

Chapter 1

PHYSIOLOGY OF INJURY AND EARLY MANAGEMENT OF COMBAT CASUALTIES

DIEGO VICENTE, MD*; BENJAMIN K. POTTER, MD[†]; AND ALEXANDER STOJADINOVIC[‡]

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*Lieutenant, Medical Corps, US Navy; Department of General Surgery; Walter Reed National Military Medical Center, 8901 Wisconsin Avenue, Bethesda, Maryland 20889-5600

[†]Major, Medical Corps, US Army; Department of Orthopaedic Surgery, Walter Reed National Military Medical Center, 8901 Wisconsin Avenue, Bethesda, Maryland 20889-5600

[‡]Colonel, Medical Corps, US Army, Department of General Surgery, Walter Reed National Military Medical Center, 8901 Wisconsin Avenue, Bethesda, Maryland 20889-5600

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 enzymes¹⁶⁻¹⁸ 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

blasts²³ 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 tourniquets³⁷⁻³⁹ and

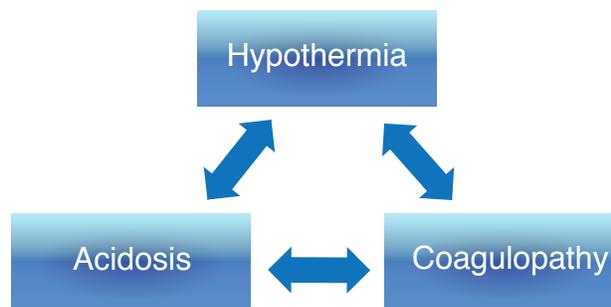


Figure 1-1. Lethal triad.

