CHAPTER

39

Anesthesia for Trauma & Emergency Surgery

Brian P. McGlinch, MD

KEY CONCEPTS

1. All trauma patients should be presumed to have "full" stomachs and an increased risk for pulmonary aspiration of gastric contents.
2. Cervical spine injury is presumed in any trauma patient complaining of neck pain, or with any significant head injury, neurological signs or symptoms suggestive of cervical spine injury, or intoxication or loss of consciousness.
3. In the multiple-injury patient, providers should maintain a high level of suspicion for pulmonary injury that could evolve into a tension pneumothorax when mechanical ventilation is initiated.
4. In up to 25% of major trauma patients, trauma-induced coagulopathy is present shortly after injury and before any resuscitative efforts have been initiated.
5. Administering blood products in equal ratios early in resuscitation has become an accepted approach to correction of trauma-induced coagulopathy. This balanced approach to transfusion, 1:1:1 (red blood cell:fresh frozen plasma:platelet), is termed *damage control resuscitation*.
6. Noninfectious transfusion reactions are now the leading complication of transfusion and represent a more than 10-fold greater risk than blood-borne infection. Transfusion-related acute lung injury is the leading cause of transfusion-related death.
7. The *assessment of blood consumption* (ABC) score is an attempt to predict which patients are likely to require a massive transfusion protocol. The ABC score assigns 1 point for the presence of each of four possible variables: (1) penetrating injury; (2) systolic blood pressure less than 90 mmHg; (3) heart rate greater than 120 beats per minute; and (4) positive results of a *focused assessment with sonography for trauma* evaluation. Patients with ABC scores of 2 or higher are likely to require massive transfusion.
8. Any trauma patient with altered level of consciousness must be considered to have a traumatic brain injury (TBI) until proven otherwise. The most reliable clinical assessment tool in determining the significance of TBI in a nonsedated, nonparalyzed patient is the Glasgow coma scale.
9. Acute subdural hematoma is the most common condition warranting emergency neurosurgery and is associated with the highest mortality.
10. Systemic hypotension (systolic blood pressures <90 mm Hg), hypoxemia (Pao_2 <60 mm Hg), hypercapnia ($Paco_2$ >50 mm Hg), and hyperthermia (temperature >38.0°C) have a negative impact on morbidity and mortality following head injuries, likely because of

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their contributions to increasing cerebral edema and intracranial pressure (ICP).

11. Current guidelines recommend maintaining cerebral perfusion pressure between 50 and 70 mm Hg and ICP at less than 20 mm Hg for patients with severe head injury.
12. Maintaining supranormal mean arterial blood pressures to assure spinal cord perfusion in areas of reduced blood flow due to cord compression or vascular compromise is likely to be of more benefit than steroid administration.
13. Major burns (a second- or third-degree burn involving >20% total body surface area [TBSA]) induce a unique hemodynamic response. Cardiac output declines by up to 50% within 30 minutes in response to massive vasoconstriction, inducing a state of normovolemic hypoperfusion (*burn shock*).
14. In contrast to fluid management for blunt and penetrating trauma, which discourages use of crystalloid fluids, burn fluid resuscitation emphasizes the use of crystalloids, particularly lactated Ringer's solution, in preference to albumin, hydroxyethyl starch, hypertonic saline, and blood.
15. Carbon monoxide poisoning should be considered in all serious burn injury cases, as well as with lesser TBSA burns occurring in enclosed spaces. Unconsciousness or decreased levels of consciousness following burn injuries should be presumed to represent carbon monoxide poisoning.
16. Beyond 48 h after a major burn, succinylcholine administration is likely to produce potentially lethal elevation of serum potassium levels.

Trauma is a leading cause of morbidity and mortality in all age groups, and is the leading cause of death in the young. All aspects of trauma care, from that provided at the scene, through transport, resuscitation, surgery, intensive care, and rehabilitation, must be coordinated if the patient is to have the greatest chance for full recovery. The Advanced Trauma Life Support (ATLS) program developed by the American College of Surgeons' (ACS) Committee on Trauma has, over time, resulted in an increasingly consistent approach to trauma resuscitation. The development of criteria for level one trauma centers has also improved trauma care by directing severely injured patients to facilities with appropriate resources.

Although trauma anesthesia is sometimes thought of as a unique topic, many of the principles for managing trauma patients are relevant to any unstable or hemorrhaging patient. Thus, many common issues are addressed in this chapter.

PRIMARY SURVEY

Airway

Increasingly, emergency medical technician–paramedics and flight nurses are trained to intubate patients in the prehospital environment. More providers capable of airway management in the critically ill or injured patient are now available to intervene in the hospital setting as well. As a result, the anesthesiologist's role in providing initial trauma resuscitation has diminished in North America. This also means that when called upon to assist in airway management in the emergency department, anesthesia providers must expect a challenging airway, as routine airway management techniques likely have already proved unsuccessful.

There are three important aspects of airway management in the initial evaluation of a trauma patient: (1) the need for basic life support; (2) the presumed presence of a cervical spinal cord injury

until proven otherwise; and (3) the potential for failed tracheal intubation. Effective basic life support prevents hypoxia and hypercapnia from contributing to the patient's depressed level of consciousness. When hypercarbia produces a depressed level of consciousness, basic airway interventions often lessen the need for endotracheal intubation as arterial carbon dioxide levels return to normal.

1 Finally, all trauma patients should be presumed to have "full" stomachs and an increased risk for pulmonary aspiration of gastric contents. Assisted ventilation should be performed with volumes sufficient to provide chest rise. Some clinicians will apply cricoid pressure, although the efficacy of this maneuver is controversial.

2 Cervical spine injury is presumed in any trauma patient complaining of neck pain, or with any significant head injury, neurological signs or symptoms suggestive of cervical spine injury, or intoxication or loss of consciousness. The application of a cervical collar ("C-collar") before transport to protect the cervical spinal cord will limit the degree of cervical extension that is ordinarily expected for direct laryngoscopy and tracheal intubation. Alternative devices (eg, videolaryngoscopes, fiberoptic bronchoscopes) should be immediately available. The front portion of the C-collar can be removed to facilitate tracheal intubation as long as the head and neck are maintained in neutral position by a designated assistant maintaining manual in-line stabilization.

Alternative devices for airway management (eg, esophageal–tracheal Combitube, King supralaryngeal device) may be used if direct laryngoscopy has failed, or in the prehospital environment. These devices, blindly placed into the airway, isolate the glottic opening between a large inflatable cuff positioned at the base of the tongue and a distal cuff that most likely rests in the proximal esophagus (Figure 39–1). The prolonged presence of these devices in the airway has been associated with glossal engorgement resulting from the large, proximal cuff obstructing venous outflow from the tongue, and in some cases, tongue engorgement has been sufficiently severe to warrant tracheostomy prior to their removal. There is limited evidence that prehospital airway management in trauma patients improves patient outcomes; however, failed tracheal intubation in the prehospital environment certainly exposes patients to significant morbidity.

