BOX 66.2 Causes of Obstructed Airway or Inadequate Ventilation in a Trauma Patient

Airway Obstruction

Direct injury to the face, mandible, or neck
Hemorrhage in the nasopharynx, sinuses, mouth, or upper airway
Diminished consciousness secondary to traumatic brain injury, intoxication, or analgesic medications
Aspiration of gastric contents, blood, or a foreign body (i.e., dentures, broken teeth, soft tissue)
Misapplication of oral airway or endotracheal tube (esophageal intubation)

Inadequate Ventilation

Diminished respiratory drive secondary to traumatic brain or high cervical spine injury, shock, intoxication, hypothermia, or oversedation
Direct injury to the trachea or bronchi
Pneumothorax or hemothorax
Chest wall injury
Aspiration
Pulmonary contusion
Cervical spine injury
Bronchospasm secondary to smoke or toxic gas inhalation

BOX 66.3 Signs and Symptoms of Shock

Pallor
Diaphoresis
Agitation or obtundation
Hypotension
Tachycardia
Prolonged capillary refill
Diminished urine output
Narrowed pulse pressure

TABLE 66.1 Diagnostic and Therapeutic Options for Management of Traumatic Hemorrhage

Site of Bleeding	Diagnostic Modalities	Treatment Options
Chest	Chest x-ray Thoracostomy tube output Chest CT	Observation Surgery
Abdomen	Physical examination Ultrasound (FAST) Abdominal CT Peritoneal lavage	Surgical ligation Angiography Observation
Retroperitoneum	CT Angiography	Angiography
Long bones	Physical examination Plain x-rays	Fracture fixation Surgical ligation
Outside the body	Physical examination	Direct pressure Surgical ligation

CT, Computed tomography; *FAST*, focused assessment by sonography for trauma.

must be confirmed immediately by capnometry. Esophageal intubation or endotracheal tube (ETT) dislodgement are common and devastating if not promptly corrected. Patients in cardiac arrest may have very low end-tidal carbon dioxide (CO_2) values; direct laryngoscopy should be performed if there is any question about the location of the ETT (see also Chapter 44).

If establishment of a secure airway and adequate ventilation requires a surgical procedure such as a tracheostomy, tube thoracostomy, or open thoracotomy, this procedure must precede all others. Indeed, these procedures are commonly performed in the ED, often before the arrival of an anesthesiologist. Subsequent surgery to convert a cricothyroidotomy to a tracheostomy or close an emergency thoracotomy may then follow in the OR.

Hemorrhage is the next most pressing concern since ongoing blood loss is inevitably fatal. The symptoms of shock are presented in Box 66.3. Shock is presumed to result from hemorrhage until proven otherwise. Assessment of the circulation consists of an early phase, during active hemorrhage, and a late phase, which begins when hemostasis is achieved and continues until normal physiology is restored. In the early phase, diagnostic efforts focus on the five sites of bleeding detailed in Table 66.1, the only areas in which exsanguinating hemorrhage can occur. Immediate actions to control hemorrhage can include application of pelvic binders for bleeding associated with pelvic fractures or tourniquet application for extremity injuries. Any surgical procedure to diagnose or control active hemorrhage is an emergency case that must be brought to the OR as soon as possible. This includes exploration of the neck or pericardium to rule out hemorrhage in sensitive compartments. In the OR, the trauma surgeon focuses on anatomic control of hemorrhage, whereas the anesthesiologist is responsible for restoring the patient's physiology. Goals for early and late resuscitation are discussed in more detail later.

After management of the circulation follows the assessment of the patient's neurologic status by calculation of the Glasgow Coma Scale (GCS) score (Box 66.4)[34]; examination of the pupils for size, reactivity, and symmetry; and determination of sensation and motor function in each of the extremities. Significant abnormalities on the neurologic examination are an indication for immediate cranial computed tomography (CT) scan. Most trauma patients with a diminished GCS score will have nonoperative conditions, but for the few who require operative evacuation of an epidural or subdural hematoma, timeliness of treatment has a strong influence on outcome. Patients with unstable spinal canal injuries and incomplete neurologic deficits will also benefit from early surgical decompression and stabilization.

The final step in the primary survey is complete exposure of the patient and a head-to-toe search for visible injuries or deformities, including deformities of bones or joints, soft tissue bruising, and any breaks in the skin. The anesthesiologist can assist in this procedure by support of the head and neck, maintenance of the airway, and care in manipulating the spine.

After the primary survey, a more deliberate secondary examination is undertaken that includes a thorough history and physical examination, diagnostic studies, and subspecialty consultation. Any remaining injuries are diagnosed at this time and treatment plans established. Indications for urgent or emergency surgery may also arise during the secondary survey. The presence of a limb-threatening injury due to vascular compromise, compartment syndrome, or a severely comminuted fracture is one such indication. Although the ABCDE issues must be addressed first, a pulseless extremity, compartment syndrome, near-amputation, or massively fractured extremity must go to the OR as soon as the patient is otherwise stable.

BOX 66.4 Glasgow Coma Score

Eye-Opening Response

4 = Spontaneous
3 = To speech
2 = To pain
1 = None

Verbal Response

5 = Oriented to name
4 = Confused
3 = Inappropriate speech
2 = Incomprehensible sounds
1 = None

Motor Response

6 = Follows commands
5 = Localizes to painful stimuli
4 = Withdraws from painful stimuli
3 = Abnormal flexion (decorticate posturing)
2 = Abnormal extension (decerebrate posturing)
1 = None

The Glasgow Coma Score is the sum of the best scores in each of three categories.

INJURY PATTERNS PROMPTING URGENT OPERATIVE INTERVENTION

Fig. 66.2, an algorithm for prioritizing surgical management in trauma patients, is presented with the understanding that individual situations will vary according to available resources and the patient's response to therapy. A trauma patient will often arrive at the OR with the need for more than one surgical procedure by more than one surgical service. A trauma patient may have injuries requiring emergency surgery coexisting with injuries that can be repaired at any time. The anesthesiologist plays an important role in determining which procedures to perform, in which order, and which procedures should be postponed until the patient is more stable.

In selected cases with obvious, imminent exsanguination, patients should be directly admitted to the OR, bypassing the ED and radiology suite. Historically, it has been shown that up to a third of preventable trauma deaths may be caused by delays getting to the OR; in one registry study, mortality was increased by 1% for every 3 minutes of delay to laparotomy among hypotensive patients with abdominal injuries.[35-37] Steele and associates were among the first to describe a "direct to the OR" approach in San Diego, reporting data gathered over a 10-year period.[38] Patients with traumatic cardiac arrest, systolic blood pressure persistently lower than 100 mm Hg, amputation, or uncontrolled external hemorrhage were admitted directly to the OR for resuscitation, regardless of mechanism of injury. These triage criteria had poor sensitivity (24.1%) but high specificity (98%) in identifying patients truly in need of immediate surgery. Observed compared to predicted survival was significantly higher for the "direct to the OR"

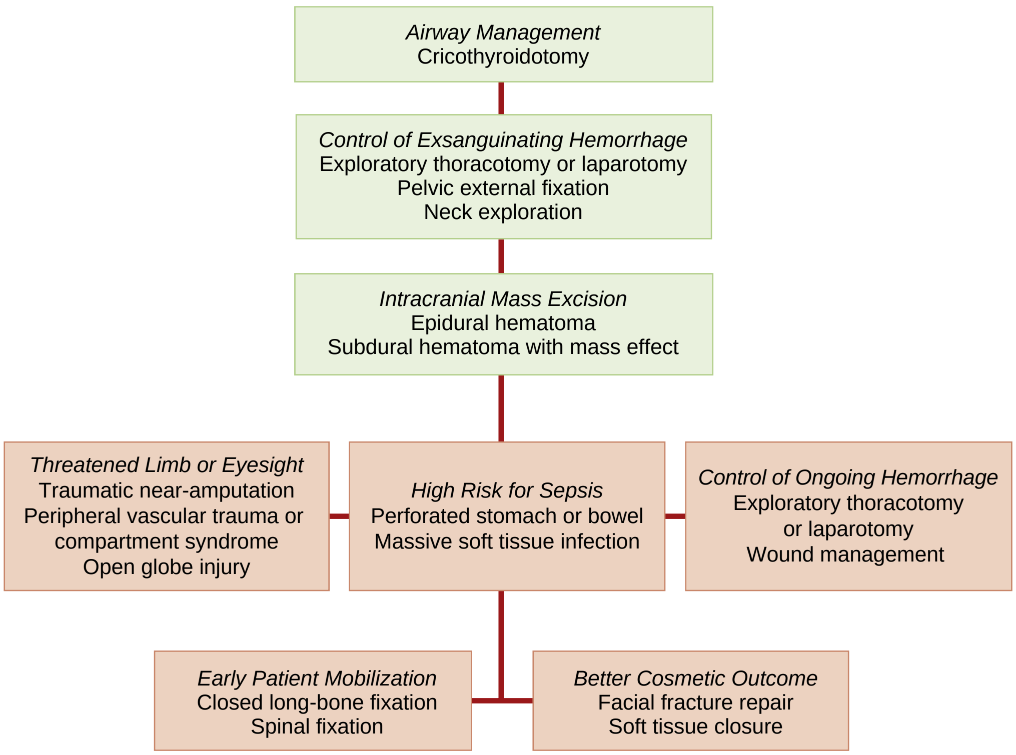


Fig. 66.2 Surgical priorities in a trauma patient. (Reprinted with permission from Dutton RP, Scalea TM, Aarabi B. Prioritizing surgical needs in the patient with multiple injuries. *Probl Anesth*. 2001;13:311.)

approach in this observational study. Similarly, Martin and associates reviewed their 10-year data from 2000 to 2009 for direct OR admission of 1407 patients (5% of all admissions) based on an expanded set of criteria including specific mechanisms.[39] After excluding patients who died on arrival (8%), 3.6% died in the OR and overall observed (5%) mortality was significantly lower than predicted (10%). Emergent surgical procedures were started within 30 minutes of arrival in 77% of patients and within 60 minutes in 92%.

Anesthesia in War and Austere Conditions

"While it is evident that the general principles of anesthesia are not affected by the circumstances of war, it is equally evident that it is our duty to assiduously seek those means in anesthesia which are especially suited to the exigencies of battle."[40]

Although written in 1942, these words are still true today, and many of the principles developed from earlier wars still apply on the modern battlefield or in a large-scale disaster (see also Chapter 68). Recent conflicts and events have allowed anesthesiologists, nurse anesthetists, and other providers to help improve management of traumatically injured patients in the areas of anesthesia, resuscitation, and damage control surgery. Management of battlefield casualties typically follows the same flow as outlined earlier, but with special consideration in the areas of prehospital interventions, resuscitation, technologic and logistic support, patient movement, mass casualty management, and surgical interventions.[41] Pain management considerations may also be affected by the nature of the injuries and transport considerations.[42]

Modern advancements in battle armor, prehospital interventions, provision of forward surgical support, and resuscitative strategies have had an impact on survival from combat injuries.[43] During the most recent conflicts in Iraq and Afghanistan, the killed-in-action rate decreased to 13.9% from 20.2% in Vietnam and World War II.[44] This is mirrored by a similar reduction in the case fatality rate. Paradoxically, the ability to get many of the severely wounded patients to a hospital (e.g., rapidly by helicopter) has led to an increase in the "died-of-wounds" rate. Most likely this rate would be even higher if not for improvements in surgical management such as damage control techniques, improved intensive care unit (ICU) care, evolving resuscitation strategies, tactical combat casualty care initiatives, and institution of a theater-wide trauma system approach.[45] More recently, transition from a 2-hour to an aggressive 1-hour target for definitive care for combat casualties has also contributed to a reduction in case fatality and killed-in-action rates.[46]

One of the major advances in battlefield medical support has been the rapid movement of patients out of the theater of operations to more comprehensive medical facilities. Even in the late l960s, wounded soldiers were evacuated out of Vietnam within 3 days of injury. In the most recent conflict, the time from injury in the Middle East until movement to more definitive medical care in Europe or North America is often within 24 to 48 hours.[47] This may include initial in-theater damage control surgery followed by one or two aeromedical evacuation missions lasting up to 12 hours with a critical care air transport team (CCATT). In preparation for such rapid movement, the anesthesiologist must ensure that perioperative interventions such as airway management, pain control, and adequacy of resuscitation are addressed before transfer. In addition, anesthesiologists are frequently assigned to a CCATT as the physician team member based on their overall skill set and ability to provide support en route to critically ill or injured patients. Beyond the support provided during wartime, the CCATT also has proved useful for the movement of critical patients during large-scale disasters such as occurred after Hurricane Katrina in 2005.[48]

Mass casualty situations are common in wartime conditions, although the role of the anesthesiologist will vary depending on the number of patients and requirements for urgent surgical interventions. Given the limited number of anesthesia providers in most combat-related scenarios, often they are not involved in the triage process. If available, however, anesthesia support can enhance emergency airway management, establishment of venous access, and supervision of resuscitative efforts. Only 10% to 20% of arriving casualties require immediate lifesaving interventions, although a much larger percentage will ultimately require surgical procedures.[49] A well-developed trauma system is persistently evolving, and management of mass casualty scenarios will become routine.[50]

Overall, anesthetic management of battlefield casualties is similar to that for patients in a civilian trauma setting; however, many factors must be considered in the perioperative plan for a combat casualty.[51] Environmental considerations such as extremes of temperature, availability of water, contamination with sand, lack of consistent electricity, and other aspects may have to be taken into consideration. Logistic support chains may be long and unable to provide sufficient supplies in the early phases of a conflict. Deployable equipment, such as drawover vaporizer systems or portable anesthesia systems, may be different from those used during peacetime, so predeployment training is vital (Fig. 66.3).[52] In addition, techniques such as total intravenous anesthesia and regional anesthesia or analgesia may be better options making familiarity with their management and associated equipment an imperative (see also Chapter 46).[53]

Optimal care of wartime casualties or victims of large disasters requires not only knowledge of a broad range of anesthetic principles and techniques but also the ability to be flexible in the face of a rapidly changing environment. With special training in airway management, provision of anesthesia and sedation, resuscitation, and pain management, anesthesiologists may find themselves involved in triage, emergency management, and perioperative and critical care.

Emergency Airway Management

The American Society of Anesthesiologists (ASA) algorithm for management of difficult airways (see also Fig. 44.1) modified for trauma is a useful starting point for the trauma anesthesiologist, whether in the ED or the OR (see also Chapter 44).[54] The concept of the algorithm is an important one. The anesthesiologist should have a plan for the initial approach to the airway and for coping with any difficulties that might develop. Fig. 66.4 is a typical algorithm for emergency

Fig. 66.3 Deployable military anesthesia equipment. (A) Drawover vaporizer and portable ventilator *(circled)* in field hospital. (B) Portable anesthesia machine. (Used with permission from CPT Bruce Baker, MD, USN.)

intubation of an unstable trauma patient. Note that it differs from the ASA algorithm in that reawakening the patient is seldom an option because the need for emergency airway control will presumably remain. Once the decision to obtain a definitive airway is made, efforts will continue until a cuffed tube is in position in the trachea, whether by conventional intubation or via a surgical approach. Failure to commit to a surgical airway soon enough results in bad outcomes more commonly than do complications of a procedure that might have been unnecessary.

INDICATIONS

The goal of emergency airway management is to ensure adequate oxygenation and ventilation while protecting the patient from the risks for aspiration. Endotracheal intubation is commonly required and is specifically indicated in the following conditions:

- Cardiac or respiratory arrest
- Respiratory insufficiency (see Box 66.2)
- Airway protection
- Need for deep sedation or analgesia, up to and including general anesthesia
- Ventilation management of patients with space-occupying intracranial lesions and evidence of increased intracranial pressure (ICP)
- Delivery of a 100% fraction of inspired oxygen (FiO_2) to patients with carbon monoxide poisoning
- Facilitation of the diagnostic workup in uncooperative or intoxicated patients

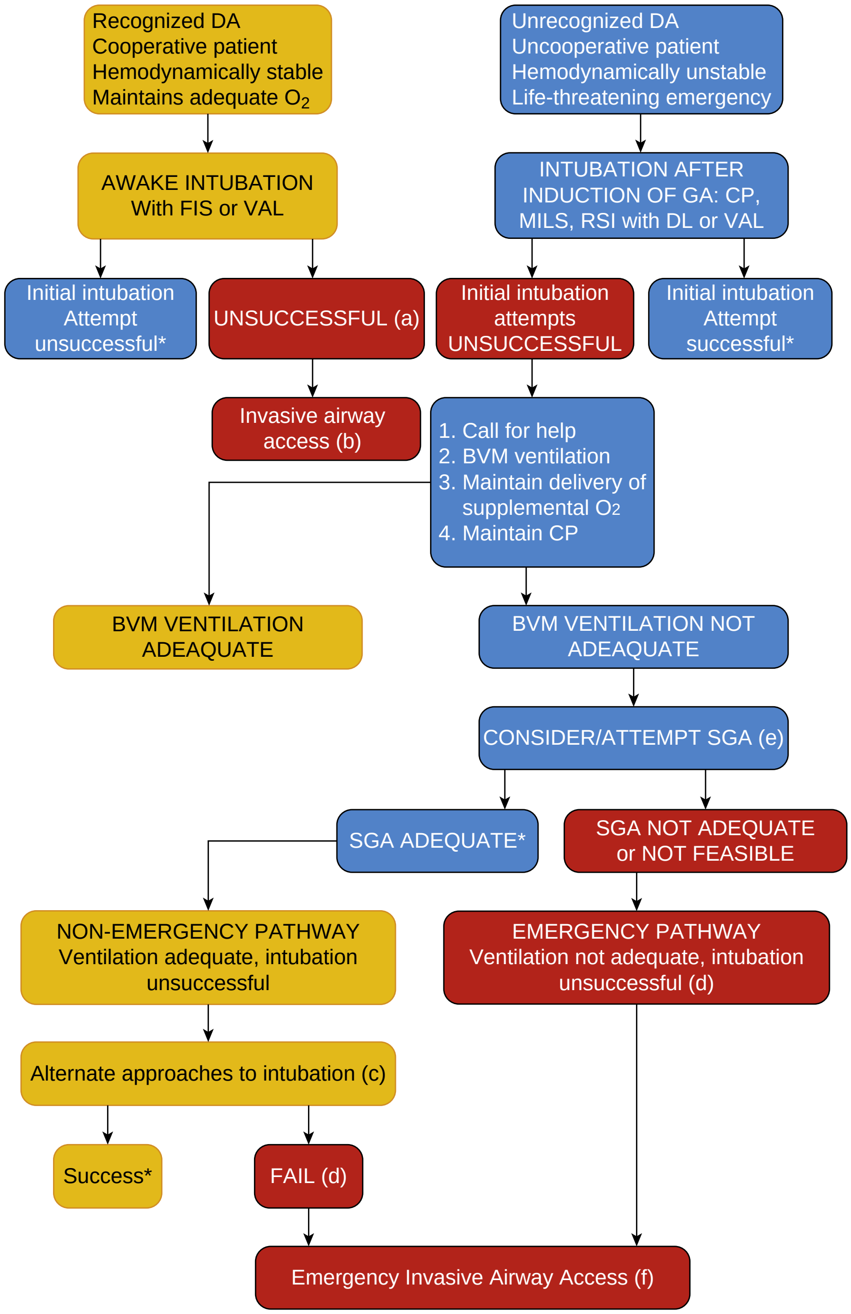
APPROACH TO ENDOTRACHEAL INTUBATION

In general, monitoring standards for airway management should be the same in the ED and OR, including an electrocardiogram (ECG), blood pressure, oximetry, and capnometry. Appropriate equipment, including an oxygen source, bag-valve-mask ventilating system, mechanical ventilator, suction, and a selection of laryngoscope blades (including video laryngoscope), ETTs, and devices for managing difficult tracheal intubations, should be available in any location where emergency intubation is likely, including the ED.

Endotracheal intubation is best accomplished in almost all cases with a modified rapid sequence approach by an experienced clinician. Although concern may exist that the use of neuromuscular blocking drugs and potent anesthetics outside the OR will be associated with a more frequent complication rate, in fact the opposite is more likely correct. Anesthesia and neuromuscular blockade allow the best tracheal intubating conditions on the first approach to the airway, which is advantageous in an uncooperative, hypoxic, or aspirating patient. Attempts to secure the airway in an awake or lightly sedated patient increase the risk for airway trauma, pain, aspiration, hypertension, laryngospasm, and combative behavior. Experienced providers, supported by appropriate monitoring and equipment, have achieved results of medication-assisted intubation outside the OR that are equivalent to those for emergency tracheal intubation within the OR.[55-57]

PROPHYLAXIS AGAINST PULMONARY ASPIRATION OF GASTRIC CONTENTS

A trauma patient should always be treated as having a full stomach and at risk for aspiration of gastric contents during induction of anesthesia (see also Chapter 44). Reasons include ingestion of food or liquids before the injury, swallowed blood from oral or nasal injuries, delayed gastric emptying associated with the stress of trauma, and administration of liquid contrast medium for abdominal CT scanning.



* Confirm ventilation, tracheal intubation or SGA placement with standard confirmatory techniques (exhaled CO_2, misting of tube, auscultation of breath sounds, improving SpO_2). If perfusion (and exhaled CO_2) absent, use additional confirmation methods (e.g., repeat laryngoscopy, bronchoscopy, esophageal detector device, chest X-ray).

(a) Other options in ASA algorithm:
- Ventilation with a face mask or SGA might be difficult or impossible in a patient with maxillofacial trauma
- Local anesthesia infiltration or regional nerve blockade are of limited value in extensive trauma surgery.

(b) Invasive airway access includes surgical or percutaneous cricothyrotomy or tracheostomy, transtracheal jet ventilation and retrograde intubation.

(c) Alternative difficult intubation approaches include (but are not limited to): VAL, SGA (e.g., laryngeal mask airway [LMA] as an intubation conduit with or without flexible scope guidance), flexible scope intubation (FSI), intubating stylet or tube changer, and light wand. Blind intubation (oral or nasal) is discouraged in patients with maxillofacial trauma and laryngeal or tracheal injury.

(d) Aborting the case and awakening the patient to optimize and re-attempt intubation via a different airway technique (e.g., awake intubation) is impractical in most trauma cases due to the emergent condition of the patient.

(e) Emergency non-invasive airway ventilation consists of SGA.

(f) Surgical airway kit should be immediately available.

Fig. 66.4 Emergency airway management algorithm in trauma. Individual practitioners and trauma hospitals should determine their own algorithm, based on available skills and resources. *ASA,* American Society of Anesthesiologists; *BVM,* bag-valve-mask; *CP,* cricoid pressure; *DA,* difficult airway; *DL,* direct laryngoscopy; *FIS,* flexible intubation scope; *GA,* general anesthesia; *MILS,* manual in-line stabilization; *RSI,* rapid sequence intubation; *SGA,* supraglottic airway; *VAL,* video-assisted laryngoscopy. (Modified from Hagberg CA, Kaslow O. Difficult airway management algorithm in trauma updated by COTEP. *ASA Newsletter.* 2014;78:56–60.)

Cricoid pressure—the Sellick maneuver—has been recommended to be applied continuously during emergency airway management from the time the patient loses protective airway reflexes until ETT placement and cuff inflation are confirmed. The Sellick maneuver consists of elevating the patient's chin (without displacing the cervical spine) and then pushing the cricoid cartilage posteriorly to close the esophagus. However, cricoid pressure may worsen the laryngoscopic grade of view in up to 30% of patients[58] without providing effective prevention of aspiration of gastric contents.[59] In a prehospital study evaluating the impact of cricoid pressure on subsequent intubation success, discontinuing cricoid pressure usually facilitated intubation of the trachea without worsening the grade of laryngoscopic view.[60] Thus cricoid pressure should be released in the trauma patient if likely to facilitate intubation attempts. The lack of evidence supporting the use of cricoid pressure and its potential to make intubation more difficult led the American Heart Association to recommend discontinuation of its use during cardiac arrest situations.[61] Additionally, the Eastern Association for the Surgery of Trauma Practice Management Guidelines for emergency tracheal intubation have removed it as a class 1 recommendation.[62]

In the traditionally defined rapid sequence induction of anesthesia, any attempt at ventilation between administration of medication and intubation is avoided, presumably because positive-pressure ventilation may force gas into the patient's stomach, leading to regurgitation and aspiration. Sellick's original paper described ventilation during cricoid pressure in patients with full stomachs with the belief that cricoid pressure during mask ventilation would prevent gastric inflation.[63] Although this may be true, cricoid pressure reduces tidal volumes, increases peak inspiratory pressure, or prevents ventilation.[64] On the other hand, the increase in oxygen consumption in trauma patients necessitates preoxygenation whenever possible. If preoxygenation is not possible because of facial trauma, decreased respiratory effort, or agitation, rapid desaturation is a possibility. Positive-pressure ventilation during all phases of induction provides the largest possible oxygen reserve during emergency airway management and will help mitigate hypoxia if intubation proves difficult. In this situation, large tidal volumes and high peak inspiratory pressures should be avoided. Application of cricoid pressure during attempts at positive-pressure ventilation should be considered to reduce gastric inflation, but it may prevent effective ventilation in some patients necessitating discontinuation.

PROTECTION OF THE CERVICAL SPINE

Standard practice dictates that all victims of blunt trauma be assumed to have an unstable cervical spine until this condition is ruled out. The airway management of these patients receives much attention from anesthesiologists because direct laryngoscopy causes cervical motion, with the theoretical potential to exacerbate spinal cord injury (SCI). Stabilization of the cervical spine will generally occur in the prehospital environment, with the patient already having a rigid cervical collar in place. This collar may be kept in place for several days before the complete gamut of tests to rule out cervical spine instability have been completed (see later discussion). The presence of an "uncleared" cervical spine mandates the use of manual in-line stabilization without application of traction throughout any attempt at intubation.[33] This approach allows removal of the front of the cervical collar to facilitate wider mouth opening and jaw displacement; however, this may slightly lengthen the time to intubation and worsen laryngeal visualization during laryngoscopy compared to intubation attempts without manual in-line stablilization.[65] Manual in-line stabilization has been tested through considerable clinical experience and is the standard of care in the ATLS curriculum. Emergency awake fiberoptic intubation, though requiring less manipulation of the neck, is generally very difficult because of airway secretions and hemorrhage, rapid desaturation, and lack of patient cooperation; and is best reserved for cooperative patients with known cervical instability under controlled conditions. Indirect video laryngoscopy offers the potential to enjoy the best of both worlds: an anesthetized patient and decreased cervical motion.[66,67] In comparative studies of direct laryngoscopy, video laryngoscopy, fiberoptic intubation, blind nasal intubation, or cricothyrotomy—in patients with known cervical cord or spine injuries, or both—there is no difference in neurologic deterioration with technique used, and no clear evidence that direct laryngoscopy worsens outcome.[68]

Fig. 66.5 Emergency intubation of a trauma patient immobilized on a long spine board. The front of the cervical collar is removed once in-line manual stabilization of the spine is established, allowing for cricoid pressure and greater excursion of the mandible. (Reprinted with permission from Dutton RP. Spinal cord injury. *Int Anesthesiol Clin.* 2002;40:111.)

PERSONNEL

Emergency endotracheal intubation requires more assistance than an intubation performed under controlled conditions (see also Chapter 6). Three providers are required to ventilate the patient and manage the airway, administer medications, and provide manual in-line stabilization (if indicated); a fourth provider may be needed to provide cricoid pressure if deemed appropriate. Fig. 66.5 is an illustration of this approach. Additional assistance may be required to restrain a patient who is combative because of intoxication or traumatic brain injury (TBI).

The immediate presence of a surgeon or other physician who can expeditiously perform a cricothyroidotomy is desirable. Even if a surgical airway is not required, additional experienced hands may prove useful during difficult intubations. The surgeon may also wish to inspect the upper airway during laryngoscopy or video laryngoscopy if trauma to the face or neck has occurred. Urgent tube thoracostomy may prove necessary in some trauma patients to treat a tension pneumothorax that develops with the onset of positive-pressure ventilation.

ANESTHETICS AND INDUCTION OF ANESTHESIA

Any intravenous anesthetic administered to a trauma patient in hemorrhagic shock may cause profound hypotension and even cardiac arrest because of inhibition of circulating catecholamines. Although propofol is the mainstay of intravenous induction in the OR, its use in trauma patients is problematic because of its vasodilatory and negative inotropic effects. Moreover, pharmacokinetic and pharmacodynamic studies in a swine hemorrhagic shock model suggest a significant reduction in propofol dosage of more than 80% to achieve the targeted effect site concentration.[69,70] Unfortunately, there is no corresponding clinical data on the impact of reduced propofol dosing in the setting of hemorrhagic shock on recall and awareness.

The most commonly used induction agent in the United States in the ED or trauma setting is etomidate.[71] Etomidate administered in a range of 0.2 to 0.3 mg/kg is associated with hemodynamic stability and has an onset/duration profile like that of succinylcholine. Its safety for use in rapid sequence induction in trauma patients has been challenged although these studies are largely retrospective with the potential for selection bias and other methodologic deficiencies.[72,73] Although etomidate is associated with transient adrenal cortical suppression after a single dose, this appears not to be clinically significant when a single dose is used for induction for intubation in both trauma and mixed surgical-medical patients undergoing rapid sequence induction.[74-76] Etomidate can cause myoclonic jerks during its onset, but use of a rapidly acting neuromuscular blocking agent, such as succinylcholine, mitigates this effect substantially.

Ketamine is also a frequently used induction agent for hypotensive trauma patients due to its centrally mediated increase in sympathetic tone and catecholamine release.[77] Its use in patients with concomitant TBI has been questioned based on older reports of associated ICP elevation.[78] More recent analysis, however, suggests that the preservation of cerebral perfusion by maintenance of mean arterial blood pressure in hemodynamically unstable patients is more important than any theoretical risk to the brain caused by ketamine's tendency to increase cerebral activity and ICP.[78,79] Some investigators have also raised concern that the psychotropic effects associated with ketamine may increase the risk of acute- and posttraumatic stress disorders in trauma patients,[80,81] although this was not found in a study examining its intraoperative use in burn patients.[82] Of more concern is the potential for barriers to its use based on institutional dispensing, tracking, and documentation procedures preventing timely access to ketamine. When barriers exist limiting its availability, ketamine may not be as readily available in the emergency setting compared to other induction agents. Due to its abuse potential, consideration has been made to reclassify ketamine as a Schedule 1 drug, potentially placing further barriers to its availability.[83] Overall, ketamine continues to be a very commonly used drug for rapid sequence induction in the trauma patient.[71]

Hypotension will likely develop in patients with hypovolemia with the administration of any anesthetic because of interruption of compensatory sympathetic outflow and the sudden change to positive-pressure ventilation. Previously healthy young patients can lose up to 40% of their blood volume before hypotension occurs, thereby leading to potentially catastrophic circulatory collapse with induction of anesthesia, regardless of the anesthetic chosen. The dose of anesthetic must be decreased in the presence of hemorrhage, including no anesthetic at all in patients with life-threatening hypovolemia. Rapid sequence induction of anesthesia and endotracheal intubation may proceed with muscle relaxants alone, although onset time may be prolonged in a patient with circulatory impairment. Subsequent patient recall of intubation and emergency procedures is highly variable and affected by the presence of coexisting TBI, intoxication, and the depth of hemorrhagic shock (see also Chapter 9). Decreased cerebral perfusion inhibits memory formation but cannot be reliably associated with any particular blood pressure or chemical marker. Administration of 0.2 mg of scopolamine (a tertiary ammonium vagolytic) may inhibit memory formation in the absence of anesthetic drugs in this situation, but it may interfere with subsequent neurologic examination because of its long half-life. Small doses of midazolam will reduce the incidence of patient awareness but also can contribute to hypotension. Although recall of ED and OR events is not unusual in this circumstance, anesthesia provider liability appears to be limited; an analysis of intraoperative awareness lawsuits in the ASA Closed Claims Database revealed no claims related to surgery in trauma patients.[84]

NEUROMUSCULAR BLOCKING DRUGS

Succinylcholine remains the neuromuscular blocker with fastest onset—less than 1 minute—and shortest duration of action—5 to 10 minutes. These properties make it popular for rapid sequence induction of anesthesia. Although the use of succinylcholine may allow return of spontaneous respiration before the development of significant hypoxia in the "cannot intubate, cannot ventilate" situation, this is unlikely to be of benefit in an emergency intubation in a trauma patient. The anesthesiologist should not rely on return of spontaneous breathing in time to salvage a difficult airway management problem but should instead proceed with efforts to obtain a definitive airway, including cricothyroidotomy if other possibilities have been exhausted.

Administration of succinylcholine is associated with several adverse consequences. Increases in serum potassium of 0.5 to 1.0 mEq/L are expected, but in certain patients K^+ may increase by more than 5 mEq/L.[85] A hyperkalemic response is typically seen in burn victims and those with muscle pathology secondary to direct trauma, denervation (as with SCI), or immobilization. Hyperkalemia is not seen in the first 24 hours after these injuries, and succinylcholine may be used safely for acute airway management. Patients at risk are those with underlying pathologic processes before their traumatic event or those undergoing subsequent surgery in the weeks to months after injury.

Succinylcholine causes an increase in intraocular pressure and should be used cautiously in patients with ocular trauma.[86] Succinylcholine may also increase ICP,[87] making its use in patients with brain trauma controversial. In both these cases, however, hypoxia and hypercapnia may be as damaging as the transient increase in pressure caused by the drug. If the use of succinylcholine will lead to faster intubation, its benefits may outweigh its risks. The provider must weigh the use of succinylcholine in each individual situation based on acuity, anticipated speed with which intubation can be accomplished, and likelihood that hypoxia will develop.

Alternatives to succinylcholine include rocuronium 0.9 to 1.2 mg/kg and vecuronium 0.1 to 0.2 mg/kg. Rocuronium is preferred because it has a more rapid onset of action than that of vecuronium. With the availability of sugammadex, a rapid onset selective binding agent for rocuronium, rapid sequence induction and intubation with rocuronium followed by reversal with sugammadex allows for more rapid return of spontaneous ventilation than with succinylcholine.[88] Basically, the combination of rocuronium and sugammadex provides all the advantages of succinylcholine, but none of the complications.[89,90] Because these drugs have no significant cardiovascular toxicity, large doses can be administered to achieve rapid (1- to 2-minute) paralysis.

Specific situations will always exist in which maintaining spontaneous ventilation during intubation is the preferred way to proceed. If patients can maintain their airway temporarily but have clear indications for an artificial airway (e.g., penetrating trauma to the trachea), slow induction with ketamine or inhaled sevoflurane through cricoid pressure will enable placement of an ETT without compromising patient safety.

ADJUNCTS TO ENDOTRACHEAL INTUBATION

Equipment to facilitate difficult intubation should be readily available wherever emergency airway management is performed. The equipment available depends on the preferences of the anesthesiologist; the usefulness of most special equipment depends more on previous experience than on any intrinsic properties of the device. Certain items deserve mention, however, because they are frequently cited as aids to management of a difficult airway.

The gum elastic bougie, or intubating stylet, is an inexpensive and easily mastered adjunct for management of a difficult airway. The stylet is placed through the vocal cords via direct laryngoscopy, and the ETT is then advanced over the stylet into the trachea. Placement of the bougie is easier than direct placement of an ETT because of both its smaller diameter and the ability of an experienced operator to feel it enter the trachea even when the glottic opening cannot be visualized. The bougie is passed under the epiglottis and gently advanced; if resistance is met, the bougie is withdrawn, rotated slightly, and advanced again. In this fashion the anesthesiologist can blindly palpate the larynx until the bougie advances into the trachea. The bougie also can be used with direct and indirect video laryngoscopy systems and is especially useful in the ED when the sniffing position cannot be used because of uncertainty about the cervical spine.

Supraglottic airway (SGA) devices, such as the laryngeal mask airway (LMA) (LMA North America, San Diego, CA), are recommended in the ASA algorithm for management of a patient with a difficult airway. The LMA can be used as a guide for intubation when an unsuspected difficult intubation is encountered in a trauma patient; an ETT may be placed blindly through the lumen of the LMA and into the trachea, or a fiberoptic bronchoscope may be used to guide the tube through the LMA. The LMA is an appropriate rescue device for a difficult airway situation in trauma, provided no major anatomic injury or hemorrhage is present in the mouth and larynx. In our practice the LMA has most commonly been used as a bridge to emergency tracheostomy because it allows more controlled conditions than a cricothyroidotomy.

FACIAL AND PHARYNGEAL TRAUMA

Trauma to the face and upper airway poses difficulties for the anesthesiologist.[91] Serious skeletal derangements may be masked by apparently minor soft tissue damage. Failure to identify an injury to the face or neck can lead to acute airway obstruction secondary to swelling and hematoma. Laryngeal edema is also a risk in patients who have suffered chemical or thermal injury to the pharyngeal mucosa. Intraoral hemorrhage, pharyngeal erythema, and change in voice are all indications for early intubation.

In general, both maxillary and mandibular fractures will make ventilation by mask more difficult, whereas mandibular fractures will make endotracheal intubation easier. Palpation of the facial bones before manipulation of the airway will alert the anesthetist to these possibilities. Patients with injuries to the jaw and zygomatic arch often have trismus. Although the trismus will resolve with the administration of neuromuscular blocking agents, preinduction assessment of airway anatomy may be difficult. Bilateral mandibular fractures and pharyngeal hemorrhage may lead to upper airway obstruction, particularly in a supine patient, although intubation may be easier because of loss of skeletal resistance to direct laryngoscopy. A patient arriving at the ED in the sitting or prone position because of airway compromise is best left in that position until the moment of anesthetic induction and intubation.

THE FIELD PLACED AIRWAY

In many settings, prehospital providers work under protocols that allow for advanced airway management including intubation and placement of an SGA. It is recommended that the individuals responsible for airway management at the ED level be familiar with the protocols and devices available in their region to facilitate early assessment and airway exchange if indicated. All intubations done in the field should be immediately confirmed using capnometry. In the event of cardiac arrest and inability to confirm correct placement by observation of end-tidal CO_2, direct laryngoscopy or video laryngoscopy can be employed to visualize position of the ETT. When an SGA device is used in the prehospital setting, the positioning and ability to ventilate should be confirmed by capnometry, presence of breath sounds, and chest movement. If there is adequate ventilation, replacement of the SGA can be delayed during the initial evaluation.

Since the SGA is not considered a secured definitive airway, it should be changed out to a cuffed ETT as soon as practical. When faced with changing out an SGA, preparation is the key concept. Unfortunately, there is a paucity of literature discussing the technique of "changing out" an SGA with no single preferred method in the trauma patient. This procedure can be challenging with increased risk of losing the patient's airway. Prior to deciding which technique to use, it is extremely important to have a discussion with the prehospital care provider that managed the patient's airway in the field to determine the following information:

- What was the reason for placing an SGA?
- Were attempts at oral or nasal intubation made? How many?
- What laryngoscope/video laryngoscope was used?
- What anatomy was visualized during attempts?
- What, if any, medications were administered during the attempts?

The answers to these questions may suggest a difficult airway prompting more guarded management during the exchange process. The three options for exchange in the trauma setting are: (1) remove the SGA and replace under direct or video laryngoscopy, (2) use the SGA to place an ETT or exchange catheter, or (3) proceed to a surgical airway. The second option will largely be guided by the specific SGA, channel size, and available equipment.[92-94] Of note, there have been reports of pharyngeal, glottic, and lingual edema with the use of various SGA devices likely secondary to exaggerated anatomical distortion and indirect vascular compression.[93] It is unclear whether this is an anticipated finding in some patients with proper placement and inflation of the proximal oropharyngeal cuff or due to overinflation of the cuff. In the only published series of patients presenting with prehospital placement of the King LT (S)-D (King Systems; Noblesville, IN), 7 of 9 trauma patients ultimately underwent tracheostomy in the OR due to concerns for concomitant facial trauma, observed upper airway edema, or failed attempts at direct laryngoscopy.[93]

On occasion, prehospital personnel will perform a blind nasotracheal intubation in the spontaneously ventilating trauma patient. Successful exchange of this nasotracheal tube on arrival to the ED is consistently easier in comparison with the SGA. Nasotracheally placed tubes in the field are usually a result of the prehospital providers not being credentialed for drug-facilitated intubation. As a result, most of these patients have not had laryngoscopy performed and therefore are less likely to have airway edema from intubation attempts. Again, in this scenario, it is prudent to facilitate the intubation with muscle relaxation and adequate sedation/anesthesia prior to attempting laryngoscopy. A video laryngoscope is preferred as it allows for an expanded view of the glottis with visualization of the nasotracheal tube prior to removal of the nasal tube. Simple exchange under direct or video visualization with an oral ETT is usually all that is required. Once again, it cannot be overemphasized that a bougie be readily available in these situations for rapid placement into the glottis with various sized ETTs readily available. If one is unable to adequately visualize the glottis with laryngoscopy, it is recommended that the nasotracheal tube be left in place for a period to allow for either a controlled tracheostomy or swelling to improve and an exchange for an oral tube made later.