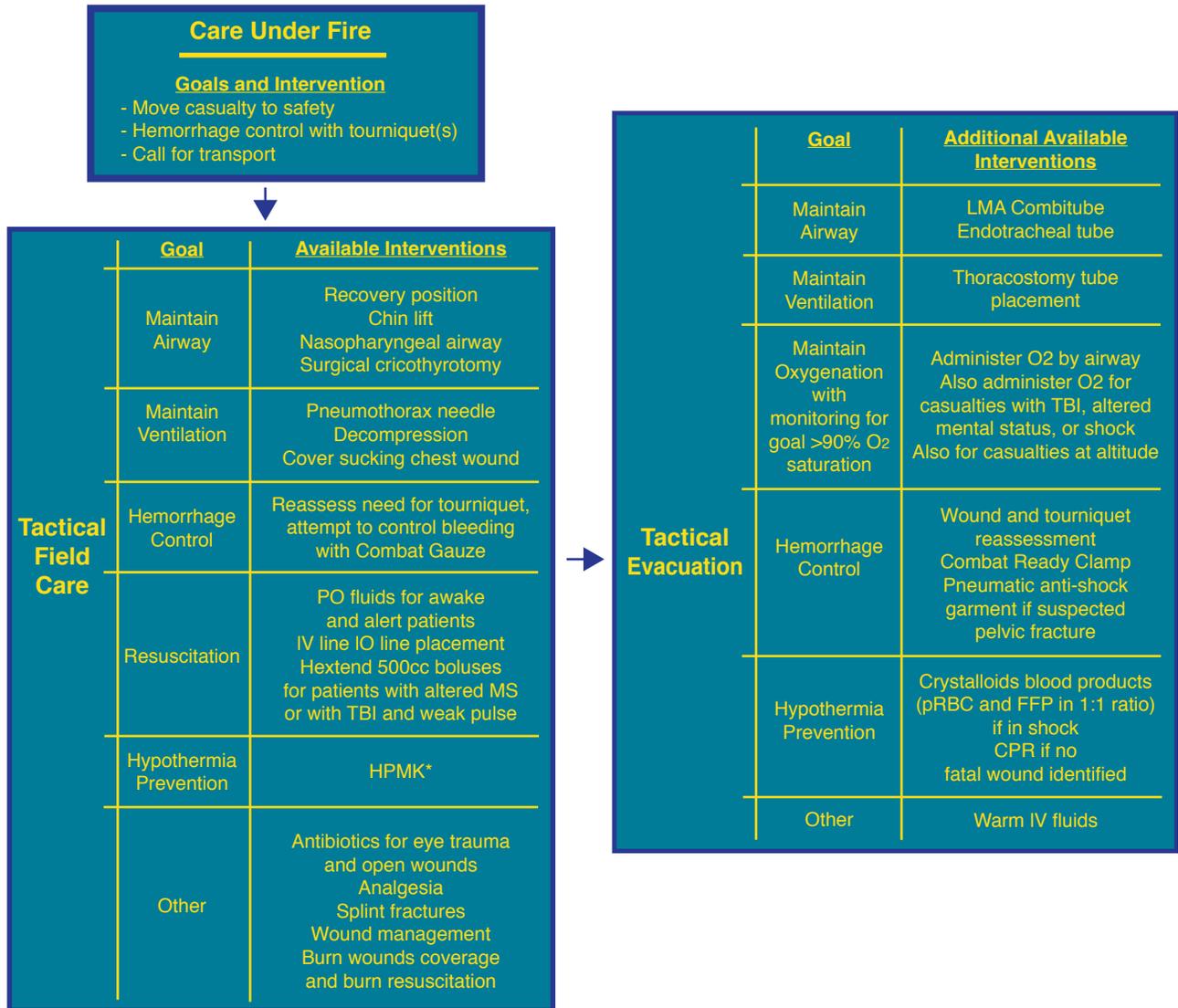


Figure 1-2. Tactical Combat Casualty Care (TCCC). As the combat casualty progresses through the stages of TCCC, he or she undergoes serial evaluations and repeated interventions as necessary. More resources and interventions are available at higher stages.

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; O₂: 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)