Airway management of the trauma patient is uneventful in most circumstances, and

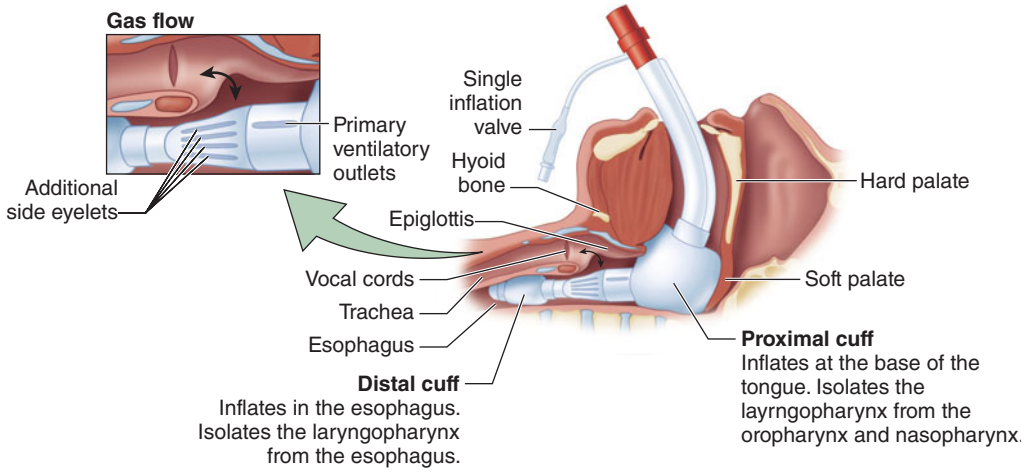

FIGURE 39–1 The King LT supralaryngeal device. The glottic opening lies between the large cuff positioned at the base of the tongue and the smaller balloon positioned in the proximal esophagus. The airway is not secured but rather isolated between the oropharynx and the proximal esophagus. (Reproduced, with permission, from King Systems Corporation, KLTD/KLTSD Disposable Supralaryngeal Airways Inservice Program, August 23, 2006, with permission.)

cricothyroidotomy or tracheostomy is rarely required to secure the trauma airway. When trauma significantly alters or distorts the facial or upper airway anatomy to the point of impeding adequate mask ventilation, or when hemorrhage into the airway precludes the patient from lying supine, elective cricothyroidotomy or tracheostomy should be considered before any attempts are made to anesthetize or administer neuromuscular blocking agents to the patient for orotracheal intubation.

Breathing

3 In the multiple-injury patient, providers should maintain a high level of suspicion for pulmonary injury that could evolve into a tension pneumothorax when mechanical ventilation is initiated. Attention must be paid to peak inspiratory pressure and tidal volumes throughout the initial resuscitation. Pulmonary injury may not be immediately apparent upon the patient's arrival at the hospital, and abrupt cardiovascular collapse shortly after instituting mechanical ventilation may announce the presence of a pneumothorax. This should be managed by disconnecting the patient from mechanical ventilation and performing bilateral needle thoracostomy (accomplished by inserting a 14-gauge intravenous catheter into the second interspace in the midclavicular line), and then by thoracostomy tube insertion. Inspired oxygen concentrations of 100% are used routinely in this early phase of resuscitation.

Circulation

During the primary trauma patient survey, signs of a pulse and blood pressure are sought. Unless the trauma patient arrives at the hospital other than by ambulance, the resuscitation team will likely have received information about the patient's vital signs from the prehospital personnel (emergency medical technicians, flight nurses). The absence of a pulse following trauma is associated with dismal chances of survival. The ACS Committee on Trauma no longer endorses the use of emergency thoracotomy in treating patients without blood pressure or palpable pulse following *blunt* trauma, even in the presence of organized cardiac activity, given the lack of evidence supporting survival following this intervention. Retrospective review of emergency thoracotomy in Europe failed to demonstrate resuscitation benefit of this procedure following either blunt or penetrating trauma in the setting of cardiac arrest. In the setting of chest trauma without detectable blood pressure or palpable pulse, current practice supports reserving resuscitative thoracotomy for patients who experience *penetrating* trauma and have preserved, organized cardiac rhythms or other signs of life.

In light of these recommendations, prompt placement of bilateral chest tubes and administration of a 500–1000 mL fluid bolus should be implemented in the pulseless victim of penetrating trauma. If return of spontaneous circulation does not occur promptly, more aggressive interventions are not indicated and resuscitation efforts can be terminated.

Neurological Function

Once the presence of circulation is confirmed, a brief neurological examination is conducted. Level of consciousness, pupillary size and reaction, lateralizing signs suggesting intracranial or extracranial injuries, and indications of spinal cord injury are quickly evaluated. As noted earlier, hypercarbia often causes depressed neurological responsiveness following trauma; it is effectively corrected with basic life support interventions. Additional causes of depressed neurological function—eg, alcohol intoxication, effects of illicit or prescribed medications, hypoglycemia, hypoperfusion, or brain or spinal injury—must also be addressed. Mechanisms of injury must be considered as well as exclusion of other factors in determining the risk for central nervous system trauma. Persistently depressed levels of consciousness should be considered a result of central nervous system injury until disproved by diagnostic studies.

Injury Assessment: Minimizing Risks of Exposure

The patient must be fully exposed and examined in order to adequately assess the extent of injury, and this physical exposure increases the risk of hypothermia. The presence of shock and intravenous fluid therapy also place the trauma patient at great risk for developing hypothermia. As a result, the

resuscitation bay must be maintained at near body temperature, all fluids should be warmed during administration, and the use of forced air patient warmers, either below or covering the patient, should be utilized.

RESUSCITATION

Hemorrhage

Certain trauma-related terminology must be understood and utilized in order to effectively communicate with surgeons during trauma resuscitations or surgeries in which blood loss is occurring. *Hemorrhage classifications I–IV*, *damage control resuscitation*, and *damage control surgery* are terms that quickly convey critical information between surgeons and anesthesia personnel, ensuring a common understanding of the various interventions that may be required to resuscitate a trauma or surgical patient experiencing bleeding. The ACS identifies four classes of hemorrhage. Understanding this classification scheme promotes more effective communication between surgeons and anesthesiologists.

Class I hemorrhage is the volume of blood that can be lost without hemodynamic consequence. The heart rate does not change and the blood pressure does not decrease in response to losing this volume of blood. In most circumstances, this volume represents less than 15% of circulating blood volume. The typical adult has a blood volume equivalent to 70 mL/kg. A 70-kg adult can be presumed to have nearly 5 L of circulating blood. Children are considered to have 80 mL/kg and infants, 90 mL/kg blood volume. Intravenous fluid is not required if the bleeding is controlled, as in brief, controlled bleeding encountered during an elective surgical procedure.

Class II hemorrhage is the volume of blood, that, when lost, prompts sympathetic responses to maintain perfusion; this usually represents 15–30% of circulating blood volume. The diastolic blood pressure will increase (a reflection of vasoconstriction) and the heart rate will increase to maintain cardiac output. Intravenous fluid or colloid is usually indicated for blood loss of this volume. Transfusions may be required if bleeding continues, suggesting progression to class III hemorrhage.

Class III hemorrhage represents the volume of blood loss (30–40% of circulating blood volume) that consistently results in decreased blood pressure. Compensatory mechanisms of vasoconstriction and tachycardia are not sufficient to maintain perfusion and meet the metabolic demands of the body. Metabolic acidosis will be detected on arterial blood gas analysis. Blood transfusions are necessary to restore tissue perfusion and provide oxygen to tissues. The patient may transiently respond to fluid boluses given in response to hemorrhage; however, if bleeding persists or given time for the fluid bolus to redistribute, the blood pressure will decline. Surgeons should be advised when this pattern persists, particularly during elective surgical cases where the development of shock is not expected. Class III hemorrhage may prompt an intervention such as a damage control procedure (see below).

Class IV hemorrhage represents life-threatening hemorrhage. When more than 40% of circulating blood volume is lost, the patient will be unresponsive and profoundly hypotensive. Rapid control of bleeding and aggressive blood-based resuscitation (ie, damage control resuscitation) will be required to prevent death. Patients experiencing this degree of hemorrhage will likely develop a trauma-induced coagulopathy, require massive blood transfusion, and experience a high likelihood of death.