Resuscitation from Hemorrhagic Shock

Resuscitation refers to restoration of normal physiology after injury. Resuscitation from hemorrhagic shock refers specifically to restoration of normal circulating blood volume, normal vascular tone, and normal tissue perfusion. Resuscitation begins immediately after injury, via the patient's own compensatory mechanisms, and continues through the prehospital, ED, OR, and ICU phases of care.

PATHOPHYSIOLOGY OF HEMORRHAGIC SHOCK

During massive hemorrhage an imbalance occurs between systemic oxygen delivery and oxygen consumption. Blood loss leads to hemodynamic instability, coagulopathy, decreased oxygen delivery, decreased tissue perfusion, and cellular hypoxia. The initial response to hemorrhage takes place on the macrocirculatory level and is mediated by the neuroendocrine system. Decreased arterial blood pressure leads to vasoconstriction and release of catecholamines to preserve blood flow to the heart, kidney, and brain, whereas other regional beds are constricted. Pain, hemorrhage, and cortical perception of traumatic injuries lead to the release of hormones and other inflammatory mediators, including renin, angiotensin, vasopressin, antidiuretic hormone, growth hormone, glucagon, cortisol, epinephrine, and norepinephrine.[95] This response sets the stage for the microcirculatory response that follows.

Individual ischemic cells respond to hemorrhage by taking up interstitial fluid, thus further depleting intravascular fluid.[96] Cellular edema may choke off adjacent capillaries and result in the no-reflow phenomenon, which prevents reversal of ischemia even in the presence of adequate macroperfusion.[97] Ischemic cells produce lactate and free radicals, which accumulate in the circulation if perfusion is diminished. These compounds cause direct damage to the cell and form the bulk of the toxic load that washes back to the central circulation when flow is reestablished. Ischemic cells also produce and release inflammatory factors—prostacyclin, thromboxane, prostaglandins, leukotrienes, endothelin, complement, interleukins, tumor necrosis factor, and others.[98] Fig. 66.6 shows the generalized inflammatory response to shock, with an emphasis on immune system amplification. This inflammatory response, once begun, becomes a disease process independent of its origin. Such alterations lay the foundations for subsequent development of multiple organ failure, a systemic inflammatory process that leads to dysfunction of different vital organs and accounts for high mortality rates.[99]

Specific organ systems respond to traumatic shock in specific ways. The CNS is the prime trigger of the neuroendocrine response to shock, which maintains perfusion to the heart, kidney, and brain at the expense of other tissues.[100] Regional glucose uptake in the brain changes during shock.[101] Reflexes and cortical electrical activity are both depressed during hypotension; these changes are reversible with mild hypoperfusion but become permanent with prolonged ischemia. Failure to recover preinjury neurologic function is a marker for a poor prognosis, even when normal vital signs are restored.[102]

The *kidney* and *adrenal glands* are prime responders to the neuroendocrine changes associated with shock and produce renin, angiotensin, aldosterone, cortisol, erythropoietin, and catecholamines.[103] The kidney maintains glomerular filtration in the face of hypotension by selective vasoconstriction and concentration of blood flow in the medulla and deep cortical area. Prolonged hypotension leads to decreased cellular energy and an inability to concentrate urine (renal cell hibernation), followed by patchy cell death, tubular epithelial necrosis, and renal failure.[104]

The *heart* is preserved from ischemia during shock because of maintenance of or even an increase in nutrient blood flow, and cardiac function is well preserved until the late stages. Lactate, free radicals, and other humoral factors released by ischemic cells all act as negative inotropes and, in a bleeding patient, may produce cardiac dysfunction as the terminal event in the shock spiral.[105] A patient with cardiac disease or direct cardiac trauma is at great risk for

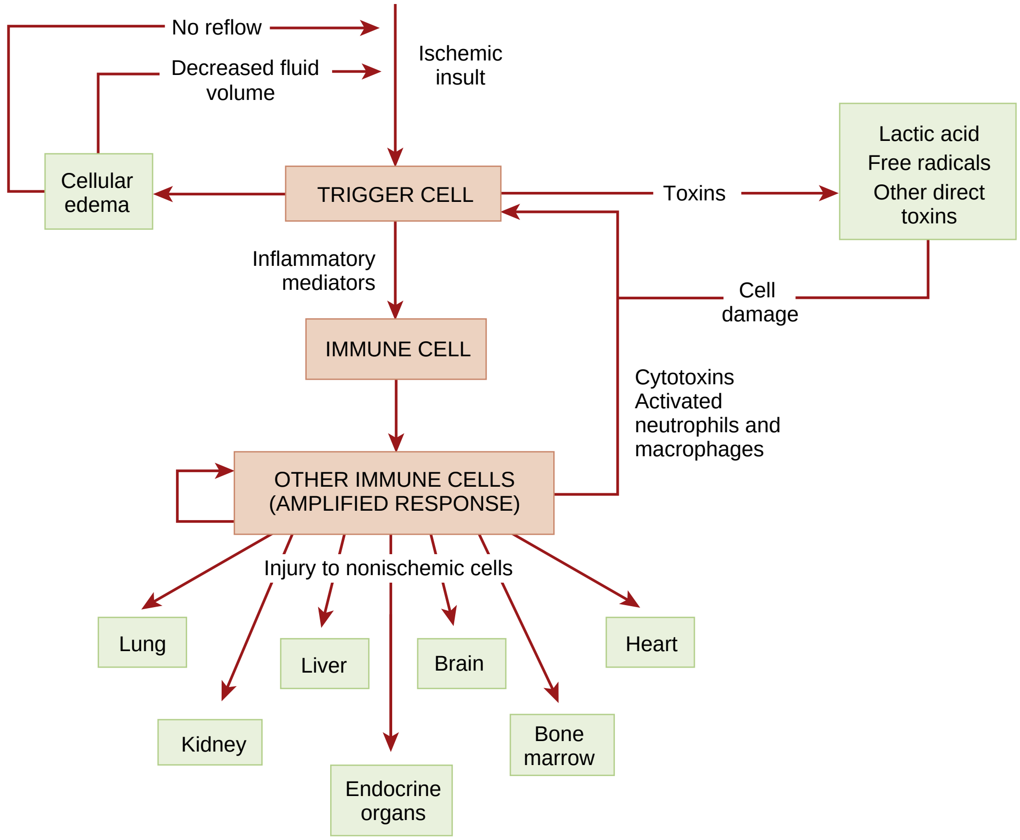


Fig. 66.6 The "shock cascade." **Ischemia of any given region of the body will trigger an inflammatory response that will impact nonischemic organs even after adequate systemic perfusion has been restored.** (Reprinted with permission from Dutton RP. Shock and trauma anesthesia. In: Grande CM, Smith CE, eds. *Anesthesiology Clinics of North America: Trauma*. Philadelphia, 1999, WB: Saunders; 83–95.)

decompensation because a fixed stroke volume inhibits the body's ability to increase blood flow in response to hypovolemia and anemia. Tachycardia is the patient's only option, with potentially disastrous consequences on the oxygen supply-demand balance in the heart. Therefore shock in older patients may be rapidly progressive and not respond predictably to fluid administration.[106]

The *lung* is the filter for the inflammatory by-products of the ischemic body. Immune complex and cellular factors accumulate in pulmonary capillaries and lead to neutrophil and platelet aggregation, increased capillary permeability, destruction of lung architecture, and acute respiratory distress syndrome (ARDS).[107,108] The lung is the sentinel organ for the development of multiple organ dysfunction (MOD) in a patient with traumatic shock.[109,110] Pure hemorrhage, in the absence of hypoperfusion, does not produce pulmonary dysfunction.[111] Traumatic shock is obviously more than just a hemodynamic disorder.

The *gut* is one of the earliest organs affected by hypoperfusion and may be one of the prime triggers of MOD. Intense vasoconstriction occurs early and frequently leads to a no-reflow phenomenon, even when the macrocirculation is restored.[112] Intestinal cell death causes a breakdown in the barrier function of the gut that results in increased translocation of bacteria to the liver and lung, thereby potentiating MOD and ARDS.[113]

The *liver* has a complex microcirculation and may experience reperfusion injury during recovery from shock.[114] Hepatic cells are also metabolically active and contribute to the ischemic inflammatory response and to irregularities in blood glucose.[115] Failure of synthetic function of the liver after shock is almost always lethal.

Skeletal muscle is not metabolically active during shock and tolerates ischemia better than do the other organs. The large mass of skeletal muscle, though, makes it important in the generation of lactic acid and free radicals from ischemic cells. Sustained ischemia of muscle cells leads to an increase in intracellular sodium and free water, with an aggravated depletion of fluid in the vascular and interstitial compartments.[116]

More recently, there appears to be a role for endothelial injury in the pathophysiology of hemorrhagic shock. The *endothelium* is one of the "largest" organs in the body with a surface area of up to 5000 m^2.[117] Under normal conditions the endothelium is anticoagulated by a number of natural anticoagulant systems including the negatively charged luminal surface layer, the glycocalyx, which is rich in heparinoids and interacts with antithrombin.[118] As noted earlier, increasing injury severity and shock lead to high catecholamine levels which can directly injure the endothelium.[119] This is evidenced by an increase in syndecan-1 levels, a marker of endothelial glycocalyx degradation.[120,121] The release of heparin-like substances in the glycocalyx may also contribute to endogenous heparinization and the coagulopathy of trauma (discussed in the next section).[122-124] The net effect of this endothelial injury results in glycocalyx shedding, breakdown of tight junctions with capillary leakage, and a pro-coagulant microvasculature that further reduces oxygen delivery due to increased tissue pressure and microvascular thrombosis.

ACUTE TRAUMATIC COAGULOPATHY

During resuscitation from hemorrhagic shock, attention must also be directed to avoidance or correction of coagulopathy. In patients with identical injury severity scores (ISS), the presence of coagulopathy is associated with at least a twofold to fourfold increase in mortality[125,126]; thus current resuscitation strategies focus on coagulopathy and shock during the initial and subsequent resuscitation. The presence of trauma-induced coagulopathy—defined as a "multifactorial, global failure of the coagulation system to sustain adequate hemostasis after major trauma"—has an endogenous component linked to hypoperfusion and tissue injury referred to as acute traumatic coagulopathy (ATC).[127] A proposed mechanism for the development of ATC is endothelial activation of protein C secondary to the traumatic inflammatory response described earlier.[128] Activated protein C (APC) is generated by thrombomodulin-thrombin complex production as a result of tissue hypoperfusion. APC inactivates factors Va and VIIIa, and in combination with the reduction in thrombin availability for fibrin formation, supports the development of ATC.[129] Additionally, degradation of the endothelial glycocalyx as a result of hypoperfusion may play a supporting role in ATC.[130]

By clinical definition, ATC starts with the early presence of reduced clot strength as demonstrated by viscoelastic monitoring and changes in laboratory-based coagulation testing associated with an increase in mortality and likelihood of receiving a massive transfusion.[131] Davenport and colleagues[131] proposed a clot amplitude threshold of less than 35 mm at 5 minutes using rotational thromboelastometry (ROTEM) (Tem Innovations, Munich, Germany) analysis, which predicted the subsequent need for a massive transfusion with a detection rate of 77% and false-positive rate of 13% (see also Chapter 50). Similar results have been obtained using RapidTEG (Haemonetics, Niles, IL) viscoelastic testing.[132] Laboratory-based coagulation testing is of limited utility in early detection of ATC because of time considerations. However, Frith and associates found patients with a prothrombin ratio of greater than 1.2 on admission to have larger transfusion requirements and increased mortality.[133] Regardless of the means used to detect coagulopathy in the severely traumatized patient undergoing resuscitation for hemorrhagic shock, the resuscitation itself should include consideration for early treatment of ATC.

In addition to the described cascade, hyperfibrinolysis occurs in some of the more severely injured patients and contributes to ATC.[134-136] The mechanism behind this early fibrinolysis is not clearly understood but may be related to hypoperfusion-induced APC formation resulting in the consumption of plasminogen activator inhibitor. The latter normally serves to downregulate tissue plasminogen activator, which promotes fibrin clot degradation. The reported incidence of hyperfibrinolysis varies significantly based on the methods and cutoffs used to diagnose fibrinolysis, but its presence is clearly associated with the increased mortality and transfusion requirements seen with ATC.

ASSESSMENT OF THE HEMOSTATIC SYSTEM

Given the importance of coagulopathy in the severely injured trauma patient, assessment of the hemostatic system is essential in the early phases of management. This is best accomplished by collecting and integrating information from the four pillars of perioperative coagulation monitoring: (1) medical history, (2) clinical presentation, (3) standard laboratory-based coagulation tests, and (4) viscoelastic monitoring.[137]

Obtaining a focused patient medical history is one of the key pieces for the evaluation of the individual bleeding risk. In some settings, standardized assessments and specific questionnaires can be deployed to raise sensitivity and specificity, resulting in superior detection of hemostasis issues compared to routine coagulation studies alone.[138] The assessment of the clinical phenotypes of bleeding is critical for the differential diagnosis and enables the clinician to differentiate and semi-quantify the underlying coagulation disorder in a very simple and quick fashion. The clinical assessment also helps to put in context abnormal coagulation studies, so that clinical interventions are not based on laboratory values alone. The clinical assessment aims to differentiate whether the cause of bleeding is "surgical" or "nonsurgical." The latter is characterized by a more diffuse pattern. Standard laboratory coagulation tests typically include prothrombin time, international normalized ratio, activated partial thromboplastin time, and platelet count. In some institutions, other laboratory values such as fibrinogen levels, factor XIII, and thrombin time may be part of a routine laboratory coagulation battery. Standard coagulation tests alone play a limited role in the initial diagnostic steps of patients with deranged hemostasis due to trauma. When used in isolation, the standard tests come with significant limitations regarding the detection of extent and origin of a given patient's coagulopathy. These tests allow only the determination of certain questions and are of restricted value. The key restrictions of standard coagulation tests are: delayed results in a dynamic situation, lack of validation, and inability to detect both hyperfibrinolysis and hypercoagulability. A recent meta-analysis concluded that standard plasma coagulation tests represent historically established parameters that are not fully supported by evidence for the management of perioperative coagulopathic bleeding.[139] This is where viscoelastic monitoring as the fourth pillar of perioperative coagulation assessment brings added value. Viscoelastic coagulation monitoring devices like RapidTEG and ROTEM assess the whole coagulation process from initial thrombin generation to maximum clot formation up to clot lysis.[140] In contrast to the standard laboratory coagulation tests, TEG and ROTEM have been better validated for the diagnosis of coagulopathy in trauma patients.[141,142] These tests can significantly improve the coagulation assessment and management of a given patient; and they may reduce unnecessary administration of procoagulant substances such as platelets, fresh frozen plasma (FFP), and coagulation factor concentrates. The rapid turnaround for results may further enable the anesthesiologist to distinguish between a surgical cause of bleeding and trauma-associated coagulopathy.

GENERAL APPROACH TO RESUSCITATION

Fluid administration is the cornerstone of resuscitation (see also Chapters 47 and 86). Intravascular volume is lost to hemorrhage, uptake by ischemic cells, and extravasation into the interstitial space. Administration of intravenous fluids will predictably increase cardiac output and arterial blood pressure in a hypovolemic trauma patient. The ATLS curriculum initially advocated rapid infusion of up to 2 L of warmed isotonic crystalloid solution in any hypotensive patient, with the goal of restoring normal arterial blood pressure. More recently, this has been revised to recognize the importance of a balanced resuscitation with elimination of the emphasis on a more aggressive approach. The current recommendation suggests initiation of resuscitation with 1 L of crystalloid and earlier use of blood and blood products for patients in shock.[33]

The change in ATLS recommendations recognizes that aggressive crystalloid fluid resuscitation during active hemorrhage may be counterproductive. Dilution of red cell mass reduces oxygen delivery and contributes to hypothermia and coagulopathy. Increased arterial blood pressure may lead to increased bleeding because of disruption of clots and reversal of compensatory vasoconstriction.[143] The result of aggressive fluid administration is often a transient increase in arterial blood pressure, followed by increased bleeding, another episode of hypotension, and the need for more volume administration. This vicious circle has been recognized since the First World War and remains a complication of resuscitation therapy today. The ATLS manual characterizes such patients as "transient responders" with active, ongoing hemorrhage.[33] Resuscitation of these patients should be considered in the following three phases (Table 66.2):

- Phase 1, Uncontrolled Hemorrhage: ongoing active bleeding with focus on damage control with pragmatic resuscitation;
- Phase 2, Controlled Hemorrhage: major bleeding sources under control with focus on goal-directed and tailored management of coagulopathy and resuscitation;
- Phase 3, Restoration of Physiology: hemorrhage has been fully controlled with focus on end-organ perfusion and optimization of physiologic state.

Managing late resuscitation (phase 3) is driven by endpoint targets and consists of giving enough fluid to optimize oxygen delivery. Early resuscitation (phase 1) is much more complex because the risks associated with aggressive intravascular volume replacement (Box 66.5), including the potential for exacerbating hemorrhage and prolonging the crisis, must be weighed against the risk for hypoperfusion and ischemia. These phases do not always have distinct transitions and tend to occur as gradual transitions from initial presentation to the OR and ultimately to the ICU.

Phase 1: Uncontrolled Hemorrhage

During the initial phase of management, the goal in trauma patients with massive hemorrhage requiring an emergent surgical procedure in the OR is to stop the bleeding as soon as possible. In this setting, there is little opportunity to perform additional studies, await test results, or evaluate for perioperative optimization. The role of the anesthesia team is to help achieve hemostasis as quickly as possible, while bridging the patient's physiologic status to allow for surgical stabilization. In phase 1, the general approach employs the concept of damage control resuscitation (DCR). DCR combines an empiric hemostatic resuscitation strategy in combination with permissive hypotension during surgical or angioembolization control of ongoing hemorrhage. In combination with surgical approaches incorporating damage control techniques, the initial goal of controlling

TABLE 66.2 Phases of Major Traumatic Resuscitation

	Phase 1	Phase 2	Phase 3
Clinical status	■ Life-threatening uncontrolled hemorrhage	■ Ongoing hemorrhage—not immediately life-threatening—partial surgical control	■ Hemorrhage controlled
Clinical priorities	■ STOP THE BLEEDING ■ Call for HELP ■ Control airway, FiO2 1.0 ■ Damage control resuscitation ■ SBP <100 mm Hg ■ MAP 50-60 mm Hg ■ Consider modifications if TBI, carotid stenosis, CAD	■ TAILORED RESUSCITATION ■ Place supportive lines (arterial/CVC) ■ Prevent hypothermia ■ Esophageal temperature probe ■ Warmed fluids ■ Warming blankets (upper/lower) ■ Increase room temperature	■ RESTORE PHYSIOLOGY ■ Rapid intravascular filling ■ Stepwise deepening of anesthesia ■ Fentanyl boluses ■ Increased volatile anesthetics ■ Additional lines (urinary catheter, nasogastric tube) ■ Communicate with all team members and ICU
Blood products	■ Activate MTP ■ Consider emergency (uncrossmatched blood products ■ Early use ■ Empiric 1:1:1 ratio (PRBC:FFP:platelets)	■ Viscoelastic monitoring to guide coagulation products ■ Hb to guide red blood cell transfusion	■ Only as required on testing ■ Deactivate MTP when appropriate
Crystalloids/colloids	■ Cautious use	■ Use for hypovolemia with normal coagulation/Hb ■ User serial lactate/BD to guide fluid requirements	■ Attempt to normalize lactate/BD
Special points	■ Consider $CaCl_2$ 1 g for every three PRBC ■ Large bore IV access (>16 G) or CVC ■ Rapid infusing system ■ Avoid vasoconstrictors	■ Consider cell salvage if appropriate ■ Aim to repeat viscoelastic testing every 30 min ■ Consider TEE for difficult cases	■ Consider vasoactive infusions if appropriate/necessary

BD, Base deficit; *CAD*, coronary artery disease; *CVC*, central venous catheter; *FFP*, fresh frozen plasma; *FiO2*, fraction inspired oxygen; *Hb*, hemoglobin; *ICU*, intensive care unit; *IV*, intravenous; *MAP*, mean arterial pressure; *mm Hg*, millimeters of mercury; *MTP*, massive transfusion protocol; *PRBC*, packed red blood cells; *SBP*, systolic blood pressure; *TBI*, traumatic brain injury; *TEE*, transesophageal echocardiography

BOX 66.5 Risks of Aggressive Volume Replacement during Early Resuscitation

Increased blood pressure
Decreased blood viscosity
Decreased hematocrit
Decreased clotting factor concentration
Greater transfusion requirement
Disruption of electrolyte balance
Direct immune suppression
Premature reperfusion
Increased risk for hypothermia

Most complications of volume resuscitation arise from increased hemorrhage volume or excessive hemodilution.

hemorrhage, physiologic stabilization, and correction of coagulopathy allows for more definitive surgical interventions later.

Permissive Hypotension. In the setting of trauma, “permissive” rather than “deliberate” hypotension is controversial and has been the focus of numerous laboratory and clinical research efforts. Deliberate hypotensive management is an accepted standard of anesthetic care for elective surgical procedures such as total joint replacement, spinal fusion, radical neck dissection, reconstructive facial surgery, and major pelvic or abdominal procedures.[144] Tolerating some degree of hypotension in the trauma patient during or prior to control of hemorrhage has been more controversial.

In 1965, Shaftan and colleagues[145] published the results of a study of coagulation in dogs that demonstrated that the formation of a soft extraluminal clot limits bleeding after arterial trauma. This study compared the quantity of blood lost from a standardized arterial injury under a variety of conditions. The least blood loss occurred in hypotensive animals (whether hypotensive from hemorrhage or from administration of a vasodilator), followed by the control group and then vasoconstricted animals. The largest amount of blood was lost in animals that underwent vigorous reinfusion during the period of hemorrhage.

Laboratory data have shown the benefits of limiting intravascular fluid volumes and blood pressure in actively hemorrhaging animals.[146-149] In the most sophisticated models, direct assessment of cardiac output and regional perfusion showed no difference between moderate-volume or large-volume resuscitation in terms of cardiac output, arterial blood pressure, or regional perfusion of the heart, kidneys, and intestines. Burris and co-workers[150] studied both conventional resuscitation fluids and various combinations of hypertonic saline and dextran and found that rebleeding was correlated with higher mean arterial pressure (MAP) and that survival was best in groups resuscitated to lower than normal MAP. The optimum target blood pressure for resuscitation varied with the composition of the fluid used. A 1994 consensus panel on resuscitation from hemorrhagic shock noted that mammalian species are capable of sustaining MAP as low as 40 mm Hg for periods as long as 2 hours without deleterious effects. The panel concluded that spontaneous hemostasis and long-term survival were maximized by reduced administration of resuscitation fluids during the period of active bleeding to keep perfusion only just above the threshold for ischemia.[151]

Hypotensive resuscitation has increasingly been accepted as a component of early DCR to avoid excessive administration of fluid until control of active hemorrhage has been achieved. This strategy uses less fluids and blood products during the early stages of treatment for hemorrhagic shock compared with the standard of care.[152] Hypotensive resuscitation has also been shown to be feasible and safe during early resuscitation of trauma patients in the prehospital and hospital settings.[154-157] Early trials suggested outcomes were improved in patients with penetrating trauma[155,156] with no evidence for worsened outcomes in those with blunt trauma.[157]

Initial work by Bickell and colleagues in 1994 randomized victims of penetrating torso trauma to one of two treatment groups: standard of care (up to 2 L of crystalloid infused in the prehospital setting) or delayed resuscitation (no fluid until the patient reached the OR).[155] This well-managed 37-month study eventually included 598 patients. Average times of transport and care were 30 minutes from injury to the ED and then 50 minutes before reaching the OR; the fluid-restricted group received an average of about 0.8 L of fluid during this time. The immediate-resuscitation group received an average of 2.5 L of crystalloid and 130 mL of blood over this same period. Although substantially different during the period of study, blood pressure on arrival at the OR was similar in both groups, which the authors took as evidence that the unresuscitated group had achieved spontaneous hemostasis. Survival to hospital discharge in the delayed-resuscitation group was significantly improved over the immediate-resuscitation group (70% vs. 62% [$P < .04$]). No data were presented on the conduct of anesthesia after arrival at the OR, but before control of hemorrhage, or on the incidence of rebleeding after volume loading and induction of anesthesia in patients who had achieved hemostasis preoperatively.

A retrospective review of trauma admissions to the Los Angeles Medical Center published in 1996 supported these findings. Patients brought to the hospital by private conveyance fared substantially better than those delivered by paramedics, even with high levels of injury severity.[156] Further corroboration was provided by retrospective examination of outcomes in a population of hemorrhaging trauma patients who received fluids via a commercial rapid infusion system (RIS, Haemonetics, Niles, IL) during initial resuscitation.[158] The survival rate of this group was compared with that predicted by the institution’s trauma registry. Patients who received fluid by the rapid infusion system, when compared with case-matched controls, had a survival rate of only 56.8% versus 71.2% for patients of similar age with similar injuries ($P < .001$).

This retrospective review was followed in 2002 by the second prospective trial of delayed resuscitation in trauma patients.[157] Patients with systolic blood pressure lower than 90 mm Hg and clinical evidence of blood loss were randomized to fluid resuscitation titrated to a systolic blood pressure of 100 mm Hg (normal group) or 70 mm Hg (study group) until the end of surgical interventions to control hemorrhage. The results of this study are summarized in Table 66.3. As in the Bickell study, hypotension allowed spontaneous resolution of hemorrhage and autoresuscitation; blood pressure would increase without exogenous fluid

TABLE 66.3 Results of a Randomized Trial of Deliberate Hypotensive Resuscitation

	Conventional	Hypotensive	Total
Patients enrolled	55	55	110
Male	46	41	87
Blunt trauma	22	31	53
Penetrating trauma	33	24	57
Injury severity score	19.65	23.62 (*P* = .11)	
Predicted survival	0.94	0.90 (*P* = .19)	
SBP during study period	114	100 (*P* < .001)	
Survived to discharge	51	51	
Died	4	4	

Probability of survival was calculated based on published historical data.
SBP, Systolic blood pressure.

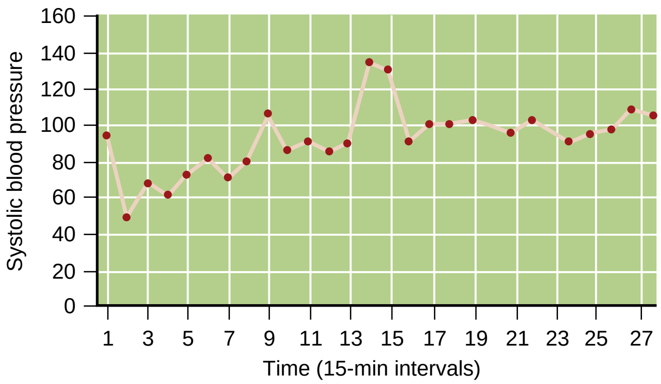


Fig. 66.7 Typical systolic blood pressure measurements of a patient undergoing damage control surgery for a grade V liver injury during deliberate hypotensive management. Oscillations of blood pressure are common during early resuscitation as a result of ongoing hemorrhage and bolus fluid administration. Once hemorrhage is controlled, blood pressure will stabilize.

administration once hemostasis was achieved. The typical patient began with a low initial pressure, followed by recovery to the vicinity of the target, overshoots and undershoots as bleeding and fluid administration continued, and an eventual rise above the target when the hemorrhage resolved, even in the absence of further fluid administration (Fig. 66.7). The 93% overall survival rate in this study was more frequent than predicted from historical data and substantially more frequent than seen in Bickell's group. This reflects the exclusion of patients who died in the prehospital phase or arrived at the trauma resuscitation unit in a moribund condition. It may also reflect improvements in overall care, an observation effect (i.e., patients in both groups received better care than did patients not in the study), or a bias in subject recruitment. Over the first 24 hours, lactate and base deficit cleared to normal in both groups and required similar amounts of fluid and blood products, thus suggesting that both groups were reaching an equivalent resuscitation endpoint. The authors concluded that administration of fluids to an actively hemorrhaging patient should be titrated to specific physiologic endpoints, with the anesthesiologist navigating a course between the Scylla of increased hemorrhage and the Charybdis of hypoperfusion.

TABLE 66.4 Differences in Presentation Between Surgical Patients Undergoing Elective Deliberate Hypotension and Emergency Trauma Cases

Aspect	Elective	Trauma
Intravascular volume	Euvolemic	Hypovolemic
Temperature	Normal	Likely hypothermic
Capillary beds	Dilated	Constricted
Level of general anesthesia	Deep	Usually light
Preexisting mental status	Normal	May be impaired
Coexisting injuries	None	May be significant
Comorbid conditions	Known and managed	Unknown

Each of these factors produces a real or perceived contraindication to the use of deliberate hypotensive technique in the trauma patient.

In a more recent study, Morrison and colleagues compared a hypotensive resuscitative strategy targeting a MAP of 50 mm Hg to one targeting a MAP of 65 mm Hg with conventional resuscitation for patients requiring emergent surgery.[152] In a preliminary report, they found that patients in the hypotensive resuscitation group had a lower, early postoperative mortality, a reduced incidence of coagulopathy, and lower mortality related to coagulopathy. Subsequently, this study was terminated early for futility and clinical equipoise with no significant survival advantage and similar rates of coagulopathy, renal failure, and infection between the low (50 mm Hg) and high (65 mm Hg) MAP target groups.[159] Of note, this study excluded blunt trauma patients included in the preliminary report.[152] Despite not identifying a difference in major outcomes, the authors did make several interesting observations. First, the need for vasopressors use in the first hour of surgery was greater in the high MAP group. Although this group also received more fluid, it was not statistically significant. Second, the intraoperative MAP was not significantly different between the low and high MAP groups (65.5 ± 11.6 mm Hg vs. 69.1 ± 13.8 mm Hg, *P* = .07, respectively). Taken in combination with available data and experience, the current consensus at major trauma centers is to allow for hypotensive resuscitation in the setting of DCR barring specific caveats discussed below. While the optimal arterial blood pressure remains controversial, a reasonable approach is to target a systolic pressure of less than 100 mm Hg with MAP between 50 to 60 mm Hg.[160]

As a component of intraoperative management, the effect of anesthetic drugs on the body's response to hemorrhage is an important difference between deliberate hypotension occurring in the elective operative setting and hemorrhagic shock occurring in the ED. Trauma patients who are hypertensive receive a minimum of anesthetics, even for induction of anesthesia, because of the obvious effect of these drugs on arterial blood pressure. A hypotensive trauma patient is thus in a state of profound vasoconstriction, as opposed to a patient undergoing elective intraoperative hypotension who is vasodilated by general anesthesia before any blood loss. Table 66.4 summarizes the physiologic contrasts between these two states. It should be noted that blood loss without shock does not produce systemic

BOX 66.6 Goals for Early Resuscitation

Maintain systolic blood pressure of 80-100 mm Hg
Maintain hematocrit of 25%-30%
Maintain prothrombin time and partial thromboplastin time in normal ranges
Maintain platelet count at greater than 50,000 per high-power field
Maintain normal serum ionized calcium
Maintain core temperature higher than 35°C
Maintain function of pulse oximeter
Prevent increase in serum lactate
Prevent acidosis from worsening
Achieve adequate anesthesia and analgesia

Fluid administration to limit hypoperfusion is balanced against an undesirable increase in blood pressure and thus bleeding.

complications such as ARDS in experimental models.[111] Based on this physiology, the recommended goals for early resuscitation are expressed in Box 66.6, and an algorithm for management is presented in Figure 66.2. The emphasis in this situation must be on rapid diagnosis and control of ongoing hemorrhage; vascular volume should be restored and anesthesia provided together by shifting the patient from a vasoconstricted to a vasodilated state while facilitating hemostasis by maintenance of a decreased arterial blood pressure.

Clinical trials of permissive hypotensive resuscitation have avoided the application of this technique to populations perceived to be at greater risk for ischemic complications,[155,157] including patients with known ischemic coronary disease, older patients, and those with injuries to the brain or spinal cord. The prohibition against hypotension in patients with TBI is especially well established because of the observed disparity in outcome between TBI patients who experience hypotension and those who do not.[161,162] Older trauma patients suffer worse outcomes from similar injuries than younger patients, presumably because of their reduced physiologic reserve.[163] Clinical care of these patients is focused on avoidance of ischemic stress and rapid correction of hypovolemia. It may well develop, however, that permissive hypotensive management to enable rapid control of hemorrhage is equally beneficial in vulnerable populations. No clinical trials to date have been conducted on this subject, but a laboratory study did find a benefit of permissive hypotension in animals with both TBI and hemorrhagic shock.[164] Absent convincing evidence in humans, permissive hypotension in older patients or patients with brain injury should generally be avoided.

Hemostatic Resuscitation. As discussed earlier, management of the early coagulopathy associated with trauma must be incorporated into the overall phase 1, Uncontrolled Hemorrhage DCR strategy—often referred to as a hemostatic resuscitation. Little utility is found in targeting endpoints of resuscitation in the face of ongoing hemorrhage. Life-threatening coagulopathy is one of the most serious complications of patients in profound shock from massive hemorrhage and is generally predictable at an early stage.[165]

Because of evidence that severely injured trauma patients are likely to develop an early and aggressive endogenous coagulopathy separate from later loss and dilution of clotting factors compounded from hypothermia and acidosis,[126,128,166] the practice of hemostatic resuscitation has become commonplace in the most severely injured patients with shock and ongoing hemorrhage. This entails the early and aggressive use of hemostatic products combined with red blood cells (RBCs) as the primary resuscitation fluids to avoid rapid deterioration into the "bloody vicious cycle" and the classic lethal triad of hypothermia, acidosis, and coagulopathy.[167] Two very distinct paradigms of hemostatic resuscitation have emerged: (1) the DCR model, which uses preemptive administration of empiric ratios of blood and hemostatic products to approximate whole blood, often according to an established institutional massive transfusion protocol (Fig. 66.8)[168-170]; and (2) goal-directed hemostatic resuscitation approaches (also often protocol based), which generally use point-of-care viscoelastic monitoring combined with the prompt administration of hemostatic concentrates.[171-173] The DCR model is typically employed during phase 1, Uncontrolled Hemorrhage, with a transition to more goal-directed hemostatic resuscitation during phase 2, Controlled Hemorrhage.

In addition to the use of permissive hypotension and limited crystalloid usage, DCR relies on the administration of empiric ratios of blood and hemostatic products to restore blood volume. In a retrospective review of combat casualties, Borgman and colleagues[174] found a mortality rate of 65% in patients receiving less than 1 unit plasma for every 4 units RBCs, but only 20% in those with a ratio of 1:2 or above. Evidence exists of a survivor bias because patients bleeding more rapidly were likely to die after receiving RBCs but before plasma could reach the bedside. Although the issue of a survivor bias does exist, this approach has been substantiated in published reviews.[174,175] Currently a ratio of 1:1:1 is most commonly adopted, although some experts believe that the amount of FFP can be reduced in most cases. In the only major, randomized trial comparing a 1:1:1 ratio with a 1:1:2 ratio of plasma, platelets, and RBCs, respectively, the transfusion of less plasma and platelets was not associated with a difference in mortality although the 1:1:1 group achieved hemostasis more rapidly and had fewer deaths due to exsanguination by 24 hours.[176]

In addition to the hypocoagulability associated with ATC, fibrinolysis is especially deleterious in severely injured trauma patients and carries an associated mortality well upward of 50%.[177,178] Many patients with primary fibrinolysis from severe hemorrhagic shock may never survive to reach the ICU. The Clinical Randomisation of an Antifibrolytic in Significant Haemorrhage 2 trial is the only class I evidence showing a 30-day survival benefit for a resuscitative therapy including tranexamic acid (TXA).[179] Subgroup analysis showed that the benefit was greatest when therapy was instituted within 1 hour of admission. A subsequent analysis, however, showed that mortality increased when therapy was instituted after 3 hours, suggesting that the risks of therapy outweighed the benefits in patients who survived beyond that timeframe.[180] Many resuscitative protocols for massive hemorrhage now include the early administration of TXA based on this and subsequent studies.[181]

Other potential drugs that may play a role in hemostatic resuscitation include recombinant activated human coagulation factor VII (rFVIIa), prothrombin complex

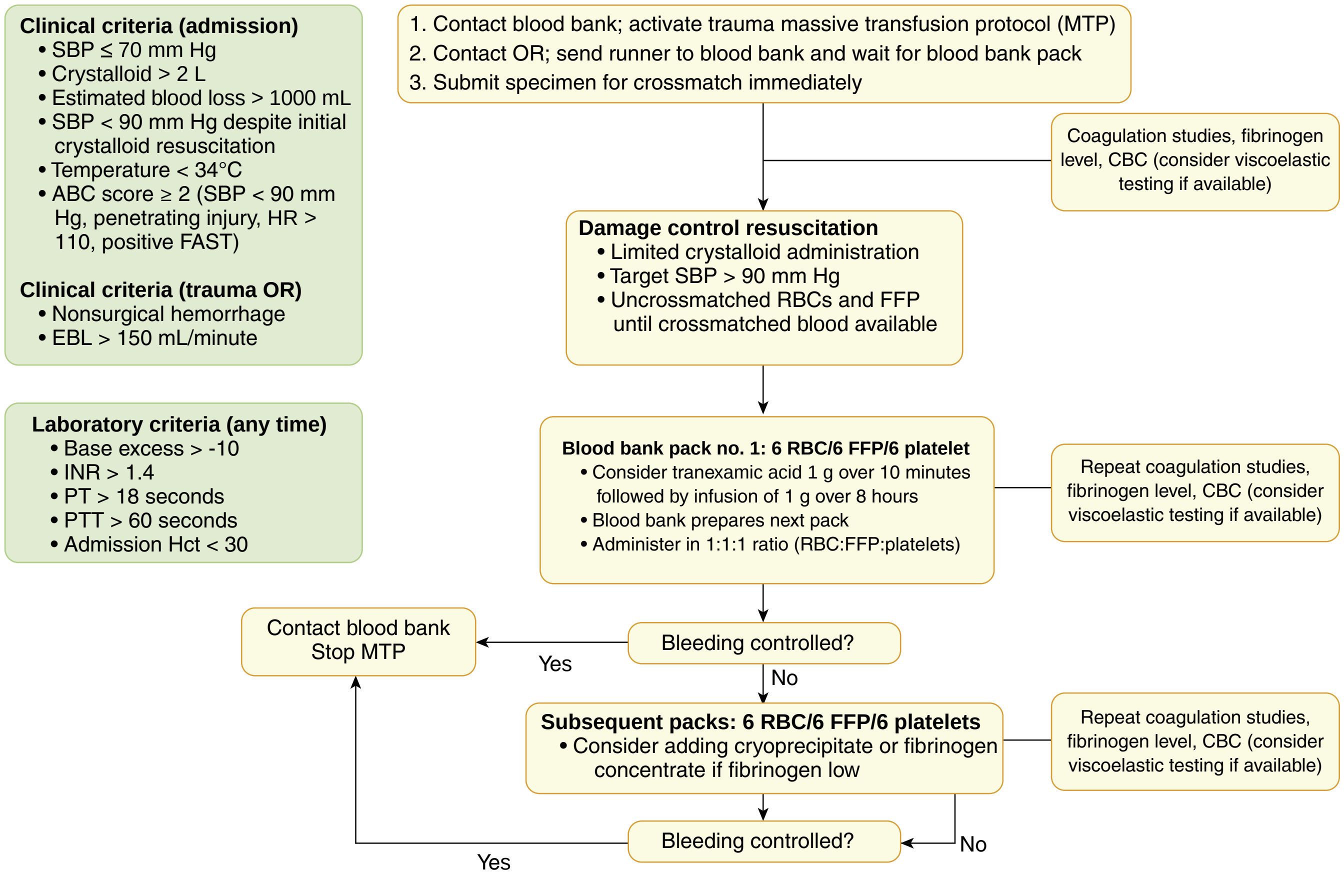


Fig. 66.8 Example of a massive transfusion protocol using specified ratios of blood products. *CBC,* Complete blood count; *EBL,* estimated blood loss; *FAST,* focused assessment with sonography for trauma; *FFP,* fresh frozen plasma; *Hct,* Hematocrit; *HR,* heart rate; *INR,* international normalized ratio; *OR,* operating room; *PT,* prothrombin time; *PTT,* partial thromboplastin time; *RBCs,* red blood cells; *SBP,* systolic blood pressure.

concentrates (PCCs), and fibrinogen concentrates. rFVIIa is licensed for the treatment of patients with hemophilia with active or anticipated hemorrhage and known antibodies to factor VIII. The observation of rapid hemostasis in this population led to the anecdotal use of rFVIIa in other congenital and acquired coagulopathies, including the dilutional coagulopathy of traumatic hemorrhage. Factor VIIa in pharmacologic doses works by triggering a burst of thrombin on the surface of platelets activated by exposed tissue factor, which produces rapid clot formation. Because tissue factor is required, coagulation is limited to the site of vascular injury, and inappropriate clotting of uninjured organs or vessels, though an acknowledged risk, occurs at only low frequency.[182] One small placebo-controlled trial of rFVIIa in hemorrhaging trauma patients has demonstrated decreased blood loss, decreased transfusion requirements, and improved outcome,[183] although a large, randomized trial failed to show a mortality benefit.[184] In combination with an increased risk for thromboembolic adverse events,[185] this lack of clear efficacy has contributed to decreased utilization of rFVIIa as an empiric therapy in the management of the bleeding trauma patient.

