The latter option requires the expansion module to be fitted below the main monitor. A range of alarms is available as well as trend data. The unit is powered from 12 to 28 volts direct current (VDC), which is compatible with vehicle and aircraft power supplies. The main unit comes in US (100–120 VAC) and European (200–240 VAC) options. There is a 3-hour battery life with all options, including end-tidal carbon dioxide, bringing the weight up to 6.1 kg. Although Propaq monitors are used at some lighter scale UK MTFs, they are not normally used as anesthetic monitors.

VENTILATORS

A limited range of ventilators is available to both forces to reduce logistics issues and the training burden for clinical and maintenance staff. For the UK DMS, the range available to field hospitals is limited to the

CompPAC 200 and the Vela (Carefusion, Yorba Linda, California). For ventilation in the emergency department and for intrahospital transfers, the CompPAC 200 is used (see The Triservice Anaesthetic Apparatus, above). The Vela is preferred for use in small children, even in the emergency department.

Vela

The Vela series comprises the Vela Comprehensive, Vela Plus, and Vela. The UK DMS has chosen the Comprehensive model (Figure 47-17), which is a fully specified, compact, mobile device. Features include a high-pressure oxygen inlet (40–85 psi) with blender, a low-pressure oxygen inlet (up to 0.5 psi, for example, from the DeVilbiss concentrator) with accumulator, and integrated monitoring and continuous display of delivered FiO_2, with high and low alarm settings. Turbine technology provides independence from compressors and wall air supplies. It can be used for pediatric or adult ventilation and has a broad range of operating modes, including volume control, pressure-regulated volume control, airway pressure release ventilation, biphasic pressure control, pressure support, noninvasive ventilation, SIMV, and continuous positive airway pressure (CPAP). There is apnea backup ventilation in SIMV and CPAP/pressure support ventilation. The ventilator tolerates a wide range of supply voltages (90–264 VAC at 47–65 Hz) and has a 6-hour internal battery. The 10.4-inch, active matrix, full-color touchscreen gives access to physiologic data and current ventilator status, including flow and volume loops and trends. The essential controls for the selected mode are displayed; inactive controls are not displayed until needed (Table 47-5).[12]

TABLE 47-5

SPECIFICATIONS FOR THE VELA VENTILATOR*

Capability	Characteristics
Tidal volume	50–2,000 mL
Rate	2–80 breaths/min
FiO_2	0.21–1.0
PEEP/CPAP	0–35 cmH_2O
PC/PS/spontaneous flow	To 140 L/min (180 L/min spont max)
PS	Off, 1–100 cmH_2O
Trigger sensitivity	Off, 1–20 L/min
PC	0–100 cmH_2O
Inspiratory time	0.3–10 s (100 L/min)
Size (H × W × D)	12″ × 13″ × 14.5″ (30.5 × 33 × 36.8 cm)
Weight	17.2 kg
Power supply	90–264 VAC, 47–65 Hz

*The Vela Ventilator is manufactured by Carefusion, Yorba Linda, CA.
CPAP: continuous positive airway pressure
FiO_2: fractioned inspired oxygen
min: minimum
max: maximum
PEEP: positive end expiratory pressure
PC: pressure control
PS: pressure support
spont: spontaneous
VAC: volt alternating current

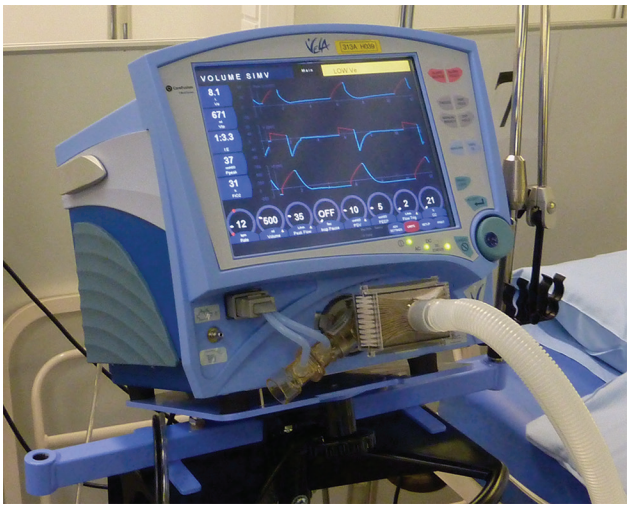

Figure 47-17. Vela ICU ventilator.
Product image used with permission from Carefusion, Yorba Linda, CA.

US Ventilators

Impact 754 Eagle Univent

The Univent (Impact Instrumentation Inc, West Caldwell, New Jersey) is a compact, versatile, portable flow generator (Figure 47-18). The ventilator has an internal battery that can be powered directly from any 11 to 15 VDC power supply. When operating on the internal compressor, the battery will last 3 hours; if external gas supplies are used, the battery can last 12 hours. There is a separate power supply unit that operates from international voltages and frequencies. Modes of ventilation include volume control, SIMV, pressure support, and CPAP (Table 47-6).

The ventilator has an internal compressor, but external oxygen and air cylinders can be attached. With an

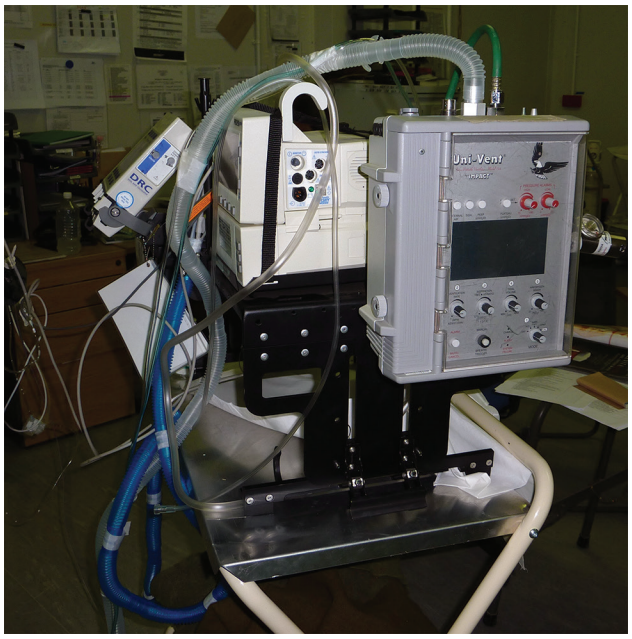

Figure 47-18. Impact 754 Eagle Univent.
Product image used with permission from Impact Instrumentation Inc, West Caldwell, NJ.

external oxygen source, the FiO_2 can be adjusted from 0.21 to 1.0(external sources must be 40 psi or more).

LTV 1000 Series ICU Ventilator

The LTV 1000 series (Carefusion, Yorba Linda, California) is used by US medical services as an ICU ventilator (Figure 47-19). UK Critical Care Air Support Teams also use it as a transport ICU ventilator, and it was used as a pediatric ventilator in ICUs prior to introduction of the Vela. With tidal volumes down to 50 mL, the device can be used for children weighing as little as 5 kg.

As with most modern ICU ventilators, there is a range of ventilation modes (volume control, pressure control, pressure support, spontaneous, SIMV, CPAP, noninvasive ventilation). The device runs from a similar power supply range as the Univent, and the main adaptor has international voltage capability (Table 47-7).

The ventilator will run on an internal battery for 45 minutes or an external battery pack (a 12 amp/h battery will last approximately 3 hours), as well as the main power supply unit. Using the same turbine technology as the Vela, the LTV 1000 will run from high- and low-pressure oxygen sources. When using a low-pressure oxygen source, the ventilator calculates delivered FiO_2 but does not measure it.

In hot climates, it is important to keep the air inlet filter damp and to check the side panel connections for the pressure- and volume-monitoring hoses, which have been known to loosen. It is an effective and versatile ventilator.

TABLE 47-6

SPECIFICATIONS FOR THE IMPACT 754 EAGLE UNIVENT*

Capability	Characteristic
Tidal volume	0–3,000 mL
Rate	1–150 breaths/min
FiO_2	0.21–1.0
PEEP/CPAP	1–20 cmH_2O
Flow rate	0–60 L/min
I:E ratio	1:1 to 1:599
Inspiratory time	0.1–3 s
Size (H × W × D)	11.5″× 8.87″ × 4.5″ (29.2 × 22.5 × 11.4 cm)
Weight	5.8 kg (13 lb)
Power supply	11–15 VDC, 100–240 VAC, 50–60 Hz

*The Impact 754 Eagle Univent is manufactured by Impact Instrumentation Inc, West Caldwell, NJ.
FiO_2: fraction of inspired oxygen
I:E: inspiration to expiration
VAC: volts alternating current
VDC: volts direct current

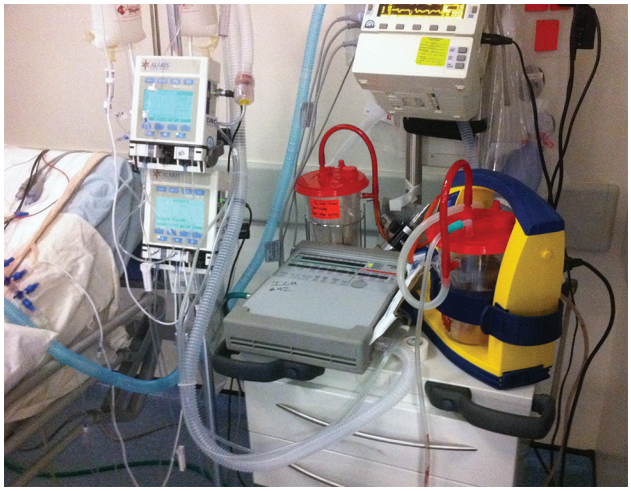

Figure 47-19. LTV 1000.
Product image used with permission from Carefusion, Yorba Linda, CA.

INTRAVENOUS INFUSION EQUIPMENT

Both the United Kingdom and the United States now use the same equipment for the aggressive volume resuscitation of casualties of conflict.

Level 1 H-1200 Fast Flow Fluid Warmer

The Level 1 H-1200 Fluid Warmer (Smiths Medical, Ashford, Kent) is an intravenous (IV) rapid infuser with fluid warming and two pressure chambers for fluids or blood products (Figure 47-20; Table 47-8). It has air detection and automatic clamping capability. The chambers pressurize the fluids, enabling rapid delivery. The infuser employs single-use, disposable administration sets that include a gas vent/filter assembly and heat exchanger to warm the IV fluids.

The installation, set-up, and replacement of Level 1 administration sets follows a four-step sequence that corresponds to numbered blocks on the device. The air detector/clamp monitors for the presence of air in the IV fluid path. When air is detected, the air detector/clamp closes off the patient line and triggers audible and visual alarms. This device has been used successfully by UK and US forces for some years, but it requires two operators per machine and occasionally must be swapped out to replace the disposable set. This means that more personnel may be involved than is sometimes ideal. The Level 1 Hotline HL-90 (Figure 47-21) is a more basic fluid warmer that is preferred to the H-1200 for pediatric massive transfusion. There is no air detection with this model (Table 47-9).

TABLE 47-7

SPECIFICATIONS FOR THE LTV 1000*

Capability	Characteristics
Tidal volume	50–2,000 mL
Rate	0–80 breaths/min
FiO_2	0.21–1.0
PEEP/CPAP	0–20 cmH_2O
PC/PS/spontaneous flow	To 160 L/min
PS	Off, 1–160 cmH_2O
Trigger sensitivity	Off, 1–9 L/min
PC	1–99 cmH_2O
Inspiratory time	0.33–9.9 s (100 L/min)
Size (H × W × D)	3″ × 10″ × 12″ (8 × 25 × 30 cm)
Weight	6.1 kg
Power supply	11–15 VDC

*The LTV 1000 is manufactured by Carefusion, Yorba Linda, CA.
CPAP: continuous positive airway pressure
FiO_2: fraction of inspired oxygen
PC: pressure control
PEEP: peak end expiratory pressure
PS: pressure support
VDC: volts direct current

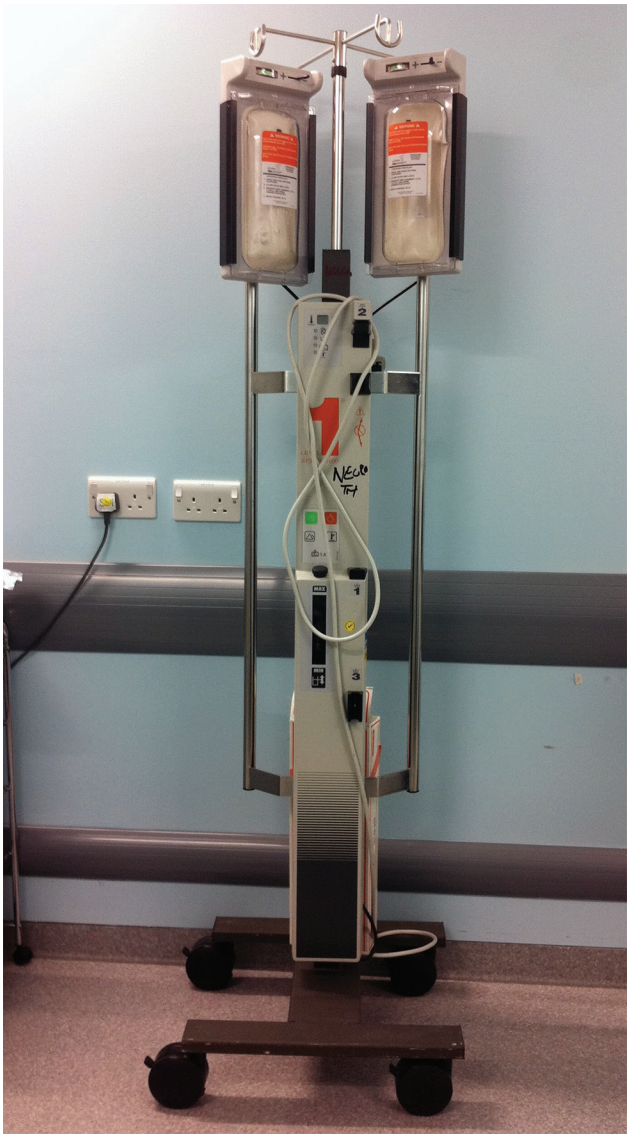

Figure 47-20. Level 1 H-1200.
Product image used with permission from Smiths Medical, Ashford, Kent, United Kingdom.