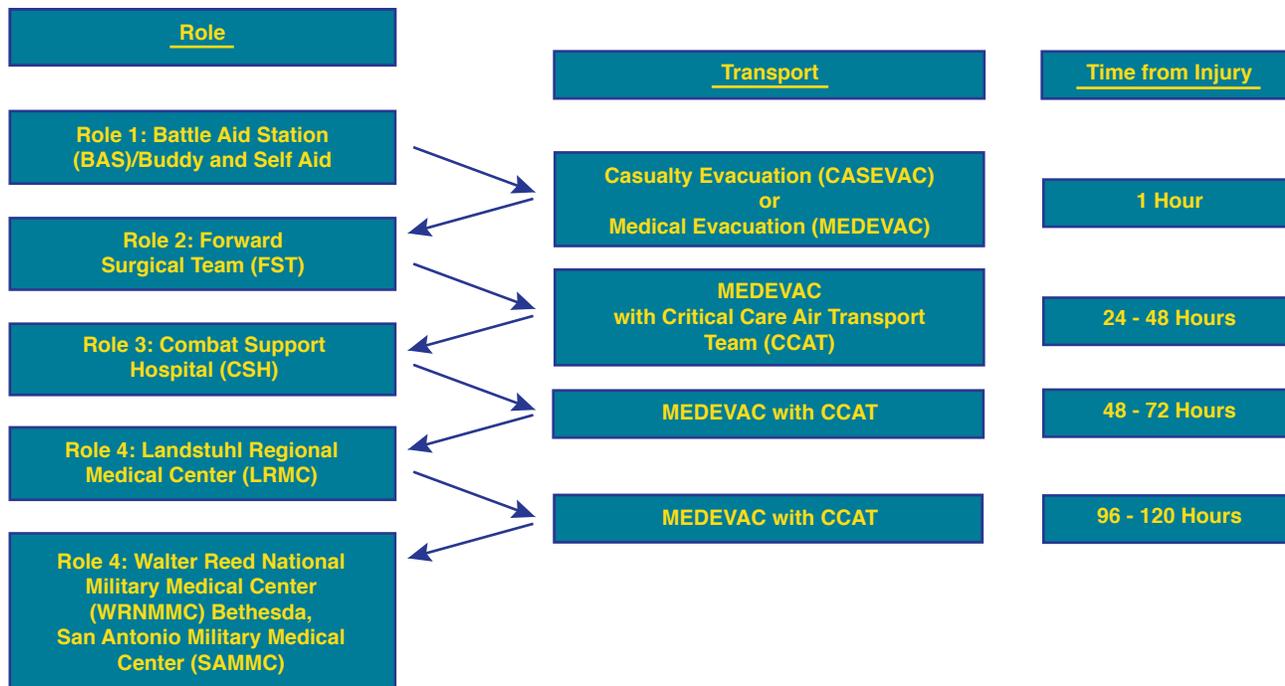


Figure 1-3. Roles of combat casualty care, transport, and time from injury.

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/mm³, 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 pre-sedation 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 syndrome⁴⁸⁻⁵¹ (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

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

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 Extremity 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.

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

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

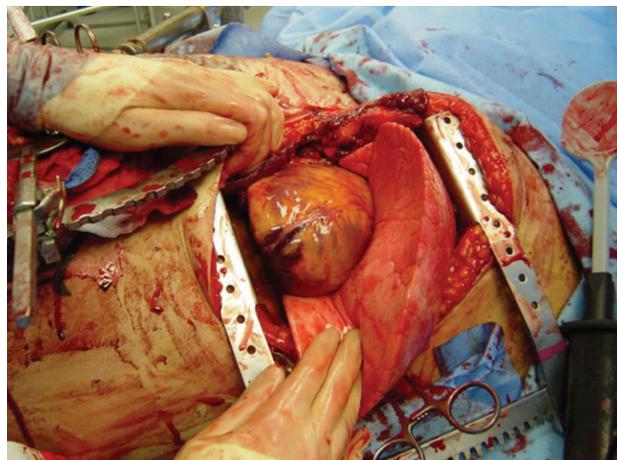


Figure 1-4. Emergency department thoracotomy can be performed for the following reasons: (1) release of pericardial tamponade, (2) hemorrhage control, (3) aortic cross clamp, (4) control of massive air embolism or bronchopleural fistula, or (5) open cardiac massage.

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 hours¹²³ 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