Experience with PCCs in clinical practice is limited. PCC has been used for many years for the treatment of congenital coagulation disorders and is recommended for reversing oral anticoagulation, particularly in the setting of traumatic intracranial hemorrhage. PCCs contain coagulation factors II, VII, IX, and X. Differences exist among products in the concentrations of these factors and other constituents, including heparin, protein C, and protein S, so results obtained with one product may not be obtained with a different formulation. Fibrinogen concentrates also may have a role in a hemostatic resuscitation for the patient with a coagulopathy with low levels of fibrinogen.[186-188]

Phase 2, Controlled Hemorrhage

Once major aspects of bleeding have been controlled, a more individualized, tailored approach to resuscitation can generally be employed by the anesthesia team. In phase 2, there should be increased utilization of point-of-care testing such as arterial blood gas and other laboratory test results, viscoelastic monitoring parameters, and physiologic data to drive decision-making. Assessment of oxygen delivery and adequacy of perfusion can start to incorporate some targeted resuscitation measures such as lactate and base deficit. Use of other monitoring modalities such as transesophageal echocardiography (TEE) or transthoracic echocardiography (TTE) may be useful in quantifying and guiding the need for further intravascular volume replacement.

While surgical bleeding may be corrected at this time, the depth and duration of shock in combination with multisystem trauma may still contribute to coagulopathy. This will require ongoing assessment such as serial evaluations with viscoelastic coagulation monitoring. Transition from empiric replacement of FFP and platelets toward a more

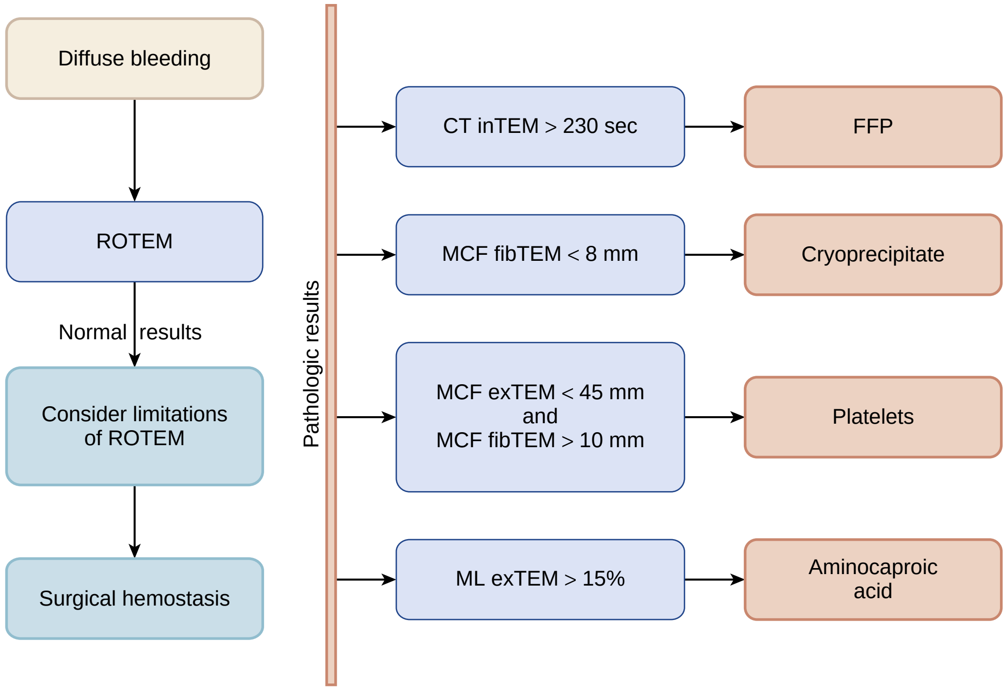


Fig. 66.9 Example of a rotation thromboelastometry *(ROTEM)* treatment algorithm for use in trauma. *CT,* Clotting time; *FFP,* fresh frozen plasma; *MCF,* maximum clot firmness; *ML,* maximum lysis. (Courtesy of San Francisco General Hospital and Trauma Center. (From Steurer M, Chang T, Lancman B. Anesthesia for trauma. In: Pardo M, Miller RD, eds. *Basics of Anesthesia*. 7th ed. Philadelphia: Elsevier; 2018:724 [Chapter 42].)

tailored approach can be accomplished using an algorithm (Fig. 66.9) to guide specific product selection and limit exposure to unnecessary blood products.

Additionally, during this time, attention to other physiologic considerations can proceed. For example, hypothermia is commonly present on initial presentation. During phase 1, all attempts should be made to initiate active fluid and surface warming although it is difficult to fully correct during a massive resuscitation. During phase 2, additional measures can be instituted as the focus shifts from control of hemorrhage to stabilization of all physiologic processes.

Phase 3, Restoration of Physiology

Box 66.7 summarizes endpoints for late resuscitation, and Fig. 66.10 presents an algorithm for management. Intravenous fluid administration is an integral, mandatory component. The adequacy of resuscitation should not be judged by the presence of normal vital signs, but by restoration of organ and tissue perfusion. The role of the anesthesiologist-intensivist is to recognize the presence of ongoing shock after traumatic hemorrhage and to resuscitate the patient with the appropriate type and amount of fluids intravenously at the appropriate time.

Late resuscitation begins once bleeding is definitively controlled by surgery, angiography, or the passage of time. The goal at this time is to restore normal perfusion to all organ systems while continuing to support vital functions. Hypoperfusion caused by hemorrhagic shock triggers a predictable cascade of biochemical events that will cause physiologic derangements persisting long after adequate blood flow is restored. The extent of hypoperfusion—the depth and duration of shock—dictates the magnitude of subsequent organ system failure. Unfortunately, traditional vital sign markers such as arterial blood pressure, heart rate, and urine output are insensitive to the adequacy of resuscitation. Occult hypoperfusion syndrome is common in postoperative trauma patients, particularly young ones.[189] This syndrome is characterized by a normal blood pressure maintained by systemic vasoconstriction, decreased intravascular volume and cardiac output, and organ system ischemia. The patient will be at frequent risk for MOD if the hypoperfusion is not promptly corrected.

The search for the optimal endpoints of resuscitation has led to several different hemodynamic, acid-base, and regional perfusion targets. Table 66.5 summarizes modalities that are available to gauge the adequacy of resuscitation, along with the shortcomings of each technique. Although the flow of blood to tissue beds is a determinant of tissue perfusion, pressure should also be an important consideration. The left ventricular stroke work index is a variable that accounts for both flow and pressure. Furthermore,

BOX 66.7 Goals for Late Resuscitation

Maintain systolic blood pressure higher than 110 mm Hg
Maintain hematocrit above individual transfusion threshold
Normalize coagulation status
Normalize electrolyte balance
Normalize body temperature
Restore normal urine output
Maximize cardiac output by invasive or noninvasive measurement
Reverse systemic acidosis
Document decrease in lactate to normal range

Fluid administration should be continued until adequate systemic perfusion is restored.

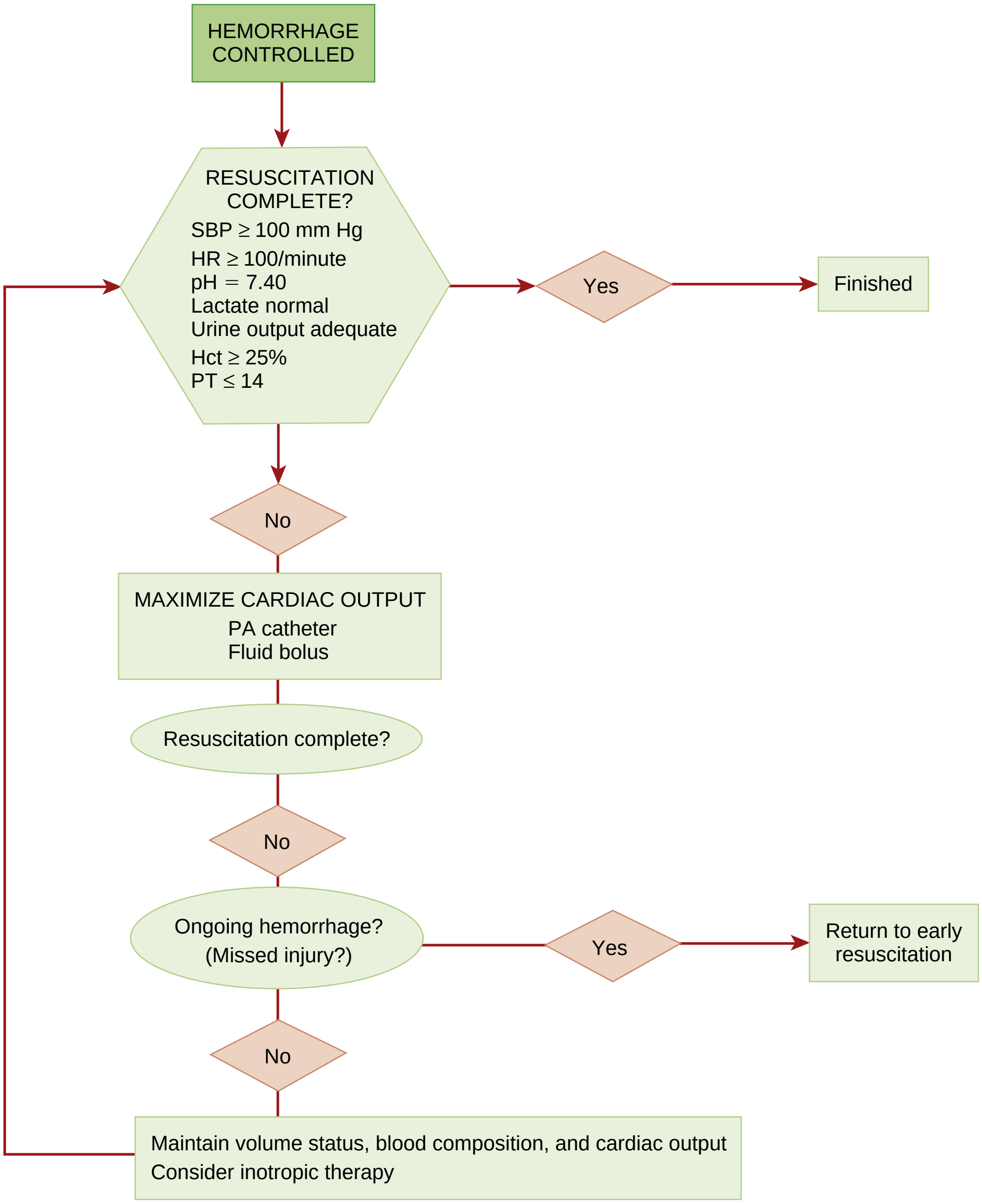


Fig. 66.10 Algorithm for management of late hemorrhagic shock. *Hct,* Hematocrit; *HR,* heart rate; *PA,* pulmonary artery; *PT,* prothrombin time; *SBP,* systolic blood pressure.

left ventricular power output has been used to quantify left ventricular performance. These indices were compared with purely flow-derived hemodynamic and oxygen transport variables as markers of perfusion and outcome in critically injured patients during resuscitation.[190] A consecutive series of 111 patients were monitored with a volumetric pulmonary artery catheter during the first 48 hours of resuscitation. The ability to clear lactate in less than 24 hours and survival were studied. Survivors exhibited significantly higher stroke work and left ventricular power output than did nonsurvivors. In addition to heart rate, these were the only variables that were significantly related to lactate clearance and survival. The higher stroke work and left ventricular power output in survivors were related to better ventricular-arterial coupling and therefore more efficient cardiac function.

Monitoring resuscitation with invasive monitors is gradually changing to noninvasive approaches that assess the return of adequate metabolism, respiration, and oxygen transport in peripheral tissue beds. One such technique is tissue oxygen monitoring (skin, subcutaneous tissue, or skeletal muscle). Skeletal muscle blood flow decreases early in the course of shock and is restored later during resuscitation, thus making the skeletal partial pressure of oxygen a sensitive indicator of decreased flow.[191,192] Stroke volume variation, the change in arterial pressure driven by the respiratory cycle, is emerging as another less invasive measure of fluid volume status; increased variation in arterial pressure during positive-pressure ventilation is a reliable predictor of decreased intravascular volume.[193] Inadequate tissue perfusion, as indicated by these specific monitors or by the traditional systemic markers of serum lactate, base deficit,

TABLE 66.5 Modalities for Assessment of Systemic Perfusion

Technique	Shortcomings
Vital signs	Will not indicate occult hypoperfusion
Urine output	May be confounded by intoxication, diuretic therapy, circadian variation, or renal injury
Systemic acid-base status	Confounded by respiratory status
Lactate clearance	Requires time to obtain laboratory result
Cardiac output	Requires placement of a pulmonary artery catheter, or use of noninvasive technology
Mixed-venous oxygenation	Difficult to obtain, but a very accurate marker
Gastric tonometry	Requires time to equilibrate, subject to artifact
Stroke volume variation	Requires an arterial line; may not be accurate in severe shock
Tissue oxygenation	Emerging technology, appears beneficial

and decreased pH, must be treated promptly once ongoing hemorrhage is controlled. The rate at which lactate returns to normal after shock is strongly correlated with outcome; failure to normalize within 24 hours is associated with a greater risk for MOD and eventual death.[189,194]

RESUSCITATION FLUIDS

Isotonic crystalloids (normal saline, lactated Ringer solution, Plasma-Lyte A) are the initial resuscitative fluids administered to any trauma patient (see also Chapter 47). They have the advantage of being inexpensive, readily available, nonallergenic, noninfectious, and efficacious in restoring total body fluid. They are easy to store and administer, they mix well with infused medications, and they can be rapidly warmed to body temperature. Disadvantages of crystalloids include their lack of oxygen-carrying capacity, their lack of coagulation capability, and their limited intravascular half-life. Of note, data have implicated specific crystalloid solutions as immunosuppressants and triggers of cellular apoptosis.[195] Unlike necrosis, apoptosis is highly regulated and involves gene modulation and complex pathways of signal transduction. It seems clear that apoptosis is an important element of reperfusion injury. In a rat model of controlled hemorrhage, animals receiving lactated Ringer solution showed an immediate increase in apoptosis in the liver and small intestine after resuscitation.[196] Neither whole blood nor hypertonic saline increased the amount of apoptosis.

Hypertonic saline solutions, with or without the addition of polymerized dextran (HS or HSD), have been extensively studied in resuscitation from hemorrhagic shock.[197] In theory, HS will draw fluid into the vascular space from the interstitium and thereby reverse some of the nonhemorrhagic fluid loss caused by shock and ischemia. A given amount of HS will thus have an enhanced ability to restore intravascular volume in contrast to an equivalent volume of an isotonic solution. This has made HS a popular choice for fluid resuscitation under austere conditions. HSD is licensed for prehospital use in some European countries and is used for resuscitation by units of the U.S. military. Multiple studies of otherwise lethal hemorrhage in animals have demonstrated improved survival after resuscitation with HSD versus either normal saline solution or the components of HSD alone. Studies of the efficacy of HSD in trauma patients have been inconclusive[198]; the most obvious benefit occurred in a subset of polytraumatized patients with both hemorrhage and TBI, in whom improved neurologic status was demonstrated in those who received HSD as a resuscitation fluid. Indeed, HS is commonly used as an osmotic agent in the management of TBI with increased ICP.[199]

Colloids, including starch solutions and albumin, have been advocated for rapid plasma intravascular volume expansion. Like crystalloids, colloids are readily available, easily stored and administered, and relatively inexpensive. As with hypertonic solutions, colloids will increase intravascular volume by drawing free water back into the vascular space. When intravenous access is limited, colloid resuscitation will restore intravascular volume more rapidly than crystalloid infusion will and at a lower volume of administered fluid. Colloids do not transport oxygen or facilitate clotting; their dilutional effect will be similar to that of crystalloids. Systematic reviews continue to show no benefit of colloids over crystalloids in the setting of trauma resuscitation,[200] although this topic continues to generate significant controversy and would benefit from several well-conducted, randomized trials.[201] Recent concerns, however, have been expressed about specific colloids such as 6% hetastarch and an adverse effect on renal function and mortality (see also Chapter 49).[202-204]

Many of the risks of aggressive intravascular fluid administration just summarized are related to dilution of the circulating blood volume. Recognition of this fact and continued improvement in the safety of donated blood led to increased use of blood products in the management of early hemorrhagic shock (see also Chapter 49). The risk for systemic ischemia is decreased by the maintenance of an adequate hematocrit, and the potential for dilutional coagulopathy can be decreased by the early administration of plasma. The composition of resuscitation fluids may be as important as the rate and timing of administration. A 4-year retrospective review of a cohort of critically injured patients who underwent emergency surgery examined the outcomes of short-term care based on the number of units of blood transfused.[205] One hundred forty-one patients received massive blood transfusions (≥20 units of RBCs) during preoperative and intraoperative resuscitation. The number of blood units did not differ between survivors (30%) and nonsurvivors (70%). Eleven variables were significantly different: aortic clamping for control of arterial blood pressure, use of inotropic drugs, time with a systolic blood pressure higher than 90 mm Hg, time in the OR, temperature less than 34°C, urine output, pH lower than 7.0, PaO_2/FiO_2 ratio less than 150, $PaCO_2$ more than 50 mm Hg, K^+ more than 6 mM/L, and calcium less than 2 mM/L. Of these, the presence of the first three variables in the face of transfusion of more than 30 units of packed RBCs was invariably fatal. Total blood loss and the amount of transfused blood were less critical than the depth and duration of shock. These concerns led to the concept of damage control surgery discussed earlier, which emphasizes rapid control of active hemorrhage.[206]

RBCs are the mainstay of treatment of hemorrhagic shock. With an average hematocrit of 50% to 60%, a unit of RBCs will predictably restore oxygen-carrying capacity and expand intravascular volume as well as any colloid solution. RBCs of blood type A, B, or AB carry major incompatibility antigens that may precipitate a lethal transfusion reaction if given to a patient with the opposite blood type. Because RBCs also carry dozens of minor antigens that can cause reactions in susceptible patients, crossmatching is desirable when time allows (typically 1 hour from the time a sample reaches the blood bank until the RBCs reach the patient). Type-specific blood requires less time for delivery from the blood bank (usually ~30 minutes) and may be an appropriate alternative in some situations. Type O blood—the universal donor type—can be given to patients of any blood type with little risk for a major reaction.[207] This is the preferred approach for patients who arrive at the ED in hemorrhagic shock. If O-positive blood is given to a Rhesus-negative woman who survives, prophylactic administration of anti-Rh_0 antibody is indicated.

Risks of RBC administration include transfusion reaction, transmission of infectious agents, and hypothermia (see also Chapter 49 for details). For example, RBCs are stored at 4°C and will decrease the patient's temperature rapidly if not infused through a warming system at the time of administration.

Plasma requires blood typing but not crossmatching; delay in availability of plasma is caused by the need to thaw frozen units before they can be administered. Busy trauma hospitals will often maintain a supply of prethawed plasma (thawed fresh plasma as opposed to FFP) that can be issued quickly in response to an emergency need; in smaller hospitals it is important to request plasma early in resuscitation if it is likely to be needed. Very busy centers are experimenting with keeping 2 to 4 units of prethawed type AB (universal donor) plasma available in the trauma resuscitation unit. Units are kept ready in this way for 2 days at a time; if not used on an emergency basis, the units are returned to the blood bank and released to the next patient needing plasma. Whether this approach improves outcomes has not yet been studied.

Platelet transfusion should normally be reserved for patients with clinical coagulopathy with a documented low serum level (>50,000 per high-power field). When the patient is in shock, however, and blood loss is likely to be substantial, platelets should be empirically administered in proportion to RBCs and plasma (1:1:1), as discussed earlier for DCR. Transfused platelets have a very short serum half-life and should be administered only to patients with active coagulopathic bleeding. Platelets should not be administered through filters, warmers, or rapid infusion systems because they will bond to the inner surfaces of these devices, thereby reducing the quantity of platelets actually reaching the circulation.

Rapid transfusion of banked blood carries the risk for inducing citrate intoxication in the recipient.[208] Harvested blood units are treated with citrate to bind free Ca^{2+} and thus inhibit the clotting cascade. Consecutive administration of multiple units of banked blood leads to a correspondingly large dose of citrate, which may overwhelm the body's ability to mobilize free Ca^{2+} and have a profound negative inotropic effect on the heart. Unrecognized hypocalcemia is a cause of hypotension in patients after massive transfusion and persists despite an adequate volume of resuscitation. Ionized Ca^{2+} levels should be measured at regular intervals in a hemorrhaging patient, and Ca^{2+} should be administered as needed (in a separate intravenous line from transfusion products) to maintain serum levels greater than 1.0 mmol/L.

RESUSCITATION EQUIPMENT

Intravascular fluid resuscitation of any kind is impossible in the absence of intravenous access. Immediate placement of at least two large-bore catheters (16 gauge or larger) is recommended during the primary assessment of any trauma patient.[33] Practitioners should have a low threshold for placement of a large-caliber central line in any patient in whom antecubital or other peripheral placement attempts have been unsuccessful. Potential sites for central line placement include the internal jugular, subclavian, and femoral veins, each of which has its own benefits and potential risks. The internal jugular approach, though familiar to most anesthesiologists, will require removal of the cervical collar and manipulation of the patient's neck and is not recommended in the acute setting unless other options have been exhausted. The femoral vein is easily and rapidly accessed and is an appropriate choice in patients without apparent pelvic or thigh trauma who require urgent drug or fluid administration. Caution should be used in patients with penetrating trauma to the abdomen because fluids infused via the femoral vein may contribute to hemorrhage from an injury to the inferior vena cava or iliac vein; these patients should have intravenous access placed above the diaphragm if possible. Femoral vein catheterization carries a high risk for the formation of deep venous thrombosis,[209] thus limiting use of this approach to the acute setting. Femoral lines should be removed as soon as possible after the patient's condition stabilizes. The subclavian vein is the most common site for early and ongoing central access in a trauma patient because the subclavian region is easily visible and seldom directly traumatized. This approach carries the highest risk for the development of pneumothorax, although many patients will already have indications for tube thoracostomy in one or both chest cavities; when possible, this is the preferred side for placement of a subclavian line. Placement of an arterial line facilitates frequent laboratory analysis and allows close monitoring of blood pressure; this should be undertaken as soon as possible but should not impede other diagnostic or therapeutic maneuvers.

The anesthesiologist should work to maintain thermal equilibrium in any trauma patient. Although deliberate hypothermia has been suggested as a management strategy for both hemorrhagic shock[210] and TBI,[211] insufficient evidence exists to support this approach. Hypothermia will potentiate dilutional coagulopathy and systemic acidosis while shivering and vasoconstriction in response to cold will demand an additional metabolic effort that may predispose the patient to myocardial ischemia. Hypothermia also greatly increases the subsequent risk for sepsis. Because many trauma patients arrive at the ED cold from exposure to the elements, early active warming measures are required. All IV fluids should be prewarmed or infused through a warming device. The patient should be kept covered with warmed blankets whenever possible, and the environment should be kept warm enough to make the patient comfortable. If hypothermia has already developed, the use of forced hot air warming is strongly indicated to restore normothermia. Even though all these measures are routine and obvious in the OR, the anesthesiologist can perform a valuable service by ensuring that they are available and applied in the ED, CT scanner, and angiography suite as well.

Commercial rapid-infusion devices are of great benefit in trauma care, particularly in the presence of hemorrhagic shock. These machines offer benefits when large quantities of fluid resuscitation are likely (Box 66.8). Early experience with these devices demonstrated higher patient temperature and reduced acidosis at the conclusion of the initial surgery,[212] although rapid infusers may contribute to over infusion of fluids, inappropriately increased arterial blood pressure, and contribute to rebleeding.[158] Application of the principles described earlier for permissive hypotension can prevent this complication; basal fluid administration should be kept low (200-500 mL/h), with fluid boluses given in response to measured systolic blood pressure less than 80 to 90 mm Hg. In practice, fluid boluses are given alternately with anesthetics, with the goal of reaching a normal depth of anesthesia without significantly increasing systolic arterial blood pressure until active bleeding is controlled.

Trauma to the Central Nervous System

CNS trauma accounts for almost half of all trauma deaths after trauma center admission and as indicated by postmortem examinations in population-based analyses (see also Chapter 57).[213,214] In the United States, 2.8 million TBI-related ED visits, hospitalizations, and deaths occurred in 2013.[215] Although not as prevalent, acute traumatic SCI also remains a significant problem.[216] As with hemorrhagic shock, CNS trauma consists of both the primary injury, in which tissue is disrupted by mechanical force, and a secondary response, in which the body's reaction to the injury plays an important role. Mitigation of secondary CNS injury depends on prompt diagnosis and early, goal-directed therapy. Although primary CNS injuries cannot be minimized other than with preventive strategies, secondary injury accounts for much of the death and disability that follows trauma.[217] The initial management of these patients can significantly affect outcome. Attention to the ABCDE approach is paramount to successful resuscitation, and the trauma anesthesiologist should be intimately involved in this process.

Trauma produces shear forces that result in primary damage to neuronal cell bodies and axons, and to the vasculature. The pathophysiologic processes of secondary injury include metabolic failure, oxidative stress, and a cascade of biochemical and molecular events leading to both delayed necrotic and apoptotic cell death.[217] Secondary injury is often exacerbated by tissue hypoxia or ischemia and by inflammatory responses, and outcomes from TBI are influenced by a large number of interacting factors. Individual drugs such as free radical scavengers, antiinflammatory agents, and ion channel blockers have been effective in animals but have had little impact or yielded disappointing results in human trials. Long-term outcome cannot be predicted from TBI; thus full resuscitative efforts should be initiated and continued, even in the most severely injured. In a recent review of the Trauma Registry of the German Society for Trauma Patients looking at over 50,000 patients, outcome prediction was most accurate using the GCS motor score and pupil reactivity. In patients with bilateral fixed and dilated pupils, a good outcome was still seen in 8% of patients.[218]

Patients with mild TBI (GCS score of 13-15) who maintain a stable GCS score for 24 hours after injury are very unlikely to deteriorate further, although they are at risk for several postconcussive effects, including headache, memory loss, emotional lability (including aggressive behavior and violence), and sleep disturbance.[219] Moderate TBI (GCS score of 9-12) may be accompanied by intracranial lesions that require surgical evacuation, and early CT is strongly indicated. Early tracheal intubation, mechanical ventilation, and close observation may be required in the management of patients with moderate TBI because of combative or agitated behavior and the potentially catastrophic consequences of respiratory depression or pulmonary aspiration occurring during diagnostic evaluation. Extubation of the trachea can be undertaken if the patient is hemodynamically stable and appropriately responsive after the diagnostic workup. Treatment of secondary brain injury is accomplished by early correction and subsequent avoidance of hypoxia, prompt intravascular fluid resuscitation, and management of associated injuries. The timing of indicated noncranial surgery in these patients with moderate injury not requiring decompressive craniectomy is highly controversial because early surgery can increase the episodes of hypoxia and hypotension.[220]

Neurologic monitoring of patients with moderate TBI consists of serial assessment of consciousness and motor and sensory function (see also Chapter 39). Deterioration of the GCS score is an indication for urgent CT to establish the need for craniotomy or invasive monitoring of ICP. If frequent neurologic monitoring is not possible because of general anesthesia continuing longer than 2 hours, aggressive analgesia, or prophylaxis against delirium tremens,

BOX 66.8 Benefits of a Fluid Infusion System in Resuscitation from Hemorrhagic Shock

- An active mechanical pump enables fluid administration rates up to 1500 mL/min
- Compatible with crystalloid, colloid, packed red blood cells, washed salvaged blood, and plasma (*not* platelets)
- Reservoir allows for mixing of products in preparation for rapid blood loss
- Fluids infused at a controlled temperature (38°C-40°C)
- Able to pump simultaneously through multiple intravenous lines
- Fail-safe detection systems to prevent infusion of air
- Accurate recording of fluid volume administered
- Portable enough to travel with patient between units

invasive ICP monitoring may be indicated.[162] Although mortality from moderate TBI is infrequent, many patients will have significant long-term morbidity.

Severe TBI is classified as a GCS score of 8 or less at the time of admission and carries a significant risk for mortality. Patients with severe TBI have mortality three times that of patients with other types of traumatic injury.[221] Early, rapid management focused on restoration of systemic homeostasis and perfusion-directed care of the injured brain will produce the best possible outcomes in this difficult population. Guidelines for all aspects of the management of patients with severe TBI have been published by the American Association of Neurological Surgeons and Brain Trauma Foundation, now in the fourth edition.[222] The clinical pathway in place at the R Adams Cowley Shock Trauma Center in Baltimore appears in Fig. 66.11.

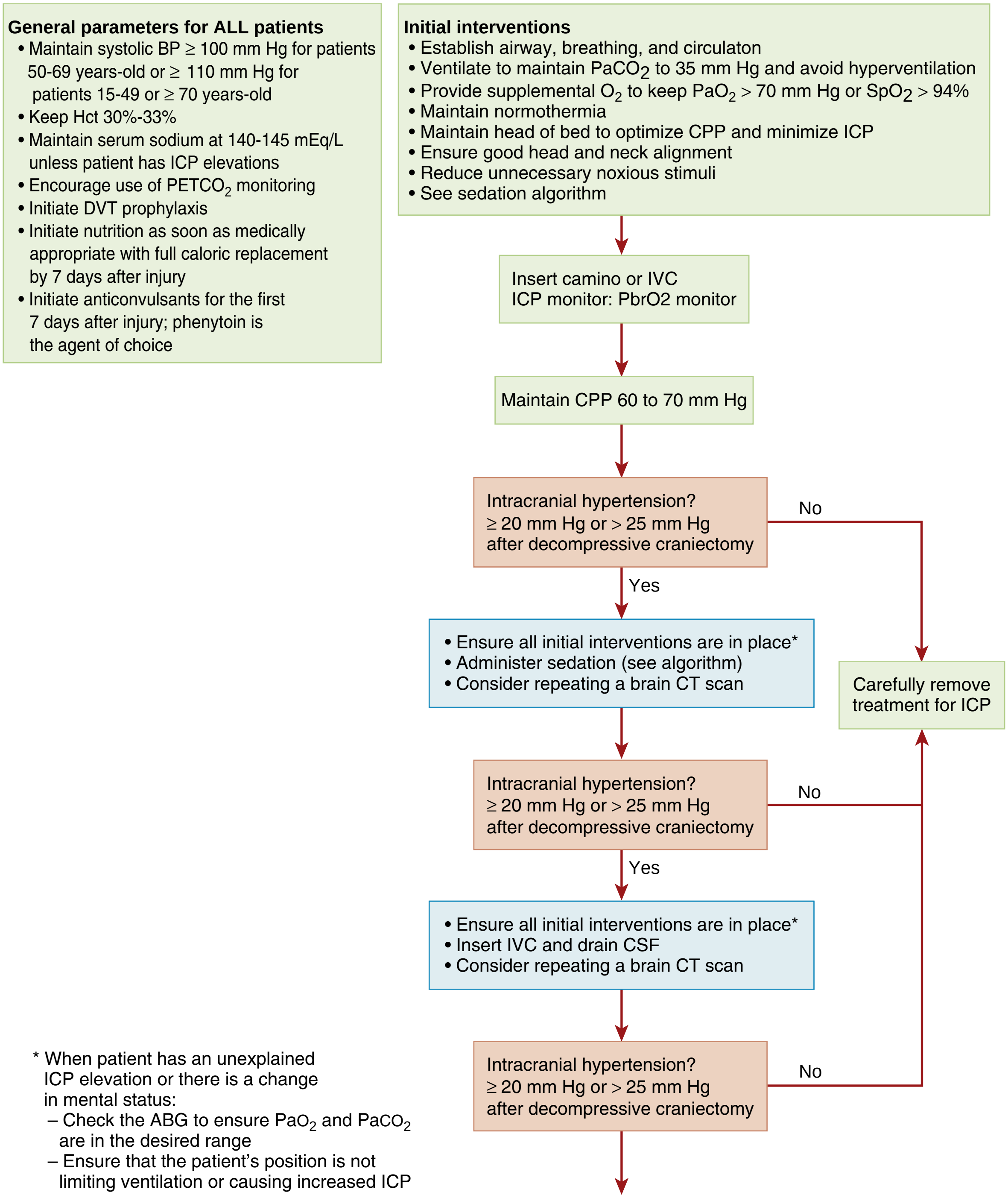


Fig. 66.11 Clinical pathway for management of severe traumatic brain injury. The goal of therapy is to maintain cerebral perfusion pressure 60 to 70 mm Hg by support of the circulation and control of intracranial pressure. Progressively more intensive therapies are added until this goal is achieved. *ABG*, Arterial blood gas; *BP*, blood pressure; *CBF*, cerebral blood flow; *CPP*, cerebral perfusion pressure; *CSF*, cerebrospinal fluid; *CT*, computed tomography; *DVT*, deep venous thrombosis; *Hct*, hematocrit; *ICP*, intracranial pressure; *IVC*, intraventricular catheter.

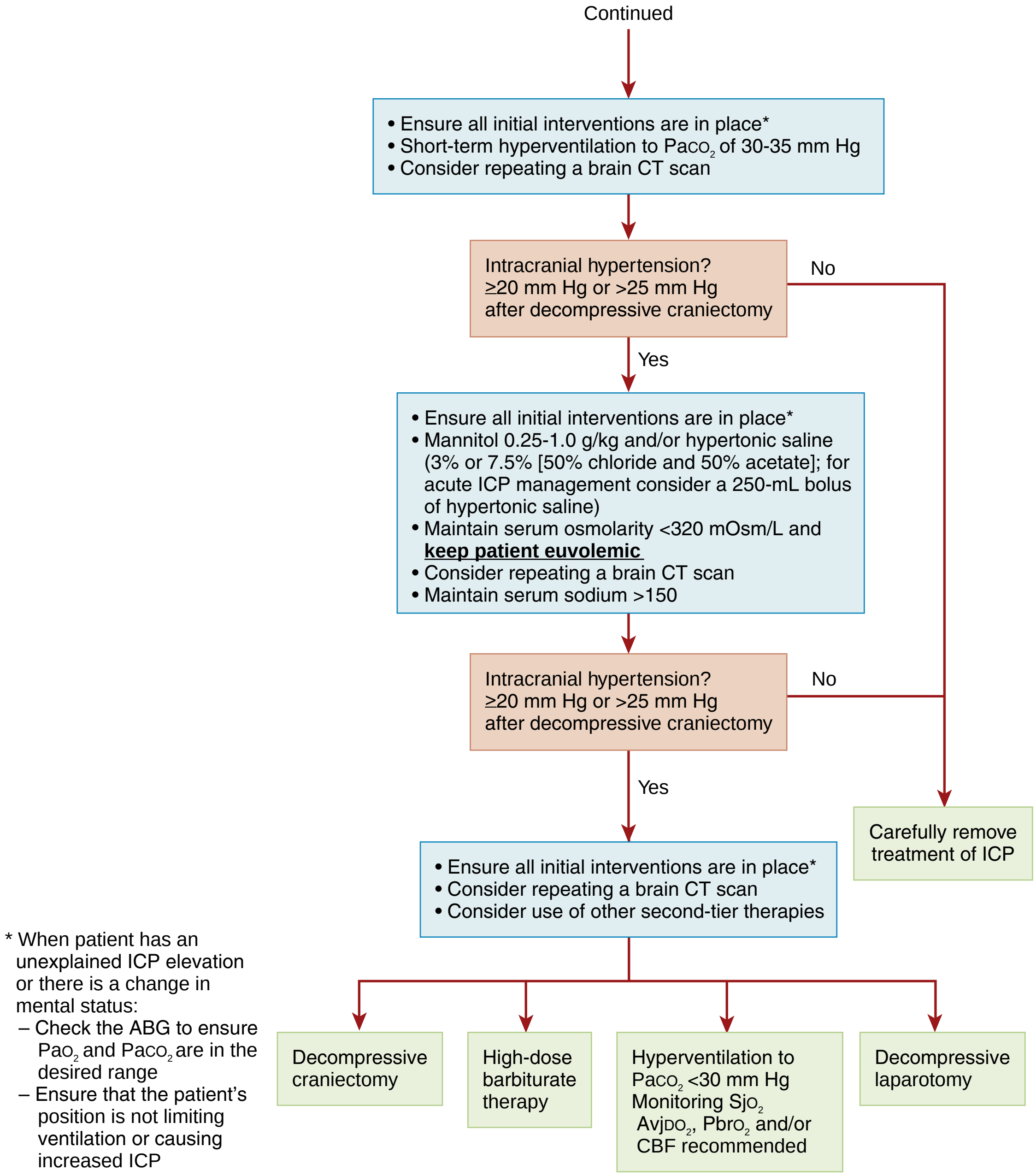


Fig. 66.11, Cont'd

A single episode of hypoxemia (PaO_2 < 60 mm Hg) occurring in a patient with severe TBI can double the incidence of mortality.[223] Prehospital tracheal intubation, nonetheless, is controversial. Previously, intubation of the trachea before arriving at the hospital was advocated because providing a definitive airway allowed adequate oxygen to be delivered to the brain, benefiting the patients. Yet worsened neurologic outcomes have been described with attempts at prehospital tracheal intubation in adult trauma patients.[224,225] The first prospective trial of prehospital intubation, conducted in urban Australia, randomized patients with severe TBI—defined as evidence of head trauma and GCS of less than 9—to intubation by paramedics in the field or by physicians on the arrival to hospital.[226] Of 312 patients, the proportion with favorable outcome was 51% in the paramedic group, compared to 39% in the hospital tracheal intubation group (P = .046). Because no international standard or consensus exists, the patient should be transported as rapidly as possible to a facility capable of managing severe TBI or to the nearest facility capable of tracheal intubation of the patient and initiation of systemic resuscitation. The sine qua non is adequacy of systemic oxygenation, by whatever means this can best be accomplished.

The patient with TBI will frequently require perioperative monitoring to include ICP, brain temperature, arterial pressure, cerebral oxygenation, and other advanced monitoring techniques (see also Chapter 39). ICP may be monitored with an intraparenchymal probe or intraventricular catheter and should be maintained at less than 20 mm Hg. A variety of monitoring devices have been used to assess adequacy of cerebral oxygenation, including jugular venous oxygen saturation, positron emission tomography, near-infrared spectroscopy, and direct brain tissue oxygenation ($Pbto_2$) monitoring.[227] Brain tissue hypoxia may be corrected by increasing FiO_2, blood transfusion, inotropic support, or sedation, if increased ICP results in decreased cerebral blood flow.[228-231] Small studies suggest an improvement in Glasgow Outcome Score and mortality with brain oxygen–targeted strategies, but no overall consensus exists.