TABLE 47-8

LEVEL 1 HOTLINE H-1200* SPECIFICATIONS

Capability	Characteristic
Flow rate (10°C input temp)	Normothermic output up to 650 mL/min
Maximum flow rate (crystalloid)	1,400 mL/min
Reservoir capacity	1.4 L
Size (H × W × D)	67″ × 20″× 20″ (1.7 m × 51 cm × 51 cm)
Weight (dry)	63 pounds (28.5 kg)
Operating temp	10°C to 40°C
Central power supply	115 VAC 60 Hz, or 230 VAC 50 Hz

*The Level 1 Hotline H-1200 is manufactured by Smiths Medical, Ashford, Kent, UK.
temp: temperature
VAC: volts alternating current

Belmont Rapid Infuser FMS 2000

The Belmont Rapid Infuser FMS 2000 (Belmont Instrument Corp, Billerica, Massachusetts) is available to US medical services and, beginning in October 2011, it was also made available to deployed UK Role 3 hospitals (Figure 47-22). Rather than rely on a pressure-bag-style infusion capability, the FMS 2000 uses a roller pump. An electromagnetic induction heating system warms fluid from 4ºC to 37.5ºC in a single pass (< 45 seconds; Table 47-10).

There are two options for large-volume blood product administration: the standard administration set has a three-way spike system, but this upper section can be replaced by a 5-spike, 3-liter cardiotomy reservoir.

The 5-by-2.5-inch display walks the operator through the setup and priming process and shows flow rate, total volume infused, fluid temperature, and in-line pressure. Infusion pressure is continuously monitored and fluid infusion is stopped when the pressure is greater than 300 mm Hg or if there is a sudden pressure spike. Pressure monitoring will automatically restrict flow, so this should be expected where fluids are being infused through smaller vascular access devices (eg, intraosseous needles).[12] There is a recirculation facility that cycles fluid through the system at 200 mL/min. This permits fluids to mix in the reservoir, and it can also be used to warm fluid before disconnecting power for transport. There is an automatic purge of air after each 500 mL of infusion to keep the system clear of the air generated as fluids are warmed.

Unlike some other rapid infusers, the FMS 2000 has a battery backup that engages as AC power is disconnected. While on battery, the maximum infusion rate is

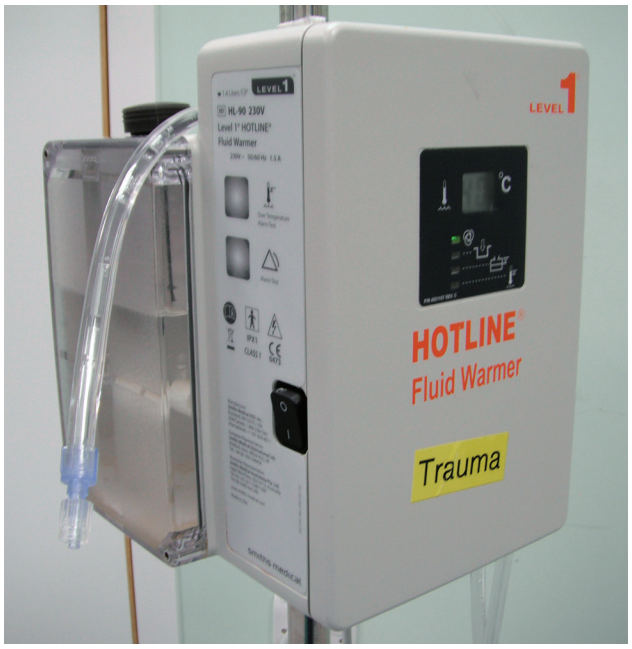

Figure 47-21. Level 1 HL-90.
Product image used with permission from Smiths Medical, Ashford, Kent, United Kingdom.

TABLE 47-9

LEVEL 1 HOTLINE HL-90* SPECIFICATIONS

Capability	Characteristics
Flow rate (10°C input temperature)	Normothermic output up to 3,500 mL/min
Maximum flow rate (crystalloid)	5,000 mL/min
Reservoir capacity	1.4 L
Size (H × W × D)	9.5″ × 8.3″ × 7″ (24.1 × 21 × 17.8 cm)
Weight (dry)	7.6 lb (3.5 kg)
Operating temperature	10°C to 45°C
Central power supply	100 VAC, 115 VAC, or 230 VAC 50/60 Hz

*The Level 1 Hotline HL-90 is manufactured by Smiths Medical, Ashford, Kent, UK.
VAC: volts alternating current

Figure 47-22. Belmont FMS200.
Product image used with permission from Genesy Medical Solutions, Ascot, United Kingdom.

restricted to 50 mL/min and there is no fluid warming. The battery provides at least 30 minutes of operation and has an 8-hour recharge time from flat.

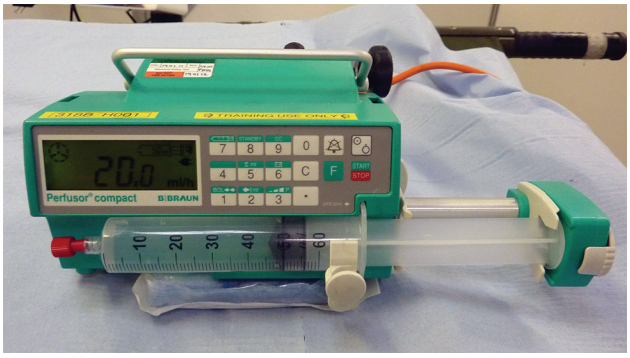

Figure 47-23. Braun Perfusor.
Product image used with permission from B. Braun, Melsungen, Germany.

TABLE 47-10

BELMONT FMS 2000 SPECIFICATIONS*

Capability	Characteristics
Fluid pump	Peristaltic roller pump
Flow rate	2.5–750 mL/min
Bolus volume	100 mL to 500 mL (50 mL increments)†
Size (H × W × D)	13.5" × 7.5" × 12"
Weight	26 lb
Operating temperature	10°C to 32°C
Central power supply	115 VAC or 230 VAC

*The Belmont FMS 2000 is manufactured by Belmont Instrument Corp, Billerica, Massachusetts.
†Maximum bolus size for UK DMS is 250 mL.
VAC: volts alternating current

For the UK version, software changes have been implemented following cases of fluid volume overload using pressure-bag infusion pumps. UK Role 3 models have a maximum bolus volume of 250 mL.

Braun Perfusor Compact S

Used by the UK DMS, the Perfusor (B Braun, Melsungen, Germany) is a battery- and central-powered syringe driver (Figure 47-23; Table 47-11). In this

TABLE 47-11

SPECIFICATIONS FOR THE BRAUN PERFUSOR COMPACT S SYRINGE DRIVER*

Capability	Characteristics
Syringe selection	2/3, 5, 10, 20, 30, 50/60 mL
Delivery rate	0.01–200 mL/h (depends on syringe size)
Size (H ×W × D)	10 × 19 × 12 cm
Weight	1.5 kg
Power supply	Batteries (disposable/rechargeable), central power
Operating time using rechargeable pack	10 h at 10 mL/h
Operating time using alkaline batteries	80 h at 10 mL/h

*The Braun Perfusor Compact S Syringe Driver is manufactured by B. Braun, Melsungen, Germany.

case, the battery power is supplied by disposable AA cells, which have ubiquitous availability. Although a rechargeable battery pack is available, the pump lasts longer with disposable batteries.

The liquid-crystal display screen shows syringe type and size, delivery rate, infused volume, pressure alarm, and central power or battery operation. There is a three-bar battery indicator (ie, three bars means full power); batteries should be replaced when the display shows one bar. A maximum of three drivers can be stacked together and locked for transport. The Perfusor has the common alarm functions, including an occlusion alarm, an adjustable pressure limit, and an automatic bolus reduction following a pressure alarm. There are also alarms for 3 minutes before the syringe will empty and 30 minutes before the battery is discharged. The user interface is not intuitive, so it is important to become familiar with the pump before using it on patients; however, it appears to be reliable and is cleared for use on UK airframes.

SUMMARY

The sophistication and capabilities of modern medical equipment are continually advancing, and military medicine must continue to review its capabilities and update equipment where appropriate. In forward austere environments, compact, lightweight, and durable equipment is necessary for surgery and anesthesia; hence, compromises have been made in equipment to deliver a medical capability. At Role 3 MTFs, the equipment capability is increasingly at or above the standard seen in Western health services, which poses challenges for training, maintenance, and logistics.

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Chapter 48

SPECIALIST EQUIPMENT FOR PAIN MANAGEMENT

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INTRODUCTION

Historically times of conflict are associated with acceleration in the evolution of medical therapies. As the enemy develops new weaponry and technology designed to inflict injury and pain, the medical community furthers advancement in surgical and resuscitative techniques, resulting in increased injury survival.[1] However, this increased survival presents further challenges to the medical community. Injury patterns have shifted so that the ratio of extremity to head and torso injuries is increasing, and soldiers are surviving complex wounds that previously would have been fatal.[2]

To meet these demands, the plethora of advanced analgesic techniques common to civilian practice must be incorporated into military medical practice and adapted to the demands of the austere military healthcare environment. The nature of combat medical treatment facilities, where patients are rapidly stabilized and moved through a casualty evacuation chain, requires advanced pain management systems that are noncomplex and efficacious, require minimal user intervention, and ideally are intuitively familiar to systems used in civilian practice. Not all systems developed for use in civilian practice, however, are suited to the military environment.

Recent advances in battlefield analgesia have been focused on the ability of regional anesthesia to provide selective sensory analgesia while limiting, if not eliminating, the cognitive and respiratory depressant effects associated with parenteral opioids. Single injection nerve blocks, continuous peripheral nerve block (CPNB) catheters, and neuraxial anesthesia have become a major component of pain management in combat-related injuries. Peripheral nerve blocks of extremities have the ability to isolate an affected limb, while transversus abdominis plane blocks, well established as effective analgesia in postsurgical patients, can be used to manage traumatic abdominal pain when neuraxial anesthesia is contraindicated.[3–5]

Specialist pain management equipment used in the deployed environment can be broadly divided into that which facilitates and delivers the targeted placement of local anesthetic solutions and that which delivers systemic analgesia. By necessity administering analgesic medication in these modalities requires specialist devices and equipment.

NERVE LOCALIZATION

Electrical Nerve Stimulator

The use of electrical stimulation has been shown to be an inexpensive, uncomplicated, and portable means of nerve localization for regional anesthesia.[6] These advantages make stimulation a particularly attractive option for use in the deployed environment. During the placement of a peripheral nerve block, stimulation needles may be used both to detect proximity to nerves and to inject the local anesthetic solution. During the procedure, the nerve stimulator electrical cable must be connected to both the stimulation needle wire and the skin electrode to assure that, once the needle tip has contacted skin, a complete circuit exists. Initial currents should be set at 1 to 2 mA with a pulse width of 0.1 millisecond and frequency of 60 to 120 Hz. Current amplitude should be variable and capable of gradual reduction to 0.1 to 0.2 mA. A stimulating current threshold of 0.2 to 0.5 mA indicates adequate proximity to the nerve, and brisk twitches at currents of less than 0.2 mA may indicate an intraneural placement of the needle. Nerve stimulator design should include audible function and failure signals.

Medical Ultrasound

Application of medical ultrasound has revolutionized many areas of battlefield trauma management. Using data gleaned from the 2006 conflict between Hezbollah and Israel, Beck-Razi et al found the positive and negative predicted value of focused assessment with sonography for trauma (FAST) to be 88.2% and 94.1%, respectively.[7] The authors hypothesized that the use of FAST at point of injury likely prevented several unnecessary emergency laparotomies and thus minimized surgical pain.

Ultrasound-guided regional anesthesia (using medical ultrasound in the targeted placement of local anesthetics) has dramatically increased in popularity over the last 5 years. Although outcome data from head-to-head clinical trials has yet to provide sufficient evidence for making ultrasound use the standard of care, its theoretical advantages over stimulation are hard to ignore.[8,9] These potential advantages include direct visualization of nerve structures, reduced incidence of accidental vascular puncture, and increased patient comfort.[10]

Identification of peripheral neural structures requires ultrasound optimized for this purpose. High-frequency probes are required (8–15 MHz) combined with machines sufficiently portable to for performing nerve blocks at the bedside. Elements of an optimum ultrasound machine for combat regional anesthesia include high image quality;

compact, lightweight, and durable design; simple and intuitive controls; and easy image storage and retrieval.[11] Modern, affordable, and portable ultrasound technology for needle placement is making its way closer to the point of injury on the battlefield, and in the near future combat healthcare providers may be equipped with these next-generation lightweight devices.