Trauma-Induced Coagulopathy

Coagulation abnormalities are common following major trauma, and trauma-induced coagulopathy is an independent risk factor for death. Recent prospective clinical studies suggest that in up to 25% of major trauma patients, trauma-induced coagulopathy is present shortly after injury and before any resuscitative efforts have been initiated. In one report, acute traumatic coagulopathy was only related to the presence of a severe metabolic acidosis (base deficit ≥6 mEq/L) and appeared to have a dose-dependent relationship with the degree of tissue hypoperfusion; 2% of patients with base deficits less than 6 mEq/L developed coagulopathy compared with 20% of patients with base deficits greater than 6 mEq/L. Although injury severity scores were likely high in those developing coagulopathy, only the

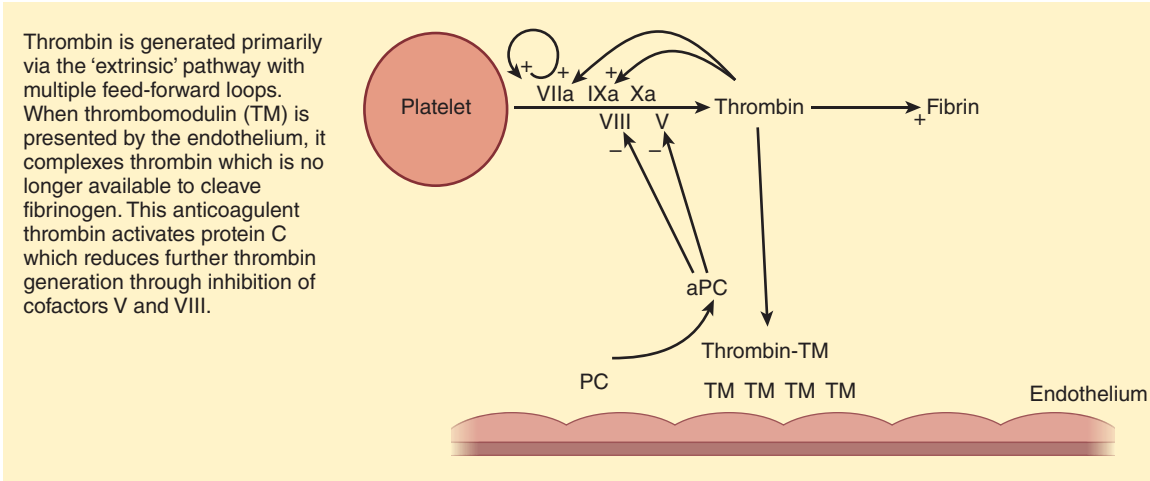

FIGURE 39–2 Mechanism of trauma-induced coagulopathy. During periods of tissue hypoperfusion, thrombomodulin (TM) released by the endothelium complexes with thrombin. The thrombin–TM complexes prevent cleavage of fibrinogen to fibrin and also activate protein C (PC), reducing further thrombin generation through cofactors V and VIII. (Reproduced, with permission, from Brohi K, Cohen MJ, Davenport RA: Acute coagulopathy of trauma: mechanism, identification and effect. Curr Opin Crit Care 2007;13:680.)

presence of the metabolic acidosis correlated to developing trauma-induced coagulopathy.

Global tissue hypoperfusion appears to have a key role in the development of trauma-induced coagulopathy. During hypoperfusion, the endothelium releases thrombomodulin and activated protein C to prevent microcirculation thrombosis. Thrombomodulin binds thrombin, thereby preventing thrombin from cleaving fibrinogen to fibrin. The thrombomodulin–thrombin complex activates protein C, which then inhibits the extrinsic coagulation pathway through effects on cofactors V and VIII (Figure 39–2). Activated protein C also inhibits plasminogen activator inhibitor-1 proteins, which increases tissue plasminogen activator, resulting in hyperfibrinolysis (Figure 39–3). One prospective clinical study found the following effects of hypoperfusion on coagulation parameters: (1) progressive coagulopathy as base deficit increases; (2) increasing plasma thrombomodulin and falling protein C (indicating activation of the protein levels with increasing base deficit), supporting the argument that the anticoagulant effects of these proteins in the presence of hypoperfusion are related to the prolongation of prothrombin and partial thromboplastin times; and (3) an influence of early trauma-induced coagulopathy on mortality.

Trauma-induced coagulopathy is not solely related to impaired clot formation. Fibrinolysis is an equally important component as a result of plasmin activity on an existing clot. Tranexamic acid administration is associated with decreased bleeding during cardiac and orthopedic surgeries, presumably because of its antifibrinolytic properties. A randomized control study involving 20,000 trauma patients with or at risk of significant bleeding found a significantly reduced risk for death from hemorrhage when tranexamic acid therapy (loading dose, 1 g over 10 min followed by an infusion of 1 g over 8 h) was initiated within the first 3 h following major trauma. Figure 39–4 demonstrates the benefit of initiating this therapy in relation to the time of injury.

Hemostatic Resuscitation

5 Early coagulopathy of trauma is associated with increased mortality. Administering blood products in equal ratios early in resuscitation has become an accepted approach to correction of trauma-induced coagulopathy. This balanced approach to transfusion, 1:1:1 (red blood cell:fresh

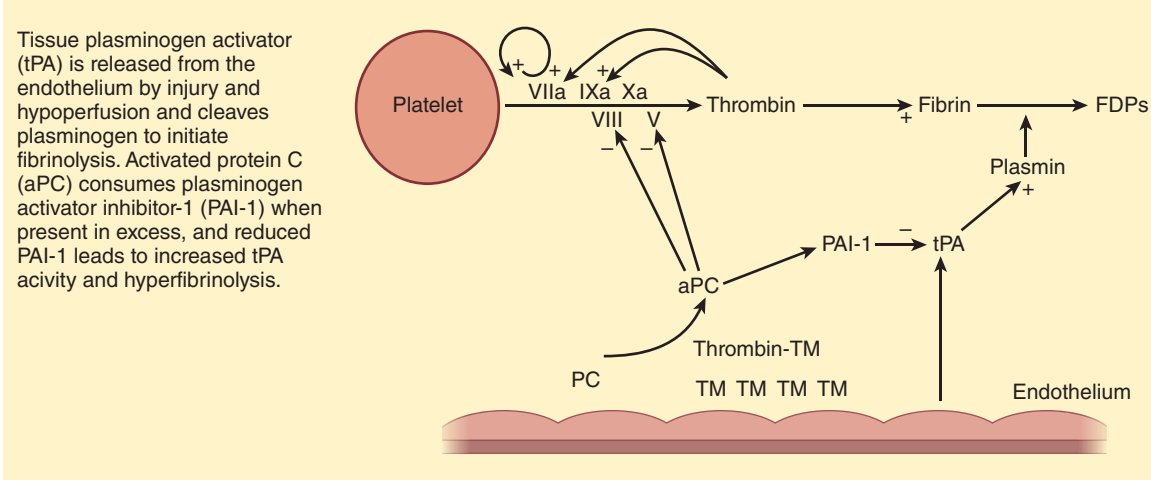

FIGURE 39–3 Mechanism of hyperfibrinolysis in tissue hypoperfusion. Tissue plasminogen activator (tPA) released from the endothelium during hypoperfusion states cleaves plasminogen to initiate fibrinolysis. Activated protein C (aPC) consumes plasminogen activator inhibitor-1 (PAI-1) when present in excess, and reduced PAI-1 leads to increased tPA activity and hyperfibrinolysis. FDPs, fibrin degradation products; PC, protein C; TM, thrombomodulin. (Reproduced, with permission, from Brohi K, Cohen MJ, Davenport RA: Acute coagulopathy of trauma: Mechanism, identification and effect. Curr Opin Crit Care 2007;13:680.)

frozen plasma:platelet), is termed *damage control resuscitation*. Although the 1:1:1 combination attempts to replicate whole blood, it results in a pancytopenic solution with only a fraction of whole blood's hematocrit and coagulation factor concentration. Red blood cells will over time improve oxygen delivery to ischemic, hypoperfused tissues. Fresh frozen plasma provides clotting factors V and VIII along with fibrinogen, which improves clotting, possibly due to overwhelming of the thrombin–thrombomodulin complex. Platelets and cryoprecipitate, although included in the 1:1:1 massive