Finally, patients with severe TBI are also at risk for non-CNS organ failure. A retrospective review of patients with isolated TBI found a frequent incidence of subsequent organ failure: 89% developed dysfunction of at least one non-neurologic organ system. Takotsubo cardiomyopathy may develop in the setting of severe TBI and lead to severe myocardial dysfunction in the patient with brain injury.[232] The reasons for development include interplay between the neuroendocrine system and the injured brain. A catecholamine surge that occurs after TBI may manifest as subendocardial ischemia, leading to biventricular heart failure, even in young, previously healthy patients. This cycle may be exacerbated during OR procedures when vasoactive drugs might be administered. β-Adrenergic blockade may be protective in human studies in patients with brain injuries. Retrospective database reviews have indicated improved neurologic outcome and reduced morbidity and mortality in patients receiving peri-injury β-adrenergic blockade although the quality of evidence is not high.[233-236]

TRAUMATIC BRAIN INJURY AND CONCOMITANT TRAUMA

Patients with isolated head injuries can be managed with traditional ventilatory strategies, but those with chest trauma, aspiration, or massive resuscitation after shock are at high risk for the development of ARDS. The classic teaching of no or low-level positive end-expiratory pressure (PEEP) to prevent increased ICP is inappropriate because hypoxemia may not be corrected. With adequate intravascular volume resuscitation, PEEP does not increase ICP or decrease cerebral perfusion pressure (CPP)[237] and may actually decrease ICP as a result of improved cerebral oxygenation.[238] Patients who develop ARDS after TBI are at risk for cerebral hypoxia.[239] A double-hit model has been proposed as the cause of ARDS secondary to CNS trauma—severe TBI leads to a systemic inflammatory response, which then primes the lungs to be more susceptible to injurious ventilator strategies or other mediators of lung injury. ARDS and cardiac insufficiency after TBI may lead to compromised systemic oxygenation and therefore a decrease in cerebral oxygen delivery.[240] Hyperventilation therapy (e.g., at a $PaCO_2$ of 25 mm Hg), long a mainstay in the management of patients with TBI, is no longer recommended as a prophylactic treatment. A range of $PaCO_2$ between 30 and 35 mm Hg should be induced with hyperventilation to 30 mm Hg only for episodes of increased ICP that cannot be controlled with sedatives, drainage of cerebrospinal fluid, neuromuscular blockade, osmotic drugs, or barbiturate coma.[161] Hyperventilation during the first 24 hours is of concern because of the critical reductions in perfusion during this timeframe. However, these recommendations should be taken in context and modified in the face of unstable clinical circumstances such as an expanding mass lesion or signs of imminent herniation.[241]

The most challenging of all trauma patients are those with severe TBI and coexisting hemorrhagic shock. A single episode of hypotension, defined as systolic blood pressure lower than 90 mm Hg, is associated with an increase in morbidity and doubled mortality after severe TBI.[242] Hypotension together with hypoxia increases mortality 30-fold. Current recommendations are to maintain a patient with severe TBI in a euvolemic state. Therefore *fluid resuscitation is the mainstay of therapy*, followed by vasoactive infusions as needed. The ideal fluid has not been defined, but perhaps hypertonic saline solutions are optimal. Correction of anemia from acute blood loss is the priority; however, an optimal target hematocrit has not been defined. Animal models and healthy human subjects support maintaining a hemoglobin lower than 7 g/dL because impaired brain function may occur with lower values, yet a hemoglobin lower than 10 g/dL may be detrimental to recovery from TBI.[243] At this time it is not clear what optimal transfusion trigger should be used (see also Chapter 49). After the initial ABCDE management of a patient with severe TBI, a stepwise approach to maintenance of CPP is initiated, with a currently recommended goal range of 60 to 70 mm Hg, as discussed in Chapter 57.

Decompressive craniectomy is a surgical procedure used to control severely increased ICP and prevent herniation after stroke, and it is now used for the same indications after severe TBI.[244] Decompressive craniectomy is indicated for selected anatomic patterns of TBI, such as when CPP cannot be maintained despite vigorous application of the previously described therapies, including barbiturate coma. Relieving ICP by removal of a piece of cranium and use of a dural patch may improve mortality and morbidity in patients who might not otherwise survive.[245] Decompressive laparotomy may be indicated in patients with severe TBI if coexisting injuries or vigorous intravascular volume infusion have increased intraabdominal compartment pressure to greater than 20 mm Hg. Increased abdominal pressure worsens pulmonary mechanics, thus necessitating higher MAP to maintain arterial oxygen saturation. This increase in ventilating pressure will increase intrathoracic pressure and impair venous drainage from the head and consequently decrease CPP. The use of decompressive celiotomy as a therapy to reduce ICP has been described[246] for managing patients with severe TBI and a potential multiple compartment syndrome.[247] Fluid therapy, ARDS, or both may increase intraabdominal pressure and intrathoracic pressure, thereby increasing ICP. Further fluid administration to support cerebral perfusion or increasing ventilatory support to treat ARDS further exacerbates this problem. This can create a cycle that ultimately produces multiple compartment syndrome and necessitates opening of the abdomen, even in the absence of primary abdominal trauma. Isolated TBI can thus become a multisystem disease.

Like hyperventilation therapy, the use of hypothermia to treat severe TBI has changed. Early studies demonstrated that moderate, systemic hypothermia reduces both the rate of cerebral edema and mortality after cortical injury in laboratory animals.[248,249] Small clinical series in humans also suggested improved outcome in patients with TBI when hypothermia was maintained for 24 or 48 hours.[250,251] However, a multicenter, randomized trial of hypothermia (33°C) versus normothermia demonstrated no improvement in outcome in a population of patients with severe TBI.[252] Patients who were hypothermic on admission and then were randomized to the normothermia group had a worse outcome than those who were left hypothermic, thus leading to the recommendation that patients with severe TBI who are hypothermic on admission not undergo active rewarming. Current Brain Trauma Foundation guidelines do not recommend early, short-term, prophylactic hypothermia.[222]

MANAGEMENT OF INTRACRANIAL PRESSURE IN THE OPERATING ROOM

Although most interventions for management of a patient with severe TBI will occur in the ICU, urgent cranial or noncranial surgery can be indicated. The previously described therapies should be continued throughout the perioperative period, including positional therapy (when possible), aggressive hemodynamic monitoring and resuscitation, administration of osmotic agents (with attention to maintaining euvolemia), and deep levels of analgesia and sedation. Appropriate anesthetic choices include opioids and low concentrations of volatile anesthetics. Pharmacologic management of TBI is the mainstay of the anesthesiologist's role in the OR. Intraoperative management of elevated ICP may be accomplished with osmotic diuretics or hypertonic saline. No difference was noted in long-term outcomes between mannitol and hypertonic saline on outcomes at 6 months, although hypertonic saline may potentiate small short-term improvements in cerebral blood flow and CPP.[253,254] A meta-analysis of common drugs used in intraoperative management of patients with TBI, including propofol, barbiturates, opioids, benzodiazepines, and corticosteroids, showed corticosteroids to be the only drug associated with an increase in mortality. The other drugs had acute benefit in reductions in ICP and sedation, but no appreciable long-term benefit.[219,222]

SPINAL CORD INJURY

SCI after trauma affects some 13,000 Americans each year.[216] Blunt trauma accounts for most of SCI: 36% from motor vehicle collisions and 42% from falls with only 4% from firearms. Cervical spine injuries occur in 1.5% to 3% of all major trauma. Over 50% of SCIs involve the cervical spine, usually between the C4 and C7 levels. Complete quadriplegia occurs in 11% of SCI cases. It is important to realize that more than 40% of trauma patients who have SCI may also have other significant injuries, including TBI.

Most spinal injuries are in the lower cervical spine, just above the thorax, or in the upper lumbar region, just below the thorax. Blunt SCI is most common in the regions of the cord that are most flexible, especially at the junctions between flexible and inflexible segments. The vertebral column is divided longitudinally into three columns: anterior, middle, and posterior; injuries to any two of these three columns suggest biomechanical instability. These are the patients who will often require urgent surgical stabilization. Patients with unstable cervical spine injuries who meet criteria for emergency intubation should undergo rapid sequence induction (see section above on protection of the cervical spine). SCI at midthoracic levels is less common because of the rotational stabilization provided by the rib cage and intercostal musculature.

SCI is commonly accompanied by radiographically visible injury to the bony spine and concomitant disruption of the muscles, ligaments, and soft tissues that support it. However, clinically significant injury to the cervical spinal cord can occur in the absence of visible skeletal injury. This entity, known as SCI without radiographic abnormality (SCIWORA), is more common in children and is presumably the result of temporary hyperdistraction or torsion of the neck insufficient to disrupt the bony skeleton.[255]

Primary injury to the spinal cord sustained at the time of injury may be exacerbated by secondary factors (Fig. 66.12). The combination of biochemical changes, vascular disruption, and electrolyte abnormalities can lead to cellular changes and worsening of SCI lesions up to 3 days after injury.[256] SCI includes sensory deficits, motor deficits, and both. Incomplete deficits may be worse on one side than the other and may improve rapidly in the first minutes after injury. Complete deficits—representing total disruption of the spinal cord at one level—are much more ominous, with generally little improvement seen over time. Spine injuries above the level of T4 to T6 are accompanied by significant hypotension because of inappropriate vasodilatation, loss of cardiac inotropy, and bradycardia resulting from denervation of the cardiac accelerator fibers (neurogenic shock). (Note the distinction between *neurogenic shock* and the incorrect term *spinal shock*, which refers to a loss of reflexes.) Functioning of the lower cord will gradually return, along with restoration of normal vascular tone. Diagnosing cervical spine instability can be difficult. The Eastern Association for the Surgery of Trauma has published guidelines regarding which patients require cervical spine radiographs, which views and studies should be obtained, and how to determine the absence of significant ligamentous injury in an obtunded patient.[257] For the obtunded patient, a high-quality CT scan without evidence of cervical SCI is considered to be adequate for clearance although this is not universally accepted. Many institutions still require magnetic resonance imaging to rule out ligamentous injury in this patient population. The most commonly missed cervical fractures are at the C1 to C2 and C7 to T1 levels, usually the result of inadequate imaging.

Early intubation of the trachea is almost universally required for patients with cervical spine fracture and quadriplegia. Ventilatory support is almost always required for patients with a deficit above C4, because they will lack sufficient diaphragmatic function. Patients

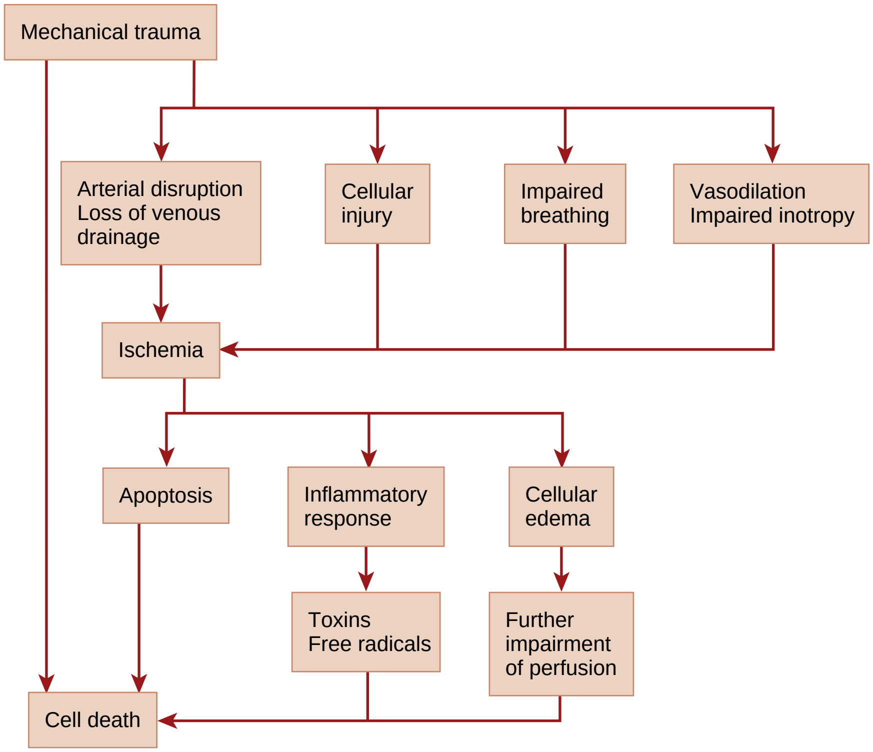


Fig. 66.12 Mechanisms of spinal cord injury. Mechanical trauma to the spinal cord is exacerbated by systemic hypoperfusion or hypoxia. (Reprinted with permission from Dutton RP. Spinal cord injury. *Int Anesthesiol Clin.* 2002;40:109.)

with levels from C6 to C7 may need support because of impairment in respiratory muscle function resulting from (1) loss of chest wall innervation, (2) paradoxical respiratory motion, (3) inability to clear secretions, and (4) decreased lung and chest wall compliance. Early tracheal intubation is recommended and, if elective, often can be accomplished by awake fiberoptic bronchoscopy or video laryngoscopy before hypoxia renders the patient anxious and uncooperative.[258,259] Also, two retrospective studies have evaluated the need for tracheal intubation in patients with cervical SCI. Como and colleagues[258] identified 119 patients, 45 of whom had complete SCI. All patients with injuries at C5 and above required intubation, and 71% of these progressed to tracheostomy. A second study of 178 patients with complete cervical SCI showed 70% of patients required tracheostomy, particularly those with an injury between C4 and C7.[259] Spontaneous ventilation and extubation of the trachea are possible after surgical stabilization and resolution of neurogenic shock, although pneumonia is a common and recurrent complication that frequently necessitates tracheostomy to facilitate pulmonary toilet.[260]

Although administration of a glucocorticoid steroid bolus in patients with blunt SCI and deficit was recommended, current guidelines suggest caution. Large-dose glucocorticoid therapy has resulted in a small, but statistically significant, improvement in neurologic level after SCI in two large multicenter trials: National Acute Spinal Cord Injury Study (NASCIS) II and III.[261,262] However, the NASCIS results have been challenged for several reasons.[263-265] The positive benefits seen with high-dose steroid administration were driven by results in a few subpopulations and were not present in a majority of patients. The improvement in spinal level seen after steroid administration has not been shown to lead to increased survival or improved quality of life, and the results have not been reproducible in other studies of acute SCI. Current recommendations by the American Association of Neurological Surgeons and the Congress of Neurological Surgeons state that there is no consistent or compelling medical evidence to justify the use of methylprednisolone for acute SCI.[266]

INTRAOPERATIVE MANAGEMENT OF SPINAL CORD INJURY

A patient about to undergo surgical reduction and fixation of a spinal column fracture presents challenges for the anesthesiologist. First and foremost is the need for intubation of the trachea in a patient with a known injury to the cervical spine. Direct laryngoscopy with in-line stabilization is appropriate in the emergency setting and in unconscious, combative, or hypoxemic patients when the status of the spine is not known.[267] Tracheal intubation can occur in the OR, in an awake, alert, and cooperative patient by several methods known to produce less displacement of the cervical spine, presumably with less risk for worsening an unstable SCI. A common technique in current clinical practice is awake fiberoptic intubation. Although the nasal route is associated with an easier path to intubation in most patients, it can lead to an increased risk for sinusitis in the ICU if the trachea is not extubated at the end of the procedure. Oral intubation may be more challenging technically but will be of greater value if the patient remains mechanically ventilated. Blind nasal intubation, transillumination with a lighted stylet, and use of an intubating LMA, videolaryngoscopy, or any of a variety of other instrument systems for indirect laryngoscopy are acceptable. Again, in comparative studies of direct laryngoscopy, videolaryngoscopy, fiberoptic examination, blind nasal intubation, or cricothyrotomy in patients with known cervical cord or spine injuries, no difference is seen in neurologic deterioration with technique used and no clear evidence exists that direct laryngoscopy worsens outcome. Clinicians are advised to use the equipment and techniques with which they are most familiar. The important concept is to successfully achieve tracheal intubation while minimizing motion of the cervical spine and preserving the ability to assess neurologic function after positioning.[68]

A patient with a partial deficit and visible spinal canal impingement on imaging studies is considered an emergency because of the potential for regaining neurologic function after decompression. Timing for surgery suggests that earlier decompression may improve outcomes in some patients, particularly those with cervical injuries, although the exact timing remains controversial.[268,269] Hemodynamic instability may complicate urgent and emergent spinal surgery. Hypotension from neurogenic shock is characterized by an inappropriate bradycardia resulting from loss of cardiac accelerator function and unopposed parasympathetic tone. However, this situation can be difficult to distinguish from hypotension resulting from acute hemorrhage, and a trial of fluid administration is still indicated, subject to the end points of resuscitation listed earlier. Once hemorrhage has been controlled or ruled out, patients who have maintenance of an increased MAP more than 85 mm Hg for 7 days after SCI may have improved functional recovery. This approach is highly controversial but remains a recommendation in treatment according to published guidelines.[270]

Orthopedic and Soft Tissue Trauma

Musculoskeletal injuries are the most frequent indication for operative management in most trauma centers (see also Chapter 64). Orthopedic trauma, like TBIs and SCIs, can be associated with long-term pain and disability. In addition to physical handicaps that these patients may endure, orthopedic injuries incurred in both civilian and military settings lead to long-term psychological trauma.[271-273] The length of many procedures, particularly in patients with multiple extremity injuries, necessitates attention to body positioning, maintenance of normothermia, fluid balance, and preservation of peripheral blood flow.

Timing of operative intervention in polytrauma patients with orthopedic injuries has been a topic of considerable academic and clinical discussion. Musculoskeletal injuries represent common injury patterns seen in patients with high-energy mechanisms of injury and are one of the more common reasons for operative intervention in the trauma population.[274,275] Historically, injuries to the extremities in multisystem trauma patients who were too critically injured for definitive operative intervention were managed with traction and prolonged immobilization. These patients experienced high rates of pulmonary failure and prolonged mechanical ventilation, developed sepsis frequently, and had high mortality rates.[276] Specifically, pulmonary complications have been a common occurrence in this population of trauma patients with nearly 30% of patients with multiple extremity injuries experiencing pulmonary morbidity. Therefore the goal of fracture management in the multisystem trauma patient revolves around restoring musculoskeletal anatomy that allows for mobilization, pulmonary toilet, and adequate pain control.

In the patient with isolated extremity or hip fracture without polytrauma, the evidence remains clear that early definitive fracture care improves outcomes. In hip fracture patients, current U.S.[277] and Canadian[278] guidelines recommend surgery within 48 hours. Within the United Kingdom, surgery within 36 hours is a quality-of-care indicator although adherence to these guidelines is incomplete.[279] These recommendations may require revision as more recent work has consistently demonstrated an increasing complication rate for surgical procedures performed over 24 hours after injury.[280-283] In the most recent study looking at the association between wait time and 30-day mortality, Pincus and associates examined the impact of wait time in hours on mortality and other complications including myocardial infarction, deep vein thrombosis, pulmonary embolism, and pneumonia.[284] Among adults requiring hip fracture surgery, a wait time of 24 hours appears to be a threshold defining higher risk. The study population, however, did not include many patients with significant trauma (<1% of patients had an ISS ≥16) or multiple fractures (4.9%) making it impossible to directly apply these results to the polytrauma patient. Similarly, fixation of femur fractures in patients with a lower ISS within 48 hours of injury is also associated with improved outcomes.[285]

SPECIFIC ORTHOPEDIC CONDITIONS

Hip fracture is a common, morbid, and costly event among older adults. Frequently, patients with isolated hip fracture present to the OR for urgent repair. Regional anesthesia is associated with decreased odds of inpatient mortality and pulmonary complications among patients with hip fracture in contrast to general anesthesia.[286] A retrospective cohort of 18,158 patients undergoing surgery for hip fracture in 126 hospitals showed that 5254 (29%) received regional anesthesia. Although unadjusted rates of mortality and cardiovascular complications did not differ by anesthesia type, regional anesthesia was associated with decreased adjusted odds of mortality (odds ratio: .710, 95% confidence interval [CI] .541, .932, $P = .014$) and pulmonary complications (odds ratio: .752, 95% CI .637, 0.887, $P < .0001$) relative to general anesthesia.[287]

Dislocation of the hip is common after high-energy trauma and is frequently accompanied by fracture of the acetabulum. Whereas the fracture itself can be safely managed on a delayed basis or nonoperatively, the dislocation is a medical emergency that must be promptly addressed if the patient is to have a good functional outcome. Failure to promptly diagnose and reduce a dislocated hip joint is a significant risk factor for avascular necrosis of the femoral head. Reduction typically requires a very deep level of sedation, which may be facilitated by nondepolarizing neuromuscular blockade. For this reason, the anesthesiologist is commonly involved.[288] Although a dislocated hip can be reduced in a spontaneously breathing sedated patient, an acutely injured patient is at high risk for aspiration of gastric contents. Any patient who will be undergoing surgery soon (such as for an open long-bone fracture or exploratory laparotomy) can be tracheally intubated at the time of reduction and maintained with appropriate sedation and analgesia until reaching the OR. Other patients who may require intubation even for uncomplicated reductions include those who are inebriated or uncooperative, hemodynamically unstable, or suffering from pulmonary dysfunction. Additionally, successful postoperative fracture reduction is more difficult to achieve in morbidly obese patients than in those with normal body mass index.[289]

Unlike acetabular fractures, fracture of the pelvic ring requires immediate recognition and management by the trauma team. Hemorrhage, even exsanguination, is common after a major pelvic ring fracture and is a leading contributor to early death after motor vehicle collisions. Bleeding occurs from multiply disrupted venous beds in the posterior pelvic bowl; if the pelvis is unstable, no anatomic barrier exists to continued expansion of this retroperitoneal bleeding. Surgical exploration by way of the peritoneum is usually unrewarding because the bleeding vessels are not easily accessed.[290] In the past, therapy consisted of supportive volume resuscitation, external fixation of the unstable pelvis, and angiography. Despite this multidisciplinary approach, mortality remained frequent in these patients, partially because of the inability of angioembolization to control venous bleeding from the rich venous plexuses associated with the most severe fractures. The technique of preperitoneal packing has been adopted by many U.S. trauma centers. Preperitoneal packing via laparotomy is a rapid method for controlling pelvic fracture–related hemorrhage that can supplant the need for emergent angiography and reduces transfusion requirements and mortality.[291] Endotracheal intubation is usually undertaken on an emergency basis in a hypotensive patient, and the anesthesiologist may remain with the patient throughout the initial hours of stabilization to manage sedation, analgesia, transport, and ongoing resuscitation needs. In the absence of an orthopedic specialist, temporary stabilization and tamponade of some pelvic fractures can be accomplished with use of a specially made pelvic binder, the pelvic portion of military antishock trousers, or a bed sheet knotted tightly around the bony pelvis.[292] Mechanically unstable pelvic ring fractures are associated with increased fluid and blood resuscitation volumes, significantly more frequent presence of concomitant injuries, and increased ventilation and ICU stay, rate of MODS, sepsis, and rate of mortality.[293]

Patients with pelvic fractures can initially be divided into two groups: (1) stable pelvic fracture; or (2) displaced pelvic ring fractures with hemodynamic instability or high risk for deterioration. This second group has the highest risk for complications and mortality related to their injuries. The presence of associated injuries to the head, chest, and abdomen can lead to conflicting priorities in management of these patients. For example, a CT finding of a high-grade splenic injury in the setting of a severe pelvic injury and hemodynamic instability may require an exploratory laparotomy before addressing the pelvic injury. These patients may present for operative procedures with or without early interventions such as placement of a pelvic binder or resuscitative endovascular balloon occlusion of the aorta. As the unstable trauma patient is moved to the angiography suite or hybrid OR, resuscitation should continue to maintain an acceptable perfusion pressure; and survival is higher if this transition occurs within 3 hours of presentation.[294] Management of these interventions in the OR requires an understanding of the anatomy, physiology, and surgical considerations involved in their employment in the operative setting as discussed above.

Open fractures should be pulse-lavaged and debrided as soon as possible after injury to minimize the risk for infectious complications. If ongoing resuscitation or unstable TBI precludes the patient from early management in the OR, this procedure can be performed at the bedside. Despite the frequency of open fractures, their management remains one of the greatest and most debated orthopedic challenges. Timing of surgical fixation in open fractures is often surgeon-specific or center-specific, because little consensus exists among orthopedic surgeons as to optimal treatment.[295]

The advantages and disadvantages of regional and general anesthesia are summarized in Boxes 66.9 and 66.10. Regional anesthesia seems to decrease postoperative morbidity and mortality[287,296,297] (including cognitive measures of function), but clinically measurable benefits may not be seen in patients receiving combined general and regional techniques.[298-300] One other prevalent concern in the management of an orthopedic trauma patient is the concomitant use of regional anesthesia, analgesia, or both and prophylaxis for venous thromboembolism, as discussed in Chapter 64.

Intraoperative TEE has shown that most patients undergoing long-bone fracture manipulation experience microembolism of fat and marrow.[301] Most patients suffer no discernible clinical impact from this phenomenon, but some will experience a significant acute inflammatory response. After long bone fractures, some lung dysfunction occurs in almost all patients, ranging from minor laboratory abnormalities to full blown fat embolism syndrome (FES). A lack of universally

BOX 66.9 Advantages and Disadvantages of Regional Anesthesia for Trauma

Advantages

Allows continued assessment of mental status
Increased vascular flow
Avoidance of airway instrumentation
Improved postoperative mental status
Decreased blood loss
Decreased incidence of deep venous thrombosis
Improved postoperative analgesia
Better pulmonary toilet
Earlier mobilization
Lower incidence of long-term pain syndromes

Disadvantages

Peripheral nerve function difficult to assess
Patient refusal common
Requirement for sedation
Hemodynamic instability with placement
Longer time to achieve anesthesia
Not suitable for multiple body regions
May wear off before procedure(s) conclude(s)

BOX 66.10 Advantages and Disadvantages of General Anesthesia for Trauma Patients

Advantages

Speed of onset
Duration: can be maintained as long as needed
Allows multiple procedures for multiple injuries
Greater patient acceptance
Allows positive-pressure ventilation

Disadvantages

Impairment of global neurologic examination
Requirement for airway instrumentation
Hemodynamic management more complex

BOX 66.11 Risk Factors for the Development of Compartment Syndrome

Orthopedic

Fractures and operative repair

Vascular

Reperfusion injury
Hemorrhage with hematoma formation
Ischemia from arterial and venous injuries

Soft Tissue

Crush injury
Burns
Prolonged compression in immobile state

Iatrogenic

Casts and circular dressing
Use of pneumatic antishock garment
Intraosseous fluid replacement in infant or child
Pulsatile lavage with extravasation
Extravasation from venous or arterial puncture site

Miscellaneous

Snakebite
Acute exertion

accepted diagnostic criteria combined with concomitant pulmonary and cardiovascular dysfunction accounts for the varying incidence reported in the literature. Clinically significant FES occurs in 3% to 10% of patients but is likely underdiagnosed in patients with multiple injuries or a high ISS.[302] Patients with coexisting lung injury are at additional risk for FES. Signs include hypoxia, tachycardia, mental status changes, and, classically, a petechial rash on the axillae, upper arms and shoulders, chest, neck, and conjunctivae. FES should be considered whenever the alveolar-arterial oxygen gradient deteriorates in conjunction with decreased pulmonary compliance and CNS deterioration. Under general anesthesia, the CNS changes will not be apparent but may be manifested as failure to awaken after surgery. If central hemodynamic monitoring is available, pulmonary artery pressure is elevated, often accompanied by decreases in the cardiac index. Diagnosis in the OR is largely based on the clinical findings after ruling out other causes of hypoxemia. Fat globules in urine are nondiagnostic, but lung infiltrates seen on chest radiography confirm the presence of lung injury and the need for appropriate ventilatory management with oxygen, higher PEEP, and possible longer-term mechanical ventilatory support.[303,304] Treatment includes early recognition, administration of oxygen, and judicious fluid management.[305] A change in the orthopedic procedure may be indicated, such as converting intramedullary nailing of the femur to external fixation. In the patient with bilateral femur fractures, it has been proposed that 1 to 2 days be allowed to pass between successive nailing procedures.[306]

Acute compartment syndrome of the extremities is defined as a "condition in which increased pressure within a limited space compromises the circulation and function of the tissues within that space."[307] The most common cause of compartment syndrome is edema secondary to muscle injury and associated hematoma formation. Although most commonly associated with traumatic injuries, compartment syndrome also can occur because of causes associated with trauma, including reperfusion injury, burns, drug overdose, and prolonged limb compression (Box 66.11). The most common fractures associated with the development of compartment syndrome are those of the tibial shaft (40%) and forearm (18%).[308,309] Forearm compartment syndrome requiring fasciotomy predominantly affects males and can occur after either a fracture or soft-tissue injury. A further 23% of cases are caused by soft-tissue injuries without fracture.[310] Although no randomized, prospective trials have been conducted, regional anesthesia does not blunt detection of compartment syndromes if there is not an associated dense motor block.[311]

Fasciotomy is usually indicated when compartment pressure approaches 20 to 30 mm Hg below diastolic pressure in any patient with a worsening clinical condition, a documented rising tissue pressure, major soft tissue injury, or a history of 4 to 6 hours of total ischemia of an extremity. Prophylactic fasciotomy may be indicated in patients with warm ischemic time more than 2 hours, ligation of the major veins in the popliteal region or distal part of the thigh, and crush injuries. Early or prophylactic fasciotomy decreases subsequent muscle loss.[312]

Crush syndrome is the general manifestation of crush injury caused by continuous prolonged pressure on one or more extremities[313] and is commonly found in patients who have been trapped in one position for an extended period. Muscle injury secondary to ischemia causes myoglobinuria, which can lead to acute renal failure and subsequent profound electrolyte disturbances. The most critical treatment consists of crystalloid fluid resuscitation; a total body fluid deficit of 15 L may occur in severe rhabdomyolysis.[314] Osmotic diuresis with mannitol and alkalization of urine with sodium bicarbonate to prevent precipitation of myoglobin in the renal tubules are controversial.[315] The preferred therapy for renal failure secondary to rhabdomyolysis at the shock trauma center is continuous renal replacement therapy and hemofiltration.[316] Most of these patients eventually have full recovery of native renal function.[317]

SOFT TISSUE TRAUMA

Assessment of soft tissue injury is critical in the management of a trauma patient. Muscular coverage is necessary for the viability of any orthopedic repair, but it may be jeopardized by avulsion at the time of injury, ischemia from elevated compartment pressure, and ongoing bacterial infection in open wounds. Acute surgical management of soft tissue injury is straightforward: all dead or devitalized tissue must be debrided, and the wound thoroughly irrigated to reduce the load of bacterial contaminants. When muscle or fascia involvement is significant, serial debridement at frequent intervals is necessary to establish a margin of completely viable tissue. Vacuum dressings for large soft tissue wounds are gaining in popularity because continuous negative pressure over the wound surface removes contaminants and encourages blood flow.[318] When serial debridement establishes viable tissue at all margins of the wound, arrangements for definitive closure can be made. Closure may be as simple as a split-thickness skin graft or as complex as free tissue transfer of muscle and fascia from an uninjured portion of the body, with attendant arterial and venous anastomoses.

A degloving injury results in significant soft tissue loss, usually in the extremities. Multiple plastic surgery and reconstructive procedures may be necessary. General anesthesia is usually required, although combining it with epidural or regional block analgesia may confer the benefits of both techniques. Close consultation with the surgical teams is suggested.

Superficial vacuum dressings can be changed at the bedside under light sedation, but patients with deep wound dressings may require general anesthesia. The need for repeated surgeries is an important consideration for the anesthetic technique. Anesthesia for free tissue transfer surgery requires meticulous attention to detail because these operations can be quite protracted. Every effort should be made to facilitate perfusion of the grafted vessels, including keeping the patient warm, euvolemic, and comfortable, and maintaining the hematocrit in the rheologically favorable range of 25% to 30%. The use of epidural anesthesia and analgesia is controversial, with some surgeons favoring it for its vasodilatory effects and others being concerned that it will induce a steal phenomenon that will actually limit flow in the denervated free tissue.[319] The use of vasopressors in microsurgery is generally avoided. Yet in one study, the vasopressor used did not cause more complications.[320]

Other Traumatic Injuries

HEAD AND NECK SURGERY

Except for emergency exploration of penetrating trauma to zone II of the neck (from the clavicles up to the angle of the mandible), most surgical repair of head and neck trauma will occur in the subacute phase, after complete resuscitation and secondary diagnostic studies. Anesthetic management of these patients is not substantially different from that for similar elective procedures, although coexisting injuries may influence patient positioning, airway management, and ventilator settings.[321,322] Surgery on the mandible and maxilla will be facilitated by nasotracheal intubation, but a secure airway should be not jeopardized by attempting to switch from an oral to a nasal tube in a patient in whom visualization of the larynx might be difficult because of traumatic swelling or body habitus. It is safer in these cases for the surgeon to either work around an oral tube secured behind the second molar (to allow dental occlusion) or place a tracheostomy if the need for intubation and mechanical ventilation is likely to be protracted. Surgery on the zygoma and the nasal, orbital, and ethmoid bones will be possible with an oral ETT. Securing the ETT to the molar with a fine-gauge wire will help stabilize it through the operation. All of these surgeries will lead to significant soft tissue swelling in the immediate postoperative period, often necessitating several days of continued intubation and sedation until sufficient venous drainage has occurred to allow safe extubation of the trachea. Though not by itself definitive, the presence of an air leak when the ETT cuff is deflated is suggestive that the airway will remain patent once the tube is removed.

CHEST INJURIES: PULMONARY

Injuries to the lung parenchyma that produce pneumothorax can be managed by tube thoracostomy to relieve tension, drain accumulated blood, and apply suction to the pleural space until the air leak spontaneously resolves. Bleeding from the low-pressure pulmonary circulation is usually self-limited. Thoracostomy is uncommon but becomes necessary when evidence exists of mediastinal injury, chest tube output exceeds 1500 mL in the first hours after injury, tracheal or bronchial injury and massive air leak are apparent, or the patient is hemodynamically unstable with evident thoracic pathology.[33] Blood collected from the pleural space is generally free of clotting factors and can be directly reinfused with any of several commercial systems[323]; however, concern has been expressed that this may be prothrombotic and potentially harmful.[324] Hemorrhage necessitating surgery may be from injured intercostal or internal mammary arteries or from the lung parenchyma. Staple resection of injured lung or even anatomic lobectomy is not uncommon, particularly after penetrating trauma.

Although double-lumen endotracheal intubation is desirable during urgent thoracotomy, this should not be the initial approach. Rapid sequence induction with a large-caliber (at least 8.0 mm in internal diameter) conventional ETT will allow diagnostic bronchoscopy if needed. The change to a double-lumen tube can then be made under controlled conditions in the presence of adequate oxygenation, anesthesia, and muscle relaxation. Tolerance of single-lung ventilation is variable in the trauma population and depends in large part on the absence of significant pathologic findings in the ventilated lung. Many patients with blunt thoracic injury have bilateral pulmonary contusions and will require increased FiO_2 and high levels of PEEP to maintain adequate oxygenation, even when both lungs are ventilated. Those with a high Thoracic Trauma Severity score in the setting of blunt trauma and pulmonary contusion are more likely to progress to ARDS potentially necessitating early, aggressive management of mechanical ventilation.[325]

Although chest trauma requiring pneumonectomy has historically resulted in mortality approaching 100%, a recent review of the National Trauma Data Bank found 261 cases of pneumonectomy following trauma (163 penetrating and 98 blunt) with an overall in-hospital mortality of 60%.[326] Intraoperative deaths are the result of uncontrollable hemorrhage, acute right ventricular failure, and air embolism. Patients who survive the initial procedure are at risk for early postoperative morbidity and mortality. Fluid management may be complicated by the need to weigh ongoing resuscitation against the treatment of right ventricular failure. Blunt thoracic trauma requiring pneumonectomy is often associated with abdominal and pelvic trauma. Volume replacement must be judicious, and the use of a pulmonary artery catheter (placed with care in a postpneumonectomy patient) or TEE may be beneficial. Echocardiography will also play an important role in assessing right ventricular function and pulmonary hypertension. Treatment of right ventricular failure after traumatic pneumonectomy is difficult.[327] During hypovolemic shock a disproportionate increase occurs in pulmonary vascular resistance with respect to systemic vascular resistance,[328] as well as frequent mortality with combined hemorrhagic shock and pneumonectomy.[329] With severe right heart dysfunction it is desirable to maintain a higher preload than normal. Several therapeutic approaches have been used to treat right ventricular failure, including close monitoring of pulmonary artery pressure, the use of diuretics for volume overload, and administration of pulmonary vasodilators. Because this injury is rare, and the number of patients reported in the literature is small, the best therapy is difficult to identify. A recent case report describes the use of nitric oxide to successfully treat pulmonary hypertension after posttraumatic pneumonectomy.[330] Extracorporeal support also has been used to sustain patients through the perioperative period, although the technical challenges are substantial and successful weaning may require days to weeks on bypass (see also Chapter 85).[331]

Tracheobronchial injury can result from either blunt force or penetrating trauma. Penetrating injuries are usually more promptly diagnosed and treated. Blunt trauma most commonly results in an injury to the tracheobronchial tree within 2.5 cm of the carina and may initially be unrecognized. The presence of subcutaneous emphysema, pneumomediastinum, pneumopericardium, or pneumoperitoneum, without apparent cause, should alert the practitioner to possible tracheobronchial injury.[332] Despite bronchoscopy and helical CT scanning, a small injury may never be delineated. If the resultant injury is an incomplete tear, it may heal with stenosis, subsequent atelectasis, pneumonia, pulmonary destruction, and sepsis. When surgery is required for a delayed, incomplete tracheobronchial injury, pulmonary resection may be required if significant tissue destruction has occurred, whereas complete transection may be amenable to reconstruction with preservation of pulmonary tissue. The level of injury dictates the surgical approach. Cervical injuries are approached through a transverse neck incision, left bronchial injuries via a left thoracotomy, and tracheal or right main bronchial injuries via a right thoracotomy. In the cervical region it is sometimes possible to access a longitudinal tear of the posterior membranous trachea by opening the anterior trachea and operating around the ETT.

CHEST INJURIES: TRAUMATIC AORTIC INJURY

Traumatic aortic injury (TAI) must be ruled out in any patient with a high-energy injury, such as occurs with a motor vehicle accident or a fall from a height. The incidence of TAI has decreased in recent years because of the increased presence of airbags in motor vehicles; most of the cases in the past decade have been caused by side-impact collisions.[333] Aortic injury occurs most commonly just distal to the left subclavian artery and is the result of sheer forces between the mobile heart and aortic arch and the immobile descending thoracic aorta. TAI is manifested as a continuum of injury from a small intimal flap to free transection contained by the surrounding mediastinum and pleura. The diagnosis is most commonly made by CT scan of the chest with intravenous contrast, which has a sensitivity comparable to aortography.[334] Surgical or endovascular repair is indicated for most patients with TAI because of the high risk for rupture in the hours and days after injury with endovascular repair being the preferred option in the absence of contraindication. Selective nonoperative management of high-risk patients with TAI has been described.[335,336] Treatment is similar to that in patients with uncomplicated type B aortic dissection and consists of β-adrenergic blockade to minimize the cardiac rate–pressure product.

CHEST INJURIES: RIB FRACTURES

Rib fractures are the most common injury resulting from blunt chest trauma. The fracture itself generally requires no specific treatment and will heal spontaneously over a period of several weeks. Therapy is directed at minimizing pulmonary complications secondary to these fractures, such as pain, splinting, atelectasis, hypoxemia, and pneumonia. Of particular concern are rib fractures in older adults (older than 55 years of age). Older patients with rib fractures have twice the mortality and thoracic morbidity of younger patients with similar injuries. Epidural anesthesia should be used liberally in patients with severe pain, older adults, and patients with preexisting compromised pulmonary function. Data support a 6% decrease in morbidity and mortality in older patients when epidural anesthesia is used[337]; however, a meta-analysis failed to identify a reduction in mortality.[338] Nonetheless, recent guidelines for pain management in the patient with blunt thoracic trauma recommend epidural analgesia over nonregional modalities of pain control.[339] Epidural analgesia may minimize or avoid complications of splinting and pain such as hypoxemia, hypoventilation, the need for tracheal intubation, and the possibility of pneumonia. Endotracheal intubation is reserved for patients who are unable to oxygenate or ventilate or who require protection of the airway.

Fracture of multiple neighboring ribs will result in the flail chest syndrome, characterized by paradoxical chest wall motion during spontaneous ventilation. Not all patients with a flail chest require positive-pressure ventilation, and endotracheal intubation should be reserved for those who meet the usual criteria. Patients who are not initially intubated should be observed closely in the ICU for signs of worsening respiratory function. Increasing cases have been reported of the use of noninvasive positive-pressure ventilation (NIPPV) for lung injury secondary to trauma.[340] For these patients who subsequently require intubation for a surgical procedure, the anesthesiologist will need to determine the safety of extubation postoperatively. NIPPV is associated with fewer cases of pneumonia, which may lead to fewer tracheostomies and decreased ICU length of stay.

A successful technique has been early extubation to a mask delivering continuous positive airway pressure or bilevel positive airway pressure.[341] Concomitant pulmonary injury, especially lung contusion, is commonly associated with flail chest. Pulmonary contusion will cause shunting, which will lead to hypoxemia. This syndrome may progress rapidly in the hours and days after injury. An initially clear chest radiograph does not exclude the possibility of a pulmonary contusion, and, again, close observation is warranted if signs of significant chest wall trauma are present. As with all patients after traumatic injury, a high degree of suspicion along with a continuous search for missed injuries is warranted. No specific therapy exists for a pulmonary contusion, and therapy is directed at the associated injuries or resultant hypoxemia. Early and aggressive implementation of a lung-protective strategy may have a role in patients with significant pulmonary contusion to minimize progression to ARDS.