NEEDLE DESIGN

Peripheral nerve block needles, as opposed to traditional hypodermic needles, are designed with a bevel angled at 20° to 30° (Figure 48-1). This blunted angle facilitates the detection of tissue planes and fascial layers as the needle tip is advanced toward its intended target. The blunted design also results in a non-cutting needle that may reduce the risk of neural tissue injury during procedures that invariably bring the needle tip within close proximity to neural structures.[12] A separate design for catheter insertion (epidural and peripheral) is the Tuohy needle, with a non-cutting tip, angled distal aperture, and graduated markings for more precise measurements. The needles may also be insulated throughout their length, excepting the tip, to facilitate nerve localization by electrical stimulation.

CATHETER TECHNIQUES

The CPNB delivers a continuous infusion of local anesthetic to a targeted site, enabling the duration of block provided by local anesthetics to be extended beyond the typical 8 to 20 hours achieved by a single injection technique. In the deployed setting a CPNB may be used to provide prolonged postoperative analgesia, including the time necessary to transfer between roles of care. Additionally, it may be used for the provision of regional anesthesia when patients require repeated surgical procedures, obviating the need for multiple general anesthetics.

Irrespective of the austere nature of the deployed environment, catheter insertion techniques should be undertaken in aseptic conditions with appropriate skin decontamination and draping with a sterile field. Ultrasound probes should be contained within a sterile sleeve when used.

Catheter kits are available for both central neuraxial and regional nerve block techniques, an example being the Braun Contiplex Touhy system (B Braun, Melsungen, Germany). Epidural sets contain a needle of Tuohy design, catheter, connector, and filter assembly, and needles for regional techniques may in addition have an insulated sheath, integrated wire for electrical stimulation, and diaphragm allowing for catheter insertion.

MEDICATION DELIVERY SYSTEMS AND PATIENT-CONTROLLED ANALGESIA

Continuous delivery of analgesic medication requires the use of a pressurized delivery system. Intravenous delivery systems are used primarily to administer opioids and ketamine, while perineural delivery systems are used for local anesthetics with or without additive medications.

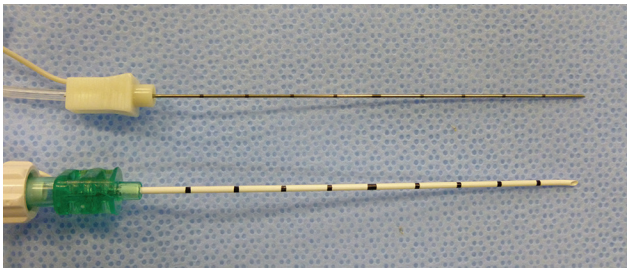

Figure 48-1. Examples of beveled nerve block needles. Above: a stimulating, non-echogenic single-shot peripheral nerve block needle; below: a non-stimulating, echogenic needle for the placement of a continuous nerve block catheter. Both products copyright B Braun Melsungen AG (used with permission).

Patient-controlled analgesia (PCA) involves the use of an infusion pump that delivers a preprogrammed dose of opioid when the patient pushes a demand button. The concept of the PCA has been in existence since the 1960s, but detailed pharmacologic work on the system began in earnest in the 1970s.[13] This pharmacokinetic and pharmacodynamic research resulted in two main concepts achieved by PCA administration of opioids: (1) individualized dosages titrated to pain relief response to achieve the MEAC (minimum effective analgesic concentration) and establish analgesia, and (2) constant plasma opioid concentrations, avoiding peaks and troughs (Figure 48-2).[14]

All modes of PCA include the following basic variables: initial loading dose, demand dose, and lockout interval. For PCA to be successful, the demand dose should produce appreciable analgesia with a single demand.[15] The lockout interval is designed to prevent

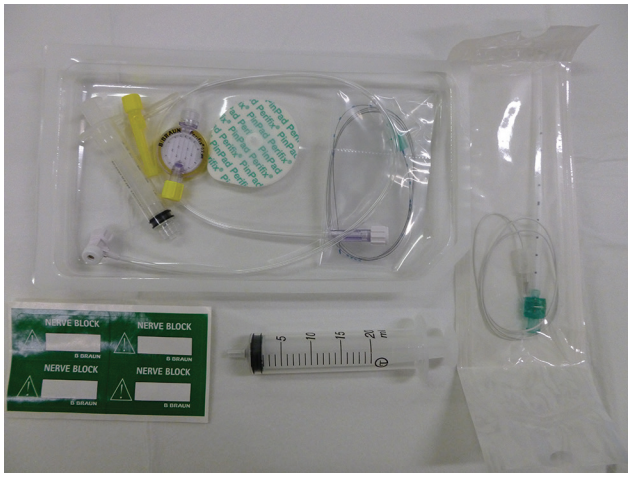

Figure 48-2. Sterile continuous nerve block set. Copyright B Braun Melsungen AG (used with permission).

overdose. Ideally, it should be long enough for the patient to experience the maximal effect of one dose before another is permitted, thus preventing "stacking" of doses.[16] All of the commonly used opioids have been successfully employed for PCA dosing. Based on the patient's individual comorbidities (eg, potentially avoiding morphine in renal failure patients) and pharmacokinetic profile of the medication, morphine, hydromorphone, and fentanyl are all reasonable choices to initiate PCA.

In addition to the targeted approach to the therapeutic window, there is additional safety in utilizing intravenous opioid PCA through a physiological negative feedback loop: the patient is likely to become too sedated to physically push the button to receive more opioid before reaching a critical point of severe respiratory depression.[17] Patient-controlled intravenous ketamine administration has been used as an alternative to opioids in the deployed setting, where the risks associated with potential respiratory depression in an austere environment are considered significant. Ketamine has a long established history of providing profound analgesia while maintaining spontaneous respiration. The most feared side effect of ketamine, especially in the combat-wounded population, is a negative hallucinogenic psychotropic effect. At low doses (10–20 mg/h basal infusion or 5–10-mg bolus with a 10-min lockout) however, this effect is typically not a problem.

Both intravenous and perineural infusion systems have the option to administer medication continuously, as a bolus, or a combination of the two. These devices can be broadly divided into those that generate the requisite infusion pressure by an electrically powered mechanism and those that power the infusion by elastomeric forces.

Electrically powered infusion devices may be driven by roller pumps or mechanical screws. Elastomeric pumps generate a pressure for administration through an elastic layer within the pump. When filled, the distension of the elastomeric layer delivers a driving pressure, and the rate of infusion is controlled by a temperature-sensitive flow restrictor downstream (solution viscosity varies with temperature, resulting in faster or slower flow rates).[18] Each system comes with unique advantages and disadvantages when used in the deployed environment (Figure 48-3).

Elastomeric infusion devices have a number of advantages when compared to electromedical equipment. They are single-use, disposable items with no requirement for servicing.[19] No external or battery power source is required, and when the patient transits through different stages of the medical evacuation chain, there is no need to change the devices, which may be assigned to a fixed location. There are also several disadvantages to elastomeric devices in the deployed environment. They typically are not reprogrammable and therefore are somewhat limited in mission capability. In addition, the pumps are sensitive to pressure differences in aircraft, which is not the case for electronic pumps.[20] Flexibility in bolus functions is also limited.[21]

Electronic devices, however, typically have incorporated pressure sensors, detecting when flow rates fall or stop, as well as a memory function, allowing information such as volumes administered and number of user interactions to be obtained from the pump.[22] This latter capability is particularly useful for acute pain issues, such as determining patient analgesic requirements. Each type of device has potential benefits, and choice should reflect both the clinical and military environment of the deployed operation while meeting regulatory body guidance and maintaining patient safety.

Medication delivery systems for local anesthetics and opioids remain an area of ongoing research and clinical development. Transdermal delivery systems utilizing the process of iontophoresis may provide a new route for opioid administration in the deployed environment, and liposomal encapsulation of local anesthetics may obviate the need for catheter infusions by significantly increasing an agent's duration of action. Potential benefits of new and novel routes of drug delivery may include eliminating the need for intravenous access, decreasing medication errors, and eliminating the risk of PCA programming errors.

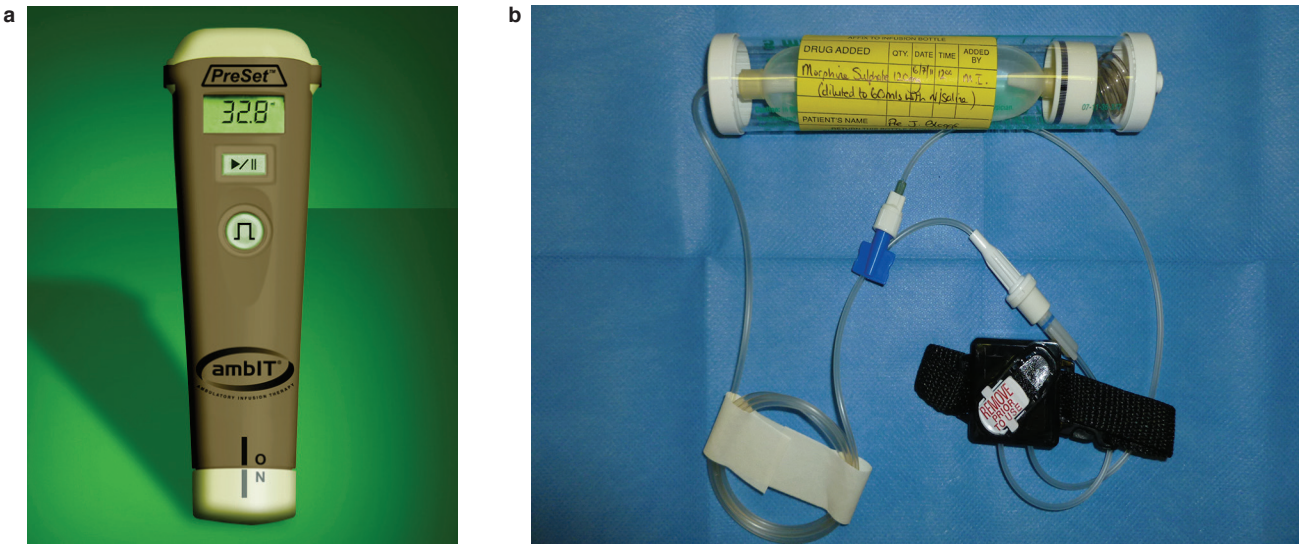

Figure 48-3. (a). ambIT electronic infusion pump (Summit Medical Products, Inc, Salt Lake City, UT); used with permission. **(b)** On-Q elastaomeric infusion device. Copyright B Braun Melsungen AG (used with permission).

SUMMARY

In most major catastrophic events, be it war or natural disaster, human innovation and medical necessity combine to promote novel, adaptable medical care to maximize casualty survival. Advanced analgesic techniques have been adapted and assimilated, becoming a fundamental part of patient management in the battlefield. By necessity there is an associated requirement for specialist equipment, both robust and suitable for the austere military environment. As we continue to improve the means by which we provide care for wounded soldiers, we will continue to increase their survival and improve their rehabilitation.

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Chapter 49

RESUSCITATION GUIDELINES

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INTRODUCTION

Cardiorespiratory arrest following trauma occurs in 1% to 4% of patients transported to major civilian trauma centers, where it is associated with a very poor overall prognosis.[1–4] Resuscitation from cardiorespiratory arrest in the military setting presents a unique challenge, with a number of important differences from civilian practice. The military population suffers a high incidence of blast and penetrating trauma as the cause of arrest,[5] and care is often delivered in a range of hostile environments. The military setting may also have significant constraints on medical resources, limiting the extent of available treatment. In enduring operations, however, resources may sometimes exceed those available to the civilian sector.

Traumatic cardiorespiratory arrest (TCRA), defined as the loss of central pulses and respiratory effort following trauma, represents the final common pathway before death due to exsanguination, pneumothorax, cardiac injury, brain injury, or asphyxia.[6] In the case of exsanguination, a period of profound hypotension with nonpalpable pulses may precede cardiac standstill, although these two phases may be indistinguishable on initial examination.

There is a lack of robust evidence for the optimal management of TCRA, in both civilian and military settings. In the absence of widely accepted, evidence-based guidelines, military practice has been guided by a combination of generic guidelines for cardiac arrest, limited civilian guidelines on prehospital resuscitation, and guidelines for resuscitative thoracotomy. A recent observational study from a United Kingdom (UK) field hospital in Afghanistan has provided some additional evidence on predictors of survivability following TCRA.[7] Although survival is rare for these patients, as in civilian practice, good outcomes can be achieved with timely, appropriate interventions, but these require access to significant resources. This chapter will review the evidence for current guidelines and, in the context of experience from current conflicts, suggest modifications to these guidelines for military TCRA.