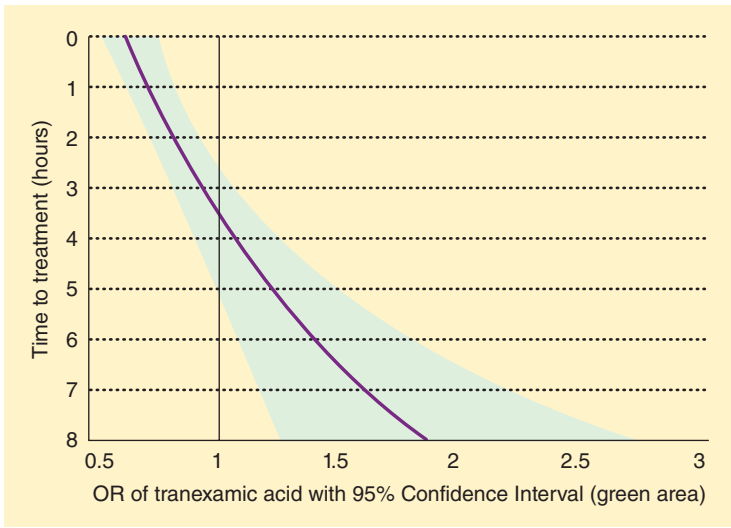

FIGURE 39–4 Influence of tranexamic acid in preventing death from bleeding. Outcomes ratios (OR) of tranexamic acid with 95% confidence interval (*green area*) on the *x*-axis and time (h) to treatment on the *y*-axis demonstrate improved survival if tranexamic acid therapy is initiated within 3 h of injury. The area of the curve to the left of OR 1.0 demonstrates the benefits of therapy, while that to the right demonstrates harm from intervention. (Reproduced, with permission, from Roberts I, Shakur H, Afolabi A, et al: The importance of early treatment with tranexamic acid in bleeding trauma patients: An exploratory analysis of the CRASH-2 randomised controlled trial. Lancet 2011;377:1096.)

transfusion protocol, are probably not necessary in the initial phase of resuscitation, given the normal platelet and fibrinogen levels noted in early coagulopathy. Additional platelet transfusions may be beneficial if the resuscitation is prolonged, as is typical for most major trauma resuscitations, or if a recalcitrant coagulopathy is noted with coagulation studies. The use of crystalloid fluids in early trauma resuscitation has markedly decreased with the increased emphasis upon early blood product administration.

Most trauma centers have early-release type O-negative blood available for immediate transfusion to patients with severe hemorrhage. Depending on the urgency of need for transfusion, administration of blood products typically progresses from O-negative to type-specific, then to crossmatched units as the acute need decreases. Patients administered uncrossmatched O-negative blood are those deemed at high risk of requiring massive transfusion. As the amount of uncrossmatched blood administered increases beyond 8 units, attempts to return to the patient's native blood type should not be pursued and type O blood should be continued until the patient is stabilized.

Military experience treating combat-wounded soldiers and civilians has provided great insight into trauma resuscitation and trauma-induced coagulopathy. As the use of blood and blood products has evolved, the 1:1:1 transfusion ratio has been uniformly adopted to address the frequent incidence of trauma-induced coagulopathy. Retrospective analysis of severely wounded solders found improved survival when this transfusion protocol was utilized. Consequently, hemostatic resuscitation has been rapidly adopted by civilian trauma centers, which have reported similar survival benefits for civilian patients with severe trauma. Nevertheless, using traditional definitions, this approach is not "evidence based" from randomized clinical trials.

Using hemostatic resuscitation (ie, damage control resuscitation), blood and blood products are administered preemptively to address a presumed coagulopathy. Often coagulation status is not assessed until the patient stabilizes. Although this treatment approach appears to be effective in controlling trauma-induced coagulopathy, patients requiring this therapy may be exposed to unnecessary additional units of blood or blood products. An alternative approach that relies on thromboelastography (TEG) may allow more goal-directed transfusion of blood and blood products and is increasingly utilized in trauma resuscitations. The formation and stability of a clot represents interactions between the coagulation cascades, platelets, and the fibrinolytic system, all of which can be demonstrated with TEG (**Figure 39–5**). As TEG use during trauma resuscitation becomes more routine, the current 1:1:1 hemostatic resuscitation ratio will likely undergo modification to proportionately less fresh frozen plasma, and the use of antifibrinolytic therapy will likely increase.

Administration of blood products must be done with consideration for potential hazards that may result from transfusion. Although blood-borne diseases such as acquired immunodeficiency syndrome, hepatitis B, and hepatitis C are usually thought of as the highest transfusion-related risks, the incidence of such infections has decreased 10,000-fold due to better screening tests of donors and donated units (see Chapter 51). Noninfectious transfusion reactions are now the leading complication of transfusion and represent a more than 10-fold greater risk than blood-borne infection. Transfusion-related acute lung injury (TRALI) is the leading cause of transfusion-related death reported to the U.S. Food and Drug Administration. However, although the bleeding trauma patient is at risk for a transfusion-related reaction, that risk is minimal compared with the far greater likelihood of death from exsanguination. The most prudent approach for blood product utilization in the bleeding trauma patient is to administer the blood products that are necessary, based on laboratory studies, clinical evidence of significant bleeding, and the degree of hemodynamic instability that can be directly attributed to hemorrhage.

Massive Transfusion Protocols

Delay in obtaining blood products other than red blood cells is common in both civilian and military settings. Clinical evidence supports the need for, and benefit of, established massive transfusion protocols (MTPs), allowing the blood bank to assemble blood

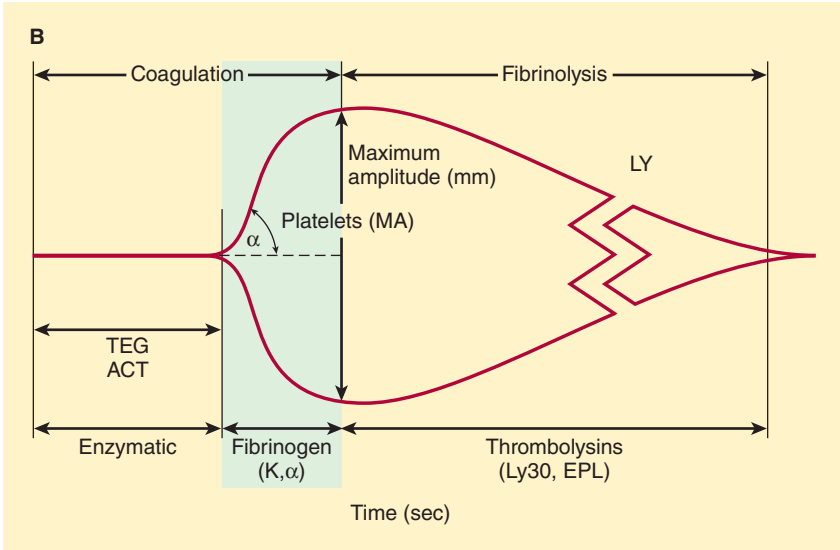

FIGURE 39–5 Thromboelastograph (TEG). The graph begins as a straight line until clot formation begins (the enzymatic stage of clotting). As a clot forms, increasing resistance develops on the strain gauge, creating a splaying of the graph. The pattern of the graph suggests the status of fibrinogen stores (α angle) and platelet function (maximum amplitude, MA). Eventually, fibrinolysis will occur as demonstrated by decreasing MA. Deficiencies of various clotting components will affect each phase of the TEG whereas increased fibrinolysis will be demonstrated by an earlier decline in the maximum amplitude. ACT, activated clotting time; EPL, Ly30, K, R, values related to rate of clot breakdown. (Reproduced, with permission, from Kashuk JL, Moore EE, Sawyer M, et al: Postinjury coagulopathy management: Goal directed resuscitation via POC thrombelastography. Ann Surg 2010;251:604.)

products in prescribed ratios to support hemostatic resuscitation. With MTPs in place, hemostatic resuscitation can continue until the demand for blood products stops. An MTP-driven, blood-based resuscitation, rather than a crystalloid-based resuscitation, improves survival from trauma, reduces total blood product utilization in the first 24 h following injury, reduces acute infectious complications (severe sepsis, septic shock, and ventilator-associated pneumonia), and decreases postresuscitation organ dysfunction (an 80% decrease in odds of developing multisystem organ failure).

It is important to establish which personnel are empowered to invoke use of the MTP, given the expense and implications for the blood bank in terms of blood inventory, personnel training and availability, and disruption of routine blood bank duties. Establishing an MTP benefits both the patient, through improved survival and fewer complications, and the institution, through more efficient and effective processes for utilizing critical blood bank resources.

Initiating an MTP for all trauma patients is impractical; however, delaying request for an MTP until the patient has undergone a thorough trauma evaluation may increase the risk of morbidity and mortality. The *assessment of blood consumption* (ABC) score is an attempt to predict which patients are likely to require an MTP. The ABC score assigns 1 point for the presence of each of four possible variables: (1) penetrating injury; (2) systolic blood pressure less than 90 mmHg; (3) heart rate greater than 120 beats per minute; and (4) positive results of a *focused assessment with sonography for trauma* (FAST) evaluation. The FAST evaluation is a bedside ultrasonography screening examination performed by surgeons and emergency department physicians to assess the presence or absence of free fluid in the perihepatic and perisplenic spaces, pericardium, and pelvis.