CHEST INJURIES: CARDIAC INJURY

Blunt cardiac injury is a rare and poorly understood phenomenon that must be considered in any patient who has sustained a frontal impact to the chest. Bruising or edema of the myocardium is functionally indistinguishable from myocardial ischemia and may be causally related in that the pathophysiology of cardiac contusion may involve forcible dislodgement of unstable atherosclerotic plaque. If the patient is hemodynamically stable and the ECG does not demonstrate conduction disturbances or tachyarrhythmias, blunt cardiac injury can be safely excluded.[342] If either a new tachyarrhythmia or conduction disturbance subsequently develops or the patient has unexplained hypotension, other causes (hypovolemia, renal failure) should be ruled out first. If the workup is negative, TTE should be performed. Right ventricular dysfunction resulting in hypotension may be overlooked while more common causes of hypotension in the trauma patient are being evaluated. TEE is superior to TTE in obese patients or those with injuries to the chest wall that make it difficult to obtain adequate acoustic windows, but it will generally require intubation and deep sedation to perform. Once diagnosed, blunt cardiac injury should be managed as ischemic cardiac injury, with completion of resuscitation and then careful control of fluid volumes, administration of coronary vasodilators, and monitoring and symptomatic treatment of rhythm disturbances. Anticoagulation with aspirin or heparin should be approached on a case-by-case basis as determined by the patient's other injuries. Cardiology consultation is appropriate if the patient may benefit from coronary angiography followed by angioplasty or stenting of stenotic vessels.

Patients with penetrating cardiac trauma and blunt trauma causing rupture of one or more chambers (usually the atria) are often not seen by the trauma center because of a frequent rate of prehospital mortality.[343] Those who do not die immediately of free exsanguination into the thoracic cavity will have pericardial tamponade and can be extremely unstable in the first minutes after admission. This condition is diagnosed by clinical suspicion, focused assessment with sonography for trauma (FAST), and direct inspection during ED thoracotomy. Relief of the tamponade and clamp or suture control of the cardiac injury may allow restoration of spontaneous circulation and necessitate urgent transition to the OR for definitive hemostasis and chest closure. Cardiopulmonary bypass may be required for support during repair of cardiac injuries.

ABDOMINAL INJURY

The need for exploratory laparotomy, once the mainstay of the trauma surgeon, has declined because FAST and high-resolution CT have reduced the incidence of negative abdominal explorations, whereas angiographic embolization of hemorrhaging vessels in the liver and spleen has reduced the need for open procedures. Urgent celiotomy, when it does occur, will typically follow the principles of damage control, as described earlier.[344] The abdomen is opened and packed tightly in all four quadrants. Systematic exploration is undertaken in each quadrant in turn, with time taken only for control of hemorrhage and rapid staple closure of open gastrointestinal injuries. The abdomen is packed open at the end of the procedure, a sterile drape is used to cover exposed viscera, and the patient is moved to the ICU to complete resuscitation. Definitive treatment of nonlethal injuries and restoration of bowel continuity are deferred until a second operation 24 to 48 hours later.

Anesthetic management of emergency celiotomy should follow the principles of early resuscitation outlined earlier. Adequate intravenous access is required, as well as continuous arterial pressure monitoring. Use of cell salvage devices can reduce the patient's exposure to banked blood, although reinfusion is generally deferred if there has been significant contamination by bowel contents. A rapid infusion system is advantageous for preservation of intravascular volume and normothermia during periods of heavy bleeding. Subsequent abdominal surgeries will be needed in hemodynamically stable patients and should not present unusual anesthetic challenges. Subsequent reconstructive procedures can become technically difficult as scarring and adhesions develop; the anesthesiologist should be prepared for a long anesthetic procedure with the potential for significant hemodynamic compromise.

Selected Patient Populations

TRAUMA AND PREGNANCY

Trauma to pregnant patients is associated with a frequent risk for spontaneous abortion, preterm labor, or premature delivery, depending on the location and magnitude of the mother's injury.[345] Trauma is the leading nonobstetric cause of death in pregnant women with nearly double the mortality rate.[346] Early consultation with an obstetrician is desirable for any pregnant trauma patient, for both immediate management and long-term follow-up. The best treatment of the developing fetus consists of rapid and complete resuscitation of the mother. Trauma patients in the first trimester of gestation may not realize that they are pregnant; for this reason, human chorionic gonadotropin testing is part of the initial laboratory studies for any injured woman of childbearing age. Serious trauma occurring during the period of fetal organogenesis may induce birth defects or miscarriage because of the effects of medications, pelvic irradiation, or hemorrhage leading to placental ischemia. Indicated radiologic tests should not be deferred but shielding of the pelvis should be provided whenever possible.[347] Patients who do not spontaneously miscarry should be advised of the potential risks for birth defects and be referred for counseling if desired.[348] Dilatation plus curettage of the uterus is advisable after miscarriage to avoid toxicity arising from retained products of conception.[349]

Trauma occurring in the second or third trimester of pregnancy necessitates early ultrasonographic examination to determine fetal age, size, and viability. Monitoring of the fetal heart rate is indicated if the pregnancy is sufficiently advanced that the fetus would be viable if delivered. Preterm labor is very common in this population and should be treated with β-adrenergic agonist drugs or magnesium at the direction of the obstetrician. Delivery should be delayed if the fetus is not an unacceptable metabolic stress on the mother. Delivery by cesarean section is indicated if the mother is in extremis, if the uterus itself is hemorrhaging, or if the gravid uterus is impairing surgical control of abdominal or pelvic hemorrhage.[350] Placental abruption can occur in response to substance abuse or abdominal trauma and can precipitate life-threatening uterine hemorrhage. Emergency cesarean section is indicated in these cases. The Kleihauer-Betke blood test can be used to determine if fetal blood has leaked into the maternal circulation[350]; if positive, administration of anti-Rh_0 immune globulin is recommended for any Rh-negative mother carrying a Rh-positive fetus. By the third trimester the uterus is sufficiently enlarged to compress the inferior vena cava when the patient is positioned supine, thereby impairing venous return to the heart and contributing to hypotension. Left lateral uterine displacement is indicated to treat this problem. If the patient is immobilized on a long spine board because of concern for a thoracic or lumbar spinal fracture, the whole board can be tipped to the left. Because the gravid uterus displays abdominal contents upward, elevating the head of the bed may also be required to improve ventilation.

PEDIATRIC TRAUMA PATIENTS

While the overall proportion of pediatric trauma patients compromises a minority of trauma center admissions, traumatic injuries remain the leading cause of death for children aged 1 to 14 years of age.[2] Motor vehicle crashes, drowning, falls, and burns constitute most of the mechanisms for traumatic injury.[351] A disproportionate number of pediatric trauma patients (~80%) have an associated TBI; such injuries are responsible for more than half of all deaths.[351,352] The initial priorities of trauma assessment and management are the same for children and adults; nonetheless, several particularities must be considered, including differences in airway anatomy, the normal range of physiological variables, and other anatomical differences (see also Chapter 77).[33,352]

Successful management of the pediatric airway constitutes the foundation and initial step in pediatric trauma resuscitation because hypoxia and respiratory compromise are the most common causes of cardiac arrest.[352,353] Preceding airway management, a high degree of caution must be exercised to immobilize the cervical spine in patients with dangerous mechanisms that increase the risk of spinal injuries. Some of these mechanisms include: falls from height greater than one meter or five stairs, axial loading to the head, high-speed and/or rollover motor vehicle crashes, ejection from a motor vehicle, or accidents involving motorized recreational vehicles and bicycle collisions.[354] SCIWORA may be present in up to 35% of children with high-risk mechanisms, and this increased risk precludes clearance of the cervical spine in the same manner as adults.[354,355] Children have a high oxygen consumption rate and react to hypoxia with bradycardia, sometimes even with very brief periods of apnea.[356] The anesthesiologist should be prepared to encounter the following anatomical differences in pediatric patients when managing the airway: a large, protuberant occiput; a small oral cavity; a large tongue; presence of large adenoids and tonsils; and a short and anteriorly displaced larynx.[352,356] These anatomical features can predispose to obstruction, bleeding, and a difficult airway. Pediatric patients should always be considered to have a full stomach, and rapid sequence induction and intubation is the gold standard for airway management.[356] In cases where intubation and mask ventilation are impossible, surgical cricothyroidotomy is considered unsafe in children younger than 12 years of age due to the small size of the cricothyroid membrane and proximity to vocal cords.[33] Needle cricothyroidotomy is preferred as a temporizing solution until other preparations are made. Emergent venous access in children can be challenging; if unable to obtain peripheral access, intraosseous access is recommended.[33] Determination of weight is mandatory to dose medications appropriately and guide fluid and blood component resuscitation. If weight is unknown, a length-based resuscitation tape or specialty stretcher with an integrated scale may be used. Throughout the perioperative period, vigilance for hypothermia should be maintained. Owing to a large surface-to-weight ratio, immature thermoregulation mechanisms, and the effects of general anesthetics, pediatric trauma patients are prone to develop hypothermia.[357] Hypothermia can be particularly detrimental for pediatric trauma patients because up to a 10% decrease in clotting factor activity has been observed for every degree Celsius below normothermia.[357,358]

There are several unique aspects of pediatric trauma resuscitation that anesthesiologists should be familiar with. Due to differences in pediatric coagulation profiles (i.e., decreased levels of procoagulatory proteins and fewer coagulation inhibitors), thromboelastography has an unclear role for diagnosing and treating coagulation disorders.[357] There are no validated scoring systems to determine the need for massive transfusion in children, and a DCR strategy (i.e., transfusion-packed RBCs to FFP to platelets in a 1:1:1 ratio) has not been studied adequately in pediatric patients. Hypotensive resuscitation has not been studied in children and may be hazardous because baseline blood pressures in children closely approximate the lower limits of cerebral autoregulation.[357,359,360] The use of adjuncts such as TXA rFVIIa may have utility in limiting bleeding, but large studies in pediatric patients are presently lacking.[359,361]

OLDER TRAUMA PATIENTS

As the world population continues to increase in age, elderly trauma patients continue to become a growing part of the overall trauma population.[362] Overall mortality among elderly trauma patients is higher than among younger adults; injured patients over age 74 years are at considerably higher risk for mortality.[363] Elderly trauma patients frequently present with lower ISS, but more often sustain severe injuries to the head and lower extremities.[364]

When managing elderly patients with trauma, the effects of aging on physiological function and the impact of preexisting conditions and medications must be carefully considered. Equivalent traumatic injuries will have a markedly more serious outcome in the elderly than in younger victims (see also Chapter 65).[363,365] Elderly patients may be taking multiple medications such as anticoagulants and β-blockers, each of which may worsen traumatic shock by causing severe bleeding and blunting of compensatory responses, respectively. Elderly patients may not have teeth or dentures to help maintain airway patency; edentulous patients are often difficult to mask ventilate, and dentures may loosen and obstruct the airway. Many older adults have decreased chest wall and pulmonary compliance, diminished functional residual capacity, and decreased mucus clearance—all of which have been shown to contribute to a mortality that is double that found in younger patients.[366] In elderly patients with blunt thoracic trauma, each additional rib fracture increases mortality and the risk for pneumonia by over 20%. Intubation may be complicated by degenerative spine changes and kyphoscoliosis. The patient may have a pacemaker or automatic implantable cardioverter-defibrillator in place, as well as artificial cardiac valves or stents. Older adults have thinner skin and are more prone to hypothermia and early development of pressure ulcers. Great care must be taken during intraoperative positioning to avoid secondary pressure injuries. Potential causes of an altered mental status may include delirium, dementia, pain medication, sedatives, increased ICP, and decreased CPP, the latter two of which are most deadly.

A tailored evaluation and management of elderly trauma patients is a crucial factor for improving outcomes.[367] A higher hematocrit, achieved by tighter control of administered fluid, is generally recommended to maintain maximized tissue oxygen delivery. Posttraumatic myocardial dysfunction is a significant risk, particularly if the heart rate is elevated secondary to blood loss, pain, or anxiety. Complex operations or large blood loss should encourage the use of TEE, invasive arterial monitoring, and noninvasive monitors of cardiac output and volume status to guide fluid and inotropic therapy.[163] Older patients will have diminished requirements for postoperative analgesia and may respond to sedative medications with inappropriate agitation.

JEHOVAH'S WITNESS PATIENTS

A trauma patient who refuses blood products requires special management. Early identification and control of hemorrhage are obviously important (see also Chapter 49). Deliberate hypotension to limit bleeding may be helpful. Preoperative and intraoperative phlebotomy should be minimized. The use of salvaged red cells (from intraoperative collection or from a chest drainage system) should be discussed with the patient because some Jehovah's Witness patients will allow the use of salvaged blood provided that the entire system remains in continuity with the vasculature.[368] The use of albumin and other substances derived from circulating proteins should be addressed. Early hemodynamic monitoring is indicated to help determine the role of colloid therapy, vasopressors, and inotropes in maintaining tissue oxygen delivery at the highest possible level. Case reports have described the successful use of rFVIIa and hemoglobin-based oxygen carriers in Jehovah's Witness patients to rapidly correct bleeding and support oxygen delivery, but these agents are not approved for these uses and large case series are not available.[369] In the postacute phase, the use of erythropoietin to promote red cell growth may shorten the period of relative anemia although there is limited evidence.[370]

Postoperative Care

EMERGENCE AND TRACHEAL EXTUBATION

Initial surgery in a trauma patient is followed by a period of monitoring and ongoing treatment in which the anesthesiologist is closely involved, either in the postanesthesia care unit (PACU) or in the ICU (see also Chapter 80). The adequacy of posttraumatic resuscitation must be confirmed, as outlined earlier, and diagnostic studies of the secondary survey completed. Rapid termination of general anesthesia is highly desirable, particularly in patients with an altered level of consciousness or other evidence of TBI before surgery. Change in mental status from the preoperative baseline is an indication for repeat cranial CT and a search for possible metabolic or toxic derangements.

Although neurologic function should be assessed postoperatively, early extubation of a trauma patient should not be taken for granted. Many patients will require continued ventilator support because of CNS trauma, direct pulmonary or chest wall trauma, massive transfusion, upper airway edema, or ongoing intoxication. Box 66.12 lists the criteria for extubation after urgent or emergency trauma surgery. If any doubt exists about the patient's ability to meet these criteria, the patient should be transported to the PACU or ICU with the ETT in place. Appropriate analgesic medication should be administered, with sedation if necessary. Twelve to 24 hours of support allows confirmation of successful resuscitation and surgical repair, hemodynamic equilibration, titration of appropriate analgesia, and resolution of intoxication. Many patients can be tracheally extubated easily and safely at this time; those who cannot are at high risk for the development of MOD—heralded by the development of posttraumatic ARDS—and will usually require days to weeks of subsequent intensive care.

ACUTE PAIN MANAGEMENT

Trauma patients present significant pain management challenges to the clinician because of multiple sites of injury, protracted episodes of care, complicating psychological and

BOX 66.12 Criteria for Operating Room or Postanesthesia Care Unit Extubation of the Trachea in a Trauma Patient

Mental Status

Resolution of intoxication
Able to follow commands
Noncombative
Pain adequately controlled

Airway Anatomy and Reflexes

Appropriate cough and gag
Ability to protect airway from aspiration
No excessive airway edema or instability

Respiratory Mechanics

Adequate tidal volume and respiratory rate
Normal motor strength
Required fraction of inspired oxygen (FiO_2) less than 0.50

Systemic Stability

Adequately resuscitated (see earlier)
Small likelihood of urgent return to the operating room
Normothermic, without signs of sepsis

emotional issues, and prior or ongoing substance abuse (see also Chapter 81). As with pain management practice in other diseases, trauma patients are frequently undertreated, thus creating a significant source of dissatisfaction. Because trauma patients run the gamut of physiology from healthy young athletes to debilitated older adults, anesthesiologists providing pain management for trauma patients must be prepared for a wide range of needs.

Individual trauma patients will have widely varying requirements for pain medication, so induction of analgesia must be titrated carefully, ideally in a closely monitored environment such as the PACU. Administration of rapidly acting intravenous agents in small doses at frequent intervals until pain relief is achieved is recommended. This allows the practitioner to determine the patient's basal requirements before starting long-acting medications or patient-controlled analgesia. Hypotension in response to analgesics is most commonly indicative of hypovolemia and should prompt a search for occult hemorrhage while further resuscitation occurs.

The need for analgesic medication and the duration of requirement for analgesics will be minimized if a comprehensive emotional support system is available to the patient. Trauma, because of its unexpected nature, carries with it a strongly negative psychological overlay that can have a profound effect on how anatomically based pain is perceived by the brain[371] and on how the patient reacts. After an injury the patient may have legal, financial, and family-based concerns, without the ability to immediately address them. The availability of counselors—religious, financial, or legal—who can help the patient and family with these issues is of enormous benefit. The anesthesiologist can help by communicating to the patient a clear description of the injuries, the probable time required for recovery, and the plan for managing pain throughout the course. The anesthesiologist should refer the patient to counseling services as needed and should be alert to the potential for posttraumatic stress disorder (PTSD) in any traumatized patient.[372] Referral to an experienced psychiatrist or psychologist is appropriate if PTSD is hindering the patient's recovery.

The need for analgesic medication is also influenced by the schedule of physical therapy prescribed for the patient. In general, the more active a patient can be after traumatic injury, the lower the risk for pulmonary complications, venous thrombosis, and decubitus ulcers. Though painful in the short term, the sooner the patient is mobilized, the lower are the analgesic requirements in the long term. Early mobilization demonstrates to the patient the path to recovery and contributes to an improved emotional state. One of the goals of analgesia, therefore, is provision of adequate medication to facilitate physical therapy without so sedating the patient that participation is impossible.

Neuropathic pain arises when direct injury occurs to a major sensory nerve and is common after spinal cord trauma, traumatic amputations, and major crush injuries. Neuropathic pain is characterized by burning, intermittent electrical shocks, and dysesthesia in the affected dermatomal distribution. Neuropathic pain must be identified because it responds poorly to the analgesics used for somatic pain. This diagnosis should be considered whenever pain control is poor or the patient has a rising requirement for medications unexplained by anatomic injuries. First-line therapy for neuropathic pain has been revolutionized by the widespread use of gabapentin, an antiepileptic drug with very strong specificity for this problem.[373] Gabapentin therapy is typically initiated at a dose of 200 mg three times daily, with daily titration upward to a maximum of 2 to 3 g/day. If neuropathic pain persists, selective regional anesthesia or analgesia may be indicated in an effort to break the cycle of spinal cord receptor recruitment.[374]

Regional analgesia (see also Chapters 45 and 46) provided through an epidural or brachial catheter should be considered for any trauma patient with a potential to benefit from this approach because it will spare the use of systemic opioids and facilitate early mobilization.[375] Epidural analgesia produces high levels of patient satisfaction and improved pulmonary function after major thoracoabdominal and orthopedic surgery in elective populations[376]; this is very likely true for the trauma population as well. Regional techniques are less useful when the patient has multiple sites of injury or when fractures or open wounds make placement difficult. Although epidural placement in anesthetized patients is relatively contraindicated because of the potential for occult SCI, the risk-to-benefit ratio in many trauma patients favors placement during surgery, when general anesthesia facilitates appropriate positioning and patient cooperation.

Summary

Trauma touches all ages and classes of patients, from young and vigorous to older and frail. Because of its high prevalence, practicing anesthesiologists will encounter trauma patients throughout their careers. Increased public recognition of the consequences of injury has sparked interest in trauma research and education, with the result that diagnostic and therapeutic techniques have evolved rapidly in recent years. The anesthesiologist, as a perioperative physician, is in the ideal position to understand and apply these new techniques across the trauma continuum.

Acknowledgment

The editors, publisher, and Dr. Thomas E. Grissom would like to thank Drs. Maureen McCunn and Richard P. Dutton for their contribution to this chapter in the prior edition of this work. It has served as the foundation for the current chapter.

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67 Prehospital Care for Medical Emergencies and Trauma

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KEY POINTS

- After World War II, the subspecialty of prehospital emergency medicine evolved with leadership from doctors in anesthesiology. In many countries, prehospital emergency medicine is considered the fourth pillar along with anesthesiology, critical care, and pain therapy.
- Emergency medical service (EMS) systems differ among and within countries. When these differences were put together, two primary models evolved. In the United States, paramedics provide prehospital care for all patients (single-tiered system). In many European countries, EMS-physicians lead the prehospital care for patients requiring advanced life support (two-tiered system).
- The core approach of managing prehospital emergencies involves basic life support and advanced life support.
- Rapid, simultaneous assessment and triage form the cornerstone of prehospital care—the use of a primary survey and limited diagnostic adjuncts can ensure transport to the most appropriate care setting.
- In major trauma, prehospital care must limit the time spent on the scene, control hemorrhage, and expedite transport to a trauma center, ideally via a rescue helicopter. Although this approach has been used both in the military (e.g., Vietnam) and in civilian locations, it is not always possible. Prehospital intravenous fluid resuscitation for major trauma varies in approach. Patients with penetrating torso injuries and hemorrhagic shock may benefit from limited intravenous fluid resuscitation and permissive hypotension, in particular in urban settings. Prevention of the lethal triad of hypothermia, acidosis, and coagulopathy is of paramount importance.
- In acute coronary syndrome and stroke, achieving rapid reperfusion of the ischemic tissue is the priority. Because only specialized centers provide 24-hour cardiac catheter service or stroke teams, rapid transport to acute myocardial infarction or stroke centers is critical. In response to a myocardial infarction, morphine, oxygen, nitrates, and aspirin are the main components of prehospital therapy. Fibrinolysis for myocardial infarction has been used with significant success in the prehospital setting but requires very close supervision by EMS physicians.
- The future of EMS will likely see an increased use of telehealth, which can narrow the time gap between the hospital and the field. This will facilitate improved field and hospital diagnostics and treatments, and ensure more efficient handoffs when arriving at the receiving hospital.

Background

The heritage of the modern emergency medical service (EMS) can be traced to the late 1700s with the approach to triage and forward patient retrieval of Napoleon's chief surgeon, Dominique Jean Larrey. Later, in 1832, in London, transport carriages were introduced for cholera patients. The rationale for the introduction of such carriages was that the "curative process commences the instant the patient is put into the carriage."[1] Americans adopted this concept during the American Civil War, when General Jonathan Letterman, a Union military surgeon, created the first organized system in the United States to transport injured patients. Civilian EMS systems in the United States evolved subsequently—with the first one forming in Cincinnati in 1865.

It was nearly a century later in the 1960s when changes in medical technology and knowledge joined forces with political will to formalize the concept of prehospital care. In the early 1960s, two major clinical advances occurred—cardiopulmonary resuscitation (CPR) for life support of the patient in cardiac arrest, and the development of portable external defibrillators. These two advancements provided the foundation of advanced cardiac life support (ACLS). This, in turn, led to the concept of trained community members to respond to emergencies to improve outcome.

In 1965, President Lyndon Johnson created the President's Committee for Traffic Safety, which published the report *Health, Medical Care, and Transportation of the Injured*. It identified motor vehicle crashes as a significant public health concern. The committee recommended a national program to reduce highway deaths and injuries.

Additionally, in 1966, a report was released by the National Academy of Sciences titled *Accidental Death and Disability: The Neglected Disease of Modern Society*. It emphasized the need to address the quality of prehospital emergency medical care as it recognized that ambulances were ill-equipped and inappropriately staffed.

These two documents paved the way for the Highway Safety Act of 1966, which was passed by Congress and ultimately led to the formation of the National Highway Traffic Safety Administration (NHTSA) within the Department of Transportation. The NHTSA developed a national EMS curriculum which, in 1969, became the standard for emergency medical technician (EMT) training in the United States. In 1973, Congress further passed the EMS Systems Act, granting funds for the development of regional EMS systems. As a result, various states established a total of approximately 300 EMS regions with associated federal support. In the United States, the responsibility of EMS then shifted from federal to state level in 1981, which created heterogeneity in the system.

During the same period of time, EMS and prehospital medical systems were similarly evolving in most developed countries outside the United States. All had unique aspects and points of difference, mostly influenced by the local geography, political will, origin, and resources. The fundamental mission of EMS systems remained common—to deliver the best possible prehospital care to the right patients, in the right timeframe, and to transport them safely to a higher level of care.

Some systems evolved to be predominantly staffed by physicians, while others were staffed almost exclusively by paramedics with no to very minor physician involvement, and most fell somewhere between the two—with at least the capacity for a combined physician-paramedic crew.

BASIC VERSUS ADVANCED LIFE SUPPORT AND BEYOND

CPR was developed in 1960 when the American Heart Association started a program to train physicians in mouth-to-mouth resuscitation (expired air resuscitation) with associated external chest compressions. This led to the development of a tiered system: basic life support (BLS) and advanced life support (ALS) to denote the different skillsets of responders in an organized EMS.

First responders (such as police officers, firefighters, EMTs, and paramedics) are generally the first to arrive to an emergency and can provide medical assistance. A tiered system with different skillsets helps to make the scope of practice and role delineation clearer in the often chaotic prehospital environment.

BASIC LIFE SUPPORT

The adult BLS sequence is circulation-airway-breathing (C-A-B). The goal is to ensure continuous blood (and by proxy, oxygen) supply to major organs. Firefighters, lifeguards, and police officers are often BLS certified because they are often the first to a scene and the application of BLS does not require specialized medical knowledge, per se.

CPR and the use of an automated external defibrillator (AED) are considered BLS skills and the increasing availability of automated defibrillators makes it ever more likely that "unskilled" members of the community will be the first to deliver defibrillation in the event of an out-of-hospital cardiac arrest (OHCA). BLS also includes simple airway maneuvers such as chin lift, jaw thrust, and oxygen administration. For trauma care, basic skills include airway management such as simple airway maneuvers, oropharyngeal and nasopharyngeal airways, and bag-mask ventilation.

Besides CPR and automated external defibrillation, BLS also includes hemorrhage control, and fracture and cervical spine immobilization. In the trauma patient who has suffered a primary traumatic cardiac arrest, CPR is unlikely to be of benefit and may, in fact, detract from useful life saving interventions. This relative controversy will be discussed later in the chapter. The decision not to perform CPR in the resuscitation of the arrested trauma patient should be made by personnel specifically trained at least at the ALS level—and probably only in advanced prehospital systems that include suitably skilled physicians or intensive care paramedics.

ADVANCED LIFE SUPPORT, INTENSIVE CARE–LEVEL PREHOSPITAL CARE

The knowledge and skills necessary to successfully perform ALS are built upon the firm foundations of BLS. For example, even the most experienced prehospital or trauma practitioner relies upon basic airway maneuvers to maintain oxygenation when more sophisticated techniques have failed.

In the prehospital environment, ALS is most often provided by paramedics (advanced EMT practitioners). In many jurisdictions, however, there is a level of prehospital care above ALS, and, outside the United States, combined physician-paramedic crewed helicopter emergency medical services (HEMS) are common. In places where doctors are not part of a HEMS response, paramedics with advanced and intensive care skillsets are often used. This enables the resuscitation bay to essentially be taken to the patient, and, in addition, for advanced resuscitation to be maintained throughout transport. The rationale for these advanced prehospital teams is to maximize the opportunity, while minimizing the time delay for delivery of the advanced trauma care that severely ill or injured patients need.

The provision of advanced, hospital-level interventions (e.g., advanced airway management such as endotracheal intubation, thoracostomy, resuscitative thoracotomy, and commencement of blood product transfusion) remain areas of significant controversy when applied to the prehospital environment. The question of whether it is better to prioritize caring for the patient in the field ("stay and play") or to prioritize expedited transport ("scoop and run") is an old and polarizing one. In environments where transport times are prolonged due to expansive distances (such as Australia), delivering advanced interventions early can be lifesaving. On the other hand, prolonging scene times and delaying presentation to definitive trauma care in order to initiate treatment in the field is unlikely to be in the patient's best interest in other settings. In all mature trauma systems (incorporating the prehospital and in-hospital phases of care), a balance will need to be struck between these two competing priorities.

The reality is that on a population-wide scale, it is rare for anyone to need the advanced services of an EMT, let alone an HEMS prehospital resuscitation team. It is difficult, therefore, to adequately power a study to show a difference between the various models of prehospital care, if such a difference exists. As such, individual prehospital and in-hospital trauma systems have adapted to fulfill the perceived requirements of their population and account for areas of operational limitation.

Training for ALS EMTs now requires a rigorous educational background, and includes skills in advanced and invasive monitoring, diagnostics, and management. Systems functioning at or above the ALS level provide a more comprehensive assessment and stabilization of the trauma patient. In the United States, providers certified at the paramedic level (EMT-P) can perform any of these interventions, whereas providers certified at the intermediate level (EMT-I) may perform a select subset of these skills. Other jurisdictions have comparable delineations of skillset.

Emergency Medical Technician and Paramedic-Based Emergency Medical Service Systems

Specially trained EMTs and paramedics form the bulk of the workforce of EMS systems in North America and in many other developed nations. Under U.S. law, physicians, functioning as EMS directors, are required to approve the medical operations of the EMS systems they oversee. This includes communication, clinical operations, and governance.

In the United States, there are three levels of EMTs based on their level of education and training: EMT-basic (EMT-B), EMT-intermediate (EMT-I), and EMT-paramedic (EMT-P). The primary focus of the EMT-B is to provide basic emergency medical care (using BLS skills) and transportation to a healthcare system. They typically staff nonemergency ambulances and may also respond to nonemergency calls. EMT-I practitioners, or advanced EMTs, provide basic and limited advanced emergency medical care and transportation. They are regulated differently and their skills range between BLS and ALS depending on state regulations. EMT-P is the highest skillset within EMTs in the United States. They undergo intensive 1- to 2-year training in advanced prehospital emergency care. They are ALS trained and can perform procedures such as endotracheal intubation, administration of drugs, and manual defibrillation.

In many other countries, paramedics are required to complete bachelor-level university degrees and are nationally or regionally registered by regulatory bodies—in a similar way that medical practitioners are licensed. In some regions, such as in most states of Australia and in many countries in Western Europe, intensive care paramedics have advanced skills in complex medical and trauma management as well as casualty access and rescue skills.

Primary Survey and Initial Assessment at the Scene

The primary survey is usually performed in the same ABCDE (airway-breathing-circulation, disability-exposure) mnemonic format that is widely known from ACLS. Since 2010, the American Heart Association has recommended changing their ABC approach (airway-breathing-compressions) to CAB (compressions-airway-breathing), emphasizing circulation prior to airway. This is particularly relevant for someone in cardiac arrest to bring focus to chest compressions. It is also relevant to the trauma patient suffering critical bleeding. If you can hear the bleeding, you should stop it first!

The approach, therefore, in the prehospital major trauma patient is C-ABCDE. Strategies for prehospital management of critical hemorrhage are discussed in more detail elsewhere in the chapter, but broadly, options include direct pressure, deep wound packing, use of novel hemostatics (there are several on the market), and tourniquets for extremity hemorrhage (the Combat Application Tourniquet [CAT] is the most widely used). Specific techniques can be used for major maxillofacial hemorrhage, which include nasal packing, or balloon tamponade devices (such as the Rapid Rhino), dental splints, and immobilization of the jaw with a hard, cervical spinal collar.

Novel approaches using resuscitative endovascular balloon occlusion of the aorta (REBOA) to manage critical bleeding in pelvic trauma in the absence of chest trauma are not yet widespread. Indeed, prehospital REBOA has been used successfully on only one documented occasion by London's Air Ambulance. It remains an area of significant controversy and of limited evidence.

Once critical bleeding has been identified and temporarily controlled, moving through the remainder of the primary survey in a rapid yet methodical way is warranted. If there is airway (A) obstruction, the obstruction should be cleared and the airway secured if necessary. While it will often be appropriate to perform a rapid sequence intubation in the prehospital environment, it may not always be necessary or beneficial. A balance must be struck between delaying transfer, likely disposition once in hospital, distance to definitive care, and skillset available. Most prehospital services continue to secure the cervical spine with a hard collar for transport and this would be applied at this time.

Breathing (B) becomes the next priority; after the airway is cleared and possibly secured, a patient's breathing is assessed by observing respiratory rate, pattern, and degree of chest rise and fall. Supplemental oxygen and assisted ventilation may be indicated. Immediately reversible causes of potentially fatal chest trauma, such as tension pneumothoraces, may also be identified and treated with minimal delay to patient transfer. In the case of tension pneumothoraces, many advanced prehospital services (especially those with medical-paramedic HEMS crews) have adopted a finger thoracostomy approach to chest decompression, rather than needle thoracostomy, to achieve a more definitive endpoint early and minimize the risk of reaccumulation of the pneumothorax in transit. Of course, this definitive care may not be available prehospital in all jurisdictions.

Circulation (C) is next assessed by palpating pulses, checking for heart rate, pulse quality and regularity, measuring blood pressure, and again assessing for sources of hemorrhage. As an approximation, a palpable carotid pulse corresponds to a systolic blood pressure of at least 70 mm Hg, and a palpable radial pulse to a systolic blood pressure of 80 to 90 mm Hg.

Also, as part of the primary survey, large-bore intravenous access is obtained. Early fluid management in the trauma patient has been a contentious issue in trauma management for many years. There has been much discussion in the scientific and prehospital literature in relation to the role of crystalloid fluid in the early resuscitation of the trauma patient. Over the past decade there has been a significant shift away from the aggressive use of crystalloid and a shift toward early administration of blood products in the prehospital environment. Some prehospital retrieval services carry red cells only, whereas some (particularly in the United Kingdom) also carry freeze-dried plasma products. In the trauma patient with no concomitant head injury, achieving a systolic blood pressure of 90 mm Hg is ideal (so-called "permissive hypotension"). The only caveat to this blood pressure target is in the head-injured patient. If head injury is present or suspected, then hypotension should be avoided. Indeed, available evidence suggests that in the presence of severe head injury, a single episode of systolic hypotension below 90 mm Hg may double mortality.

As part of the assessment of circulation, suspected long bone fractures can be immobilized with CT-6 (or equivalent) splints and the patient may be packaged in a vacuum mat for transport. If the prehospital team has the capability and expertise, an extended focused assessment with sonography in trauma (e-FAST) exam can be performed as part of the circulatory assessment. This may assist in identifying pneumothoraces before transport and may also reveal other major sources of bleeding that can be relayed to the receiving center.

Disability (D) is measured using the Glasgow Coma Scale (GCS). The GCS is made up of three parts: eyes, verbal response, and motor response. The best patient response is recorded. In the GCS, a maximum of 4 points are allocated for eye opening, 1 being unresponsive and 4 being eyes open; a maximum of 5 points are allocated to verbal response, with 1 being unresponsive and 5 being alert and oriented; and a maximum of 6 points are allocated to motor response, with 1 being unresponsive and 6 for the patient who obeys commands. The highest possible score is 15 and the lowest score possible is 3. As a general rule, patients with GCS score of 8 to 9 have alteration in their conscious state significant enough so as to no longer have the ability to protect their own airway. Assessment of exposure and environment (E) and measures to defend core body temperature completes the primary survey and patient packaging process.

It is important to note that the management of the trauma patient in the prehospital environment relies on concurrent activity within the team. The key to prehospital diagnosis and treatment is the initiation of important treatment as problems are identified, while minimizing unnecessary time spent at the scene. The focus of professional prehospital teams is avoiding the "therapeutic vacuum," or time where nothing useful is happening for the patient.

On arrival, and having assessed the scene, the prehospital care team must rapidly obtain a relevant and focused history of the patient and the events surrounding the incident. In the case of trauma, the ATMIST and AMPLE mnemonics provide a useful framework for gathering key initial information. ATMIST stands for age, time of incident, mechanism of injury, injuries sustained, vital signs (initial and subsequent), and treatment given so far. The AMPLE approach can then be applied to gather specific and relevant history. AMPLE stands for allergies, medications (regular and acute), past history, last meal (menses, tetanus injection) and the events surrounding the injury.

MONITORS

Standard monitors in the prehospital setting include pulse oximetry, noninvasive arterial blood pressure monitoring, electrocardiography (ECG), temperature, and capnography. Indeed, the standards of patient monitoring in the mature EMS or prehospital retrieval service are as high as in most critical care areas of a major hospital. The main difference, of course, is that the monitor display itself must be capable of withstanding the environmental rigors of the prehospital environment, be easily carried, and have a long battery life. There are many commercially available monitors (and ventilators, infusion pumps, and other equipment for that matter) that are approved for flight and designed to be robust across a range of transport platforms and environmental extremes.

POINT OF CARE ULTRASOUND

Portable and affordable ultrasound machines now make point-of-care (POC) ultrasound in the prehospital setting common. In Europe, the United Kingdom, and Australia, where physicians are active in the prehospital management of patients, this becomes even more practical to use. A Dutch observational study showed that 61% of ultrasound examinations impacted decisions in 88% of patients both in prehospital and once they reached definitive care.[2] POC ultrasound is not limited to cardiac arrest; as mentioned earlier, abdominal ultrasound has also been shown to impact treatment decisions and in experienced hands, does not significantly delay treatment.[3]

Out-of-Hospital Cardiac Arrest

Survival from sudden cardiac arrest is highly dependent on early CPR and early defibrillation. Communities have placed an increasing emphasis on bystander CPR, community accessible AEDs, and rapid response of EMS providers.[4]

When the EMS arrives on the scene, the "Universal Treatment Algorithm" is started.[5] Chest compressions continue until cardiac monitoring is placed and it is determined whether defibrillation is appropriate. Pharmacotherapy and airway management remain poorly defined. Optimal airway management in OHCA has not been established, and studies have shown that resuscitation with medications increases likelihood of the return of spontaneous circulation (ROSC) without improved outcome.[6]

The likelihood of ROSC will depend in large part on the etiology of the cardiac arrest as well as the presence of adequate bystander CPR. By far the most common cause of OHCA is from cardiac pathology.

ACUTE CORONARY SYNDROME

Over 5.5 million patients in the United States present to emergency departments with chest pain as their primary complaint. Almost 50% of these patients arrive by ambulance.[7] Any mature EMS system needs to be well equipped to manage this common and potentially life-threatening presentation. For a patient presenting with chest pain in the prehospital setting, three things need to happen: (1) diagnosis be made, (2) treatment commenced, and (3) triage to facility.

PREHOSPITAL DIAGNOSIS AND ELECTROCARDIOGRAPHY

The prehospital ECG is essential in the assessment and triage of patients with chest pain and making the diagnosis of ST-segment elevation myocardial infarction (STEMI). It has been shown through multiple studies that prehospital ECGs are not only technically feasible but they also decreased the time from presentation to reperfusion.[8] This finding has been replicated in rural settings.[9] A recent study looking at clinical outcomes with use of prehospital ECGs showed that the adjusted risk of mortality was lower in cases where ECGs were used.[10]

PREHOSPITAL TREATMENT AND FIBRINOLYSIS

Primary percutaneous coronary intervention (PCI) is the treatment of choice for STEMI patients if this therapy can be performed within 90 minutes of onset or less. When a patient is unable to receive mechanical reperfusion within that time window (either because of geographic isolation or arrival at a non-PCI community hospital) then evidence supports the use of fibrinolysis followed by PCI within 24 hours.[11] It was subsequently demonstrated that a paramedic-based prehospital fibrinolysis program was practical and feasible in reducing time to treatment with favorable clinical outcomes.[12]

MECHANICAL CIRCULATORY SUPPORT

Prehospital Extracorporeal Cardiac Life Support

Recently extracorporeal cardiac life support (ECLS) has been introduced to the prehospital environment for treatment of OHCA patients in some very select settings. The use of mechanical ECLS in the prehospital setting remains controversial. Whether initiated in the field or in the emergency department, there is insufficient evidence to guide broad-based adoption of this practice. Multiple case reports have demonstrated some utility, but this therapy should not detract from high-quality ACLS.[13-15]

Respiratory Distress

Respiratory distress is a common complaint necessitating prehospital medical intervention.[16] Management requires rapid identification of emergent conditions and subsequent intervention to prevent further deterioration. Unfortunately, dyspnea, the perception of respiratory distress, can be influenced by a variety of factors and patient complaint alone is insufficient to identify the underlying cause or its severity. Early stabilization of respiratory function regardless of the underlying pathology is important to avoid significant morbidity and mortality. Even for anesthesiologists who are not participating in prehospital care, it is important to have a familiarity with these conditions as emergent or urgent surgical patients may present with dyspnea that is not fully evaluated and their initial management will fall on the anesthesia team in the perioperative period.