EXISTING GUIDELINES

Cardiac Arrest Guidelines

Currently, the only internationally recognized guidelines for the treatment of cardiac arrest are those produced by the European Resuscitation Council and adopted by both the Resuscitation Council (UK) and the American Heart Association.[6] Although the core adult resuscitation algorithm (Figure 49-1) offers simplicity and standardization, it was produced principally for cardiac arrest from primary cardiac causes, not from trauma (the pediatric algorithm is also shown, in Figure 49-2, for reference). Consequently, the guidelines are mostly based on evidence from nontrauma resuscitation and place the greatest emphasis on early defibrillation for ventricular fibrillation or pulseless ventricular tachycardia, cardiac rhythms rarely encountered in TCRA.[7]

The 2010 update of the European Resuscitation Council guidelines does, however, include a useful discussion of TCRA in the "Special Circumstances" section.[8] Here, the authors recognize the lack of robust evidence for the treatment of TCRA and make a number of recommendations relevant to military resuscitation, including:

- Undertake only life-saving interventions at the scene, with immediate transfer to the nearest hospital.
- Do not delay transfer for unproven interventions such as spinal immobilization.
- Standard cardiopulmonary resuscitation (CPR) should not delay the treatment of reversible causes.
- Chest compressions are still considered "the standard of care in cardiac arrest irrespective of aetiology," but they are of limited value in hypovolemia and cardiac tamponade.
- Pericardiocentesis is not recommended, because it is usually ineffective and delays thoracotomy.
- For tension pneumothorax, anterior or lateral thoracostomy is more effective than needle decompression and quicker than inserting a chest tube.
- For assisted ventilation, it may be useful to limit tidal volumes and respiratory rate in order to reduce the effect of raised intrathoracic pressure on venous return.
- The role of vasopressors in TCRA remains unclear.

Trauma Resuscitation Guidelines

A number of sources of guidelines are available to military clinical staff; however, it must be recognized that even the currently published military

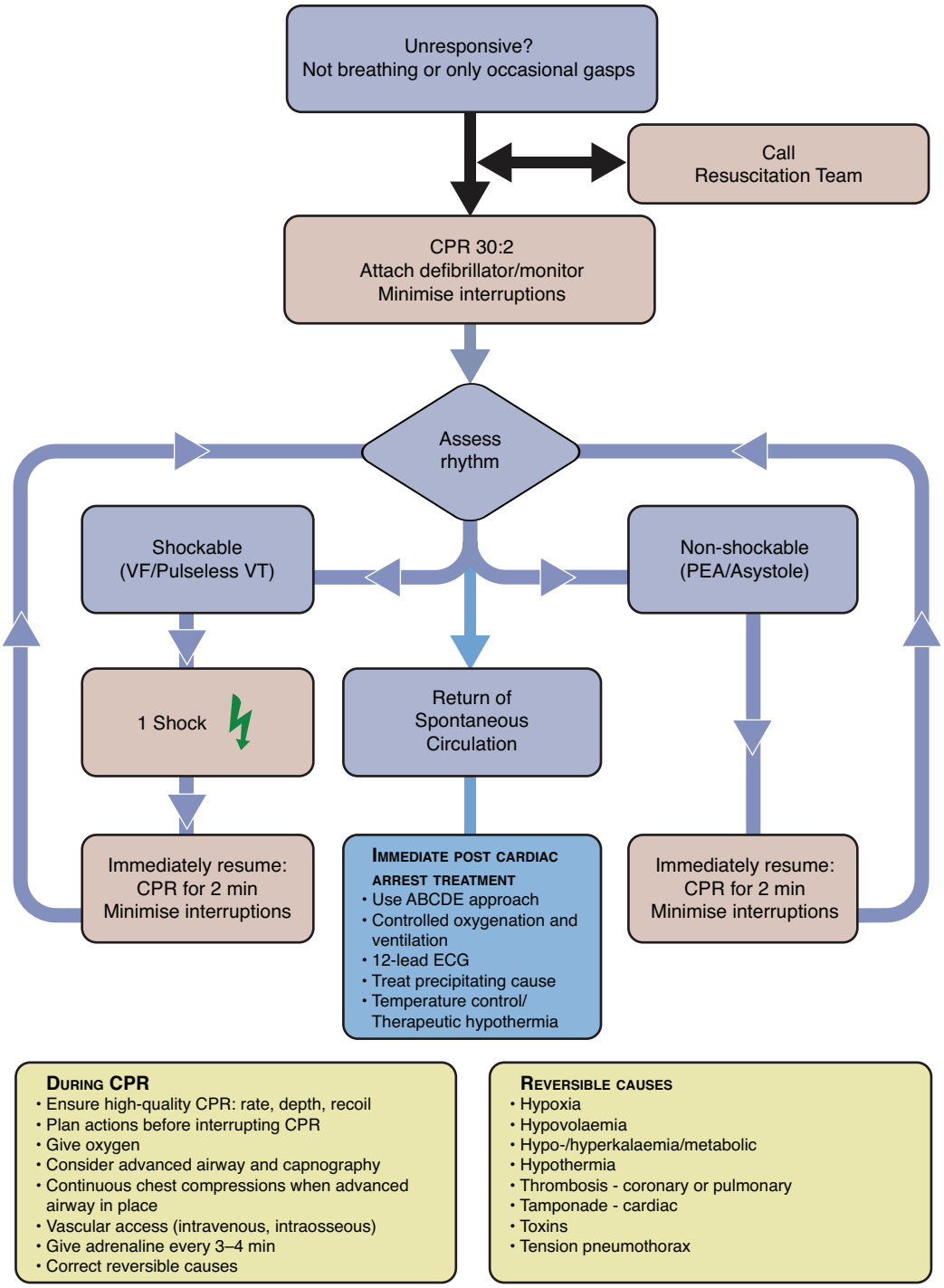

Figure 49-1. Adult Advanced Life Support algorithm. Copyright European Resuscitation Council—www.erc.edu—2012/003.
ABCDE: airway, breathing, circulation, disability, exposure
CPR: cardiopulmonary resuscitation
ECG: electrocardiogram
VF: ventricular fibrillation
VT: ventricular tachycardia
PEA: pulseless electrical activity
Reproduced with permission from: Nolan JP, Soar J, Zideman DA, et al. European Resuscitation Council Guidelines for Resuscitation 2010 Section 1. Executive summary. *Resuscitation*. 2010;81:1232.

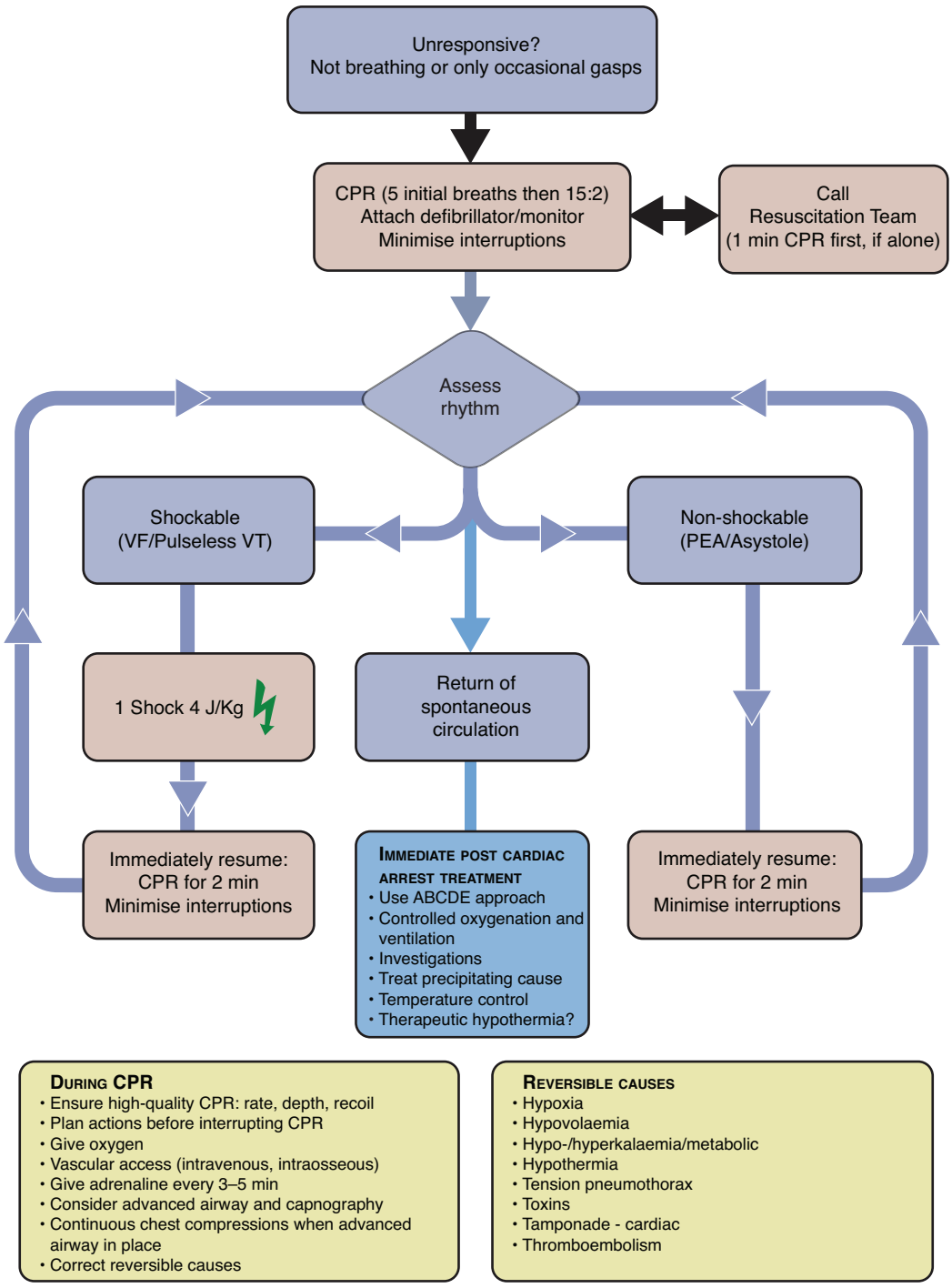

Figure 49-2. Pediatric Advanced Life Support algorithm. Copyright European Resuscitation Council—www.erc.edu—2012/003.
ABCDE: airway, breathing, circulation, disability, exposure
CPR: cardiopulmonary resuscitation
ECG: electrocardiogram
VF: ventricular fibrillation
VT: ventricular tachycardia
PEA: pulseless electrical activity
Reproduced with permission from: Nolan JP, Soar J, Zideman DA, et al. European Resuscitation Council Guidelines for Resuscitation 2010 Section 1. Executive summary. *Resuscitation*. 2010;81:1249.

guidance is based on civilian data. Sources include the following:

The American College of Surgeons' Advanced Trauma Life Support (ATLS)

ATLS is a well-recognized approach to trauma resuscitation, aimed at members of a civilian emergency department trauma team.[9] Although the course provides a widely accepted paradigm for managing trauma patients, there is little specific guidance on the management of TCRA.

Battlefield Advanced Trauma Life Support (BATLS)

BATLS is a course designed by the UK Defence Medical Services (UK DMS) to provide standardized training in emergency trauma care for military practitioners.[10] BATLS adapts civilian practice for the military setting by applying emerging medical evidence and practical military experience from recent conflicts. A key feature of BATLS is the military paradigm of <C>ABC, where catastrophic external hemorrhage, represented by <C>, is dealt with before attending to the airway, breathing, and circulation. For military TCRA, the main advice offered by BATLS concerns the appropriateness of attempting resuscitation for these patients. The 2008 BATLS manual makes the following recommendations:

- It is appropriate to start resuscitation for witnessed TCRA, particularly when hypovolemia is the underpinning cause and volume is rapidly restored.
- It is not appropriate to start resuscitation for TCRA in the case of blunt trauma with absent vital signs at the scene of injury.
- It is not appropriate to start resuscitation for TCRA in the case of penetrating trauma with absent vital signs for 5 minutes at the scene of injury.

Clinical Guidelines for Operations (GCOs)

CGOs, published by the UK DMS, represent current policy for emergency medical care in the military setting.[11] The main areas of TCRA guidance within CGOs relate to triage (Figure 49-3) and resuscitative thoracotomy (Figure 49-4). According to the CGOs, the triage status of a military TCRA victim varies according to the presence of effective enemy fire: if enemy fire is present the casualty is considered dead, but if enemy fire is absent it may be appropriate to commence basic life support.