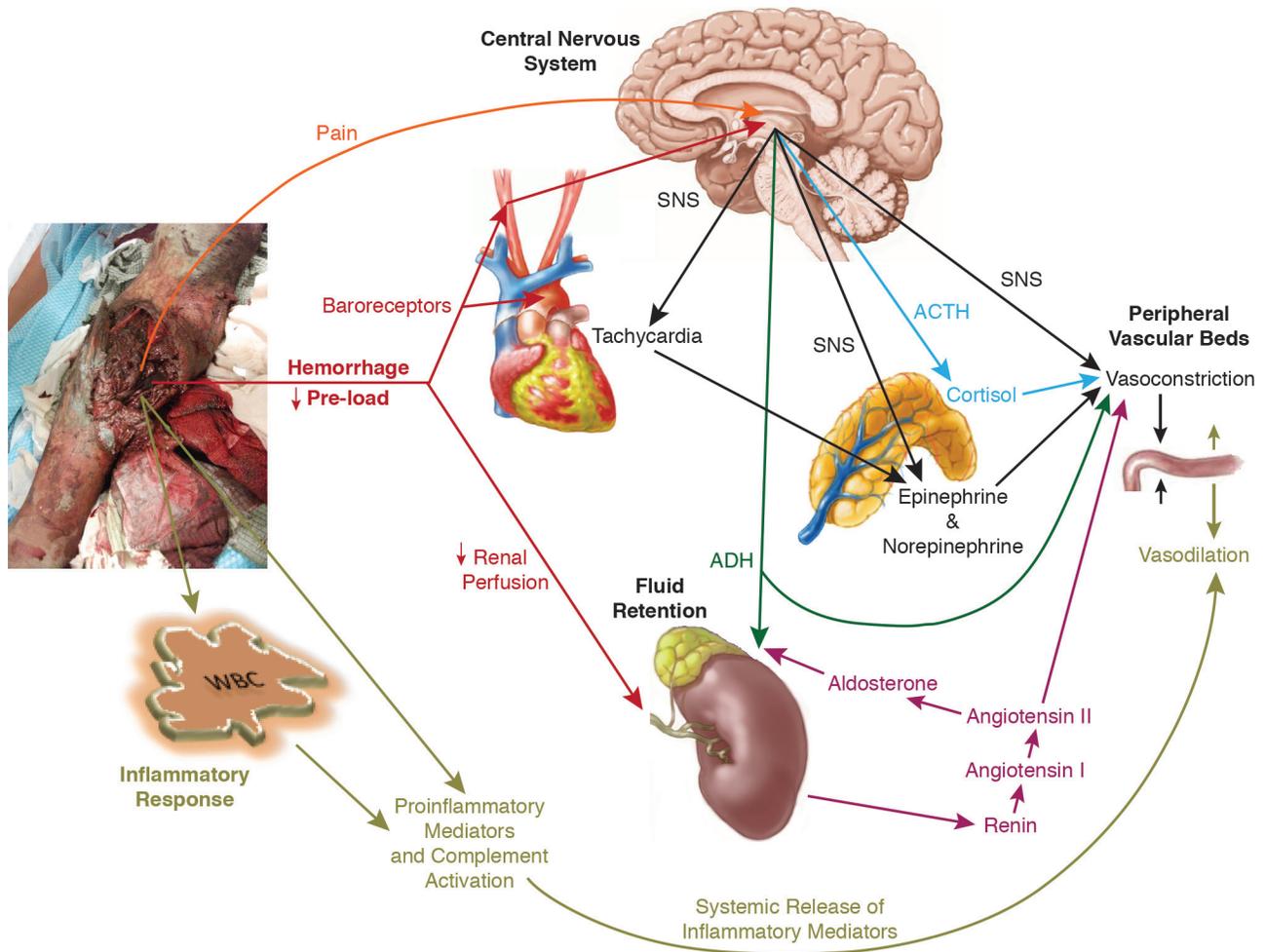


Figure 1-5. Effect of neurohormonal and inflammatory response to trauma on cardiovascular system. ACTH: adrenocorticotropic hormone; ADH: antidiuretic hormone; SNS: sympathetic nervous system; WBC: white blood cell. Images adapted with permission from Vesalius: <http://www.vesalius.com/welcome.asp>. 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 indicated¹⁴⁷ at a Role 3 facility. Support of myocardial contractility should include resolving hypothermia^{148,149} and acidosis,^{150,151} as well as inotropic 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 bronchopulmonary 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 chest¹⁵⁶; 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.

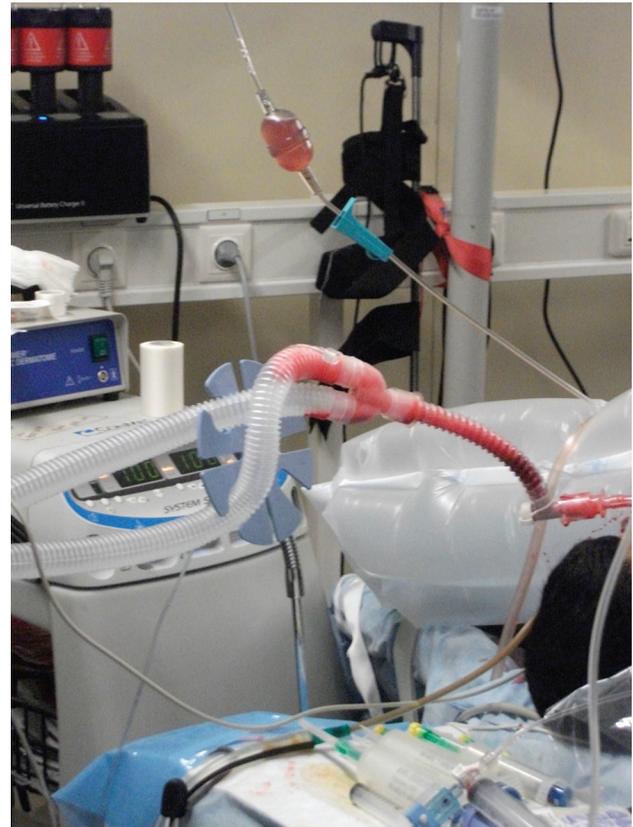


Figure 1-7. Blood in endotracheal tube of combat casualty with blast lung injury.