EVALUATION

Initial evaluation of the patient in respiratory distress is structured to identify patients at risk for rapid progression to respiratory failure and identify the need for invasive support. Respiratory rate is an easily obtainable vital sign, and a rate of more than 30 breaths per minute would be defined as abnormal. It is important to consider that other factors, such as anxiety and intoxications, can affect the respiratory rate. Other physical signs include stridor, upper airway obstruction, inability to speak in full sentences, and cyanosis. Pulse oximetry has emerged as a standard monitor for detecting hypoxemia, although it should be noted that a patient can have significant respiratory pathology while maintaining a clinically acceptable oxygen saturation.[16] Developing a differential of dyspnea can be challenging but may be simplified if the clinician considers the underlying organ system likely to be the culprit. Table 67.1 provides a comprehensive, but not exhaustive, list of causes of dyspnea that an anesthesiologist should be familiar with in the emergent and prehospital setting.

MANAGEMENT

Initial management of the patient with dyspnea in the field involves stabilization when possible and rapid transport to a facility that can provide definitive care. Interventions in the prehospital setting can range in complexity depending on the needs of the patient and the skillset of the providers. Common interventions include supplemental oxygen for the management of hypoxia, inhaled bronchodilators for wheeze, and bag-mask ventilation or intubation for fulminant respiratory failure. Prehospital care also will often include needle decompression for suspected tension pneumothorax (although increasingly finger thoracostomy is performed) and epinephrine administration for suspected anaphylaxis. Comprehensive management of these conditions is beyond the scope of this chapter; however some pearls related to respiratory distress are in Box 67.1.

Neurologic Injury and Head Injury

Diseases of the central nervous system pose a significant challenge to the prehospital provider. Many of the familiar techniques used by clinicians to gather important information are unavailable. An unconscious or post-ictal or post-seizure patient will not be able to provide useful clinical history and even gathering basic identifying information can be a challenge. These issues are magnified in the prehospital environment. This section will provide an overview of the major priorities and challenges that face the prehospital provider when confronted with a neurologic event in the field. For further clinical detail on the management of these conditions please refer to the Chapters 84 and 66.

TABLE 67.1 Etiologies of Dyspnea

Organ System	Conditions
Pulmonary	Airway obstruction Pulmonary embolus Noncardiogenic pulmonary edema Acute respiratory distress syndrome Pneumonia Anaphylaxis Obstructive sleep apnea Asthma Cor pulmonale/pulmonary hypertension Aspiration Pleural effusion Malignancy Chronic obstructive pulmonary disease
Cardiac	Cardiogenic pulmonary edema Acute coronary syndrome Tamponade Pericarditis Congenital cardiac disease Valvular heart disease Cardiomyopathy
Neurologic	Cerebrovascular accident Intracranial hemorrhage Organophosphate poisoning Multiple sclerosis Guillain-Barré syndrome Tick paralysis Amyotrophic lateral sclerosis Polymyositis Porphyria
Trauma	Pneumothorax (tension vs. simple) Hemothorax Diaphragm injury Tamponade Rib fractures
Abdominal	Acidosis/shock from abdominal catastrophe Splinting from abdominal pain Pregnancy Ascites
Psychogenic	Panic attack Somatization disorders Hyperventilation syndrome
Metabolic/endocrine	Toxic ingestion Diabetic ketoacidosis Renal failure/metabolic acidosis Electrolyte abnormalities Fever Thyrotoxicosis
Infectious	Pneumonia Epiglottitis
Hematologic	Carbon monoxide poisoning Cyanide poisoning Anemia Sickle cell crisis (acute chest syndrome)

Adapted from Braithwaite S, Perina D. Dypsnea. In: Walls R, Hockberger R, Gausche-Hill M, eds. *Rosen's Emergency Medicine: Concepts and Clinical Practice*. 9th ed. Philadelphia: Elsevier; 2018.

TRAUMATIC BRAIN INJURY

Traumatic brain injury is a major cause of morbidity to patients who have been traumatically injured. From the moment of injury all care should be focused on maximizing the survival of remaining neuronal tissue. Specific parameters outlined in Chapter 66 are followed with particular emphasis on minimizing secondary injury.

BOX 67.1 **Pearls (Precepts, Dicta) for Respiratory Distress**

- Rule out life-threatening, rapidly progressive causes such as tension pneumothorax or acute coronary syndrome prior to initiating a more thorough workup for causes of shortness of breath
- A normal oxygen saturation does not mean a patient is without significant respiratory pathology
- The majority of patients with respiratory distress are related to pulmonary or cardiac causes; however, the astute clinician should always keep in mind additional causes in the appropriate clinical context
- Chronic conditions tend to be better tolerated than acute ones. For example, chronic mitral regurgitation is well tolerated until it becomes severe whereas acute mitral regurgitation is a medical emergency
- The acuity of onset of the condition is helpful for determining the cause. For example, a patient with a chronic neuromuscular disorder who presents with an acute worsening likely has an additional explanation for his or her shortness of breath
- There is no clinical finding that rules out pulmonary embolism and a low threshold for further workup should be maintained in the appropriate patient
- Unnecessary supplemental oxygen does not have a benefit and likely leads to preventable harms

SPINAL CORD INJURY

Patients with spinal cord injury (SCI) present unique challenges to the prehospital provider. Patients rarely injure themselves in locations where access and maintenance of spinal precautions is easy. Balancing the risk of worsening or causing injury must be weighed against the necessity of moving the patient to a suitable transport platform. It is important to note that transporting a patient as carefully as practical is very unlikely to generate enough force to cause an injury that is not already present.

With this in mind, the safest approach for handling potential SCI cases is to assume such an injury is present in any mechanism that could result in significant SCI, but also not to delay lifesaving treatment in preference of spinal precautions.

Spinal immobilization techniques have received increased scrutiny over the past decade. The hard-cervical collar is no longer considered the "magic bullet" of cervical spine immobilization. Hard spinal boards are also falling out of favor with many emergency services around the world. Regardless of the jurisdiction, if "hard" immobilization devices are used, there is a shift toward using them for as short a period of time as possible. Depending on the expected transport time from the scene there may be a role for early application of Philadelphia- or Aspen-style collars with transport in a vacuum mat. The main advantage of cervical spine collars is that they alert the receiving hospital that this patient might have c-spine injury and needs according attention. It is possible that this can be achieved with a soft collar, avoiding some of the risks of prolonged hard collar immobilization. This is particularly relevant for the older patient with fragile skin or for the patient requiring prolonged transfer.

ACUTE STROKE MANAGEMENT

Stroke is a much feared and potentially debilitating condition. While it can be more difficult to identify the location of a particular stroke without a detailed neurologic exam, the ability to provide targeted reperfusion therapy for large vessel stroke has seen a drastic change in the expected outcome for those patients. Much like percutaneous interventions for heart attacks, interventional neuroradiology is able to drastically change the clinical trajectory of a "brain attack." Minimizing the ischemic time for neuronal tissue becomes the priority. Thus, early diagnosis, activation of neuroradiology, and timely transport become essential to achieving optimal clinical outcomes. This has resulted in some jurisdictions deploying thrombolysis in the field. Recent major studies were halted early due to evidence of benefit for those patients who received thrombectomy. As such, transporting these patients to a center that can provide clot retrieval is important. To maximize the speed of diagnosis, mobile stroke units have been developed that contain a portable computed tomography scanner and capacity for "teleradiology" to make a radiologic diagnosis in the field. While these platforms have been launched with significant enthusiasm, they remain the exception across the developed world, and potentially of questionable cost-benefit.

SEIZURE

Seizure is a common presenting complaint and makes up approximately 1% of all emergency department admissions. First responders are therefore often required to provide emergency medical management and, where appropriate, supportive care during transport to hospital. The detailed management of status epilepticus is covered in Chapter 57, but patients with status epilepticus have improved outcomes when treatment is commenced in the field by EMS providers.

ALTERED MENTAL STATUS

Altered mental status covers a broad range of conditions affecting a patient's cognition. In the prehospital environment, first responders may be required to assess, treat, and safely transport people with a range of underlying disorders. Patients may be drug affected, head injured, delirious, or suffering from a wide range of medical or neurosurgical disorders. Prehospital providers may need to treat patients with altered mental status in their own home, at a motor vehicle accident scene, or in any number of complex environments. As with any scene, providing a safe environment for the providers as well as for the patient is of utmost importance.

When the need arises, pharmacologic sedation may be utilized to facilitate safe transport to a healthcare facility. Providers are expected to assess and treat any easily reversible causes of altered mental status such as hypoglycemia or hypoxia, but many precipitants are outside the scope of a field diagnosis. Collateral history is increasingly important to determine the timeline for the changes in mental status and how far the patient's current altered state has departed from normal.

Prehospital Trauma

Trauma remains a significant burden of disease globally and is the greatest cause of death for those under 40 years of age. As has been demonstrated in previous chapters in this text, early access to definitive care is the single greatest modifier in the survivability of a major traumatic event. Trauma patients' need for advanced and specialized trauma care begins at the moment of injury, hence delays in transport or delivery of definitive care contribute to poor outcome. Accordingly, the prehospital management of trauma patients has the potential to start the process of recovery from the point of injury. It is essential that anesthesiologists working in receiving hospitals understand the capacity, unique challenges, and limitations of providing care in the prehospital environment so they can be prepared to continue with ongoing care. Understanding what can (and cannot) be done in the prehospital environment minimizes duplication and stagnation of care in-hospital, and is essential to avoiding the therapeutic vacuum.

TRIAGE

Triage is the process of prioritizing and allocating resources based on clinical need and urgency. Routinely, higher-level responses are dispatched to the most acutely sick or injured patients. First responder systems and processes differ within and among nations. Depending on the specific first responder system of the country, the first response providers could be any combination of road ambulance (staffed by paramedics/EMTs of variable skillsets or physicians), higher acuity "fast-cars" containing a physician or advanced practice EMTs (sometimes referred to as intensive care paramedics), or HEMS crews. The triage system in most developed EMS systems is activated via a standard phone number. This number varies from region to region and can be reached via landline or cellular networks (911 in the United States, 999 in the United Kingdom, 000 in Australia, and 112 from cellular telephones worldwide). Once activated, trained nonmedical dispatchers utilize a standardized script to determine urgency and location, and provide bystander advice and support. An appropriate emergency medical resource is tasked to respond. Alternatively, the system may be activated for a partner emergency service such as police or fire to respond to an incident, or all three emergency services may be required at an incident site.

SCENE SECURITY

For providers who predominantly practice in-hospital, it can be easy to take the relative luxuries of lighting, environmental control, and personal security for granted. For first responders and others who deliver trauma care in the prehospital environment, there are many hazards that need to be considered and addressed to ensure as safe an environment as possible—for themselves and their patient.

Some examples of hazards that need to be considered and/or addressed are included in Table 67.2.

The priority on arrival at the scene is to ensure safety for self, the crew, bystanders, and then the patient. It is also important to rapidly assess the number and nature of injured, so a further triage can occur. If the number or severity of injured exceeds the capacity of the on-scene crew, then additional resources should be requested. For the purposes of this chapter we will consider only one injured patient. For considerations in the mass casualty context, refer to Chapter 68.

TABLE 67.2 Environmental and Human Hazards

Environmental Hazards	Human Hazards
■ Live power cables	■ Active shooters
■ Fire	■ Distressed family/friends
■ Spilled chemicals/fuel	■ Intoxicated bystanders
■ Other vehicles/cars/traffic	■ Panicked/distressed crowds
■ Enclosed spaces/inadequate ventilation	■ Complex or insecure scenes (e.g., subsequent to terrorist attack, conflict zones)
■ Unsafe/stable buildings/trees	
■ Extreme weather (sun/rain/snow)	
■ Biological hazards	

PRIMARY AND SECONDARY SURVEYS

First responders have been trained to approach the primary survey with the same mindset and using the same principles of Advanced Trauma Life Support (ATLS). The focus of the prehospital primary survey is to perform a rapid initial assessment identifying, assessing, and treating life-threatening injuries. It differs from the in-hospital primary survey in that there is the inherent imperative to secure and package the patient for transport at the same time as assessing and treating. In some circles providers have looked to dichotomize the clinical approach in the field between the two extreme positions of "stay and play" and "scoop and run." In reality, neither approach presents a one-size-fits-all, and providers need to consider the individualized requirements of their patient based on injury, skillset, transfer time, resources, and environmental constraints—and then adapt the clinical approach to balance these priorities and optimize the patient's care.

STAY AND PLAY

The stay and play concept refers to the approach of maximizing the amount of definitive clinical treatment that occurs in the field before transport to the receiving hospital. By providing maximal appropriate treatment in the field, the intent is to minimize the clinical insult of the injury or illness. The rationale for stay and play is that if the patient needs advanced trauma care from the moment of injury, then that care should be delivered to them early on. In reality, it is not as simple as merely transporting a one-person trauma resuscitation bay to the patient. Treatments provided in the field can be more difficult to achieve. Despite ever-increasing standards of prehospital resuscitation and more easily available diagnostic equipment, reduced sterility and limited diagnostics and monitoring remain an issue for prehospital teams. As such, each treatment needs to be considered in relation to its potential benefits weighed against the possible harm in delayed primary transfer. In recognition of this tension, many high-performing teams have developed focused, standard operating procedures to maximize their chance of first attempt success and minimize the time taken to perform tasks. A commonly performed intervention is rapid sequence intubation. "Kit-dumps" (Fig. 67.1) are commonly used both as a cognitive aid and as a workspace tool to improve team performance in the field. The strongest clinical prehospital team is one that possesses complementary and overlapping skills. It remains an inescapable reality that teams that are jointly staffed in physician/advanced paramedic configuration tend to have a broader scope and extended range of practice.

Fig. 67.1 "Kit-dumps" are commonly used both as a cognitive aid and as a workspace tool to improve team performance in the field. (Courtesy MedSTAR, Adelaide, South Australia.)

SCOOP AND RUN

The focus with the scoop and run approach is to minimize scene time and transport the patient to definitive care as soon as possible. Proponents of scoop and run argue that the time to definitive care is what makes the difference, hence the patient should be moved as quickly as possible to a trauma center—with only the barest of essential lifesaving interventions performed at the scene. Depending on the expected transport time, this philosophy may be entirely appropriate for many trauma patients. Any interventions performed in this context would be for immediate lifesaving interventions only.

FURTHER CONSIDERATIONS

Prehospital providers are also expected to gather as much useful information from the scene as possible to guide ongoing care. Current medications, collateral medical history, and photos of the accident scene are all tasks that are expected to be undertaken whenever possible.

Transport destinations are often predetermined. Effort is made in most systems to deliver patients to the appropriate clinical location wherever possible. For example, a suspected myocardial infarction should be transported to a center where PCI is possible. Other acute subspecialty centers include stroke, extracorporeal membrane oxygenation, trauma, pediatrics, and burns. Factors such as patient preference and, in certain jurisdictions, level of insurance can also be considered where the clinical setting allows.

BALANCED RESUSCITATION

The early control of major hemorrhage has become the focus of any significant trauma resuscitation. As we gain greater understanding of the consequences of acute traumatic coagulopathy as well as the downstream impact of our clinical resuscitation, there has been a transition away from crystalloid-based fluid resuscitation toward "hemostatic" resuscitation fluids. Many ambulance services around the world now carry fractionated blood products—usually packed red blood cells, but an increasing number are carrying plasma. There is active research underway on the early administration of tranexamic acid (TXA) and the impact this may have on clinical outcomes after injury. The CRASH-2 trial demonstrated a mortality benefit when TXA was administered within 3 hours of injury. The hypothesis is that early application may result in greater benefits, although the trial was justifiably criticized for nonhomogeneity of trial sites. Challenges that still need to be addressed include issues such as blood crossmatching, fluid warming, stock control, and cost. There may also be a role for administration of clotting factor concentrates, but this has yet to translate into evidence.

HEMORRHAGE CONTROL

Together with the increased use of blood products in the field there has been a growing array of interventions available for stopping bleeding. The American College of Surgeons (ACS) has been coordinating a nationwide "Stop the Bleeding" campaign in the United States to improve the use and knowledge of hemorrhage control by bystanders. Techniques such as direct pressure, wound packing, and tourniquet application have been introduced to the bystander population.

ANTIFIBRINOLYTICS

The debate around the efficacy and safety of TXA use in the acute severe trauma patient continues. The results of the CRASH-2 trial have been adopted in many trauma systems. On the other hand, many remain unconvinced given the significant heterogeneity of trial sites in the study. The CRASH-2 trial did show a reduction in death from bleeding in trauma patients who received a 1-g intravenous dose of TXA within 3 hours of injury. This benefit did not extend if the first dose was given beyond 3 hours, where the trend was toward complications from thrombosis. There is also some concern that fibrinolysis is not occurring in all bleeding trauma patients and conceptually antifibrinolytic therapy may not be indicated. Additional trials such as the PATCH study are investigating whether targeted use of TXA conveys any survival benefit.

TOURNIQUETS

A CAT correctly placed can significantly reduce the volume of blood loss and arrest hemorrhage in time to transport a patient to definitive care.

RESUSCITATIVE ENDOVASCULAR BALLOON OCCLUSION OF THE AORTA

London's Air Ambulance (known more commonly as London HEMS) has pioneered the use of REBOA in the field for the management of patients with severe bleeding that previously would have been unlikely to survive to hospital. They have field-deployed REBOA multiple times with an overall positive result. The majority of the cases where REBOA had been used involved significant pelvic trauma. The evidence base for REBOA use is expanding. Yet, on the other hand, in a mature trauma system, patients eligible for REBOA may in fact receive definitive care in the operating room instead. The main question now is whether this type of highly specialized intervention is beneficial to implement in other contexts—or whether it truly has a mortality benefit. Perhaps there may come a time when REBOA, or something similar, will become the standard for hemorrhage control in the field.

FUTURE DIRECTIONS AND CHALLENGES

The burden of trauma is growing. With an aging population, more densely populated urban centers, heavier road use, and accelerating technological possibilities for care, the challenges (and opportunities) for first responders are immense. Below are some of the challenges and active areas for investigation currently being explored. As anesthesiologists we need to remain informed and open to the evolution of clinical fields that we partner with and work within.

TRAUMA TRIAGE

The time from incident or injury to the notification of emergency services can either be immediate or significantly delayed. Efforts have been made to automate part of the process. Many modern cars have crash detection and emergency notification built in. Companies such as OnStar provide timely notification to emergency services in the event of a vehicle crash. As more cars are fitted with detailed analytics, there is potential for emergency services to be notified directly when a vehicle is involved in an incident of sufficient force to result in injury.

Point-of-Care Diagnostics

As POC devices become smaller, more reliable, and more affordable there is potential for front-line medical providers to perform rapid assessments to further triage and prioritize patients and their medical needs. Ultrasound is the first device that is likely to be widely adopted at the front line—and already has been deployed in many prehospital services around the world. It is relatively cheap, portable, robust, and has a growing body of evidence to support its use.

The impact of POC diagnostics could be further enhanced with greater use of telemetry and telemedicine. The transmission of live feeds from emergency services to the receiving hospital could provide hospital staff with the information necessary to expedite appropriate care—for example surgical services. Combining field and telemedicine in this way has garnered significant interest from organizations that provide care in the most remote and austere of environments, such as mining companies and the military. The potential application of advanced telemedicine and realtime clinical support for a severely injured or unwell patient aboard a ship at sea is immediately obvious. The two-way nature of telemedicine would also enable hospital staff to support operators in the field, potentially reducing the need for every front-line provider to be an expert in interpretation. Although telemedicine will never replace technical skills, it may greatly enhance clinical decisions.

Overdose/Toxicology/ Environmental Exposure

In contrast to the relatively controlled confines of a modern hospital, prehospital providers are truly confronted with the unknown. In addition to the clinical presentation of the patient there are significant issues that need to be considered when ensuring no further harm occurs to either patients, bystanders, or first responders.

Environmental toxins, chemical exposures, armed patients, or nearby assailants are an unfortunate reality we need to confront. First and foremost, it is the responsibility for any prehospital provider to ensure they are safe. This may require that access to patients be delayed to allow time for police, fire, and/or utility providers to secure the scene. Many EMS units (particularly in the United States) have responded to this challenge with the development of specialized "tactical units." These providers are given additional training to allow them to operate in potentially hostile environments.

The remaining discussion will assume that scene safety has been achieved and that front-line providers have access to the patients.

EVALUATION

An anesthesiologist should have an appreciation of common environmental exposures and toxidromes that may be present, particularly because often these patients can have a concomitant surgical condition that will require management. Evaluation of the intoxicated patient is often challenging and limited to the clinical exam and history obtained from nearby persons. Empty pill containers and other environmental clues can be helpful, but may not be present.[17] If an environmental exposure is suspected, care should be taken to avoid exposure to the emergency response personnel and the patient's clothing should be placed in a secure enclave to prevent further exposure.[17] In the event of significant biologic/chemical/radiation exposure, expert consultation should be obtained to ensure safety of first responders and treating clinicians. While many laboratory tests exist to detect ingested substances, it is important that prehospital providers be familiar with common clinical toxidromes as these may be the only clue to the etiology of the overdose.

MANAGEMENT

As with most emergency management, initial treatment is directed toward stabilizing vital signs before progressing to specific management of any environmental or toxic exposures. Decontamination is also indicated in environmental exposures; this includes removing the patient from the exposure, removal of the patient's clothing, and surface decontamination when indicated.[17] Once these are stabilized further interventions can be attempted. In addition to vital signs, other rapid tests in the field include a 12-lead ECG and a blood glucose. Finally, with the current epidemic of opioid abuse, empiric administration of naloxone should be considered in the appropriate patients. If transportation to a hospital is likely to be delayed, then consultation with toxicology experts should be considered. Table 67.3 is a substantial list of different agents by presenting clinical finding. Table 67.4 lists common ECG findings of selected agents.

ENVIRONMENTAL EXPOSURES

Anesthesiologists should further be familiar with environmental exposures. These include hypothermic and hyperthermic injury. Frostbite refers to the phenomenon of tissue freezing leading to severe microvascular injury and eventual cell death. This can lead to significant morbidity including limb loss and substantial pain. Prehospital management is limited as one of the most devastating treatment failures of frostbitten extremities is freezing-rewarming-refreezing. Therefore, rewarming should not be attempted in the field if there is any chance of it being interrupted. Wet clothing should be removed and the affected area immobilized and protected from physical trauma if possible. If significant cold injury is present and re-warming is attempted, care must be taken as the sudden return of cold blood to the heart can precipitate ventricular dysrhythmias and subsequent cardiac arrest.[18]

Burn injuries are very common but fortunately the majority are only minor injuries. Detailed anesthetic considerations for management of the patient with burns are addressed elsewhere in this book (give chapter here). Specific prehospital management for severe burns focuses on stopping any ongoing burn injury, securing an airway where necessary, and providing adequate analgesia. Many jurisdictions will have a specialist burns center that EMS providers can transfer patients to for definitive care, weighed against the need for transport to the nearest hospital. Considerations for transport to a tertiary burn center are outlined in Fig. 67.2.

Horizons in Prehospital Care

The trend in prehospital care over the last several decades has been toward bringing high-quality care to the roadside. By reducing the therapeutic vacuum, patients receive lifesaving interventions at an earlier time in their disease process hopefully translating to reduced morbidity and mortality. The increasing use of real-time data, predictive analytics, and telemedicine will enable front-line providers to assist in earlier diagnostics and more tailored therapies, and enable hospital-based services to implement plans based on needs identified in the field. The capacity to save lives is going to continue to increase as prehospital and hospital-based providers work in concert for the benefit of their patients.

TABLE 67.3 Clinical Findings and Associated Agents

Clinical Finding	Agent
Miosis	Cholinergics, clonidine, carbamates, opioids (EXCEPT meperidine), organophosphates (pesticides), phenothiazines, pilocarpine
Mydriasis	Anticholinergics, sympathomimetics (albuterol), withdrawal from narcotics
Coma (important to rule out hypotension first)	Alcohols (ethanol, ethylene glycol), tricyclic antidepressants, thallium, toluene, heavy metals (lead), lithium, hypoglycemics (check blood glucose), arsenic, SSRI/SNRIs, antihistamines, opioids, benzodiazepines, barbiturates, carbon monoxide, cyanide, clonidine, insulin (check blood glucose), isoniazid, other sedative-hypnotics
Seizure (history of seizure disorder will increase likelihood)	Hypoglycemics/insulin (check blood glucose), organophosphates, sympathomimetics, salicylates, tricyclic antidepressants, bupropion, cocaine, camphor, chlorinated hydrocarbons, propranolol, phencyclidine, heavy metals (lead), lidocaine, lithium, withdrawal from anti-epileptics, methylxanthines (theophylline, caffeine), methanol, nicotine, amphetamines, ethanol withdrawal
Diaphoresis	Sympathomimetics (cocaine, amphetamines), organophosphates, salicylates, phencyclidine/ketamine
Dry skin	Antihistamines, anticholinergics
Blisters	Barbiturates, mustard gas, snake and spider envenomation
Flushed skin	Niacin, anticholinergics, carbon monoxide
Cyanosis (check oxygen saturation)	Nitrates, nitrites, ergotamine, aniline dyes, dapsone, phenazopyridine
Bradycardia	β blockade, opioids, anticholinesterases, anti-arrythmias (amiodarone, digoxin), calcium channel blockers, clonidine, cholinergics, alcohols
Tachycardia	Cocaine, amphetamines, anticholinergics, antihistamines, alcohol withdrawal, phencyclidine, ketamine, theophylline, caffeine, thyroid hormones, tricyclic antidepressants
Hypothermia	Opioids, carbon monoxide, insulin, hypoglycemics, sedatives, alcohols
Hyperthermia	Neuroleptic malignant syndrome (decreased reflexes, rigidity), serotonin syndrome (hyperreflexia, clonus), salicylates, sympathomimetics (cocaine, amphetamines), alcohol withdrawal, anticholinergics, antipsychotics, antidepressants, nicotine
Hypotension	Calcium channel blockers, clonidine, arsenic, cyanide, aminophylline, anti-hypertensives, antidepressants, sedatives, opioids
Hypertension	Thyroid supplements, cocaine, amphetamines, anticholinergics, nicotine, caffeine, sympathomimetics
Tachypnea	Salicylates, nerve agents, metabolic acidosis secondary to ingestion, pneumonitis from inhalation
Bradypnea	Sedatives, alcohols, opioids, marijuana

Adapted from Meehan TJ. Approach to the poisoned patient. In: Walls R, Hockberger R, Gausche-Hill M, eds. *Rosen's Emergency Medicine: Concepts and Clinical Practice*. 9th ed. Philadelphia: Elsevier; 2018.

TABLE 67.4 EKG Findings in Overdoses

Segment/Interval	Appearance	Agent
P wave	Absent	Digoxin, cholinergics
PR interval	Prolonged	Calcium channel blockers, β-blockers, magnesium
QRS interval	Prolonged	Type 1 anti-arrhythmics (lidocaine), antihistamines, cocaine, tricyclic antidepressants
ST segment	Scooped	Digoxin
QT interval	Prolonged	Many agents, methadone, antipsychotics most relevant.
T wave	Peaked	Acidosis leading to hyperkalemia
T wave	Flattened	Lithium
U wave	Present	Lithium, caffeine, theophylline, albuterol,

Adapted from Meehan TJ. Approach to the poisoned patient. In: Walls R, Hockberger R, Gausche-Hill M, eds. *Rosen's Emergency Medicine: Concepts and Clinical Practice*. 9th ed. Philadelphia: Elsevier; 2018.

Courtesy of the
American Burn Association
Advanced Burn Life Support (ABLS)
Learn more about the ABA and ABLS at **www.ameriburn.org**

Burn Center Referral Criteria

A burn center may treat adults, children, or both.

Burn injuries that should be referred to a burn center include:

1. Partial thickness burns greater than 10% total body surface area (TBSA).
2. Burns that involve the face, hands, feet, genitalia, perineum, or major joints.
3. Third degree burns in any age group.
4. Electrical burns, including lightning injury.
5. Chemical burns.
6. Inhalation injury.
7. Burn injury in patients with preexisting medical disorders that could complicate management, prolong recovery, or affect mortality.
8. Any patient with burns and concomitant trauma (such as fractures) in which the burn injury poses the greatest risk of morbidity or mortality. In such cases, if the trauma poses the greater immediate risk, the patient may be initially stabilized in a trauma center before being transferred to a burn unit. Physician judgment will be necessary in such situations and should be in concert with the regional medical control plan and triage protocols.
9. Burned children in hospitals without qualified personnel or equipment for the care of children.
10. Burn injury in patients who will require special social, emotional, or rehabilitative intervention.

Excerpted from Guidelines for the Operation of Burn Centers (pp. 79-86), Resources for Optimal Care of the Injured Patient 2006, Committee on Trauma, American College of Surgeons

Severity Determination

First Degree *(Partial Thickness)*
Superficial, red, sometimes painful.
Second Degree *(Partial Thickness)*
Skin may be red, blistered, swollen. Very painful.
Third Degree *(Full Thickness)*
Whitish, charred or translucent, no pin prick sensation in burned area.

Percentage Total Body Surface Area (TBSA)

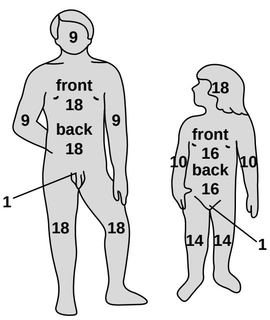

Fig. 67.2 Indications for transfer to a tertiary burn center. (Courtesy American Burn Association. https://ameriburn.org/public-resources/burn-center-referral-criteria/.)

Acknowledgment

The editors and publisher would like to thank Drs. Peter Nagele and Michael Hupfl for contributing a chapter on this topic in the prior edition of this work. It has served as the foundation for the current chapter.

Complete references available online at expertconsult.com.

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68 Biological, Natural, and Human-Induced Disasters: The Role of the Anesthesiologist

DANIEL W. JOHNSON, WILLIAM P. MULVOY, and STEVEN J. LISCO

KEY POINTS

- Natural disasters such as earthquakes, hurricanes, floods, tsunamis, and tornadoes have the ability to cause massive interruptions in the normal provision of health care.
- Anesthesiologists possess a combination of knowledge and procedural skills that make them extremely valuable in the immediate and ongoing response to large-scale disasters; to do so, they and their colleagues must be willing to modify their traditional workflow in order to best meet the needs of victims while also serving the needs of other patients.
- Anesthesiologists who travel from resource-rich to resource-limited environments to provide rescue medical care must cope with a number of unanticipated challenges, including lack of familiarity with infectious diseases which have been eradicated from first-world nations, and the management of these diseases.
- Anesthesiologists who travel to disaster-ravaged areas are exposed to a variety of physical and mental stressors that can have an impact on them for months or years after an event.
- Because of the very high early mortality following some natural disasters such as tsunamis, local healthcare systems can be overwhelmed and many local healthcare workers are either lost or displaced. As a result, anesthesiologists who are able to travel to victim countries and provide temporary aid can provide much needed support for the community.
- Although hurricanes tend to have fewer immediate fatalities than earthquakes or tsunamis, their severity and the subsequent flooding can cripple the healthcare system and the physical plants, including clinical and educational facilities.
- When disasters induce shortages of medications, fluids, and supplies, as resulted from Hurricane Maria in 2017, global conservation of existing supplies and avoidance of waste are essential.
- Following a massive terrorism-induced disaster such as the September 11, 2001 attack on New York City, health systems were encouraged to develop more formal triage plans, with particular emphasis on clinical access centers in areas surrounding potential disaster sites.
- In addition to natural disasters, mass shootings are also challenging the healthcare systems. In the United States, 1123 people have been killed by mass shootings since 1966. Accordingly, all anesthesiologists and other providers must be prepared to provide care in the aftermath of these heinous events.
- As acts of terrorism have become more aggressive in recent decades, anesthesiologists may be more likely to provide care following a chemical, biological, radiological, or nuclear (CBRN) event. To do so, they must have an understanding of the implications of each of these disasters on the community and the providers, and be prepared to modify their practices to best meet the needs of patients and the community.
- One of the highest priorities for healthcare workers after a CBRN event is to take precautions against becoming the next victim. Personal protective equipment (PPE) is of the greatest importance in these situations.
- During an outbreak of a highly dangerous infectious disease, anesthesiologists must understand the mechanisms of transmitting the disease and its management; they are often able to serve as consultants in the design of optimal care pathways to minimize risk to patients and healthcare workers.

Introduction

Large-scale disasters force the human population to address complicated issues often never confronted before, in an attempt to address death and injuries and restore order. For decades, many countries have had in place national, regional, and hospital disaster management plans that focus on preparedness, immediate response, and recovery efforts. The objective of these plans is to minimize further injury or damage by optimizing the use of resources and personnel to respond to mass casualties resulting from both natural and human-induced disasters. Healthcare providers have a special role to play in all such events. Anesthesiologists, because of their unique combination of skills and knowledge, are an extremely valuable part of the overall effort to optimize the health and recovery of disaster victims. In-depth knowledge of pharmacology and physiology, expertise in resuscitation, experience in critical care medicine, and the ability to extinguish pain represent some of the critical knowledge and skills anesthesiologists are able to provide in response to these disasters.

In order to provide the support needed during and after these events, anesthesiologists must be prepared to modify their traditional role in patient care and apply their unique abilities to care for patients in a wide variety of scenarios. If, after a massive natural disaster, a given hospital system has adequate personnel to provide intraoperative anesthesia care, but inadequate physicians to triage and provide resuscitation in the Emergency Department (ED) or in the field, anesthesiologists should volunteer to provide care in these settings. Similarly, for those victims requiring surgical interventions, during the postoperative phase of care, most hospitals will not have sufficient critical care physicians to care for all of the critically injured patients. Anesthesiologists are uniquely suited to temporarily assume the role of intensivist, as they so often do in the operating room. Drawing from their training experiences as well as their backgrounds in intraoperative management of complex surgical patients, all anesthesiologists, whether formally trained in critical care medicine or not, should expect to play a key role in the management of patients in the intensive care unit (ICU) and other settings following a disaster.

To address some of the opportunities and challenges associated with management during and after disasters, this chapter is organized into four distinct sections. Whereas consistent themes exist among all of these realms, each section reveals unique roles for the anesthesiologist in addressing patient and community needs after each type of disaster. The four sections of the chapter are:

1. Natural Disasters
2. Acts of Terrorism
3. Chemical, Biological, Radiological, and Nuclear Warfare
4. Epidemic and Pandemic Infectious Outbreaks

Section 1: Natural Disasters

Natural disasters such as earthquakes, hurricanes, floods, tsunamis, and tornadoes can cause massive disruptions in communities, while also interfering with the normal provision of health care. Prehospital care can be completely disrupted due to damage to roads, and hospital care can be brought to a halt due to lack of water, oxygen, fuel, electricity, and other necessary utilities. Hospitals themselves can be damaged or destroyed by these types of events. Communication can be difficult or impossible following natural disasters due to a combination of damage to telecommunication systems and simultaneous usage overload as families and victims try to locate one another. In this section of the chapter, several historical examples of natural disasters will be examined in order to highlight the critical role of the anesthesiologist in the recovery from these devastating events.

EARTHQUAKES

Earthquakes are capable of inducing an incredible amount of damage in a very brief period of time. Resource-limited areas of the world, suffering from poor underlying infrastructure at baseline, are particularly vulnerable to severe damage following an earthquake. The magnitude 7.0 earthquake that struck Haiti on January 12, 2010 was one of the most devastating natural disasters in modern history, and was an example of how badly a nation's healthcare system can be damaged by this type of event. The death toll in Haiti is difficult to calculate accurately, but nearly all analyses place the number of lives lost at more than 130,000, with 1.5 million people immediately displaced following the earthquake.[1,2] More than 80% of Haitian schools and more than 50% of Haitian hospitals were destroyed.

Healthcare professionals from around the world recognized that a resource-limited nation such as Haiti would require an incredible amount of assistance to recover from the earthquake. Surgeons, anesthesiologists, and healthcare providers from the United States and other nations traveled to Haiti in order to provide initial trauma care, and subsequently to assist in backfilling the jobs of Haitian healthcare workers who were lost in the earthquake. Médecins Sans Frontières, or Doctors without Borders, stated in 2013 that its response to the Haitian earthquake was the largest relief operation in the history of the organization.

Hundreds of healthcare workers from around the world wanted to participate in the recovery effort from the earthquake, however a major limiting factor was the inability to access the sole airport in Haiti. The United States Air Force assumed leadership of the airport and provided air traffic control until Haitian authorities could recover adequately to resume control.[3] Additional immediate support came with the deployment of one of the two United States Navy Hospital ships, the USNS *Comfort*. The USNS *Comfort* and her sister ship, USNS *Mercy*, are enormous oil tankers that have been converted to hospital ships designed to deliver health care to U.S. military personnel during wartime. In reality, they are now more commonly deployed to provide humanitarian aid to disaster-ravaged areas of the world. In 2010, the USNS *Comfort* deployed to the coast of Haiti within 72 hours of the earthquake and subsequently embarked upon the largest disaster-relief operation in the history of these floating hospitals. More than 850 patients received care on the ship during the weeks following the earthquake, including 237 children and one premature newborn.[4] The most common condition treated on the ship was musculoskeletal extremity injury, composing about 40% of the overall reasons for admission.[5] The average length of stay was 8 days and surgery was performed 843 times on 454 patients over the course of 5 weeks. Fifty-eight of the operations were amputations.[6] Anesthesiologists from various U.S. hospitals provided care on the USNS *Comfort*, some as new volunteers and some with prior experience in military and disaster relief (Figs. 68.1 and 68.2).

Fig. 68.1 USNS *Comfort* off the coast of Haiti in 2010. (Photo courtesy Dr. Paul G. Firth, Massachusetts General Hospital, Boston, MA.)

Fig. 68.2 Patient ward on board the USNS *Comfort* in Haiti in 2010. (Photo courtesy Dr. Paul G. Firth, Massachusetts General Hospital, Boston, MA.)

Fig. 68.3 University of Miami anesthesiologist working in a makeshift operating room on the island of Haiti following the 2010 earthquake. (Photo courtesy Dr. Ralf E. Gebhard, University of Miami Department of Anesthesiology, Miami, FL.)

Provision of medical and surgical care aboard the floating hospital was relatively "normal" when compared with the task of providing care on the island of Haiti itself. Nearly all medical facilities in Port Au Prince were either damaged or destroyed by the earthquake, forcing volunteer healthcare teams to work in makeshift clinics, hospitals, and tent facilities throughout the city. One of the largest such facilities was based out of the United Nations compound at the airport.[7] In the initial days after the earthquake, surgical care was very limited because of a lack of supplemental oxygen, unsterile conditions, and a lack of resources to provide anesthesia. Amputations were performed under local anesthesia until a team of anesthesiologists skilled in single-shot block techniques arrived.[7] Following their arrival, these anesthesiologists were able to facilitate the performance of 1000 operations, including major orthopedic surgery. They were also instrumental in the postoperative care and analgesia of the surgical patients (Fig. 68.3).

Anesthesiologists who travel from resource-rich to resource-limited environments to provide rescue medical care must contend with a lack of familiarity with infectious diseases that have been eradicated from first-world nations. During the Haitian earthquake recovery, this situation confronted anesthesiologists who encountered patients suffering from tetanus. Although not completely eradicated from the United States, few anesthesiologists have any experience caring for patients with tetanus. Caused by the anaerobic bacterium *Clostridium tetani*, tetanus causes severe neck rigidity, trismus, and chest wall immobility. All of these problems create difficulties for anesthesiologists in terms of airway management and maintenance of ventilation. The Haitian population is at an elevated risk to develop tetanus following contamination of wounds because of relatively lower rates of tetanus vaccination compared with other nations. Anesthesiologists who provided care for surgical patients on the USNS *Comfort* reported two cases of tetanus that had impact on their anesthetic care.[8] In one case, the patient was noted to have a small mouth opening as a result of trismus. Following induction of general anesthesia with inhaled sevoflurane, the patient's total body rigidity was markedly improved. Unfortunately, after receiving multiple anesthetics with muscle relaxation, despite receiving appropriate care, the patient had persistent significant muscle weakness, retention of pulmonary secretions, and ultimately pneumonia resulting in his death. In another case, the patient received repeated general anesthetics, consisting of inhaled sevoflurane without neuromuscular blocking drugs. In this case, the patient fully recovered. The anesthesiologists involved in the care of these tetanus victims noted that though the mouth opening is limited, the nasal airway is unaffected by tetanus, so mask ventilation is possible despite the trismus. They also emphasized the importance of conserving medications such as neuromuscular blocking drugs when one is practicing medicine in a severely resource-limited environment so that the drugs are available for the patients most in need. By applying their expertise in pathophysiology and pharmacology, these anesthesiologists were able to safely and effectively deliver surgical anesthesia to high-risk patients without the use of neuromuscular blocking agents by taking advantage of the neuromuscular blocking properties of potent inhaled anesthetic agents.