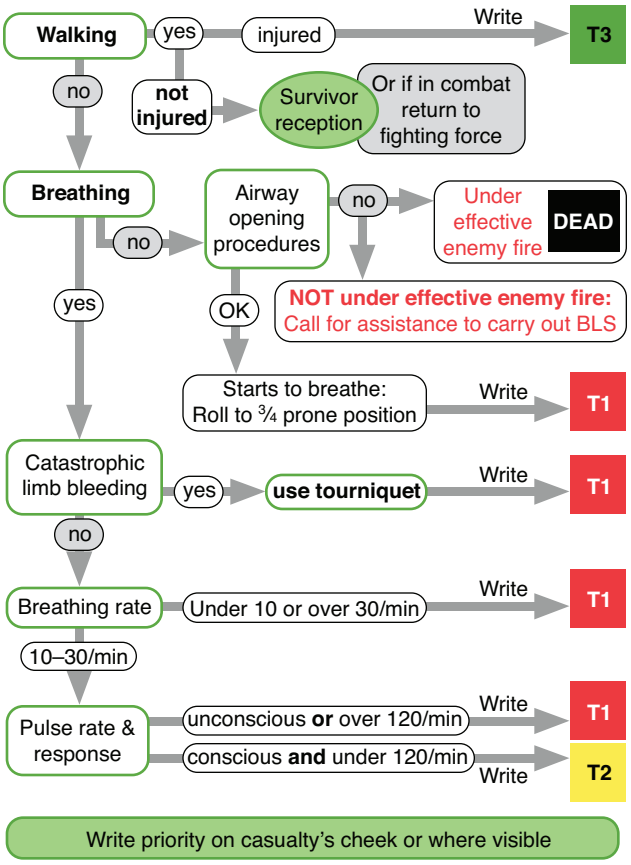

Figure 49-3. Triage flowchart for incident management. The NATO triage categories of T1, T2, and T3 indicate treatment priorities as follows: T1: immediate, T2: urgent, and T3: delayed.
Reproduced from: *Clinical Guidelines for Operations*. London, England: Defence Concepts and Doctrine Centre, 2008: Section 2: 51. Joint Doctrine Publication 4-03.1.

Guidance within CGOs on the use of resuscitative thoracotomy reflects recommendations made by the American College of Surgeons Committee on Trauma in 2001.[12] The committee states that for TCRA due to blunt injury, thoracotomy may be considered only if signs of life were present on arrival in the emergency department. For TCRA due to penetrating trauma, thoracotomy may be considered if signs of life were present until 5 minutes before presentation. Again, this guidance is based on civilian data and guidelines.

Prehospital Resuscitation Guidelines

In 2003 the US National Association of EMS Physicians and the American College of Surgeons published guidelines on withholding resuscitation for prehospital

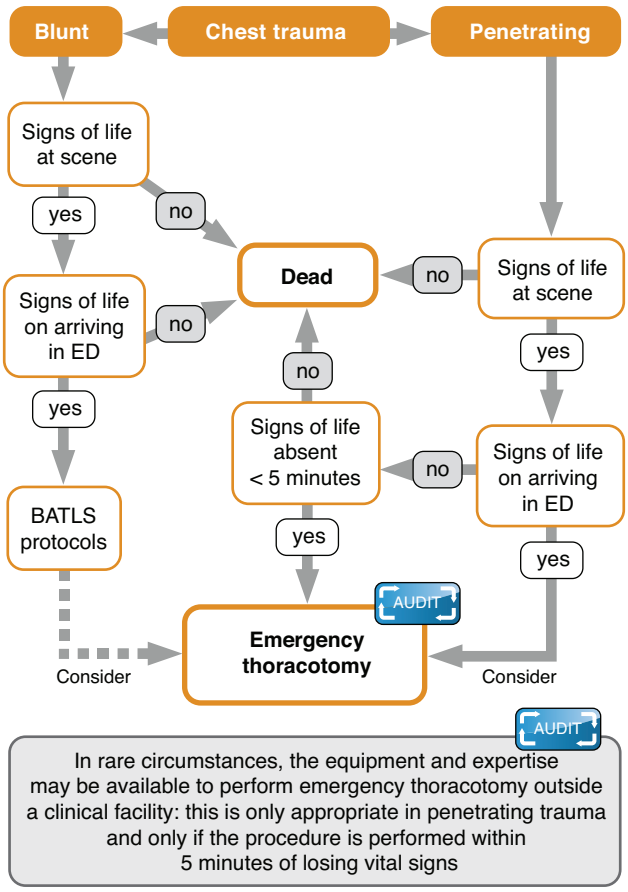

Figure 49-4. Emergency thoracotomy treatment guidelines flowchart.
ED: emergency department
Reproduced from: *Clinical Guidelines for Operations*. London, England: Defence Concepts and Doctrine Centre, 2008: Section 3: 16. Joint Doctrine Publication 4-03.1.

TCRA.[4] Based on a number of observational studies, the guidelines were aimed at limiting futile care and recommended that treatment should be withheld or withdrawn in the case of:

- blunt trauma with asystole at the scene,
- penetrating trauma with asystole and no signs of life at the scene,
- 15 minutes of unsuccessful CPR, or
- anticipated transfer time of more than 15 minutes.

The acceptance of these guidelines has been limited, however, by subsequent reports of a number of TCRA victims who survived despite meeting these criteria for withdrawal of care. Therefore, while these guidelines offer useful information on factors associated with poor outcome, it is not recommended that they be strictly applied in all cases of TCRA, especially not military TCRA.

EVIDENCE FOR MILITARY TRAUMATIC CARDIORESPIRATORY ARREST

Although there is little published evidence for the management of military TCRA, a recent observational study from a UK field hospital in Afghanistan provided some evidence on indicators of survivability.[7] Over a period of 6 months, the hospital received 52 adult patients who suffered TCRA beginning either in the field (29 patients), during helicopter transport to hospital (16 patients), or in the hospital (7 patients). Four of these patients survived to Role 4 hospital discharge and were neurologically intact.

In contrast to civilian studies of TCRA, these patients were principally injured by blast and fragmentation injuries, and the cause of arrest in over 80% of patients was exsanguination—a mechanism normally associated with very poor outcomes in civilian studies. Despite the prevalence of this cause of arrest, the overall rate of survival, at 8%, was broadly in keeping with civilian studies.

Comparing survivors with nonsurvivors, the following factors were identified as potential predictors of survival:

- **Arrest beginning after transfer to hospital.** Three of the four survivors arrested in hospital and one arrested during helicopter transfer. Although 5 of 29 patients who arrested on the ground achieved return of spontaneous circulation (ROSC), none of these patients survived to hospital discharge.
- **Electrical activity on electrocardiogram (ECG) during arrest.** All four survivors had some electrical activity on ECG during arrest—three were in sinus-based rhythms and one had an agonal rhythm. Only 1 of the 29 patients with asystole achieved ROSC, and this person did not survive to discharge.
- **Cardiac movement on ultrasound during arrest.** When performed in the emergency department as part of the initial assessment of a TCRA victim, brief ultrasound examination of the heart appeared to be a useful tool in assessing salvageability. All six of the patients with cardiac activity on ultrasound during

arrest exhibited ROSC, with two surviving to discharge. In the other patients ultrasound was not performed because equipment or skilled ultrasound practitioners were unavailable. Conversely, ROSC was not achieved in any of the 18 patients without cardiac activity on ultrasound.

Importantly, these results were achieved in a well-established field hospital with an advanced prehospital service, rapid access to blood products and emergency surgery, and a well-organized critical care air-evacuation service. In total, over 900 units of packed red blood cells and fresh frozen plasma were used on these 52 patients, with each survivor requiring an average of 47 units of blood and 45 units of plasma.

Resuscitative thoracotomy may also have contributed to the survival of these patients. All four of the survivors received a thoracotomy, during which a number of important interventions were made. In addition to open-chest CPR, pericardial tamponade was released in one patient, and at least two other survivors were treated with direct compression of the descending thoracic aorta, while distal control of hemorrhage was achieved.

AREAS OF CONTROVERSY

The traditional paradigms of resuscitation from cardiac arrest may be challenged in a number of controversial areas, particularly in the context of TCRA. Three of these areas are discussed below.

Epinephrine and Other Vasopressors

The use of epinephrine (adrenaline) is well established in the European Resuscitation Council guidelines, where a bolus dose is recommended every 3 to 5 minutes. However, the guidelines themselves state that this suggestion is not supported by any robust human studies, and that although epinephrine may increase coronary and cerebral perfusion pressure during CPR, it may also impair microcirculation and contribute to post–cardiac-arrest myocardial dysfunction.[6] Recent work has shown a deleterious effect, with the use of vasoconstrictors being associated with increased mortality in trauma patients.[13] In military TCRA, the priority must be aggressive volume resuscitation with blood and blood products. Although epinephrine and other vasopressors may have a role, their use should not be a priority and excessive doses should be avoided.

Intubation, Ventilation, and Chest Compressions

Securing a definitive airway and performing intermittent positive-pressure ventilation (IPPV) and external chest compressions have been considered fundamental to resuscitation from cardiac arrest. In TCRA, however, these procedures may prove detrimental if applied incorrectly. Compressing an empty heart (in the case of hypovolemia) is ineffective and may interfere with other, more useful procedures.[8] In addition, IPPV may increase intrathoracic pressure, further reducing venous return to the heart.[14] A pragmatic approach for military TCRA is emphasizing the restoration of circulating blood volume ahead of chest compressions and limiting the frequency and tidal volume of invasive ventilation until circulation is restored. Early thoracotomy may assist in this context by minimizing the effect of IPPV on intrathoracic pressure and hence venous return, as well as allowing the correction of reversible causes (eg, tamponade) and allowing internal cardiac massage to be performed.

Capnometry as a Guide to Resuscitation

In addition to its role in guiding IPPV and preventing hyperventilation, capnometry may also prove useful as evidence of residual cardiac output in an apparently pulseless patient. This is the subject of current research by the UK DMS, with the aim of providing a guide to the salvageability of future TCRA patients.

SUMMARY

A summary of the treatment priorities for the military TCRA patient is shown in Figure 49-5. Key points are as follows:

- Although survival from military TCRA is rare, good outcomes are possible, especially in the context of in-flight or in-hospital arrest when there is organized ECG activity, cardiac movement on ultrasound examination, and a persisting end-tidal CO_2 trace.
- Resuscitation from military TCRA demands a large amount of healthcare resources, including blood products and rapid access to emergency surgery.

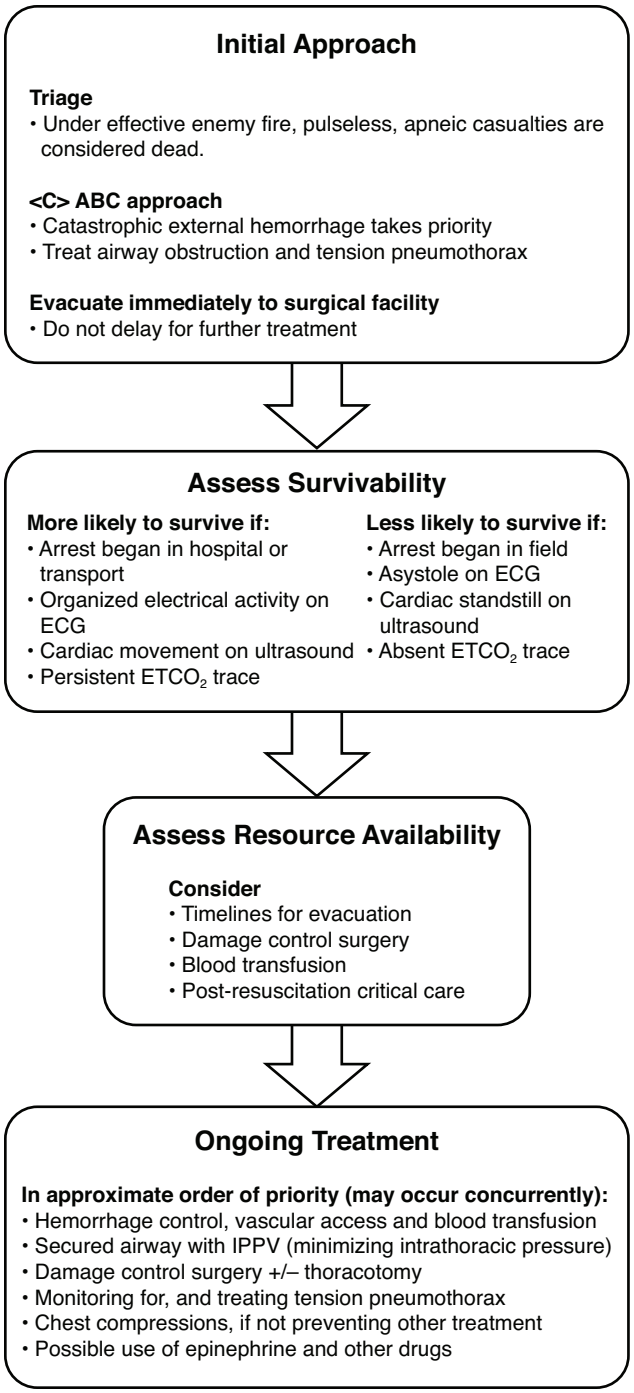

Figure 49-5. Summary of management of traumatic cardiorespiratory arrest.
ECG: electrocardiogram
$ETCO_2$: end-tidal carbon dioxide
IPPV: intermittent positive-pressure ventilation

- Restoration of circulating volume, immediate transfer for surgery, and the correction of reversible causes (including catastrophic hemorrhage, tension pneumothorax, and cardiac tamponade) should take priority over the use of vasopressors and possibly also external chest compressions. Resuscitative thoracotomy may also be emerging as a recommended approach in this situation.

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Chapter 50

THE HOME BASE: LANDSTUHL, GERMANY, AND HOSPITALS IN THE CONTINENTAL UNITED STATES

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INTRODUCTION

Before leaving the combat theater, injured soldiers are surgically stabilized, although considerable surgery usually remains to be done. In the typical combat-wounded veteran, most restorative surgical procedures are performed at Role 4 facilities. The network of US military hospitals in the continental United States, as well as in Landstuhl, Germany, provides a robust healthcare system capable of definitive care for all of the combat casualties who survive to evacuation from the combat theater. This system is also supported by a small number of civilian or Veterans Affairs (VA) medical centers that provide unique or specialty care for some soldiers (based on need).