Patients with ABC scores of 2 or higher are likely to require massive transfusion. This scoring system has been validated in multiple level 1 trauma centers and is now relatively commonplace in trauma evaluations.

DEFINITIVE TRAUMA INTERVENTIONS

The physical examination, emergency procedures, and evaluations used to determine the extent of injury, need for an MTP, and surgical intervention all occur outside the operating room. The decision to proceed to the operating room may be the first point in the trauma resuscitation process at which an anesthesiologist is involved. Key issues in the anesthetic management of trauma patients include the need to avoid vasopressors and minimize crystalloid infusions until bleeding is controlled. Blood products are the fluids of choice for trauma resuscitation.

Anesthetic Induction & Maintenance

Conscious and oriented trauma patients arriving for emergent surgery should have an abbreviated interview and examination, including emphasis on consent for blood transfusions and advice that intraoperative awareness may occur during emergency surgery. This discussion should be documented in the patient's record.

The operating room should be as warm as is practical. Intravenous fluid warmers and rapid infusion devices should be used. All patients arriving for trauma surgery should be presumed to have full stomachs and thus to be at increased risk for aspiration. As noted earlier, the presence of a C-collar may increase the difficulty of intubation. Accordingly, robust suction equipment and alternative airway devices (eg, fiberoptic bronchoscopes, videolaryngoscopes) should be immediately available for use.

Intravenous access is usually established in the prehospital setting or in the emergency department. If the existing peripheral intravenous lines are of sufficient caliber and quality for infusing blood under pressure (ie, a 16-gauge or 14-gauge catheter), a central line is usually not necessary for the initial surgical intervention. Patients may arrive in the operating room so profoundly hypotensive and hypovolemic that peripheral intravenous access is impossible. In this circumstance, a subclavian or an intraosseous catheter should be inserted and blood-based resuscitation initiated. The subclavian vein is often preferred for central venous access in profoundly hypotensive patients owing to its position between the first rib and the clavicle, which tends to stent the vein open. An intraosseous catheter is usually seated into the bone marrow of the proximal tibia or humerus, a process that is facilitated by use of a bone drilling device. Use of intraosseous access requires that the bone distal to the intraosseous catheter to be intact; otherwise; extravasation of infused fluid through the fracture site, the path of least resistance, will occur. A pressure bag must be used for infusing any fluid through the intraosseous catheter due to resistance to passive flow from the bone marrow, although the intraosseous space is intimately connected with the venous system and transfused blood readily enters the central circulation via this route.

Major blood loss and hemodynamic instability create a dangerous situation for the conscious trauma patient and a challenging decision for the anesthesiologist planning the induction of general anesthesia. Trauma patients with severe injuries are poor candidates for induction with propofol, given the likelihood of profound hypotension following even modest doses (0.25–0.5 mg/kg intravenously). Etomidate preserves sympathetic tone, which makes it a modestly safer choice than propofol. Ketamine is also a reasonable choice, particularly if given in 10-mg intravenous boluses until the patient becomes unresponsive. Scopolamine, 0.4 mg intravenously, should be considered as an amnestic agent for the hemodynamically unstable but conscious patient at high risk for hemodynamic collapse on induction of anesthesia who arrives in the operating room for emergency surgery. What is most important is not the particular intravenous anesthetic induction agent chosen, but recognition that the hemodynamically unstable trauma patient will require significantly less anesthetic medication than in normal circumstances.

An arterial line will be helpful but insertion may prove difficult in the hypotensive, hypoperfused trauma patient. Attempts at placing invasive monitors can continue as the patient is prepped for

surgery and the surgeon begins the operation. If halted, attention should focus on transfusion-related efforts.

Damage Control Surgery

If the trauma patient requires emergent laparotomy for intraabdominal hemorrhage, the trauma surgeon will perform an abbreviated procedure termed *damage control surgery* (DCS), which is intended to stop hemorrhage and limit gastrointestinal contamination of the abdominal compartment. After making a midline incision, the surgeon quickly searches for sources of bleeding through a quadrant-by-quadrant examination. Communication between the surgeon and the anesthesiologist is essential in DCS; the surgeon must know if the patient is becoming unstable, hypothermic, or coagulopathic in spite of ongoing resuscitation during the operative procedure. The surgeon will usually compress or pack the area of bleeding if the patient is hypotensive, an intervention that usually improves hemodynamics by slowing hemorrhage and allowing more rapid restoration of circulating blood volume. If direct compression of the hemorrhaging intraabdominal tissue fails to improve hemodynamic stability, the surgeon can also slow the rate of hemorrhage by compressing the aorta. Compression of the aorta also provides tactile information to the surgeon. Particularly in circumstances where invasive arterial monitoring was not accomplished, the surgeon's fingers on the aortic pulse can provide useful information regarding volume status: a soft, compressible aorta represents profound hypovolemia, whereas a firm, pulsatile aorta suggests more normal volume status.

Definitive repair of complex injuries is not part of DCS. Identification and control of injured blood vessels and solid organs, as well as inspection of injuries in areas relatively inaccessible to midline approaches (eg, deep liver lacerations, retroperitoneal hemorrhage) but potentially amenable to interventional radiology techniques, occurs during DCS laparotomy. Hollow viscus injuries are addressed with resection or stapling, or both, to prevent abdominal contamination, often leaving the intestines disconnected until the patient is more stable. At that later time, bowel continuity can be restored or colostomy can be performed. At any time during DCS, if the patient becomes unstable or profoundly hypothermic, or if transfusions are insufficient in maintaining perfusion, the operation should be interrupted, the bleeding areas packed, and a decision should be made as to whether the patient can be transferred to the interventional radiology suite to treat bleeding from surgically inaccessible sites or transferred to the intensive care unit to allow warming, treatment of hemodynamic or hemostatic abnormalities, and continuation of resuscitation.

The interventional radiology suite is increasingly utilized as part of the DCS sequence, because interventional radiology techniques can reach essentially any bleeding vessel and deposit coils or foam to control hemorrhage. Most notably, liver, kidney, and retroperitoneal injuries, pelvic ring fractures, and major thoracic and abdominal vascular injuries are potentially controlled by interventional radiology procedures. Following DCS, trauma patients will frequently be transferred to the interventional radiology suite to assess blood flow and hemostasis of organs either injured by the initial trauma or potentially compromised as part of the DCS.

TRAUMATIC BRAIN INJURY

8 Any trauma patient with altered level of consciousness must be considered to have a traumatic brain injury (TBI) until proven otherwise (see Chapter 27). The presence or suspicion of a TBI mandates attention to maintaining cerebral perfusion and arterial oxygenation during all aspects of care. The most reliable clinical assessment tool in determining the significance of TBI in a nonsedated, nonparalyzed patient is the Glasgow coma scale (GCS, Table 27–2). A declining motor score is suggestive of progressing neurological deterioration, prompting urgent neurosurgical evaluation and possible surgical intervention. Although trauma patients frequently have head injuries, few head injuries require emergent neurosurgical intervention.

TBIs are categorized as either *primary* or *secondary*. Primary brain injuries are usually focal injuries directly related to trauma, disrupting normal anatomy or physiology, or both. Four categories of primary brain injury are seen: (1) subdural

hematoma; (2) epidural hematoma; (3) intraparenchymal hemorrhage; and (4) nonfocal, diffuse neuronal injury disrupting axons of the central nervous system. These injuries potentially compromise cerebral blood flow and elevate intracranial pressure (ICP). Death occurring soon after significant head trauma is usually a result of the primary brain injury.

9 **Acute subdural hematoma** is the most common condition warranting emergency neurosurgery and is associated with the highest mortality. Small bridging veins between the skull and brain are disrupted in deceleration or blunt force injuries, resulting in blood accumulation and compression of brain tissue. The accumulation of blood raises ICP and compromises cerebral blood flow. Morbidity and mortality are related to the size of the hematoma and magnitude of the midline shift of intracranial contents. Midline shifts of intracranial contents may exceed the size of the hematoma, suggesting a significant contribution of cerebral edema. Acute subdural hematomas should be surgically evacuated, particularly in patients with elevated ICP.