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 PaO₂:FiO₂ ratio less than 200. Risk fac-

tors 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 FiO₂ less than 60%.¹⁶⁴ To reduce FiO₂, peak end-expiratory pressure (PEEP) can be increased while keeping peak pressure at less than 35 cm to 40 cm H₂O to prevent lung injury.¹⁶⁵ It may be difficult to obtain eucapnia (Pco₂ 40–45 mm Hg)¹⁴ with these settings; permissive hypercapnia (Pco₂ > 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 (PaO₂ < 60 mm Hg or SaO₂ < 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.

Prompt initiation of medical management with intubation, appropriate oxygenation (goal PaO₂ > 60 mm Hg), ventilation (goal Paco₂ 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-

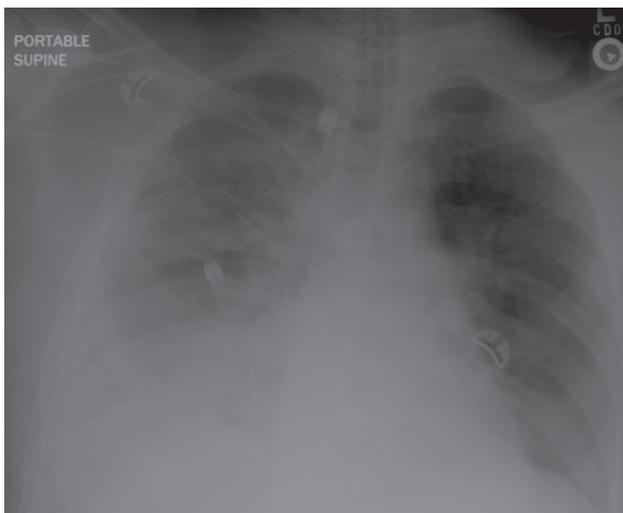


Figure 1-8. Patient with acute respiratory distress syndrome with bilateral patchy infiltrates on chest x-ray.

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 ≥ 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 thromboembolism (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 frac-

tures, acute lung injury, GCS less than 10, a need for mechanical ventilation with PEEP greater than 6, and rhabdomyolysis with creatinine phosphokinase 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 CO₂ 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:

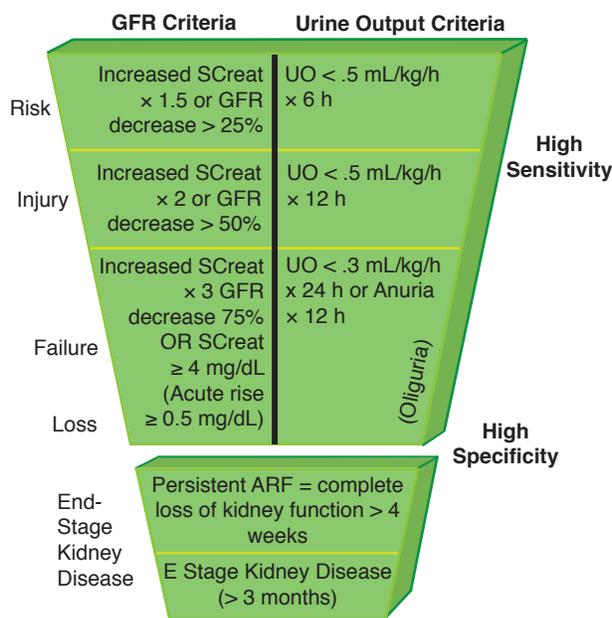


Figure 1-9. “RIFLE” criteria for acute renal failure.

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 / mm³ 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 Moni-

tor 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 myoglobinuria-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 urine²⁰² 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 deter-

mine 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 en-

zymes, 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

**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: platelets; 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.

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

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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