The Haitian earthquake response highlighted another critical issue associated with all major disasters: the psychological impact on healthcare workers caring for victims. After returning to the United States following humanitarian missions to Haiti, several anesthesiologists wrote thoughtful essays on the care that they had provided.[9] Anesthesiologists who travel to disaster-ravaged areas are exposed to a variety of physical and mental stressors that can have an impact on them for months or years after the event. Prolonged work hours, inadequate rest, exposure to gruesome traumatic injuries, and witnessing the prolonged pain and suffering of children are some examples of what disaster responders endure. Despite these challenges, healthcare providers are expected to provide optimal medical care, while simultaneously providing psychological support to devastated family members. During the busy hours and days of the immediate response to a disaster, healthcare providers stay focused on the call of duty, which allows them to keep personal emotions at bay. As the critical needs resolve, anesthesiologists and other healthcare providers are forced to deal with the physical, mental, and emotional trauma that they themselves have sustained. One might wonder why healthcare providers would leave the safety of their home nation to purposely expose themselves to this kind of potential anguish. Consistent qualities appear to be present among responders to massive natural disasters: they understand that all human beings have dignity, they sympathize with people who are suffering, and they desire to be part of the healing process.

TORNADOES

While tornadoes tend to be shorter in duration than other natural disasters, they are able to create horrendous damage within seconds to minutes. One of the most destructive storms in American history was the tornado rated EF-5 on the Enhanced Fujita scale that ravaged Joplin, Missouri on May 22, 2011. During the ensuing power outage, some hospitals' backup generators failed, requiring anesthesia care and surgery to be performed by flashlight alone.[10] When hospitals are directly destroyed by natural disasters, as occurred at St. John's Mercy Medical Center in Joplin, one of the biggest challenges for the community is the need to restore the healthcare infrastructure, including access to the electronic health record. With proper disaster preparedness, including contingencies for complete losses of hospitals and clinics, normal patterns of care can be restored within a reasonable timeframe.[11]

Opportunistic infections are another common downstream effect from all natural disasters, including tornadoes. Following the 2011 tornado in Joplin, a cluster of patients suffering from necrotizing cutaneous mucormycosis was observed and required extensive treatment.[12] Anesthesia providers are critically important during "outbreaks" such as this since emergency surgeries to control rapidly expanding infectious diseases are critical to patient survival (Figs. 68.4 and 68.5).

Fig. 68.4 Necrotizing cutaneous mucormycosis of the flank following the Joplin, MO tornado. (From Neblett Fanfair R, Benedict K, Bos J, et al. Necrotizing cutaneous mucormycosis after a tornado in Joplin, Missouri, in 2011. *N Engl J Med.* 2012;367[23]:2214–2215. Published by the Massachusetts Medical Society.)

TSUNAMI

A tsunami is a very large sea wave that results from a submarine earthquake, volcanic eruption, or other significant geologic movement. On December 26, 2004, a massive earthquake, magnitude 9.1 on the Richter Scale, occurred beneath the Indian Ocean and caused one of the deadliest natural disasters in recorded history. The tsunami that resulted from the earthquake created enormous waves that made their way to the shores of 14 countries, including: Indonesia, Sri Lanka, Malaysia, Bangladesh, India, Thailand, and Myanmar. Tsunamis produce a very high ratio of mortality to injury. These waves result from the influx of water on the shores of victim countries causing the immediate death of many people secondary to drowning or the direct impact of waves or debris. The aftermath of a tsunami tends to have three phases. During phase 1, a high number of people are instantly killed. During phase 2, in the hours and days following the initial impact, relief healthcare workers are able to provide life- and limb-saving care to victims of blunt trauma and water exposure.[13] During phase 3, recovery is impeded by a lack of healthcare personnel and a lack of infrastructure needed to provide longer term posttrauma care.

The 2004 tsunami killed more than 230,000 people, injured tens of thousands, and was estimated to have immediately displaced more than 5 million people. One factor in its extreme lethality was the fact that December is the peak tourist season for many of the beaches of Southeast Asia. As is the case with many tsunamis, there was essentially zero warning that deadly waves were about to make landfall. The Aceh province of the Indonesian island of Sumatra was by far the hardest hit area, with waves reaching greater than 25 m in height and with over 100,000 people killed. People on the beach at the time of the tsunami were killed directly, while people farther inland were killed as a result of drowning or impact with massive floating debris (Fig. 68.6).

While there is essentially no role for relief healthcare workers during phase 1 after a tsunami, healthcare workers from nearby regions can and should respond to care for patients during phase 2. Following the 2004

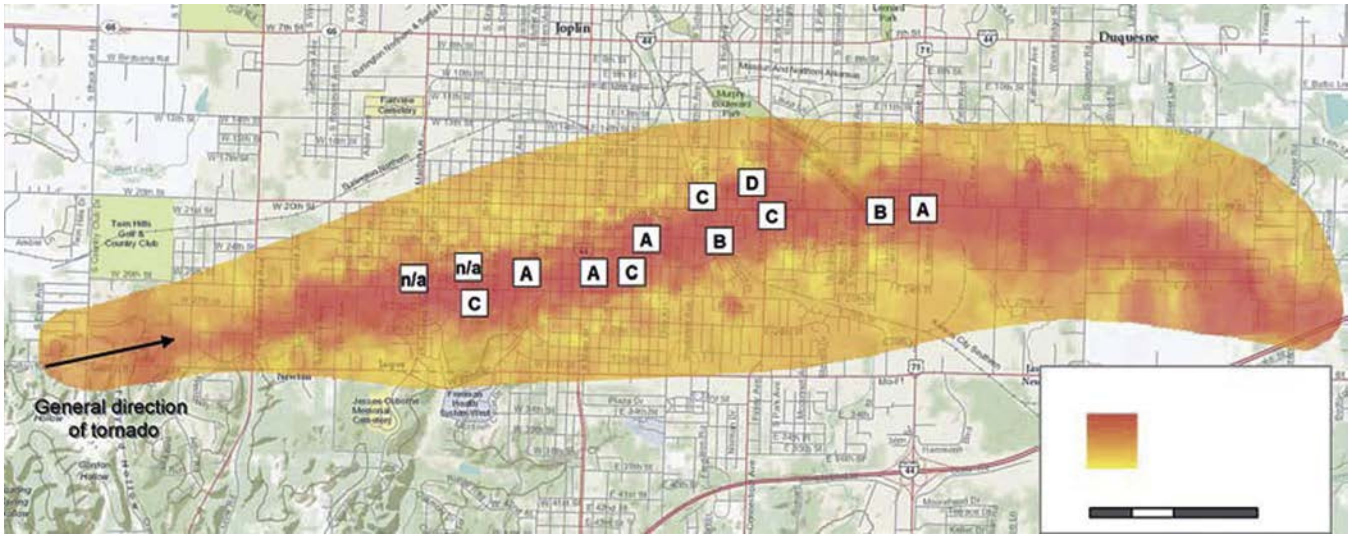

Fig. 68.5 Case locations of mucormycosis with relation to the Joplin tornado course. (Data are from the US Army Corps of Engineers and Esri. From Neblett Fanfair R, Benedict K, Bos J, et al. Necrotizing cutaneous mucormycosis after a tornado in Joplin, Missouri, in 2011. *N Engl J Med.* 2012;367[23]:2214–2215. Published by the Massachusetts Medical Society.)

Fig. 68.6 Debris in the streets of the Aceh province after the December 2004 tsunami. (Photo courtesy Dr. Michael G. Fitzsimons, Massachusetts General Hospital, Boston, MA.)

tsunami, a team of 17 surgeons, 6 anesthesiologists, and other healthcare workers traveled as part of the Thai Red Cross Society for humanitarian mission from Bangkok, Thailand to Phang-Nga, Thailand to provide care. The multidisciplinary team was led by one surgeon and one anesthesiologist. They arrived just 1 day after the tsunami. Over the course of 3 days, the team provided surgical care for 107 patients. The injury profile consisted predominantly of soft-tissue wounds and bone fractures. Halothane was the only inhalational anesthetic available, highlighting the need for anesthesiologists to be nimble in their ability to use older medications during disaster relief.

Most surgical procedures performed in Phang-Nga included general anesthesia following rapid sequence induction. Spinal anesthesia, regional anesthesia, and local anesthesia were also utilized for selected cases. The providers noted an increased incidence of intraoperative oxygen desaturation as compared with findings during other relief operations. The cause for the desaturation is thought to be related to aspiration of seawater and lung contusion secondary to direct impact of waves or debris. In addition to the challenges associated with performing procedures near sites of destruction, providing care can be challenging even if it is being provided remotely relative to the tsunami itself. One common problem is the lack of access to laboratory services. As in post-earthquake anesthetic care, the anesthesiologist may be required to rely on clinical judgment without additional laboratory data to guide decision making in the administration of fluids, electrolytes, antibiotics, and blood products after a tsunami or other natural disaster (Fig. 68.7).

In addition to the regional response, a massive international relief effort was launched in response to the tsunami. In January 2005, Operation Unified Assistance was started as part of the United States response to the catastrophe. Organized by Project HOPE and the U.S. Public Health Service, the USNS *Mercy* was sent from San Diego, California to the Aceh province in Sumatra, representing the first joint effort of military and civilian relief aboard a U.S. Navy ship.[14] U.S. responders to Indonesia were required to demonstrate up-to-date vaccination records for all typical immunizations, receive vaccines for typhoid and hepatitis A, and take prophylactic medication to protect from malaria. Additionally, negative airflow pods were present on the ship to allow for safe care of patients with active tuberculosis. Prior to arrival, all staff members were briefed on the differences in culture they would encounter, and the possible implications for providing care. More than 90% of Indonesian citizens are Muslim, and Islamic Law plays an important role in the day-to-day decision making, including consent for treatment, end-of-life procedures, and other issues for many of the families impacted by the tsunami.

Another serious outcome after a tsunami is often the loss of life of healthcare workers. An estimated 60% of all healthcare workers in the Aceh province died or were

Fig. 68.7 **Aceh, Indonesia operating room destroyed by December 2004 tsunami.** (Photo courtesy Dr. Michael G. Fitzsimons, Massachusetts General Hospital, Boston, MA.)

Fig. 68.8 **Satellite photo of Hurricane Katrina over the Gulf of Mexico.** (Used with permission from the National Oceanic and Atmospheric Administration, National Environmental Satellite, Data, and Information Service. Available from www.nesdis.noaa.gov. Accessed October 26, 2018.)

missing following the tsunami. Thus, Operation Unified Assistance was prepared to backfill the roles of lost healthcare workers in addition to providing care directly related to the effects of the tsunami. While the USNS *Mercy* was designed for enormous overall capacity, for this operation its clinical footprint consisted of staffing for 3 operating rooms and 50 inpatient beds.

Operation Unified Assistance provided surgical care for 154 patients, but only 8.4% of these were in need of surgery as a direct result of the tsunami.[14] Because of the high mortality to injury ratio in a tsunami, the critical need in Aceh was to compensate for the loss of healthcare workers rather than require large numbers of providers to support patients needing surgical procedures. This differentiates the tsunami experience from the Haitian earthquake experience, with its higher relative incidence of survivable injuries.

For those patients requiring surgery, general anesthesia with inhaled agents supplemented by opioids and neuromuscular blocking drugs was the anesthetic technique of choice. Although access to equipment appropriate for regional anesthesia was limited, one of the primary reasons for this approach was related to the significant language barrier between patients and anesthesiologists. In addition, the clinicians felt postanesthesia recovery from general anesthesia would be easier and safer than for neuraxial or regional, considering all of the circumstances. Another commonly needed and often limited resource includes blood and blood products. In this situation, a total of 122 units of packed red blood cells, 13 units of plasma, and 4 units of cryoprecipitate were transfused during the relief operation.[14] While response to a tsunami has some similarities to the response to a large earthquake, the presence of a modern floating hospital allows the anesthesiologist to employ general anesthetic techniques much more readily than in a tent-based operating suite during inland recovery from an earthquake. At the conclusion of Operation Unified Assistance, a portable monitor and anesthetic medications were donated by the team to the local healthcare facility.

HURRICANES

A hurricane is a tropical storm with maximum sustained winds of 74 mph (119 km/h) or higher. Hurricanes have the potential to disrupt the normal function of the healthcare system in a variety of ways. When hurricanes reach landfall, the high wind speeds and excessive rainfall make any outdoor movement unsafe, thus paralyzing emergency medical systems and preventing patients from seeking necessary care. With major hurricanes, extensive flooding is a common complication because of the large volumes of rainfall combined with seawater entrained onto land as part of the storm surge. These storms have fewer immediate fatalities than earthquakes or tsunamis, however the ability of hurricanes to cripple the provision of medical and surgical care has numerous downstream consequences. In this section, Hurricane Katrina will be examined as an example of the destructive forces of hurricanes in general.

In August 2005, Katrina started as a storm over the Bahamas, then gained strength over Florida before reaching the Gulf of Mexico. Over the Gulf of Mexico, Katrina reached Category 5 status with top wind speeds of 175 mph. At the time of landfall, the storm was a category 3 hurricane. It produced 8 to 14 inches of rain over southern Louisiana, Mississippi, and Alabama. At some sites on the Mississippi coast, the storm surge was 25 to 28 feet higher than normal tide level and destroyed the majority of structures in its path. In New Orleans, Louisiana, the storm surge sent water over the levees protecting the city, causing flooding in 80% of the city and the need for widespread evacuation. Hurricane Katrina remains the costliest storm in the history of United States at greater than $100 billion in losses, and it was the deadliest hurricane since 1928 (Fig. 68.8; Table 68.1).[15]

The flooding and subsequent displacement of thousands of New Orleans inhabitants following Hurricane Katrina highlight one of the key elements of major natural disasters. In addition to the impact of the hurricane on the healthcare delivery infrastructure, a storm of this magnitude is capable of completely disrupting medical education at the undergraduate and postgraduate levels. New Orleans is home to two major medical schools with

TABLE 68.1 Saffir-Simpson Hurricane Wind Scale

Category	Sustained Wind Speeds	Damage Potential
1	74-95 mph 119-153 km/h	**Very dangerous winds will produce some damage:** Well-constructed frame homes could have damage to roof, shingles, vinyl siding, and gutters. Large branches of trees will snap, and shallowly rooted trees may be toppled. Extensive damage to power lines and poles likely will result in power outages that could last a few to several days.
2	96-110 mph 154-177 km/h	**Extremely dangerous winds will cause extensive damage:** Well-constructed frame homes could sustain major roof and siding damage. Many shallowly rooted trees will be snapped or uprooted and block numerous roads. Near-total power loss is expected with outages that could last from several days to weeks.
3	111-129 mph 178-208 km/h	**Devastating damage will occur:** Well-built framed homes may incur major damage or removal of roof decking and gable ends. Many trees will be snapped or uprooted, blocking numerous roads. Electricity and water will be unavailable for several days to weeks after the storm passes.
4	130-156 mph 209-251 km/h	**Catastrophic damage will occur:** Well-built framed homes can sustain severe damage with loss of most of the roof structure and/or some exterior walls. Most trees will be snapped or uprooted, and power poles downed. Fallen trees and power poles will isolate residential areas. Power outages will last weeks to possibly months. Most of the area will be uninhabitable for weeks or months.
5	157 mph or higher 252 km/h or higher	**Catastrophic damage will occur:** A high percentage of framed homes will be destroyed, with total roof failure and wall collapse. Fallen trees and power poles will isolate residential areas. Power outages will last for weeks to possibly months. Most of the area will be uninhabitable for weeks or months.

Adapted from the National Hurricane Center of the National Oceanic and Atmospheric Administration. Available from: www.nhc.noaa.gov accessed October 30, 2018.

accompanying academic and clinical enterprises. Immediately following Hurricane Katrina, the academic and clinical buildings at Tulane University School of Medicine and Louisiana State University (LSU) School of Medicine were both badly damaged by flood waters.[16] Tulane University collaborated with Baylor University College of Medicine in Houston, Texas to use facilities for its medical student programs. LSU utilized facilities on its flagship campus in Baton Rouge, Louisiana for preclinical classes. Housing for students was a challenge, and most Tulane students were hosted by members of the Houston community, while LSU students either found their own housing in Baton Rouge or stayed on a large ferry coordinated by the Federal Emergency Management Agency. Remarkably, both Tulane and LSU were able to resume medical education within 4 weeks of the hurricane.[16]

Relocating preclinical students proved to be easier than identifying suitable clinical education sites for third and fourth-year medical students. Both schools were forced to find new clinical sites within Louisiana and beyond. Compounding the challenge was the closure of the damaged Veterans Affairs Medical Center in New Orleans. This degree of disruption had an impact on the medical school admissions process for both schools, yet both schools were able to enroll incoming classes very much on par with prior years. With regard to the residency match process, Tulane did experience a slight increase in the number of unmatched medical school graduates, likely owing to the reduction in residency spots at Tulane after the hurricane.[16] Despite all of these formidable challenges, both Tulane and LSU were able to keep their entire medical school enterprises alive, though with some impact on faculty needs. Both institutions were able to improve several physical and academic aspects of their campuses in the years following Katrina. In a sense, these two universities have provided other academic centers with a road map for how to continue to thrive following the harrowing experience of an all-encompassing natural disaster. Their perseverance was essential to the future health care of patients in Louisiana and beyond.

While the medical schools' survival was a triumph, Hurricane Katrina and subsequently Hurricane Rita had a distinctly negative effect on the field of anesthesiology in Louisiana. The two largest teaching hospitals in New Orleans closed after Katrina, and the largest of these—Charity Hospital—never reopened. These closures and other factors led to a decline in the number of anesthesiologists, as documented by a 2006 statewide survey.[17] Statewide residency positions in anesthesiology fell from 24 graduates per year in 2004 to 13 graduates per year in 2007. Like many states, Louisiana is fairly dependent on new anesthesiology graduates remaining in the state to take the place of retiring physicians. Of the respondents, 37% of anesthesiologists reported that they had difficulty filling open positions within their group. In this setting, 92% reported that they had experienced an increase in the number of daily cases, and that obstetric cases for patients with no prenatal care had risen out of proportion to other cases. This phenomenon likely represents a glimpse into the impact of natural disasters on all medical specialties: the same stresses experienced by anesthesiology programs were felt by other residency programs, including the obstetrics and gynecology program. Compared with the complete annihilation of medical infrastructure caused by the earthquake in Haiti, the plight of post-Katrina New Orleans may seem insignificant. Still, the fact that a large city within the wealthiest nation on earth can have its medical infrastructure completely disrupted for months after a storm attests to the impact of large-scale natural disasters.

In September 2017, Hurricane Maria struck Puerto Rico and effectively interrupted the island's access to clean

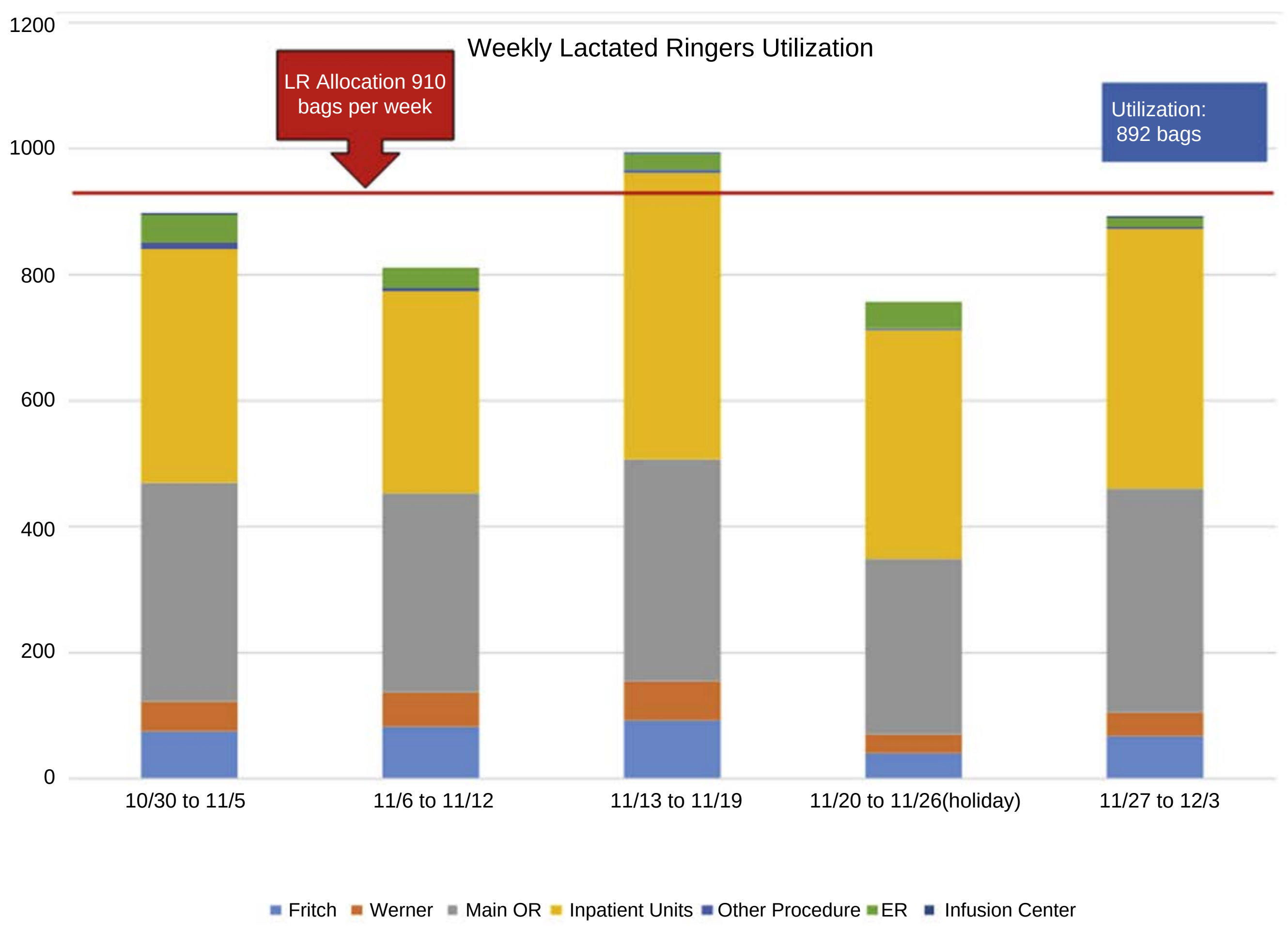

Fig. 68.9 UNMC tracking utilization of lactated Ringers during crystalloid shortage from Hurricane Maria.

water, electricity, telecommunications, and transportation. At the time Maria made landfall on Puerto Rico, it had slightly decreased in intensity from a Category 5 to a Category 4 hurricane, yet it damaged or destroyed nearly every structure in its path and the death toll is estimated by different agencies at between 1000 and 2800.[18,19] Drug and fluid shortages in the continental United States following Hurricane Maria exposed a major vulnerability of the overall healthcare system, and provided anesthesiologists with an opportunity to help lead the way out of a potential nationwide crisis.

Puerto Rico is a massive producer of pharmaceuticals and medical devices. Baxter, a large multinational company had factories in Puerto Rico, producing approximately 50% of all of the 0.9% normal saline bags used each day in U.S. hospitals.[20] The shutdown of these factories, and all other major pharmaceutical factories in Puerto Rico, resulted in an immediate shortage of fluids and medications throughout the United States. The initial response at many mainland hospitals was to identify alternative sources for products in other countries. This approach simply expanded the shortages of fluids and medications internationally.

The experience with shortages of supplies and materials made healthcare leaders recognize the interdependence of all health systems and the importance of conservation of existing supplies and avoidance of waste. In response to the shortages created by the events in Puerto Rico, anesthesiologists at many medical centers were asked to help create strategies to maximize the efficient use of fluids and medications. At the University of Nebraska Medical Center, anesthesiologists worked with other physicians, pharmacists, and leaders in nursing and administration to cocreate robust strategies to avoid critical shortages of necessary fluids and medications. Figs. 68.9 and 68.10 offer a glimpse at some of these strategies. Note that intravenous fluids administered in the operating rooms were required to be on an infusion pump, a major change from the typical "gravity drip" employed by anesthesia providers. The shortages forced healthcare workers across the nation to (1) think more critically about every milliliter of fluid they give to patients, and (2) use different fluids and medications than what they use during times of nonshortage. While the shortages induced by Hurricane Maria were problematic, their net effect has likely been toward positive changes within the U.S. healthcare system. In the aftermath, many have come to realize that our excessive reliance on one U.S. territory for fluids and pharmaceuticals is neither wise nor sustainable.

Section 2: Acts of Terrorism

SEPTEMBER 11, 2001 ATTACKS

On September 11, 2001, an organized team of terrorists commandeered three U.S. airliners with the intention of crashing them into prominent buildings in New York City and Washington, DC. Two of the aircraft were flown directly into the twin towers of the World Trade Center in New York, causing massive fires and eventually causing both of the 110-story buildings to collapse. Nearly 3000 people died as a result of the September 11th attacks, making it the deadliest act of terrorism in recorded history.

An anesthesiologist-intensivist from New York University, Dr. J. David Roccaforte, was on duty at Bellevue Hospital 2.5 miles away from the site of the attack. Dr. Roccaforte's paper describing the hours and days following the 9/11 attack remains essential reading for healthcare professionals in disaster preparedness.[21] In the paragraphs that follow, the key points from this important paper are summarized.

Hospital phone lines were nonfunctional in the hours following the attack, prompting a recommendation that hospitals be equipped with radio communication equipment and satellite-based communication devices. While cellular communication has clearly improved many-fold between 2001 and 2018, it is difficult to predict the performance of modern networks in a situation where millions of users are simultaneously trying to connect with other users. Based on an assumption that hundreds of victims would be arriving rapidly to the operating rooms and EDs, thousands of dollars worth of fluids, medications, and kits were opened and prepared, much of which went to waste. In retrospect, these resources should have been used only with confirmation of patient need. Considering the number of capable hospitals in New York outside of lower Manhattan, a pre-made plan to triage patients from Bellevue and other nearby hospitals out to other trauma centers would have been beneficial. A field hospital was set up in a warehouse near the World Trade Center, intended to be a site for emergency trauma surgery in the event that hospitals were overwhelmed. Unfortunately, the 100-bed field hospital lacked sufficient resources for the provision of anesthesia, and thus was not entirely usable.

Due to the lack of functional telecommunications, medical students were employed as runners and assigned to attending physicians. All workers were encouraged to wear a label on their shirt with name, specialty, and title, to expedite face-to-face communication. While the 9/11 attack did not induce a major failure of the physical plant at Bellevue, the need for hospital engineers was noted. In the event of a failure of electrical or oxygen supply, head engineers would be needed to restore normal state or provide a contingency plan. A traditional triage system using color-coding was used effectively for incoming patients: green signified non-urgent; yellow for potentially urgent; and red for immediate, life-threatening injuries. Any patient in the yellow category with potential need for airway management or sedation was assigned a senior anesthesiology resident or a critical care fellow. As the initial surge of patients slowed, the team at Bellevue wisely mandated a shift system, sending healthcare workers home to rest and avoid the certain burnout that results from ongoing 24/7 care following a major disaster.

Fig. 68.10 Partial timeline of fluid conservation strategies at UNMC after Hurricane Maria.

MASS SHOOTINGS

In the United States, mass shootings continue to increase in frequency and severity. The Centers for Disease Control and Prevention (CDC) has not been allowed to investigate gun violence as a matter of public health, which has hampered high quality research on this topic. Using a definition of a mass shooting where four or more people are killed by a lone shooter, from 1966 through November 2018 there have been 158 mass shootings in the United States.[22] No region appears to be safe from these events, as they have occurred in schools, churches, offices, and military bases in all parts of the country. The number of people killed in these mass shootings is 1135, with 186 of them being children or teenagers.[22] Accordingly, all anesthesiologists must be prepared to provide care in the aftermath of these heinous events.

The lethality of these attacks appears to be increasing with each passing year, in part because more and more of these shootings are carried out with military-style semiautomatic rifles as opposed to conventional guns. The AR-15 has emerged as the weapon of choice for the criminals who perpetrate these senseless acts. Following the Parkland, Florida, shooting where 17 people were killed by a former student at Marjory Stoneman Douglas High School in February 2018, a radiologist with many years of experience diagnosing gunshot wounds in a Level 1 trauma center penned an article about the drastically different injury pattern visualized with gunshot wounds from an AR-15 versus those from a typical handgun.[23] With a normal gunshot wound, the radiologist can trace a laceration through affected organs that is roughly the same width as the bullet itself. Following the Parkland shooting with an AR-15, radiologists and trauma surgeons observed that tissue had been destroyed in wide swaths near the pathway of bullets. Organs were found to be "smashed" and "shredded," where traditionally they are found to be merely lacerated following handgun injuries. This extensive damage to surrounding structures occurs as a result of the much higher bullet velocities and thus higher energy levels transmitted by assault rifles as compared with typical handguns.

On October 1, 2017, the worst mass shooting in the history of the United States took place in Las Vegas, Nevada. A lone gunman armed with multiple rifles fired more than 1000 rounds into a large music festival crowd on the Las Vegas strip. His position on the 32nd floor of a nearby hotel and his modification of semiautomatic weapons into automatic weapons likely increased the terrible lethality of the attack. Fifty-eight people were killed and more than 400 were injured by gunfire that night.

In the hours immediately following the mass shooting in Las Vegas, dozens of anesthesiologists were called to respond and provide care for the victims. A friend and colleague, Dr. Devin Kearns, describes the harrowing night:

"I was at home on call for pediatric anesthesia for the Level 1 trauma center here in Las Vegas. Shortly after falling asleep I was awoken by the phone ringing and the familiar voice of my partner. However, this call was anything but routine as he proceeded to say there had been a mass shooting.

The next thing I remember was jumping out of bed while telling my wife there had been a shooting on the strip. As I drove to the hospital I was listening to the radio for updates, not knowing what to expect. Upon arrival I was asked to go to OR 2 and set up for a critically injured, hemodynamically unstable patient who would be arriving shortly. As I checked the machine and prepared medications the situation still seemed surreal. We see a lot of penetrating and non-penetrating trauma at our facility. It is simply part of what we do. But could anything have prepared me for a mass casualty shooting?

As I waited for this critical patient to arrive, the time continued to pass. About 5 or 10 minutes later I called the front desk for an update… the patient didn't survive.

I then went to the preoperative area and as I entered I witnessed a scene I will never forget. It was a scene I had not prepared for, and a scene I never expected to witness on my soil as I am not a member of the military. I saw multiple fatally wounded patients with their loved one by their side hoping, praying, and longing for the moments prior to the terror and fear they experienced at this senseless act.

Additional victims were triaged as they arrived. We cared for many of them in the operating room and this continued for the days and weeks to follow. These victims were not merely wounded physically, but emotionally, and spiritually as well."

Dr. Sher's and Dr. Kearns' personal accounts of caring for mass shooting victims serve as a reminder to all anesthesiologists. We all must be prepared to provide optimal care for shooting victims with injury patterns that once were limited to military settings. Given the devastating tissue injury induced by assault rifles such as the AR-15, special consideration should be given to obtaining additional vascular access before or during surgery, and to requesting additional blood products prior to losing the tamponade effect following the opening of the abdomen or the chest.

BOSTON MARATHON BOMBING

On April 15, 2013, two homemade bombs made of pressure cookers filled with pellets and nails were detonated 12 seconds apart near the finish line of the Boston Marathon. The unusual nature of the bombs in this incident caused an unusual injury pattern: 3 victims were killed by the blasts and 264 were injured, 66 of whom sustained lower extremity injuries.[24] The close proximity of multiple major trauma centers to the site of the bombing likely played a role in reducing the mortality rate of this event. Despite a collaborative effort among the Boston city trauma centers, EDs were overwhelmed when 78% of the patients treated at city trauma centers arrived within 90 minutes of the initial event. Of these high acuity patients, 45 were admitted directly to operating rooms, 11 to ICUs, and 12 required emergency airway management.[25] Of the 127 patients receiving care at Boston's trauma centers, over 100 likely had some facet of their immediate care provided by an anesthesiologist. The unique perspective and skills of the anesthesiologists allowed them to provide a wide variety of clinical services within what, in fact, served as the epicenter of terrorism disaster management.

Unlike other terror attacks in the United States, the bombs discharged in Boston caused injuries similar to those observed in the Afghanistan and Iraq wars, caused by improvised explosive devices (IEDs). This injury pattern in wars has prompted a resurgence of interest in the use of tourniquets in the field to prevent exsanguination from major injuries of the extremities.

Of the 66 patients with extremity injuries, 29 of them had life-threatening bleeding at the point of injury.[24] Twenty-seven of these 29 had a tourniquet applied in an attempt to reduce bleeding. All of the 27 tourniquets were improvised, that is, they were not commercially produced tourniquets designed expressly for the purpose of arterial occlusion. The most common type used was rubber tubing wrapped around the extremity combined with a Kelly clamp. Sixty-three percent of the tourniquets were applied by non-emergency medical service (EMS) personnel, and several were not applied tightly enough to actually occlude arterial flow. Experts in battlefield trauma care have discussed the use of tourniquets after the Boston Marathon bombing and have suggested an educational campaign to teach the correct technique for application of purpose-driven tourniquets, and suggested that all EMS providers be provided with these devices. Anesthesia providers have extensive experience with the proper application of arterial tourniquets, as a result of their widespread use during orthopedic operations, so their role is critically important in reducing blood loss and saving lives. In the event of another mass casualty event resulting from IEDs, anesthesiologists, certified registered nurse anesthetists, anesthesia assistants, and anesthesiology residents should instruct healthcare workers and volunteer first aid workers on the application of tourniquets. In the probable absence of purpose-driven commercially produced devices, emergency medicine, trauma, and anesthesia providers should inspect applied tourniquets to ensure they are applied in a manner that will occlude arterial flow.

The two perpetrators of this vicious crime were eventually apprehended and taken to a hospital with severe injuries. One died from his injuries while the other survived. This scenario brings to light one of the challenges of working as an anesthesiologist following a terrorist attack: the physician must continue to provide the best care possible even when the patient has done tremendous harm to other human beings. In situations like this, the physician benefits from focusing on the anatomic and physiologic data at hand, while ignoring the surrounding emotional elements. All physicians must remember their oath to "do not harm," under any circumstances.

Section 3: Chemical, Biological, Radiological, and Nuclear Warfare

In today's global political environment the threat of a chemical, biological, radiological, or nuclear (CBRN) attack combined with massive casualties cannot be disregarded. Many people assume that sophisticated and organized individuals initiate these types of attacks; this is not generally the case. Breaking CBRN into two groups, we can see that chemical and biological are more common than radiological and nuclear incidents, which in recent history have been a direct result of natural disasters. Regardless, medical professionals must address, prepare for, and train for these types of disasters and attacks to better care for those negatively impacted. After reviewing the history of CBRN warfare, this section will analyze, define, demonstrate, and outline the U.S. military's approach to CBRN protocols, procedures, and standard operating procedures (SOPs). Using the structure, function, and focus of the U.S. military's approach to CBRN will help delineate the anesthesiologist's role in the civilian sector to help improve patient care while minimizing collateral damage and casualties.

The U.S. military has established a hierarchy, structure, and SOPs for CBRN situations. As acts of terrorism have become more aggressive in recent decades, civilians may be more likely to see the effects and aftermath of a CBRN act of terrorism. Figs. 68.11 and 68.12 outline the Department of Defense (DOD) strategy and hierarchical structure for CBRN attacks on domestic soil, military installations, and abroad.[26] Attempting to adapt the military's training protocols may help reduce unrest and exemplify the vital role of the consultant anesthesiologist during a CBRN attack.

An increased public awareness of possible CBRN attacks by terrorist groups started in the fall of 2001, when the biological agent, anthrax powder, was mailed to two U.S. senators at their congressional offices in Washington, DC. Civilian medical responders were forced to deal with and experience the toxic effects. As these CBRN terrorist threats have become more apparent toward the civilian populations, anesthesia providers have become more involved with on-site and prehospital medical management. Understanding the toxic effects and pathophysiologic effects of CBRN agents can help reduce the collateral damage to first responders. Anesthesia providers are essential in resuscitation efforts at the scene of an attack and with continued life support measures during sustained CBRN and terrorist attacks.

HISTORICAL ASPECT OF CHEMICAL, BIOLOGICAL, RADIOLOGICAL, AND NUCLEAR WARFARE

Chemical, biological, radiological and nuclear warfare is deeply embedded in the history of world conflicts. The search for tactical wartime and war-fighting advantages have been documented since ancient Babylon. Chemical weapons can be traced back to 10,000 BC when the San society of Southern Africa used natural poisons from snakes on the tips of their spears to hunt antelope. One of the first suspected uses of chemical agents in war can be found between the Persians and Romans in the year 256.[27] According to archeologic evidence, the Persian military exposed Roman soldiers to a noxious gas, killing them before ever having to face them in battle. During the conflict between the Romans and Persians, it was noted that the Persians used a suspected sulfurous gas in a tunnel, quickly killing 20 Roman soldiers by asphyxiation. This primitive form of combusting sulfur crystals in a confined space may have been the very first chemical or biological material employed as an offensive weapon during a conflict. This was centuries before the indoctrination of the Geneva Protocols in 1925, which outlawed the use of weaponized chemicals during wartime.

Biological weapons, and eventually nuclear weapons, have evolved with advancing technologies. In fact, World War I saw the overutilization of chemical agents as offensive weapons. These chemical agents included phosgene gas, chlorine gas, and mustard gas during the infamous trench

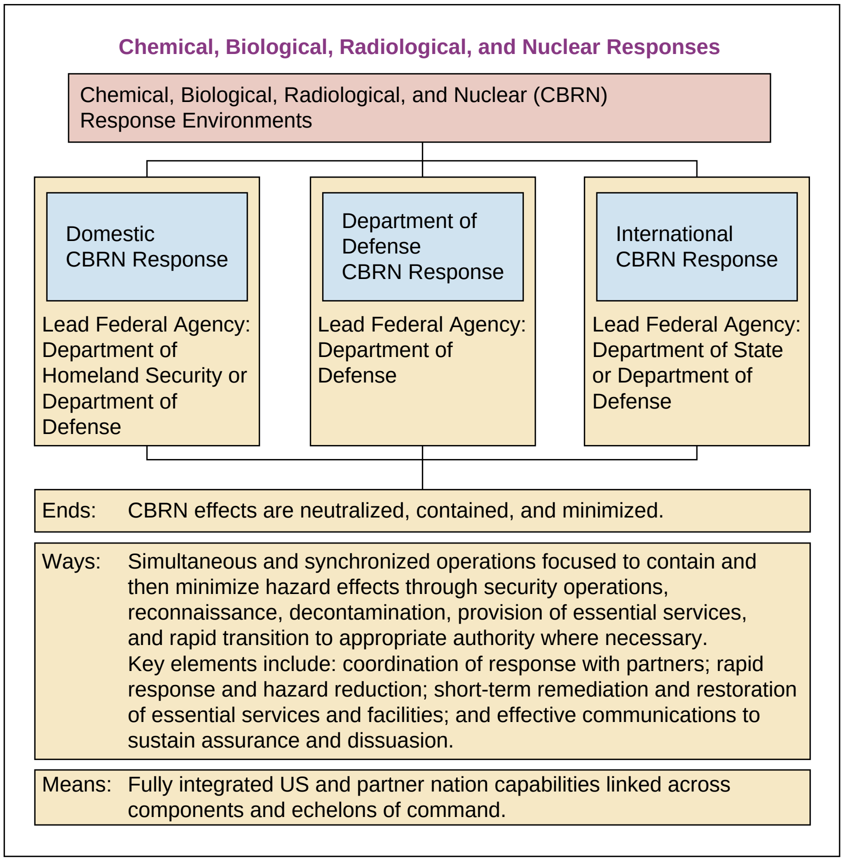

Fig. 68.11 U.S. Dept. of Defense CBRN Response table. (Adapted from Joint Publication 3-41, Chemical Biological, Radiological and Nuclear Response, September 9, 2016.)

warfare encounters between 1915 and 1918. Chemical weapons were very effective and the release of these gases during battle had substantial impact and accounted for a large number of casualties (Figs. 68.13 to 68.15). These heinous attacks against unprepared troops led to chemical and biological weapons being considered weapons of *mass destruction* by the British government, because they led to catastrophic and painful loss of life. During World War I, more technologically advanced chemical and biological weapons continued to be used, resulting in approximately 1.3 million casualties related to these explosive devices.[28] This is why many historians consider World War I as the "Chemist's War." The toxic effects of chemical weapons during World War I were compounded by the fact that many of these weapons were combined with ballistic explosive shells to increase lethality of the chemicals. The combination of toxin and ballistic injuries led to compounding casualties on and away from the battlefield, as a consequence of the crippling nature of the chemical weapons on those that survived the chemical attacks.