Epidural hematoma occurs when the middle cerebral artery or other cranial vessels are disrupted, most often in association with a skull fracture. This injury accounts for less than 10% of neurosurgical emergencies and has a much better prognosis than acute subdural hematoma. The patient with an epidural hematoma may initially be consciousness, followed by progressive unresponsiveness and coma. Emergent surgical decompression is indicated when supratentorial lesions occupy more than 30 mL volume and infratentorial lesions occupy more than 10 mL volume (brainstem compression may occur at much lower hematoma volumes). A small epidural hematoma may not require immediate evacuation if the patient is neurologically intact, if close observation and repeated neurological examinations are possible, and if neurosurgical resources are available should emergent decompression become necessary.

Intraparenchymal injuries are caused by rapid deceleration of the brain within the skull, usually involving the tips of the frontal or temporal lobes. They represent nearly 20% of neurosurgical emergencies following trauma. These injuries tend to be associated with significant edema, necrosis, and infarcts in the tissue surrounding the damaged tissue. Intraparenchymal injury may coexist with a subdural hematoma. There is no consensus regarding the surgical interventions that should be performed for intraparenchymal hemorrhage, but surgical decompression may be necessary to reduce dangerously sustained increased ICP.

Diffuse neuronal injury results from events resulting in rapid deceleration or movement of the brain tissue of sufficient force to disrupt neurons and axons. This form of brain injury is more common in children than in adults. The extent of the injury may not be obvious in the period soon after injury but will become apparent with serial clinical and radiographic (magnetic resonance imaging) examinations. The greater the extent of diffuse neuronal injury following trauma, the higher will be the mortality and severe disability. Surgical interventions are not indicated for these injuries unless a decompressive craniectomy is required for relief of refractory elevated ICP (see below).

Secondary brain injuries are considered potentially preventable injuries. Systemic hypotension (systolic blood pressures <90 mm Hg), hypoxemia (Pa_{O_2} <60 mm Hg), hypercapnia (Pa_{CO_2} >50 mm Hg), and hyperthermia (temperature >38.0°C) have a negative impact on morbidity and mortality following head injuries, likely because of their contributions to increasing cerebral edema and ICP. Hypotension and hypoxia are recognized as major contributors to poor neurological recovery from severe TBI. Hypoxia is the single most important parameter correlating to poor neurological outcomes following head trauma and should be corrected at the earliest possible opportunity. Hypotension (mean arterial blood pressure <60 mm Hg) should also be treated aggressively, using fluid or vasopressors, or both, to assure cerebral perfusion.

10

Management Considerations

A. Intracranial Pressure

In the absence of a clot requiring evacuation, medical interventions are the primary means of treating elevated ICP following head trauma.

Normal cerebral perfusion pressure (CPP), the difference between mean arterial pressure (MAP, discussed in Chapter 26) and ICP (ie, MAP – ICP = CPP), is approximately 10 mm Hg. ICP monitoring is not required for conscious and alert patients; in addition, patients who are intentionally anticoagulated or who have bleeding diathesis in response to trauma should not have ICP monitoring. However, an ICP monitor should be placed when serial neurological examinations and additional clinical assessments reveal impairment, or when there is an increased risk for elevated ICP (Table 39–1). Interventions to reduce ICP are indicated when readings are higher than 20–25 mm Hg. Although multiple studies have evaluated interventions aimed at improving CPP and managing ICP without finding obvious outcomes benefit for any treatment scheme, current Brain Trauma Foundation guidelines recommend maintaining CPP between 50 and 70 mm Hg and ICP at less than 20 mm Hg for patients with severe head injury.

TABLE 39–1 Indications for intracranial ICP monitoring.[1,2]

Severe head injury (defined as GCS score ≤8 after cardiopulmonary resuscitation) *plus* (a) Abnormal admitting head CT scan *or* (b) Normal CT scan plus ≥2 of: age >40 y, systolic blood pressure >90 mm Hg, decerebrate or decorticate position
Sedated patients; patient in induced coma after severe TBI
Multisystem injury with altered level of consciousness
Patient receiving treatment that increases risk of increased ICP, eg, high-volume IV fluids
Postoperatively after removal or intracranial mass
Abnormal values in noninvasive ICP monitoring, increased dynamics of simulated values, or abnormal shapes in transcranial Doppler blood flow velocity waveform (increased pulsatility) with exclusion of arterial hypotension and hypocapnia

[1]ICP, intracranial pressure; GCS, Glasgow Coma Scale; CT, computed tomographic; TBI, traumatic brain injury.

[2]Reproduced, with permission, from Li LM, Timofeev I, Czosnyka M, et al: Review article: The surgical approach to the management of increased intracranial pressure after traumatic brain injury. Anesth Analg 2010;111:736.

Cerebral blood flow is related to arterial carbon dioxide concentration in a dose-dependent relationship. As arterial carbon dioxide levels decrease, cerebral vasoconstriction occurs, reducing ICP. Conversely, as arterial carbon dioxide levels rise, cerebral vasodilation occurs, increasing ICP. Changes in arterial carbon dioxide levels exert a prompt cerebral blood flow and ICP response, making hyperventilation an effective intervention when brain herniation is suspected or proven. However, this intervention must be appreciated in the context of TBI: hyperventilation in the presence of systemic hypotension increases the risk of neurological ischemia and should be avoided in the early stages of resuscitation for patients with TBI.

Osmotic diuretic therapy is another commonly used and widely accepted method for reducing elevated ICP. Intravenous mannitol doses of 0.25–1.0 g/kg body weight are effective in drawing intravascular fluid into the vascular system. As extravascular fluid is drawn into the vascular system, brain edema and ICP will decrease. Because this intervention is very effective for inducing brisk diuresis, serum osmolarity and electrolytes (particularly potassium) must be monitored.

Barbiturate coma is an intervention that attempts to decrease cerebral metabolic rate, cerebral blood flow, and cerebral oxygen demand in order to reduce elevated ICP and suppress the metabolic rate of ischemic cells until cerebral perfusion improves. Hypotension is commonly associated with this therapy, which should limit its use in the hemodynamically unstable patient. Vasopressors may be used in order to maintain CPP between 50 and 70 mm Hg. The pentobarbital dose administered is based upon electroencephalographic evidence of burst suppression in order to maximally reduce the cerebral metabolic rate of oxygen.

B. Severe TBI & Multiple Trauma

The presence of a severe head injury in the presence of other major traumatic injuries and ongoing hemorrhage creates a situation in which patient management goals may conflict. As noted above, in the head-injured patient requiring emergent decompression, mean blood pressures must be maintained between 50 and 70 mmHg to assure

adequate CPP and prevention of secondary ischemic neurological injuries. In patients without brain injury, hemorrhage is usually treated with a more hypotensive goal until bleeding is controlled. Deference is paid to the most life-threatening condition as the priority intervention with the expectation that CPP be maintained throughout, even if this approach results in greater transfusion requirements.

SPINAL CORD INJURY

The normal spine comprises three columns: anterior, middle, and posterior. The anterior column includes the anterior two thirds of the vertebral body and the anterior longitudinal ligament. The middle column includes the posterior third of the vertebral body, the posterior longitudinal ligament, and the posterior component of the annulus fibrosis. The posterior column includes the laminae and facets, the spinous processes, and the interspinous ligaments. Spine instability results when two or more of the three columns are disrupted. The trauma patient with a relevant mechanism of injury (typically blunt force involving acceleration–deceleration) must be approached with a high degree of suspicion for spine injury unless it has been ruled out radiographically.

A lateral radiograph of the cervical spine demonstrating the entire cervical spine to the top of the T1 vertebra will detect 85–90% of significant cervical spine abnormalities. Cervical spine radiographs should be examined for the appearance and alignment of the vertebral bodies, narrowing or widening of interspinous spaces and the central canal, alignment along the anterior and posterior ligament lines, and appearance of the spinolaminar line and posterior spinous processes of C2 through C7. The presence of one spinal fracture is associated with a 10–15% incidence of a second spinal fracture.