LANDSTUHL REGIONAL MEDICAL CENTER

History

Located in southwestern Germany in the state of Rheinland-Pfalz, Landstuhl Regional Medical Center (LRMC) is the largest American military medical center outside the United States. The US Army has maintained a hospital presence in the town of Landstuhl since November 28, 1951, when the 320th General Hospital took over operational control from an existing German military hospital. Soon thereafter, construction began on a new hospital, and the 320th General Hospital moved patients into the new facility on March 9, 1953. This hospital has been continuously operational since that day, although the name has changed three times. It was renamed the 2nd General Hospital in 1954 and Landstuhl Army Medical Center in 1994. In November 2003, it was redesignated Landstuhl Regional Medical Center.

Throughout its history, LRMC has been a key medical resource for the European theater and the Middle East. Among the service men and women treated at Landstuhl were the Marines injured during the 1980 hostage rescue attempt in Iran and in the 1983 Beirut barracks bombing, as well as 500 casualties of the 1988 disaster at the Ramstein Air Show.[1]

In the post-9/11 era, the bed capacity at LRMC was expanded by almost 50%. The greatest expansion was a tripling of the intensive care unit beds from 6 to 18, and an increase in the number of inpatient psychiatry beds, from 12 to 22. A smaller increase was seen in the number of medical-surgical beds (now 74). There are eight main operating rooms, two obstetric operating rooms, and two urologic operative procedure rooms. From January 1, 2004, through January 5, 2011, LRMC treated 64,892 patients, returning 20.9% back to duty within Central Command (CENTCOM).

Current Capabilities

LRMC's top priority, as published by the commander at the time of this writing, is casualty reception for wounded warriors from across the CENTCOM (which includes Iraq and Afghanistan), African Command (AFRICOM), and European Command (EUCOM) areas of responsibility. Since the Cold War ended, medical capabilities have been consolidated at LRMC because 23 other hospitals in Europe have closed, making LRMC the sole US tertiary referral facility for military forces, their families, and other beneficiaries in EUCOM, CENTCOM, and AFRICOM. EUCOM alone comprises 245,000 beneficiaries (Table 50-1). In addition to being a tertiary referral center, LRMC is a primary care facility serving 100,000 beneficiaries, with the remaining 145,000 EUCOM beneficiaries receiving primary care at outlying clinics. The specialties represented at LRMC are myriad. In addition to the surgical specialties listed in Exhibit 50-1, many medical specialties and ancillary services are offered, including addiction treatment, nutrition care, physiatry, physical and occupational therapy, and social work.

The specific capabilities of each of these services tend to be weighted toward military combat care

TABLE 50-1

A TYPICAL DAY AT LANDSTUHL REGIONAL MEDICAL CENTER (BASED ON AVERAGES FROM DECEMBER 2009 TO NOVEMBER 2010)

Admissions	25
Outpatient visits	2,908
Operating room cases	31
Intensive care unit census	9
Laboratory services	2,396
Radiology services	789
Births	3
Pharmacy products	1,297
Meals served	1,769

EXHIBIT 50-1

LANDSTUHL REGIONAL MEDICAL CENTER SURGICAL SPECIALTIES

- anesthesiology/pain clinic
- otolaryngology
- general surgery
- plastic surgery
- hand surgery
- podiatry
- neurosurgery
- spine surgery
- obstetrics/gynecology
- trauma
- ophthalmology
- urology
- oral surgery
- thoracic surgery
- orthopedics

needs to a greater degree than most civilian or stateside facilities of similar size. For example, the LRMC neurosurgical service is geared to provide state-of-the-art care for complex spine injuries and traumatic brain injuries, but is more limited in its ability to care for intracranial vascular cases and tumors due to imaging limitations and an absence of interventional neuroradiologists. Urgent cases that require such services are immediately transferred to local German hospitals with these capabilities.

The consultants at LRMC and stateside medical centers frequently interact with medical staff in the combat zone, either as part of a formal consultation program or an informal peer-to-peer communication. For instance, primary care physicians examining an uncommon skin lesion can take a digital photo and email it, along with a case description, to derm.consult@us.army.mil. A telemedicine administrator at Fort Sam Houston in San Antonio, Texas, screens the request and sends it to a dermatologist on call at LRMC or one of the stateside medical centers, who will then send recommendations back to the originating doctor within a few hours. This process allows many service men and women to avoid unnecessary travel within or from the combat theater,[2] and it is available for many other specialties. Modern communications also support peer-to-peer communication between surgeons in the field and LRMC receiving surgeons. This exchange has facilitated LRMC surgical teams in anticipating the logistical and medical needs for a patient before his or her arrival.

Specialty Services

Because penetrating and blast injuries to the eye are common in combat trauma, ophthalmologists have active roles in maintaining or restoring eyesight to injured service members. The ophthalmology service providers perform initial, mid-term, and long-term management of all anterior segment trauma, and mid-term and long-term management of vitreoretinal and orbital trauma; that is, they can perform initial globe repair, but subsequent vitreoretinal surgery is sent to a local hospital if urgent and deferred until arrival in the United States if not urgent. Routine nontrauma cases include all commonly performed refractive surgeries, cataract surgeries, strabismus, and oculoplastic procedures. The LRMC ophthalmology service typically receives one to eight telephone calls or emails daily from optometrists, primary care physicians, and physician assistants in the combat zone via an Army Medical Department telemedicine site. In addition, providers participate in a monthly teleconference with forward-deployed and stateside sites to discuss cases.

LRMC has dedicated an extracorporeal membrane oxygenation (ECMO) team to support cases of devastating lung injuries. The team consists of physicians, intensive care nurses, and respiratory technicians who have undergone specific ECMO training. Although the majority of ECMO cases have been initiated at LRMC, there have been several ECMO cannulations in the combat theater, with ECMO care continuing during evacuation to LRMC. The first ECMO cannulation in the combat theater was performed in Kandahar in October 2010. Regardless of where ECMO is initiated, the ECMO team cares for the injured service member until he or she arrives at University Hospital Regensburg, a German facility with significant ECMO expertise. Because of its role in the care of combat casualties, LRMC is also actively involved in the organ donation program with its European counterparts.[3]

The vast majority of US patients received from the combat theater spend only a short time at LRMC before continuing their journey to the United States. There are typically three scheduled air evacuation/critical care air transport team flights to the United States from Germany per week, and there is capability to "spin up" additional flights, if necessary. Thus, the average US combat surgical patient receives one to two surgeries at LRMC. The destination of patients departing from LRMC is determined by several factors, such as type of injury (eg, all major burns go to the burn center in San Antonio; see Institute of Surgical Research, below), location of the service member's unit, the location of the service member's family, and potentially by the available bed space at stateside hospitals.

MILITARY HOSPITALS IN THE UNITED STATES

Military Medical Centers

Military medical treatment facilities (Exhibit 50-2) are any medical care sites, including clinics, hospitals, and medical centers. Military medical centers (MEDCENs) are Army medical facilities that offer tertiary care (sophisticated diagnosis/treatment of any ailment) as well as primary and secondary care. Each MEDCEN has a hospital and other services (preventive medicine, blood bank, etc). MEDCEN hospitals are the largest MTFs, have the most sophisticated equipment and most specialized staffs, and offer the widest arrays of specialty care in the military health system. All MEDCENs offer graduate medical education (internships, residencies) for physicians. Army hospitals that offer complex, resource-intensive secondary care (eg, inpatient care, surgery under general anesthesia) but do not provide all the services required for a MEDCEN are called Army community hospitals (ACHs). ACHs also deliver primary care at outpatient clinics inside and outside the hospital (eg, at troop clinics and outlying clinics at small posts). A facility that offers all ACH services except inpatient care is called an Army health center. A clinic is defined as an outpatient facility offering primary care or simple specialty care (ie, routine exams, tests, and treatments supervised by a larger entity such as a Medical Department activity (see Military Medical Activities, below). A clinic may be a stand-alone site (eg, an Army health clinic) or part of a major health facility (family practice clinic, pediatric clinic, and so forth, within a hospital).

Combat casualty care is delivered in a variety of US military hospitals; the major centers are the Walter Reed National Military Medical Center (the old Walter Reed Army Medical Center combined with the National Naval Medical Center); San Antonio Military Medical Center (Brooke Army Medical Center and Wilford Hall Medical Center); and San Diego Naval Medical Center. These MEDCENS provide initial treatment for the majority of combat casualties. Injuries requiring less complex care may be treated in other MEDCENS closer to the service member's duty station or home.

The individual services have unique programs aimed at providing the highest quality of care to the injured warrior. The Army Wounded Warrior Program (AW2) is the official US Army program that assists and advocates for severely wounded, ill, and injured soldiers, veterans, and their families, wherever they are located and regardless of military status. Warriors in transition who qualify for AW2 are assigned to the program as soon as possible after arriving at the Warrior Transition Unit. AW2 supports these soldiers and

EXHIBIT 50-2

US MILITARY MEDICAL TREATMENT FACILITIES

Army Medical Treatment Facilities

- Madigan Army Medical Center, Joint Base Lewis-McChord, WA
- William Beaumont Army Medical Center, Fort Bliss, TX
- Brooke Army Medical Center, Fort Sam Houston, TX
- Carl R. Darnall Army Medical Center, Fort Hood, TX
- Dwight D. Eisenhower Army Medical Center, Fort Gordon, GA
- Walter Reed National Military Medical Center, MD
- Womack Army Medical Center, Fort Bragg, NC

Navy Medical Treatment Facilities

- Naval Hospital, Beaufort, SC
- Naval Hospital, Bremerton, WA
- Naval Hospital, Camp Lejeune, NC
- Naval Hospital, Camp Pendleton, CA
- Naval Hospital, Jacksonville, FL
- Naval Hospital, Lemoore, CA
- Naval Hospital, Oak Harbor, WA
- Naval Hospital, Pensacola, FL
- Naval Hospital, Twenty-nine Palms, CA
- National Naval Medical Center, MD
- Naval Medical Center, Portsmouth, VA
- Naval Medical Center, San Diego, CA

Air Force Medical Treatment Facilities

- 3rd Medical Group, Elmendorf Air Force Base, AK
- 6th Medical Group, MacDill Air Force Base, FL
- 14th Medical Group, Columbus Air Force Base, MS
- 20th Medical Group, Shaw Air Force Base, SC
- 56th Medical Group, Luke Air Force Base, AZ
- 47th Medical Group, Laughlin Air Force Base, TX
- 75th Medical Group, Hill Air Force Base, UT
- 96th Medical Group, Eglin Air Force Base, FL
- Keesler Medical Center, Keesler Air Force Base, MS
- Wilford Hall Medical Center, Lackland Air Force Base, TX
- Wright-Patterson Medical Center, Wright-Patterson Air Force Base, OH

their families throughout their recovery and transition, even into veteran status. This program, through the local support of AW2 advocates, strives to foster the independence of warriors in transition.

The Marine Wounded Warrior Regiments (WWRs) are strategically placed assets that have thus far contacted or provided support in some degree to nearly 25,000 marines, whether they are assigned to the regiment or returned to their parent units. "Once a Marine, always a Marine" is an enduring commitment the WWR upholds. Whether marines are wounded in combat, fall ill, or are injured in the line of duty, the WWR serves the total force: active duty, reserve, retired, and veteran marines. The regiment maintains administrative and operational control of two wounded warrior battalions located at Camp Pendleton, California, and Camp Lejeune, North Carolina. These battalions have detachments located at medical treatment facilities and at VA polytrauma rehabilitation centers. The span of the regiment extends across 23 locations from Landstuhl, Germany, to Okinawa, Japan, and throughout the continental United States. The regiment's nerve center is the Wounded Warrior Operations Center (located at Pendleton and Marine Corps Base Quantico, VA), which serves as the central point of contact for all nonmedical care management issues.

Institute of Surgical Research

The US Army Institute of Surgical Research, part of the US Army Medical Research and Materiel Command, is collocated with Brooke Army Medical Center and dedicated to both laboratory and clinical trauma research. Its mission is to provide requirements-driven combat casualty care and medical solutions and products for injured soldiers, from self-aid through definitive care across the full spectrum of military operations. The Institute also provides state-of-the-art trauma, burn, and critical care to Department of Defense beneficiaries around the world and civilians in the south Texas trauma region as well as burn special medical augmentation response teams.

Military Medical Activities

The US military has made tremendous progress in the rehabilitative care of injured combatants. The medical personnel of the combined services are doing outstanding work to develop and implement the Military Health System (MHS) rehabilitative programs necessary to return severely injured service members to duty or to a productive civilian life. Severely injured service members often require prolonged treatment, time to heal, and rehabilitative care before a decision can be rendered about their ability to remain on active duty. The MHS is meeting this challenge by improving the coordination of healthcare for service members and veterans with the Veterans Health Administration. MHS is dedicated to ensuring that service members are provided outstanding clinical care and streamlined administrative processes to return them to duty status or to transition them from MHS care to the VA healthcare system in an effective and timely manner. Five MHS Specialty Centers of Excellence, listed in Exhibit 50-3, provide specialized care with unique capabilities in their assigned sphere.[4]

Veterans Affairs Medical Centers

VA medical centers represent the most comprehensive hospitals within the Veterans Health Administration network. There are 152 medical centers, located across the 50 states, the District of Columbia, and Puerto Rico, as well as a multitude of smaller facilities such as community-based outpatient clinics and living centers. As detailed on the VA website (http://www.va.gov/health/MedicalCenters.asp), VA medical centers provide all of the traditional hospital-based services including surgery, critical care, physical therapy, and mental health. Many centers provide additional specialty services such as neurology, prosthetics, and vision care. In some VA medical centers, plastic surgery and organ transplantation are offered.