Thoracolumbar injuries most commonly involve the T11 through L3 vertebrae as a result of flexion forces. The presence of one thoracolumbar spinal injury is associated with a 40% chance of a second fracture caudal to the first, likely due to the force required to fracture the lower spine. Bilateral calcaneus fractures also warrant a thorough thoracolumbar spine evaluation due to the increased incidence of associated spinal fractures associated with this injury pattern.

Cervical spine injuries occurring above C2 are associated with apnea and death. Lesions of C3–5 impact phrenic nerve function, impairing diaphragmatic breathing. High spinal injuries are often accompanied by neurogenic shock due to loss of sympathetic tone. Neurogenic shock may be masked initially in major trauma because hypotension may be attributed to a hemorrhagic, rather than a neurologic, cause. The presence of profound bradycardia 24–48 h after a high thoracic spinal cord lesion likely represents compromise of the cardioaccelerator function found in the T1–4 region.

The principal therapeutic objectives following spinal cord injury are to prevent exacerbation of the primary structural injury and to minimize the risk of extending neurological injury from hypotension-related hypoperfusion of ischemic areas of the spinal cord. In patients with complete spinal cord transection, very few interventions will influence recovery. In patients with incomplete spinal cord lesions, careful management of hemodynamic parameters and surgical stabilization of the spine are critical in preventing extension of the existing injury.

Methylprednisolone is often administered for spinal cord injury to reduce spinal cord edema in the tight confines of the spinal canal, although there is scant evidence that this intervention improves outcomes following spinal cord injury in humans. While not considered a standard of care, it is included in the current clinical recommendations of the American Association of Neurological Surgeons as a treatment option. Maintaining supranormal mean arterial blood pressures to assure spinal cord perfusion in areas of reduced blood flow due to cord compression or vascular compromise is likely to be of more benefit than steroid administration. Hypotension must be avoided during induction of anesthesia and throughout surgical decompression and stabilization of a spinal injury.

Surgical decompression and stabilization of spinal fractures are indicated when a vertebral body loses more than 50% of its normal height or the spinal canal is narrowed by more than 30% of its

normal diameter. Despite outcome studies from animal models of traumatic spinal cord injury demonstrating benefit from early surgical intervention or steroid therapy, or both, current human studies have failed to demonstrate significant benefit from either intervention. Currently, the presence of a decompressible lesion in the area of an incomplete spinal cord transection is not an indication for early operative intervention unless other, more life-threatening, conditions are present.

The elderly are at greater risk for spinal cord injury due to decreased mobility and flexibility, a higher incidence of spondylosis and osteophyte formation in the degenerative spine, and decreased intracanal space accommodating spinal cord edema following cord trauma. The incidence of spinal injury from falls in the elderly is rapidly approaching that of spinal cord injury from motor vehicle accidents in younger patients. Mortality following spinal cord injury in the elderly, particularly those over the age of 75 years, is higher than that in younger counterparts with similar injury.

The unique injury pattern of penetrating spinal cord injury warrants consideration. Unlike blunt spinal trauma, penetrating trauma of the spinal cord due to bullets and shrapnel is unlikely to induce an unstable spine. As a result, C-collar and longboard immobilization may not be indicated. In fact, C-collar placement in the presence of a cervical spine penetrating injury may hinder observation of soft tissue swelling, tracheal deviation, or other anatomic indications of imminent airway compromise. Unlike blunt trauma, penetrating injuries of the spinal cord induce damage at the moment of injury without risk of subsequent exacerbation of the injury. Like other spinal cord injuries, however, maintenance of spinal cord perfusion using supranormal mean arterial pressures is indicated until spinal cord function can be more fully evaluated.

BURNS

Burns represent a unique but common traumatic injury that is second only to motor vehicle accidents as the leading source of accidental death. Temperature and duration of heat contact determine the extent of burn injury. Children (because of a high body surface area to body mass ratio) and the elderly (whose thinner skin allows deeper burns from similar thermal insult) are at greater risk for major burn injury. The pathophysiological and hemodynamic responses to burn injuries are unique and warrant specialized burn care that can be optimally provided only at burn treatment centers, particularly when more than 20% of a patient's body surface area is involved in second- or third-degree burns. A basic understanding of burn pathophysiology and of resuscitation requirements, especially early initiation of therapies such as oxygen administration and aggressive fluid resuscitation, will improve patient survival.

Burns are classified as first, second, or third degree. *First-degree* burns are injuries that do not penetrate the epidermis (eg, sunburns and superficial thermal injuries). Fluid replacement for these burns is not necessary, and the area of first-degree burns should not be included in calculating fluid replacement requirements when extensive, more significant burns are also present. *Second-degree* burns are partial-thickness injuries (superficial or deep) that penetrate the epidermis, extend into the dermis for some depth, and are associated with blistering. Fluid replacement therapy is indicated for patients with second-degree burns when more than 20% of total body surface area (TBSA) is involved. Skin grafting also may be necessary in some cases of second-degree burns, depending upon size and location of the wounds. *Third-degree* burns are those in which the thermal injury penetrates the full thickness of the dermis. Nerves, blood vessels, lymphatic channels, and other deep structures may have been destroyed, creating a severe, but insensate, wound (although surrounding tissue may be very painful). Debridement and skin grafting are nearly always required for recovery of patients from third-degree burns.

13 Major burns (a second- or third-degree burn involving >20% TBSA) induce a unique hemodynamic response. Cardiac output declines by up to 50% within 30 minutes in response to massive vasoconstriction, inducing a state of normovolemic hypoperfusion (*burn shock*). Survival depends on restoration of circulating volume and infusion of crystalloid fluids according to recommended protocols (see below). This intense hemodynamic response may be poorly tolerated by patients with

significant underlying medical conditions. If intravenous fluid therapy is provided, cardiac function returns to normal within 48 h of the injury, then typically progresses to a hyperdynamic physiology as the metabolic challenge of healing begins. Plasma volume and urine output are also reduced early on after major burn injuries.

14 In contrast to fluid management for blunt and penetrating trauma, which discourages use of crystalloid fluids, burn fluid resuscitation emphasizes the use of crystalloids, particularly lactated Ringer's solution, in preference to albumin, hydroxyethyl starch, hypertonic saline, and blood. Following burn injuries, kidney failure is more common when hypertonic saline is used during initial fluid resuscitation, death is higher when blood is administered, and outcomes are unchanged when albumin is used in resuscitation.

Fluid resuscitation is continuous over the first 24 h following injury. Two formulas are commonly used to guide burn injury fluid resuscitation, the Parkland and the modified Brooke. Both require an understanding of the so-called *rule of nines* (**Figure 39–6**) to calculate resuscitation volumes. The (adult) *Parkland* protocol recommends 4 mL/kg/% TBSA burned to be given in the first 24 h, with half the volume given in the first 8 h and the remaining amount over the following 16 h. The (adult) *modified Brooke* protocol recommends 2 mL/kg/% TBSA, with administration of half the calculated volume beginning in the first 8 h and the remainder over the following 16 h. Both formulas use urine output as a reliable indicator of fluid resuscitation, targeting (adult) urine production of 0.5–1.0 mL/kg/h as indications of adequate circulating volume. If adult urine output exceeds 1.0 mL/kg/h, the infusion is slowed. In both protocols, an amount equal to half the volume administered in the first 24 h is infused in the second 24-h period following injury, with continued attention to maintaining adult urine output at 0.5–1.0 mL/kg/h. The formula for fluid resuscitation of children is the same as that for adults, but children weighing less than 30 kg should receive 5% dextrose in Ringer's lactate as their resuscitation fluid and target urine output should be 1.0 mL/kg/h. The target urine output for infants younger than 1 year of age is 1–2 mL/kg/h.

Management Considerations

The Parkland and modified Brooke protocols both use urine output as an indicator for adequate fluid resuscitation. However, circumstances may arise in which the volume of fluid administered exceeds the intended volumes. For example, initial fluid resuscitation volumes may be miscalculated if first-degree burns are mistakenly incorporated into the TBSA value. Prolonged use of sedatives and sedative infusions may also result in hypotension that is treated with additional fluids rather than vasoconstrictors. The phenomenon of *fluid creep* occurs when intravenous fluid therapy volumes are increased beyond intended calculations in response to various hemodynamic changes. Fluid creep is associated with abdominal compartment syndrome and pulmonary complications, which represent resuscitation morbidity.

A. Abdominal Compartment Syndrome

Abdominal compartment syndrome is a risk for pediatric patients, adults with circumferential abdominal burns, and patients receiving intravenous fluid volumes greater than 6 mL/kg/% TBSA. Intraabdominal pressure can be determined by measuring intraluminal bladder pressure using a Foley catheter. The transducer is connected to a 3-way stopcock at the point where the Foley catheter connects to the drainage tube. After the transducer is zeroed at the pelvic brim, 20 mL of fluid is instilled to distend the bladder. Intraabdominal pressure readings are taken 60 s after fluid installation, allowing the bladder to relax. Intraabdominal pressures exceeding 20 mmHg warrant abdominal cavity decompression. However, an abdominal surgical procedure places the burn patient at high risk for intraabdominal *Pseudomonas* infection, particularly if the laparotomy incision is near burned tissue.

B. Pulmonary Complications

Excessive resuscitative fluid volumes are associated with an increased incidence of pneumonia. Patients with severe burns frequently have pulmonary injury related to the burn. Decreased tracheal ciliary activity, the presence of resuscitation-induced pulmonary edema, reduced immunocompetence, and tracheal intubation predispose the burn patient to

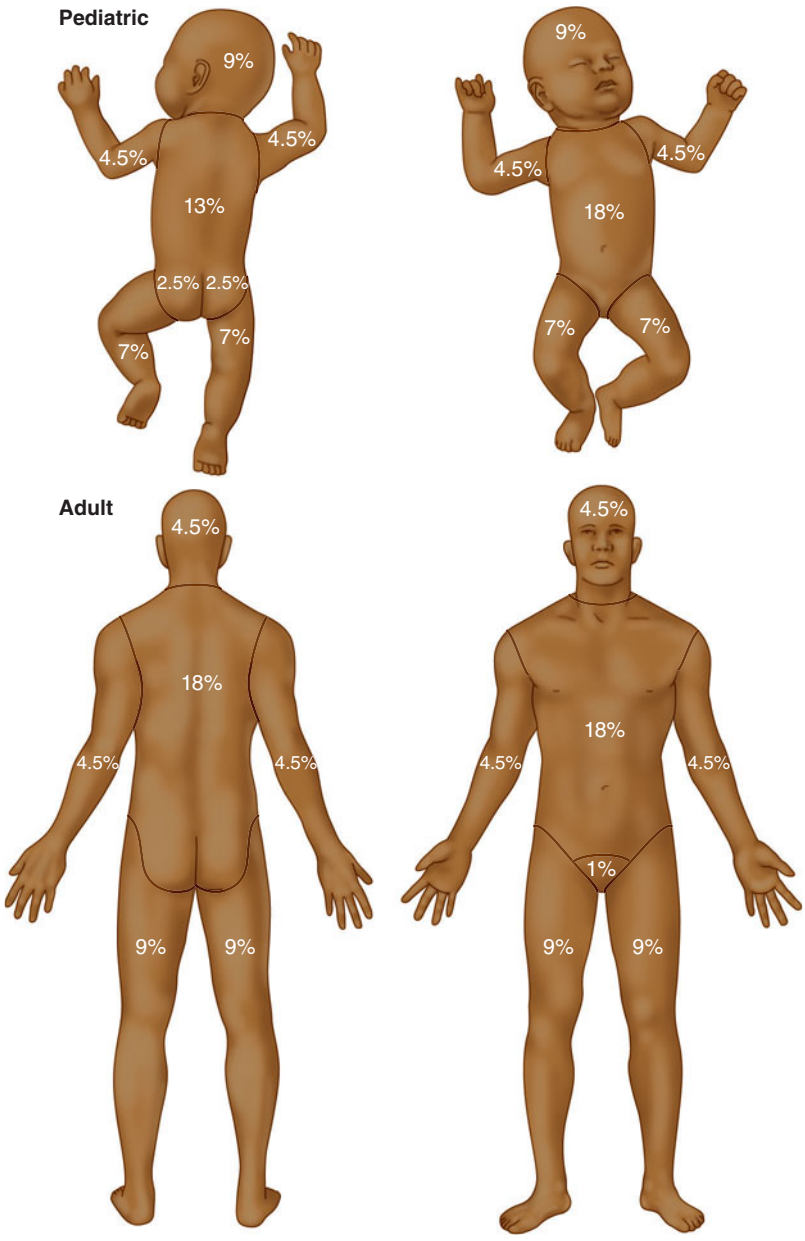

FIGURE 39–6 The rule of nines, utilized to estimate burned surface area as a percentage of total body surface area (TBSA). (Reproduced, with permission, from American College of Surgeons: *ATLS: Advanced Trauma Life Support for Doctors (Student Course Manual)*, 9th ed. ACS, 2012.)

pneumonia. Abdominal compartment syndrome can have an adverse impact on pulmonary function. Intravenous fluid administration volumes must be monitored closely and documented to be consistent with American Burn Association recommendations (ie, the Parkland or modified Brooke protocol). Fluid administration that exceeds recommendations warrants careful review of the rationale for the increased fluid therapy volume, including assessment of possible causes for hypotension (eg, sepsis) or reduced urine output (eg, abdominal compartment syndrome).

C. Carbon Monoxide Poisoning

15 Carbon monoxide poisoning should be considered in all serious burn injury cases, as well as with lesser TBSA burns occurring in enclosed spaces. Unconsciousness or decreased levels of consciousness following burn injuries should be presumed to represent carbon monoxide poisoning, prompting endotracheal intubation and mechanical ventilation with high inspired concentration oxygen therapy. Carbon monoxide binds to hemoglobin with an affinity approximately 250 times that of oxygen. The resultant carboxyhemoglobin (HbCO) leaves less hemoglobin available to bind with oxygen (HbO_2) and shifts the O_2–Hb dissociation curve to the left; both of these processes result in impaired availability of oxygen molecules at the local tissue level. Pulse oximetry provides a falsely elevated indication of oxygen saturation in the setting of carbon monoxide exposure because of its inability to distinguish between HbO_2 and HbCO. If carbon monoxide poisoning is suspected, HbCO can be directly measured via arterial or venous blood gas analysis. HbCO concentrations below 10% are usually not clinically significant. However, with high inspired oxygen concentrations, HbCO levels of 20% correspond to a hemoglobin oxygen saturation of 80%; intubation and mechanical ventilation is indicated in such circumstances to improve local tissue oxygenation and enhance carbon monoxide elimination. Death from carbon monoxide poisoning occurs at HbCO levels of 60%.

Anesthetic Considerations

A primary characteristic of all burn patients is an inability to regulate temperature. The resuscitation environment must be maintained near body temperature through the use of radiant warming, forced air warming devices, and fluid warming devices.

Assessment of the patient begins with inspection of the airway. Although the face may be burned (singed facial hair, nasal vibrissae), facial burns are not an indication for tracheal intubation. The need for urgent airway management, mechanical ventilation, and oxygen therapy is indicated by hoarse voice, dyspnea, tachypnea, or altered level of consciousness. Arterial blood gases should be obtained early in the treatment process to assess HbCO levels. Mechanical ventilation should be adjusted to afford adequate oxygenation at the lowest tidal volumes.

Tracheal intubation in the early period following burn injury (up to the first 48 h) can be facilitated with succinylcholine for paralysis. In patients with significant burns (>20% TBSA), injuries and disruption of neuromuscular end plates occur followed by upregulation of acetylcholine receptors. 16 Beyond 48 h after a major burn, succinylcholine administration is likely to produce potentially lethal elevation of serum potassium levels.

Analgesia for burn patients is challenging because of concerns about opioid tolerance and psychosocial complications. Multimodal approaches are often advantageous. Regional analgesia may provide benefit, although in the early postburn period this technique may mask the symptoms of compartment syndrome or other clinical signs and symptoms.

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