Together, these hospitals and clinics are designed to provide continuity of treatment for service members after their departure from active duty service. The VA has established care management teams consisting of

EXHIBIT 50-3

US MILITARY HEALTH SYSTEM SPECIALTY CENTERS OF EXCELLENCE

- Walter Reed National Military Medical Center Amputee Care Center and Gait Laboratory
- National Naval Medical Center's Traumatic Stress and Brain Injury Program
- Center for the Intrepid and Brooke Army Medical Center Burn Center at Joint Base San Antonio, Texas
- Naval Medical Center San Diego Comprehensive Combat Casualty Care Center
- Department of Defense/Veterans Affairs Defense and Veterans Brain Injury Center (multiple sites)

case managers and patient advocates to help newer veterans initiate and coordinate their care upon exiting active duty and when relocating from one geographic region to another. In recent years, the VA has taken numerous steps to increase dissemination of information to beneficiaries, with many VA medical centers using social media such as Facebook and Twitter to help the beneficiaries stay informed.

SUMMARY

The military and VA hospitals comprise a geographically vast and technically capable network reaching from Germany to the United States. Every conceivable medical specialty and all ancillary services are contained within the system. The temporal span of interaction with the patient lasts decades—from days after injury through the rest of the service member's military career and beyond.

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Chapter 51

THE HOME BASE: QUEEN ELIZABETH HOSPITAL BIRMINGHAM AND OTHER HOSPITALS IN THE UNITED KINGDOM

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INTRODUCTION

The essential challenge for clinicians at Role 4 is to integrate with and extend the care given in the operational theater. In the United Kingdom (UK), responsibility for continuity of acute care rests primarily with the University Hospitals Birmingham National Health Service (NHS) Foundation Trust, a comprehensive publicly funded acute care provider as defined within NHS legislation. Military patients are admitted to the Trust's teaching hospital, the Queen Elizabeth Hospital Birmingham (QEHB) (Exhibit 51-1), which hosts the Royal Centre for Defence Medicine (RCDM), whose primary role is to support deployed operations. At any one time, military patients constitute only 1% of total patient numbers, but the resource-intensive nature of their injuries imposes a significant surgical workload that necessitates considerable planning to ensure their clinical care while avoiding interruption to the hospital's usual NHS activity. The logistical requirements include a system of command and communication that cascades from the care given at Roles 1 through 3 to the receiving teams at Role 4 (Exhibit 51-2).

PATIENT ADMISSIONS AND DISPOSITION

Injured military personnel are transported by aeromedical evacuation teams or, in the case of the critically ill, by specially trained and tasked critical care air support teams. Repatriation of the casualty to Birmingham can be expected within 24 to 48 hours of injury. Given this condensed timeline, seriously injured personnel are often still in the "damage control phase" of their surgical management.[1]

On admission to the QEHB, ventilated patients will be transferred to critical care by the critical care air support team staff, where an immediate assessment of their injuries and physiological status is undertaken before continued stabilization or immediate surgery. In many cases this will be the "second look" following resuscitative surgery at Role 3.[2] Such surgery is limited by a patient's physical status and is often the first of repeated visits to the operating room until the patient's clinical condition improves enough to consider definitive surgery for function restoration.

The signature injury of the conflict in Afghanistan has been blast injury from improvised explosive devices. The secure medical signals received at Role 4 will

EXHIBIT 51-1

THE QUEEN ELIZABETH HOSPITAL BIRMINGHAM

- Commissioned in June 2010.
- Contains 1,213 beds.
- Serves as a tertiary referral teaching hospital undergoing accreditation as a Role 1 trauma center.
- The department of anesthesia has 70 National Health Service consultant staff, 24 of whom are also part of the cadre of 32 intensivists.[1]
- Has a total of 100 intensive care beds; 25 each are designated for trauma, neurological sciences, cardiothoracics, and general critical care.[2]
- Has a designated military ward with 32 beds, and 40% are single patient rooms.
- Staff includes embedded military clinicians from the Royal Centre for Defence Medicine, including four anesthetists, four orthopedic surgeons, three plastic surgeons, and one general surgeon.
- The Royal Centre for Defence Medicine clinicians are supported by military academic departments of anesthesia and critical care, surgery, and emergency medicine.

(1) Hodgetts TJ, Mahoney PF, Kirkman E. Damage control resuscitation. *J R Army Med Corps.* 2007;153:299–300. (2) Jones CP, Chinery JP, England K, Mahoney P. Critical care at role 4. *J R Army Med Corps.* 2010;156(Suppl 1):S342–348.

EXHIBIT 51-2

ESSENTIAL REQUIREMENTS FOR RECEIVING CASUALTIES AT ROLE 4

- Secure signals detailing the numbers of expected casualties and nature of injuries are necessary.
- Trauma coordinators should ensure that key clinical personnel are aware of incoming casualties and operating theater space and organize critical care beds as necessary.
- Sufficient and appropriately trained staff must be available to receive patients at ward and critical care locations.
- Dedicated laboratory support should also be available, particularly to maintain blood and blood product supplies for critical care patients.

EXHIBIT 51-3

ANESTHETIC CONSIDERATIONS FOR THE CRITICALLY INJURED MILITARY PATIENT

- Continue anesthesia as part of the damage control philosophy, and pay rigorous attention to the physiological control of tissue oxygenation to avoid or correct the lethal triad of coagulation, hypothermia, and lactic acidosis.[1]
- Consider the evolution of the patient's wounds and physiology during transfer from Role 3. Upon arrival at the Queen Elizabeth Hospital Birmingham, patients' pathophysiology is frequently complicated by the systemic inflammatory response syndrome.[2]
- Use blood/fresh frozen plasma and other specialized hemostatic therapies as practiced at Role 3. Hemostatic resuscitation is guided by standard laboratory tests plus near patient assessment with thromboelastometry.[3]
- Restrict crystalloid use (unless specific indications exist) to allow better hemostatic resuscitation and decrease edema.
- Be aware of evolving blast lung injury and use appropriate ventilator strategies.[4]
- Change Role 3 resuscitative central lines and ensure nasogastric feeding tube in situ.

(1) Wood PR, Haldane AG, Plimmer SE. Anaesthesia at role 4. *J R Army Med Corps*. 2010;156(Suppl 1):S308–310. (2) Jones CP, Chinery JP, England K, Mahoney P. Critical care at role 4. *J R Army Med Corps*. 2010;156(Suppl 1):S342–348. (3) Midwinter MJ, Woolley T. Resuscitation and coagulation in the severely injured trauma patient. *Philos Trans R Soc Lond B Biol Sci*. 2011;366:192–203. (4) Brower RG, Morris A, MacIntyre N, et al. Higher versus lower positive end expiratory pressures in patients with the acute respiratory distress syndrome. *N Eng J Med*. 2004;351:327–336.

EXHIBIT 51-4

CLINICAL AND ANESTHETIC CONSIDERATIONS FOR PATIENTS ADMITTED TO THE MILITARY TRAUMA WARD

- The nature of the wounds may not be entirely clear from the signals sent from Role 3, so patients receive a surgical assessment on admission.
- Patients may require preoperative intravenous fluids following extended "nil by mouth" status (surgery anticipated during aeromedical evacuation). Depending on the proposed surgical procedure, patients also may need cross-matching.
- A military pain team that prescribes and monitors multimodal analgesia including neuropathic agents oversees analgesic considerations. When patients arrive, the effectiveness of in-transit analgesia is assessed because continuous peripheral nerve block/epidural catheters may need revision or de novo insertion perioperatively. Patients are subsequently assessed daily on pain rounds[1] (see Figure 51-2).
- A subgroup of patients recently arrived on the ward from the critical care unit may still be high dependency.
- Two military trainees supported by consultant staff manage the patients holistically. Military mental health and regimental welfare services also provide routine support.

(1) Devonport L, Edwards D, Edwards C, Aldington DJ, Mahoney PF, Wood PR. Evolution of the role 4 UK military pain service. *J R Army Med Corps*. 2010;156(Suppl 1):S398–401.

detail a patient's specific injuries so that the appropriate clinicians can assess him or her upon arrival. The clinical group will include those responsible for performing a secondary survey, including ophthalmologists and ear, nose, and throat surgeons. If significant head or chest injuries exist, the patient will be directed to the appropriate surgical specialists in neurosurgical or cardiac critical care.

Less severely injured casualties are admitted directly to a trauma ward, where an early review of the patient's wounds is undertaken. Those admitted to critical care are also commonly sent to an operating room within 2 to 8 hours of their arrival to have their wounds inspected. Again, this may be the first procedure in a lengthy series.[3]

The anesthetic considerations for these two groups are distinct. The essential elements are detailed in Exhibit 51-3 and Exhibit 51-4, including comments (Exhibit 51-4) relevant to patients recently discharged from critical care.

COORDINATING CLINICAL CARE

Military patients are reviewed at a weekly multidisciplinary team ward round. This review occurs in two parts: (1) a sit-down multidisciplinary team meeting followed by (2) a conventional ward round consisting

of key clinical personnel. The first multidisciplinary team component is attended by more than 20 staff from clinical and support disciplines. Every military patient's overall progress is reviewed, including arrangements for discharge for continued rehabilitation at the Defence Medical Rehabilitation Centre, Headley Court, Surrey.

All NHS and military patients requiring surgery are prioritized twice daily in the "bunker," a secure room within the QEHB main theater complex. At these meetings theater lists and patients are managed so that all necessary urgent and emergency activity continues without affecting normal scheduling.

OPERATING ROOM ACTIVITY

The complex nature of ballistic wounds means that as the patient's condition stabilizes, multiple surgical interventions are the norm. Frequent and often prolonged surgical episodes have implications for operating room logistics. During February 2010, six military patients were in the operation room for over 10 hours, including one who required 45 hours of surgery, and his operative interventions continued into the next month.

EXTERNAL RELATIONSHIPS

The QEHB is a central component of the UK Role 4, but it cannot work in isolation. Occasionally, clinical issues require discussion between the physicians and care providers at Role 3 and 4 facilities. The joint theater clinical case conference, a secure pan-operational teleconference, is conducted on a weekly basis and facilitates these discussions.

Care at the QEHB precedes the extensive rehabilitation needed for many of the injured personnel subsequently undertaken at the Defence Medical Rehabilitation Centre. A further consideration is managing surges in patient activity. The Trust, in conjunction with the Department of Health, has agreements with neighboring trusts to displace patients, if necessary.

New clinical lessons are continually being absorbed, often in coordination with research conducted by the Defence Science and Technology Laboratory, Porton Down, which is translated into practical combat protection and casualty care. The critical relationships between the QEHB and external partners are summarized in Figure 51-1.

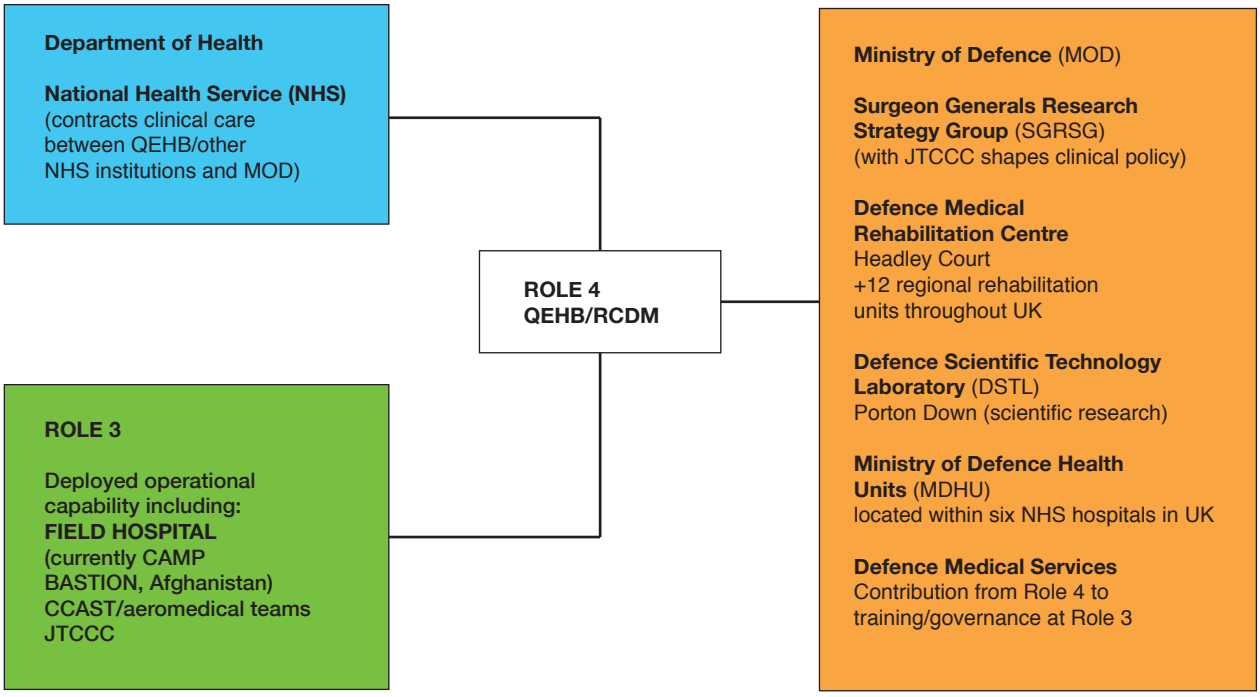

Figure 51-1. Relationship of Queen Elizabeth Hospital Birmingham to external bodies.
CCAST: Critical Care Air Support Team; JTCCC: joint theater clinical case conference; RCDM: Royal Centre for Defense Medicine

SUMMARY

Considerable effort is used to manage the complex injuries received at Role 4. A highly experienced multidisciplinary clinical team of NHS and military staff has developed during the years of conflict, and a close relationship exists with the deployed field hospital. The UK National Audit Office has scrutinized the clinical pathway from point of injury through rehabilitation in its report *Ministry of Defence–Treating Injury and Illness Arising on Military Operations*.[4] This publication remains one of the most extensive reviews of the care provided to injured UK military personnel. It concluded that overall, the "treatment for seriously injured personnel is highly effective," which reflects favorably on the contribution from both UK Role 4 elements—QEHB and the Defence Medical Rehabilitation Centre. Future progress will continue to depend on close support and cooperation between civilian clinicians and the defense medical services.

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ABBREVIATIONS AND ACRONYMS

A

ABC: *Acinetobacter baumannii-calcoaceticus* complex
ABC: airway, breathing, and circulation
ABC: assessment of blood consumption
AC: alternating current
ACCP: American College of Chest Physicians
ACH: Army community hospital
ACRM: Anesthesia Crisis Resource Management
ACS: abdominal compartment syndrome
ACS: acute compartment syndrome
ACTH: adrenocorticotropin
AE: aeromedical evacuation
AECC: Aeromedical Evacuation Co-ordination Centre
AFRICOM: African Command
AKI: acute kidney injury
ALI: acute lung injury
AMEDD C&S: Army Medical Department Center and School
AMPA: alpha-amino-3-hydroxyl-5-methyl-4-isoxazole-propionate
AMPLE: allergies, medication, pregnancy, last eaten
AMR: advanced medical retrieval
APP: abdominal perfusion pressure
APRV: airway pressure-release ventilation
ARDS: acute respiratory distress syndrome
APLS: Advanced Paediatric Life Support
APS: acute pain service
APTR: activated partial thromboplastin time ratio
ATC: acute trauma coagulopathy
ATICE: Adaptation to the Intensive Care Environment (instrument)
ATLS: Advanced Trauma Life Support
ATN: acute tubular necrosis
ATP: adenosine triphosphate
AW2: Army Wounded Warrior Program

B

BABT: behind-armor blunt trauma
BAS: battalion aid station
BATLS: Battlefield Advanced Trauma Life Support
BIPAP: biphasic positive airway pressure ventilation
BK: bradykinin
BPF: bronchopleural fistula
BVM: bag-valve masks

C

<C>ABC: catastrophic hemorrhage, airway, breathing, and circulation
CASEVAC: casualty evacuation
CASF: contingency aeromedical staging facility
CAT: combat application tourniquet
CBF: cerebral blood flow
CBRN: chemical, biological, radiological, or nuclear
CC: combat casualty
CCAT: critical care air transport
CCAST: critical care air support team
CCPG: Canadian clinical practice guideline
CCR: Canadian C-Spine Rule
CENTCOM: Central Command
$CMRO_2$: cerebral metabolic rate of oxygen
CO: cardiac output
CONUS: continental United States
COTS: coagulopathy of trauma shock
COX: cyclooxygenase
CPAP: continuous level of positive airway pressure
CPG: clinical practice guideline
CPK: creatine phosphokinase
CPNB: continuous peripheral nerve block
CPP: cerebral perfusion pressure
CPR: cardiopulmonary resuscitation
CRM: Crew Resource Management
CRRT: continuous renal replacement therapy
CSH: combat support hospital
CSI: cervical spine injury
CT: clotting time
CT: component therapy
CT: computed tomography
CVC: central venous catheter
CVP: central venous pressure
CVVH: CRRT with a veno-venous technique of hemofiltration
CXR: chest x-ray

D

DLEBT: double-lumen endobronchial tube
DCR: damage control resuscitation
DLT: double-lumen endobronchial tube
DMS: Defence Medical Services (United Kingdom)
DNBI: disease non-battle-injury
DoA: depth of anesthesia
DPL: diagnostic peritoneal lavage
DR: disaster relief
DVPRS: Defense and Veterans Pain Rating Scale
DVT: deep venous thrombosis

E

EAST: Eastern Association for the Surgery of Trauma
ECCN: en-route critical care nurse
ECG: electrocardiogram
ECMO: extracorporeal membrane oxygenation
ED: emergency department
EDOCS: expeditionary deployable oxygen concentrator system
EMT-B: emergency medical technician-basic
EN: enteral nutrition
ESBL: extended spectrum β-lactamase
ESPEN: European Society for Parenteral and Enteral Nutrition
$ETCO_2$: elevated end-tidal carbon dioxide
ETT: endotracheal tube
EUCOM: European Command

F

F: French gauge
FiO_2: fraction of inspired oxygen
FAST: focused assessment with sonography for trauma
FFP: fresh frozen plasma
Fr: French gauge
FRC: functional residual capacity
FST: forward surgical team
FW: fixed wing
FWB: fresh whole blood

G

G: gauge
Ga: gauge
GABA: γ-aminobutyric acid
GAWS: Guardian Angel Weapon System
GCOs: Clinical Guidelines for Operations
GCS: Glasgow coma scale

G6PD: glucose-6-phosphate dehydrogenase

H

HA: humanitarian assistance
HbCO: carbon monoxide hemoglobin
HD: hemodialysis
HELLP: hemolysis, elevated liver enzymes, and low platelets (syndrome)
HEMS: Helicopter Emergency Medical System
HFOV: high-frequency oscillatory ventilation
HFV: high-frequency ventilation
HIV: human immunodeficiency virus
HMAP: high mean arterial pressure
HR: heart rate

I

IAP: intraabdominal pressure
ICO: infection control officer
ICP: intracranial pressure
ICU: intensive care unit
ID: internal diameter
IED: improvised explosive device
IJV: internal jugular
iLA: interventional lung assistance
ILV: independent lung ventilation
IM: intramuscular
IMV: intermittent mandatory ventilation
IN: intranasal
INR: international normalized ratio
IO: intraosseous
IOP: intraocular pressure
IPPV: intermittent positive-pressure ventilation
IRI: ischemia-reperfusion injury
IRT: immediate response team
ISO: International Organization for Standardization
ISS: injury severity score
IV: intravenous
IVC: inferior vena cava
IVCF: inferior vena cava filter

J

JTTR: Joint Theater Trauma Registry

L

LAST: local anesthetic systemic toxicity
LMA: laryngeal mask airway
LMAP: lower mean arterial pressure
LMWH: low molecular weight heparin
LOP: limb occlusion pressure
LRMC: Landstuhl Regional Medical Center
LSI: life-saving intervention
LTP: long-term potentiation

M

MAC: minimum alveolar concentration
MAP: mean arterial pressure
MASF: mobile aeromedical staging facilities
MCF: maximum clot firmness
MDCT: multidetector row spiral computed tomography
MDR: multidrug-resistant
MEAC: minimum effective analgesic concentration
MEDCEN: military medical center
MEDCOM: US Army Medical Command
MEDEVAC: medical eva
MERT: medical emergency response team
MERT(E): medical emergency response team (enhanced)
MH: malignant hyperthermia
MILS: manual inline stabilization
MIST-AT: mechanism of injury, injuries sustained, symptoms and signs, treatment given–age (adult/child) and time of injury
MHS: Military Health System
MODS: multiorgan dysfunction syndrome
MRAP: mine-resistant ambush-protected
MRI: magnetic resonance imaging
MRSA: methicillin-resistant *Staphylococcus aureus*
MRSN: Multidrug Resistant Organism Repository and Surveillance Network
MTF: medical treatment facility
MTP: massive transfusion protocol

N

NAPQI: *N*-acetyl-p-benzoquinine imine
NASA: National Aeronautics and Space Administration
NBI: nonbattle injury
NEXUS: National Emergency X-Radiography Utilization Study
NGF: nerve growth factor
NGO: nongovernmental organization
NHS: National Health Service (United Kingdom)
NK_1: neurokinin-1
NMBA: neuromuscular blocking agent
NMDA: *N*-methyl-d-aspartate
NMS: neuroleptic malignant syndrome
NS: normal saline
NSAID: nonsteroidal antiinflammatory drugs

O

OEF: Operation Enduring Freedom
OI: oxygenation index
OIF: Operation Iraqi Freedom
OL-ILV: one-lung independent ventilation
OR: operating room
OSCAR: High Frequency OSCillation in ARDS (trial)
OSCILLATE : Oscillation for Acute Respiratory Distress Syndrome Treated Early (trial)
OTFC: oral transmucosal fentanyl citrate

P

PACCOM: Pacific Command
PAG: periaqueductal grey
PCA: patient-controlled analgesia
PCR: polymerase chain reaction
PD: peritoneal dialysis
PE: pulmonary embolism
PECC: patient evacuation coordination center
PEEP: positive end-expiratory pressure
$PetCO_2$: partial pressure of end-tidal carbon dioxide
PG: propylene glycol
PICU: pediatric intensive care unit
PiO_2: partial pressure of the inspired oxygen
PIS: propofol infusion syndrome
PLT: platelets
PMI: patient movement item
PMRC: patient movement requirement center
PN: parenteral nutrition
PO: per os
POGS: portable oxygen generator system
PP: pulse pressures
PPE: personal protective equipment
Ppl: plateau pressure
PPV: positive pressure ventilation
PRBC: packed red blood cells

PRIS: propofol infusion syndrome
PTSD: posttraumatic stress disorder

Q

QEHB: Queen Elizabeth Hospital Birmingham

R

RA: regional anesthesia
RAAS: rennin-angiotensin-aldosterone system
RAF: Royal Air Force
RAP: regimental aid post
RASS: Richmond Agitation-Sedation Scale
RBC: red blood cell
RCC: red cell concentrate
RCDM: Royal Centre for Defence Medicine
RCT: randomized controlled trial
REBOA: resuscitative endovascular balloon of the aorta
rFVIIa: recombinant factor VIIa
RIFLE: risk, injury, failure, loss, and end-stage disease
ROSC: return of spontaneous circulation
RSI: rapid sequence induction
RTD: return-to-duty
RRT: renal replacement therapy
RSI: rapid-sequence induction
RW: rotary wing

S

SAVe: simplified automated ventilator
SCCM: Society of Critical Care Medicine
SCD: sequential compression device
SCM: sternocleidomastoid muscle
SCV: subclavian
$ScvO_2$: central venous oxygen saturation
SI: sacroiliac
SIB: self-inflating bag
SIMV/PS: synchronized intermittent mandatory ventilation with pressure support
SIRS: systemic inflammatory response syndrome
SO: standard operating instruction
SOP: standard operating procedures
SOUTHCOM: Southern Command
STRATEVAC: strategic evacuation
SVC: superior vena cava
SVR: systemic vascular resistance

T

TACEVAC: tactical evacuation
TARGIT: Triservice Research Group Initiative on TIVA
TBI: traumatic brain injury
TBSA: total burned surface area
TCCC: Tactical Combat Casualty Care
TCI: target-controlled infusion
TCRA: traumatic cardiorespiratory arrest
TD: tracheal disruption
TENS: toxic epidermal necrolysis syndrome
TIC: toxic industrial chemicals
TIVA: total intravenous anesthesia
TL-ILV: two-lung independent lung ventilation
TLR4: toll-like receptor 4
TRPV: transient receptor potential vallinoid
TTE: transthoracic echocardiogram
TTP: tactic, technique, or procedure
TRALI: transfusion-related acute lung injury
TrkA: tyrosine kinase A
TSAA: Triservice Anaesthetic Apparatus
TST: tuberculin skin testing

U

UFH: unfractionated heparin
UK: United Kingdom
UN: United Nations
US: ultrasound
USAF: US Air Force
USAISR: US Army Institute of Surgical Research

V

VA: Veterans Affairs (Department)
VAP: ventilator-associated pneumonia
VAS: visual analog scale
VDC: volts direct current
VGA: volatile gas anesthesia
VILI: ventilator-induced lung injury
VITRIS: Vasopressin in Refractory Traumatic Hemorrhagic Shock (study)
VRE: vancomycin-resistant *Escherichia coli*
VRS: verbal rating score
Vt: tidal volume
VTE: venous thromboemoblism

W

WFWB: warm fresh whole blood
WHO: World Health Organization
WRAIR: Walter Reed Army Institute of Research
WWR: Wounded Warrior Regiment

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MERT Head Out Again by Tony Green, acrylic on paper, 2009.

Art: Courtesy of Tony Green.