There have been historical attempts to prevent and outlaw these weapons of *mass destruction* due to a review of legal implications of chemical and biological weapons. It started in 1899 with the Hague Declaration Concerning Asphyxiating Gases and again in 1907 at the Hague Convention on Land Warfare, which actually outlawed the use of poison or poisoned agents during wartime.[29] In this regard, the actions and implementation of chemical and biological agents during World War I violated these regulations. However, the landmark international regulation of 1925 in Geneva outlined, outlawed, and made the use of chemical weapons a crime against humanity, which nearly led to a cessation of chemical and biological weapons development as history moved forward toward World War II.

Under very tight secrecy, a major discovery by the German scientist Gerhard Schrader in 1936 led to the invention of anticholinesterase nerve agents. These nerve agents were not utilized in World War II, but they became more of a threat afterwards. In 1945, the former Soviet Union captured a major nerve agent manufacturing plant, subsequently giving the Soviet Union not only nuclear capabilities but biochemical weapons for mass destruction.[30] The most unfortunate aspect of the Soviet seizure of this manufacturing plant was that the Soviet military leaders began to see chemical, biological, and nerve agents as normal armaments for war. This philosophy was contrary to the Geneva convention and the worldwide ban on chemical and biological weapons.

Several other countries have been implicated in secret development and advancement of chemical and biological weapons. Most notably, the Chinese and Japanese governments denied reports of developing weapons of mass destruction, but since 1930 they have been suspected of researching and manufacturing chemical and biological weapons.[31] Unfortunately, these biological agents were tested on prisoners and the general population without proper consent, which is considered a crime against humanity under the Geneva Conventions.[32] This secret

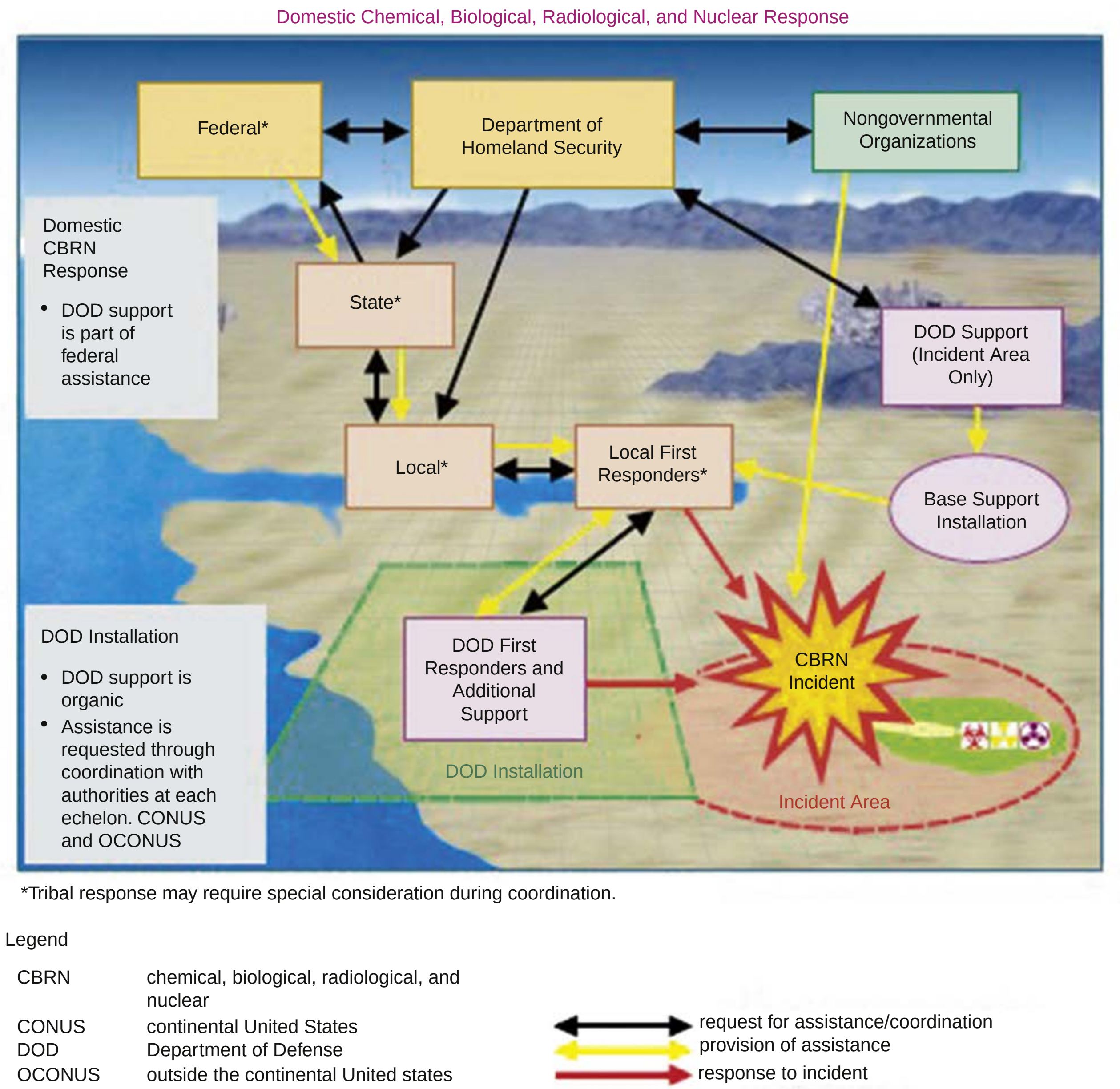

Fig. 68.12 U.S. Dept. of Defense CBRN Response Graphic. (Adapted from Joint Publication 3-41, Chemical Biological, Radiological and Nuclear Response, September 9, 2016.)

testing went on for years without notice by other world powers. Even though this is a vile and awful act against the general population, substantial research data were collected. After the capture of one of the project leaders by the United States, considerable intelligence was passed to the federal government regarding this chemical and biological research. This formed the basis for the United States' offensive biological and chemical weapons research and development as a response to other countries stockpiling these weapons. However, the Biological Weapons Convention in 1972 put an effective stop to worldwide research and stockpiling of chemical, biological, and nerve agents for weaponization.[33]

Despite the 1972 Biological Weapons Convention putting an enormous price on continued chemical, biological, and nerve agent research, the Soviet Union continued to manufacture and stockpile weapons of mass destruction. The Soviet biological warfare program hit a peak in the mid-1970s, when they developed a whole new type of biological weapon and weapon systems. The former Soviet Union's government denied these programs existed and the fact that these weapons' systems had been deployed around the world as a first strike weapon.[34] Again, continued chemical and biological weapons research was considered an inhumane act of war, but during the cold war some research actually focused on antidote-based chemical regimens to aid and manage wounded combatants. Unfortunately, with the deployment of chemical, biological, and nerve agents as offensive weapons around the world, it also contributed to these weapons being transferred to terrorist groups because of decreased accountability for these weapons by the end of the Cold War.

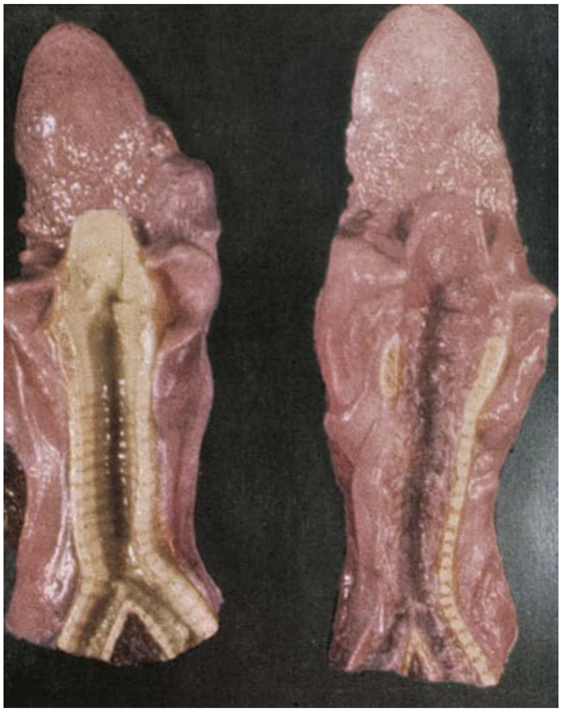

Fig. 68.13 Tracheal injury caused by mustard gas. (Courtesy Her Majesty's Stationery Office, London, United Kingdom.)

Fig. 68.15 Cutaneous vesication caused by mustard gas exposure. (Courtesy Her Majesty's Stationery Office, London, United Kingdom.)

Fig. 68.14 Eye injury caused by mustard gas. (Courtesy Her Majesty's Stationery Office, London, United Kingdom.)

The 1980s saw a shift to chemical and biological weapons as a true first-line offensive weapon. At this point in history, chemical and biological injuries could be treated more effectively on the battlefield and by first responders on the civilian side. The research into antidotes for chemical, biological, and nerve agents had finally caught up to their weaponization. The United Nations had documented the combined uses of vesicants and nerve agents, such as mustard gas and tabun toxin to increase the lethality of chemical and biological weapons.[35,36] These agents were not as effective as expected, and they accounted for only about 27,000 Iranian casualties, or about 1% of the total casualties of the Iran-Iraq war. The noted reason for the ineffectiveness or decline in lethality of these chemical weapons can be accounted for by the research into antidotes and improved care on and around the battlefield. In contrast to the organized battlefield during the Iran-Iraq war, when this agent was released on a civilian population of Halabja in Iraqi Kurdistan, it accounted for more than 5000 deaths, or a large percentage of the Kurdish village population.[37] This contrast in lethality demonstrates the improvement to battlefield medicine as well as the effectiveness of the antidotes.

The last part of the 20th century saw several civilian attacks utilizing different combinations of chemical, biological, and/or nerve agents. In one of the most notorious nerve agent attacks in Tokyo, Japan, sarin gas was used in the subway system and was responsible for multiple fatalities and injuries. Unfortunately, during this mass casualty experience, civilian medical personnel lacked knowledge, training, and understanding of appropriate medical treatments and became casualties themselves.[38,32] The lack of training for civilian medical providers and emergency staff brought home important lessons for all, especially anesthesia providers. Box 68.1 outlines the lessons learned from this nerve agent attack.[32]

Despite the progressive weaponization of chemical, biological, and nerve agents over many decades, classifying them appropriately can be challenging. In the simplest terms, they can be classified as weapons of *mass destruction*, but the application of that term was short-lived because of the advancement of antidotes and improved medical treatment. By definition, weapons of mass destruction have to have a significant destructive impact on a society, such as the atomic bombs dropped on Hiroshima and Nagasaki, Japan. In regards to chemical, biological, and nerve agents, they are defined by their effects on the populations on which they have been released, and many of these devices lack the large explosive component as compared to nuclear weapons: true weapons of mass destruction.[31] In the 1950s, because of the Tizard report, chemical, biological, and nerve agents were to be included within the term "weapons of mass destruction." In this report, the term weapon of mass destruction referred solely to the massive loss of life the weapon was capable of creating. Overall, regardless of the mechanism and explosive power of the weapon employed, chemical, biological, nerve agents, and nuclear isotopes are all considered very lethal threats to society. This holds true regardless of whether they are classified as weapons of mass destruction. The massive release of chemical, biological, or nerve agents among a very large population is possible in theory, but this would require a delivery modality such as a missile, rocket, or violent explosive vehicle.[31]

BOX 68.1 Tokyo Sarin Gas Lessons Learned for First Responders

Lesson 1	Military-grade chemical, biological, and nerve agents can be easily employed by terrorist groups
Lesson 2	HAZMAT and incidence management training for chemical, biological, and nerve agent mass causalities was not sufficient
Lesson 3	Lack of situational (scene) control, protection, and personnel decontamination contributed to additional casualties and fatalities
Lesson 4	Lack of resuscitation efforts at the site of chemical and biological release contributed to further casualties
Lesson 5	This incident lead to a significant database for medical management of patients exposed to nerve agents

CHEMICAL, BIOLOGICAL, RADIOLOGICAL, OR NUCLEAR HAZARDS DEFINED

Chemical

Historically, chemical agents were purely a military issue, because there were only a small number of people with access or knowledge to chemical agents or weapons. However, over the past 20 years the types of chemical hazards have expanded significantly because of the advanced use of toxic chemicals required for industrial manufacturing. Chemical hazards are chemicals that can cause death or harm because of their toxic properties. The characteristics of the most commonly talked about chemical agents are discussed in Table 68.2.

Most chemical agents, whether industrialized or weaponized, have the ability to severely incapacitate a person or cause immediate or imminent death. Chemical agents begin their onset of physiologic symptoms when the body comes in contact with a concentration of agent that is more than one's body can tolerate. As an anesthesiologist, traumatologist, or first responder, one must be able to rapidly identify the difference between mild and severe symptoms of chemical agent exposure, which are outlined in Table 68.3. The main categories of chemical agents are outlined here:

1. Blistering agents (vesicants)—Generally affect the eyes and mucous membranes. In addition, they damage the respiratory epithelium. Airway management in these patients can be cumbersome for anesthesia providers because of sloughing off epithelium and necrotic tissue occluding the glottic view and anatomy.[40] Overall, the effect and toxicity depend on the type of agent, the concentration of the agent, the weather/weather patterns, and exposure time.
2. Blood agents (nerve agents)—Generally inhibit oxygen transfer from blood or hemoglobin to the cells of the body, or in another way causes tissue hypoxemia. Overall, the higher the concentration of blood agents, the more rapidly fatal these agents are. Most blood agents inhibit acetylcholinesterase activity, increasing the concentration of acetylcholine at the muscarinic and nicotinic receptors, causing cholinergic toxicity.[41] This excessive acetylcholine in the body can cause a myriad of symptoms, but for the anesthesiologist airway interventions can be hindered because of

TABLE 68.2 Most Commonly Discussed Chemical Weapon Agents

Name	Type	Properties	Mechanism of Action	Physiologic Symptoms	Onset of Action
Sarin gas	Nerve	Colorless, odorless, volatile	Anticholinesterase agent	Choking, miosis, headache, nausea, respiratory distress, convulsions, death	Minutes
VX (VR) gas	Nerve	Colorless, odorless, oily liquid	Anticholinesterase agent	Choking, miosis, headache, nausea, respiratory distress, convulsions, death	<30 min
Mustard gas	Blister	*Garlic odor*, oily liquid	Vesicant, bone marrow suppression, alkylating agent, damages deoxyribonucleic acid	Blisters skin, eyes, lungs	~4-6 h
Cyanide gas	Blood	*Almond odor*, volatile	Interferes with oxygen utilization at the cellular level	Hinders oxygen transfer, *respiratory paralysis*	Minutes

Adapted from the Multi-Service Doctrine for Chemical, Biological Radiological and Nuclear Operations (FM 3-11, MCWP 3-37.1, NWP 3-11, AFTTP 3-2.42).

significant airway occlusion from secretions and profound bronchospasm, which is why atropine should be administered prior to any airway intervention.[12,42,43] Higher concentrations of agent lead to more rapid physiologic decline proceeding to cardiopulmonary arrest.
3. Choking agents (pulmonary agents)—Generally cause a shift of fluid into the small airways at lower doses, while at higher doses all the major airways become irritated and dried out, causing choking, poor gas exchange, and ultimately, death. Rapid, shallow breathing, painful cough, and cyanotic skin are indicative of fluid filling the lungs and preventing oxygenation. Commonly, these patients will present with pulmonary physiology that mimics acute respiratory distress syndrome because of the fluid shifts, airway irritation, and inflammation.
4. Riot control agents—Generally cause lacrimation or vomiting. Unlike the choking agents, they are irritants that are used to cause a brief period of misery and harassment to subdue or incapacitate a threat or aggressive persons. Most of these agents inhibit multiple different enzymes and invariably increase bradykinin release.[16] Overall, these agents do not require significant medical attention except flushing of the eyes and removal from the environment.

Biological

Biological hazards are relatively easy to produce, especially since production facilities have very little environmental signature. Production of these microbes or organisms can pose a significant threat to human and animal welfare. Biological agents differ from chemical agents in that the agents are living organisms, viruses, toxins and/or microtoxins utilized to disable, disrupt, or kill other human beings or other organisms. While most biological agents are small, they have tremendous potential for lethality and application to a large-scale attack. Most biological agents are accessible throughout the world and are relatively inexpensive to produce compared to weaponization of chemical, radiological, or nuclear agents. Today's most commonly discussed biological agents are orders of magnitude stronger and more lethal than most chemical agents. These agents are outlined in Table 68.4. A common limitation of many biological agents is that they can be degraded by changes in the environmental conditions such as ultraviolet light, temperature, and humidity.

TABLE 68.3 Typical Chemical Agent Exposure Symptoms

Mild Symptoms*	Severe Symptoms†
■ Runny nose	■ Sudden confusion
■ Sudden severe headache	■ Wheezing, labored breathing
■ Drooling	■ Miosis
■ Blurry vision	■ Painful tearing eyes
■ Chest pain	■ Sudden profound vomiting
■ Difficulty breathing	■ Severe muscle twitching/seizures
■ Sweating	■ Uncontrolled urination
■ Muscle twitching/cramps	■ Uncontrolled defecation
■ Stomach cramps	■ Profound respiratory difficulties
■ Nausea	

*Some symptoms of chemical agent intoxication can be confused with severe heat stroke.

†Adapted from the Multi-Service Doctrine for Chemical, Biological Radiological and Nuclear Operations (FM 3-11, MCWP 3-37.1, NWP 3-11, AFTTP 3-2.42).

Many of the initial symptoms of biological warfare and a biological attack are vague and nonspecific. However, in the event there is high index of suspicion for a biological attack, steps must be taken to protect first responders and medical personnel. Physical protective barriers and personal protective equipment (PPE) are essential primary preventive measures. In addition, for other hazard responders and personnel there are vaccinations and immunizations for certain biological agents. By protecting our primary responders and healthcare providers during biological attacks, they are more apt and readily able to care for those directly and immediately in danger. PPE and these concepts will be explored in more detail in the Epidemic and Pandemic Infectious Outbreaks section.

Radiological and Nuclear Weapons and Effects

Radiological hazards include electromagnetic or particulate radiation capable of causing damage, injury, or destruction through ionizing effects of neutrons, γ rays, α particles, or β particles. Radiological damage can be dispersed in many ways, but ultimately causes destruction, damage, or injury to a large population of people, while exposing an even larger population to the lingering effects of ionizing radiation.

Nuclear weapons are a complete assembly of weapons capable of independently being armed, fired, fused, and detonated to release a large-scale nuclear explosion capable of significant destruction to the land, environment, people, and animals within a large area. The intensity of nuclear weapons is greater than that of chemical and biological weapons, and the deployment of a nuclear weapon will cause significantly more death and destruction from the point of detonation, outward. Short- and long-term devastation can be attributed to nuclear fallout, which is the residual radioactive material that is propelled into the atmosphere following a nuclear detonation. Thus, nuclear and radioactive material is literally falling from the sky and is moved miles and miles away by winds within the atmosphere.[44]

Unlike chemical attacks or biological attacks, nuclear/radiological attacks rarely induce the need for immediate airway intervention, even in the harshest of exposed patients. While nuclear warfare can devastate a large area, the immediate impact can be considered similar to a high-grade explosive device. The main difference between nuclear weapons and conventional explosives is that a nuclear weapon will do greater damage by orders of magnitude. Overall, acute management of the effects of high radiation doses is prudent. Most commonly, supportive care of the hematological, gastrointestinal, neurologic, cardiovascular, and integumentary systems are required. Following a major radiological or nuclear attack, patients with significant nausea and vomiting may require intubation for airway protection.

Mass Casualty Situations and Chemical, Biological, Radiological, or Nuclear Attacks

Overall, the structure, function, and focus of the U.S. military's approach to CBRN can be adapted to the civilian sector wherever feasible to help improve patient care while minimizing collateral damage. All CBRN incidents are emergency situations that require multi-system coordination and organization. Table 68.5 demonstrates the sources and types of CBRN hazards. There are three basic reasons why CBRN situations occur:

TABLE 68.4 Most Commonly Discussed Weaponized Biological Agents

Common Name	Agent	Physiological Effects	Onset
Anthrax	*Bacillus anthracis*	Fever, fatigue, severe respiratory impairment, high fever, and excessively rapid pulse rate. Pulmonary anthrax is fatal more than 90% of the time.	1-5 days
Plague	*Yersinia pestis*	Fever, headache, and rapid heart rate, followed by pneumonia and hemorrhage of the skin and mucous membranes. Untreated plague pneumonia fatalities approach 100%, but early treatment can reduce mortality to as low as 5%.	2-3 days
Tularemia	*Francisella tularensis*	Fever, chills, headache, and muscular pain. Untreated tularemia can result in 30%-60% mortality; treated, mortality rate is reduced to 1%.	3-5 days
Botulism	*Clostridium botulinum*	Extreme weakness, nausea, headaches, and intestinal pain leading to respiratory paralysis that may cause death.	2-26 h

Adapted from the Multi-Service Doctrine for Chemical, Biological Radiological and Nuclear Operations (FM 3-11, MCWP 3-37.1, NWP 3-11, AFTTP 3-2.42).

TABLE 68.5 Chemical Biological, Radiological, or Nuclear Threats and Hazards

Chemical	Biological	Radiological	Nuclear
WEAPONS OF MASS DESTRUCTION			
■ Chemical weapons ■ Chemical agents ■ Nontraditional agents	■ Biological weapons ■ Biological agents ■ Nontraditional agents	■ Radiological dispersal devices ■ Radiological exposure devices	■ Nuclear weapons ■ Improvised nuclear device (improvised explosive devices/IND)
TOXIC INDUSTRIAL MATERIALS			
Toxic Industrial Chemicals	Toxic Industrial Biologicals	Toxic Industrial Radiologicals	
Other Sources			

Adapted from the Multi-Service Doctrine for Chemical, Biological Radiological and Nuclear Operations (FM 3-11, MCWP 3-37.1, NWP 3-11, AFTTP 3-2.42).

1. Intentional—This is a deliberate action to induce harm in the form of toxic agents, release of radiological agents, or the detonation of explosive material in an attempt to cause terror for political, religious, or ideological purposes.
2. Accidental—A human error–induced release of toxic agents, release of radiological agents into the environment, or detonation of explosive material.
3. Natural—Caused directly by or in response to a natural disaster, which leads to the release of toxic agents, release of radiological agents into the environment, or detonation of explosive material.

Currently the American Society of Anesthesiologists (ASA) has several suggested protocols for CBRN attacks, but to make these protocols most useful will require extensive and ongoing training. Disaster preparedness and coordination is not simply an ED problem to manage. For anesthesia providers, it is critically important to reinforce clinical skills and practices so that, in the event of a massive casualty event, during a natural disaster, or during a potential CBRN attack they are prepared to respond immediately and appropriately. Box 68.2 outlines the basic CBRN rules all providers should follow during a CBRN attack. Many providers do not have the adequate training to support a mass casualty situation and this leads to increased unrest and disorder. In civilian hospitals, training for CBRN as an

BOX 68.2 CBRN Basic Provider Rules and Guidelines for Safety

Rule 1	DO NOT become the casualty yourself
Rule 2	Always decontaminate the scene, situation, and patient prior to any movement to the Operating Room
Rule 3	Never assume ANY vaccine is 100% protective
Rule 4	“SAFE ZONES” should always protect themselves from contaminated “WALK-INS”
Rule 5	We all make mistakes, when in doubt call infectious disease, the CDC or WHO
Rule 6	Always decontaminate the casualties, then immediately begin ACLS, ATLS, and resuscitation measures to stabilize patients
Rule 7	Pay careful attention to sterile techniques, many patients will have compromised immune systems and/or already be neutropenic
Rule 8	Brain irradiation can cause all sorts of primary, secondary, and tertiary CNS symptoms
Rule 9	Ionizing radiation victims are less of a risk to providers compared to chemical/biological casualties

ACLS, Advanced cardiac life support; *ATLS*, advanced trauma life support; *CDC*, Centers for Disease Control and Prevention; *WHO*, World Health Organization.

entire unit is extremely challenging, because it is almost impossible to shut down an entire hospital for a day or several hours to properly practice, train, and prepare. The U.S. military, their military hospitals, and healthcare personnel have required unit, battalion, and command training exercises on U.S. soil and abroad for CBRN attacks, which help maintain order, direction, and patient care during times of turmoil and civil unrest.

Incident Management During a Chemical, Biological, Radiological, or Nuclear Attack and Personal Protective Equipment

In the face of a mass casualty event from a CBRN attack, panic and chaos are to be expected. Successful response to a CBRN attack requires command and control personnel and the medical providers to prioritize the following: personnel safety, personnel protection, command establishment, and site-wide communication. Similar to the U.S. military, the civilian sector has command control entities in the form of local, state, and federal response systems. Once the command structure is established for the incident in question, the commander is directly responsible for directing and controlling all available resources.

The first rule for CBRN casualty treatment and maintenance states, "Do not become the casualty yourself". The incident commander or officer in charge is in charge of setting the protection level to ensure maximal barrier protection and adequate PPE for all providers and first responders. Unfortunately, the appropriate PPE level to use may not be immediately known, so it is advantageous to err on the side of a higher protection during immediate decontamination and stabilization efforts. The U.S. military has adapted their PPE levels from the U.S. Environmental Agency's *Hazardous waste operations and emergency response* protocols.[45] At any time during intubation, exposure to bodily fluids, and/or airborne infection the minimum protection level should be C. In addition to this protection level, in an unknown CBRN attack all medical examinations for severe casualties or those severely and negatively impacted by the CBRN attack should be done per Table 68.6.

Protection is the key feature of incident management, and anesthesia providers should be familiar with several levels of PPE suits and masks, and the techniques of decontamination in Table 68.7. Several levels of protection are used in the management of toxic releases, but the appropriate level for medical intervention is level C, which allows reasonable tactile dexterity and contact with the patient to provide essential life support and antidote therapy onsite.[46] Level C protection is equivalent to that used by the military to provide protection against the most toxic chemical warfare agents and virulent biological warfare organisms.[47]

Decontamination

In most situations the initial hazards are unknown, however protecting oneself is one of the major keys to on-scene and in-hospital care of the afflicted patients. Many hazards are short-lived or transient, but it should be assumed that the hazard released is persistent and transmissible through the on-scene decontamination zones. Immediately establishing the level of personal protection required for medical staff, paramedics, and ancillary search and research personnel by the on-scene commander is vital to preventing an increased number of casualties among medical and rescue personnel. To safely work on search and rescue and decontamination many on-scene commanders immediately place a level C minimum for PPE for medical staff and paramedics, and upgrade or downgrade based on situational changes onsite. Triage personnel and medical personnel are vital to providing immediate: (1) triage, (2) life-support measures (TOXALS—toxicology advanced life support), and (3) antidote and other pharmacologic support.

Early life-support measures in the decontamination zone are very important.[48,49] A unique perspective to CBRN on-scene decontamination and resuscitation was introduced in 1996, and was uniquely termed TOXALS (Toxicology Advanced Life Support) to be used in conjunction with advanced cardiac life support (ACLS), advanced trauma life support (ATLS), and basic life support (BLS) in the setting of chemical, biological, and/or radiological attacks. TOXALS protocol expands the on-scene and in-hospital resuscitation efforts of trained medical personnel. and incorporates

TABLE 68.6 Initial Workup for Severely Injured Casualties During Chemical Biological, Radiological, or Nuclear Attack

Investigation	Lab Test/s
Urea and Electrolytes	▪ Arterial blood gas ▪ Glucose ▪ Lactate ▪ Calcium/phosphorus/magnesium
Full Blood Count	▪ Store sample for later analysis ▪ Consider coagulation/clotting studies
Urinalysis	▪ Store for later analysis
Electrocardiogram	
Chest Radiogram	

Store all blood samples in a safe containment area for personnel protection and for future analysis.

TABLE 68.7 Levels of Personal Protective Equipment in Chemical, Biological, Radiological, or Nuclear Incidents[45]

Level	Minimum Personal Protective Equipment Required
A	▪ Positive pressure SCBA ▪ Fully encapsulated chemical-resistant suit ▪ Double layer of chemical-resistant gloves ▪ Chemical-resistant boots ▪ Airtight seal between suit and gloves and boots
B	▪ Positive pressure SCBA ▪ Chemical-resistant, long-sleeved suit ▪ Double layer of chemical-resistant gloves ▪ Chemical-resistant boots
C	▪ Full-face air-purification device (respirator) ▪ Chemical-resistant suit ▪ Chemical-resistant outer gloves ▪ Chemical-resistant boots
D	▪ Equipment does not provide specific respiratory or skin protection and usually consists of regular work clothes

SCBA, Self-contained breathing apparatus.

principles of ACLS, ATLS, and BLS, which can be expanded to the pneumonic ABCDDEE: airway, breathing, circulation, disability, drugs, exposure, and environment.

Summary of Chemical, Biological, Radiological, or Nuclear Section

CBRN attacks and natural disasters have similar elements to medical care on the battlefield. Population control, situational control, and on-scene containment are essential to maintaining order and functionality of the civilian emergency disaster system. As anesthesiologists, the main responsibility is to be present for operating room needs, but their advanced medical knowledge, advanced pharmacology knowledge, and advanced trauma-stabilization training makes them ideal to aid and support emergency triage physicians. Not all patients during CBRN attacks or natural disasters immediately go to the operating room for stabilization. Many of the emergency responders and providers will need support either in triage, for ED consultations, pain management, or intensive care support for stabilization, pharmacology expertise, and/or procedural skills. For example, a small roving team consisting of an anesthesiologist, a critical care/ED nurse, and a respiratory therapist could manage a wide variety of critically injured patients following a CBRN massive casualty event.

An anesthesiologist's primary role is to stabilize and ensure safe operating conditions for casualties of any CBRN attack, terrorist attack, or natural disaster. However, this is not limited to preoperative assessment and intraoperative management of CBRN victims. During times of crisis, anesthesiologists cannot remain sequestered in the operating room awaiting the arrival of injured patients. They must be proactive within the hospital system or emergency response system, providing triage and management of patients prior to arrival at the operating room or the ICU.

Section 4: Epidemic and Pandemic Infectious Outbreaks

Until recently, the role of anesthesiologists in biological disasters, such as infectious epidemics and pandemics, had been discussed only in the most general terms. While the lifesaving application of positive pressure ventilation by Danish anesthesiologist Dr. Bjorn Ibsen probably saved hundreds if not thousands of lives during the Copenhagen polio epidemic of 1952, the role of North American anesthesiologists as critical members of the management team in biological disasters was not fully exploited until the severe adult respiratory syndrome (SARS) epidemic of 2003 and the West African Ebola epidemic of 2014.[50-52] During both of these worldwide healthcare crises, anesthesiologists not only played critical roles as caregivers, but risked personal safety when functioning in their role as specialists in airway management and intensive care. Knowledge of the disease, management options, and importantly, infection-control measures necessary to protect oneself, are all critical for anesthesiologists to contribute maximally and mitigate personal vulnerability during the next epidemic or pandemic.[53]

Health emergencies in the form of regional epidemics and global pandemics have the capacity to kill more than 10 million people across continents and around the world.[54] While this number sounds excessive and perhaps even alarmist, it is in fact a conservative estimate when compared to pandemics over past centuries. Smallpox is the deadliest pandemic in human history. In the 20th century alone, estimates of deaths due to smallpox exceeded 300 million people with 30% of all persons infected succumbing to the disease.[55] The causative agent, Variola virus, is thought to date back possibly as far as 3000 years to the time of the Egyptian Empire. Its global spread paralleled the advance of modern civilization, exploration, and expanding trade. Thanks to the success of worldwide vaccination, the last person to die of smallpox was in 1978 and on May 8, 1980 the 33rd World Health Assembly officially declared the world smallpox free.[56,57] Unfortunately, unlike viruses that have a human reservoir such as smallpox, polio, and measles, and hence are targets for eradication, the next pandemic is more likely to be the result of zoonotic transmission.[58]

PANDEMIC INFLUENZA A

Past influenza pandemics have occurred when genetic mutations in an enzootic cycle result in re-assortment of gene segments creating what is functionally a novel infectious agent with major changes in surface proteins, capable of sustained human-to-human transmission in a global population with little existing herd immunity.[59] Pathogenic influenza A viruses arising from this antigenic shift have been the source of influenza A pandemics for centuries. In fact, there have been no less than 10 pandemic influenza A events over the past 300 years.[60] In the preceding century alone, there were three episodes of pandemic flu. A recent analysis of the "Spanish Flu" H1N1 pandemic of 1918 to 1919 estimated that at least 50 to 100 million people died worldwide. Less severe, but also with considerable mortality (approximately 1 million globally; 100,000 in the United States), were the 1957, "Asian Flu" H2N2 and the 1968 "Hong Kong Flu" H3N2 (Fig. 68.16).[60,61] Such events underscore the pandemic threat posed by avian influenza viruses, emphasizing the need for advanced preparation necessary to augment both the clinical and research response to future global health emergencies. In fact, given the incidence and variety of influenza viruses circulating in zoonotic populations, a virus possessing the necessary properties to cause a pandemic can emerge any time.[62]

In 2009, the world experienced the first major influenza A event of the 21st century. This worldwide influenza outbreak has been described by the CDC as the 2009 H1N1 Pandemic. The virus contained a unique combination of influenza genes not previously identified in animals or people. The genes were most closely related to North American swine and Eurasian swine H1N1 influenza viruses. Because of this, initial reports referred to the virus as a swine influenza virus. However, it quickly became apparent that this new virus was circulating among humans and not among U.S. pig herds. In the United States, there were over 60 million cases over the course of a year from April 2009 to April 2010. Fortunately, loss of life attributable to this event was relatively limited with an estimated 12,500 lives lost in the United States and 284,000 globally.[63,64]

Central to disaster planning for pandemic flu is an understanding of the strategies to minimize transmission of influenza between patients and healthcare workers. Despite vast clinical experience, there is still debate over how influenza is transmitted. Possible modes of transmission included in past discussions are airborne, droplet, and contact transmission. Each requires a different infection control approach and almost certainly different PPE. Recent analysis of multiple human and animal studies concluded that natural influenza transmission in human beings occurs over short distances primarily via droplet and contact routes.[65] Transmission of influenza virus via large-particle droplets generated from the respiratory tract of infected individuals during coughing, sneezing, talking, or during procedures such as suctioning or bronchoscopy requires close contact between the infected source and the recipient (6 feet or less). Large droplets do not remain suspended in the air and hence special ventilation or airflow systems are not required since true aerosolization does not occur. Preferential airborne transmission has not been documented as a method of human-to-human influenza transmission.[66] However, many procedures such as intubation and bronchoscopy have the potential to be aerosol-generating. The current literature does not define conditions under which influenza could become opportunistically airborne. Given the paucity of data in this regard, it may be reasonable to employ airborne precautions and higher levels of PPE such as N95 masks when such events are medically necessary. This is important information for anesthesiologists caring for potential flu victims in the ICU or managing their airway in the emergency room, hospital ward, or operating room.

SEVERE ACUTE RESPIRATORY SYNDROME

Severe acute respiratory syndrome (SARS) caused by a novel coronavirus, called SARS-associated coronavirus (SARS-CoV), was first reported in Guangdong Province in China in November 2002. The first case appears to be a businessman from the city of Foshan who subsequently died from the disease. However, it was 3 months later, when a physician from the same province became ill while staying in a hotel in Hong Kong, that SARS rose to international attention. Twelve guests staying at the same hotel subsequently became infected, including seven whose rooms were on the same floor as the physician. These hotel guests became the index patients transporting the virus around the globe as they traveled home by airplane. While SARS was first identified in Toronto, Canada, in March, 2003, because of the speed of international travel, in less than 4 months about 4000 cases and 550 deaths from SARS could be traced to the guests infected in Hong Kong. Ultimately, a total of 8096 people in 29 countries became infected with SARS-CoV, of which 774 of them died (Table 68.8).[67-69]

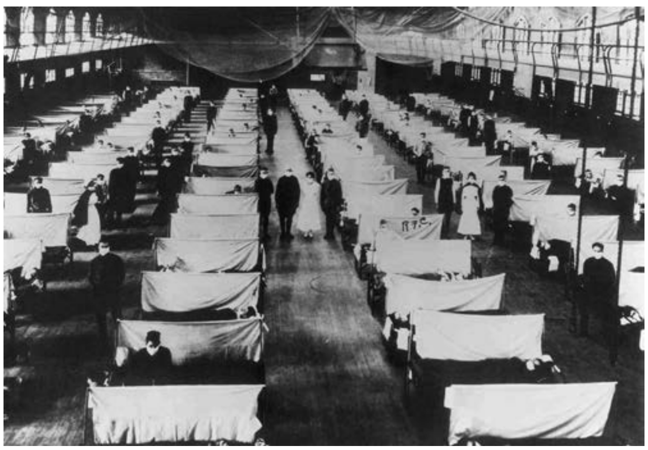

Fig. 68.16 Mass care of patients during 1918 flu pandemic. (From Clements BW, Casani J, eds. *Disasters and Public Health: Planning and Response, Second edition*. Oxford: Butterworth-Heinemann (Elsevier); 2016.)

Toronto was the epicenter of the disease in North America. The Ontario Ministry of Health reported 361 SARS cases, including 33 deaths (9%). Over half of those infected were healthcare workers including three anesthesiologists and one intensivist.[10,27,53,70] Overall case fatality was approximately 15%, but over 50% for patients over the age of 65.[9] On July 4, 2003, the World Health Organization (WHO) announced the global SARS outbreak was contained.[71]

SARS, like influenza, is of zoonotic origin. The virus is ubiquitous and has been isolated from swine, cattle, dogs, cats, and chickens. In 2002, SARS-CoV was isolated from Himalayan palm civets found in Chinese live animal markets where demand for freshly killed meat and poultry served as the source of SARS-CoV crossing species (Fig. 68.17).[72] This led to culling of the animals, although subsequent studies have not validated infection in either farm-raised or wild civets. More recent data suggest that bats are the more likely natural reservoirs of SARS-like coronavirus.[73]

Spread largely by droplets and direct contact, the virus demonstrates tropism to multiple organs and has been found not only in the lung, but the liver, kidney, sweat glands, parathyroid, pituitary, pancreas, adrenal glands, and the brain. As such, the disease is considered a true systemic illness with both pulmonary and extrapulmonary manifestations amplifying opportunity for healthcare worker exposure. This is important for anesthesiologists, intensivists, and other healthcare workers as viral shedding may occur not only in respiratory secretions but stool, urine, and even sweat.[72] Appropriate PPE providing protection beyond droplet and contact precautions is indicated when caring for SARS patients.

MIDDLE EAST RESPIRATORY SYNDROME

Like SARS, Middle East respiratory syndrome (MERS) is caused by a novel coronavirus, MERS-CoV. The first two cases of MERS-CoV infection as a cause of community-acquired pneumonia were first identified in Saudi Arabia and reported to the WHO in September 2012. As of September 2018, laboratory-confirmed cases of MERS number 2260 with 803 associated deaths (35.5%) in 27 countries across the globe.[74,75] The

TABLE 68.8 Mortality of Severe Acute Respiratory Syndrome Based Upon Age

Age (Years)	Mortality
<24	<1%
24-44	6%
45-64	15%
>65	>50%

Modified from Peng PW, Wong DT, Bevan D, et al. Infection control and anesthesia: lessons learned from the Toronto SARS outbreak. *Can J Anaesth*. 2003;50(10):989–997.

majority of cases have been reported in Saudi Arabia, United Arab Emirates, and the Republic of Korea (South Korea), although over 80% of reported cases to date have been linked to exposure in Saudi Arabia (Fig. 68.18).[76]

Fig. 68.17 Live animal markets are the interface where animal to human inter-species transmission of SARS-CoV occurred. Guangzhou government officers seize civets in Xinyuan wildlife market in Guangzhou to prevent the spread of SARS-CoV. (From Lau YL, Peiris JS. Pathogenesis of severe acute respiratory syndrome. *Curr Opin Immunol.* 2005;17[4]:404–410.)

MERS-CoV, like SARS-CoV, is a zoonotic virus. Dromedary camels appear to be the major reservoir host and animal source of MERS infection in humans through either direct or indirect contact. The specific method of transmission remains unclear, but poorly cooked camel meat and unpasteurized camel milk remain suspect, and consumption of raw or undercooked animal products carries a high risk of infection. Human-to-human contact in the non-healthcare setting appears rare with little to no sustained human-to-human transmission documented.[77]

Human-to-human transmission through close contact within the healthcare setting accounts for the majority of reported cases to date. Outside of Saudi Arabia, the largest outbreak so far occurred in South Korea in 2015.[77] In a hospital-associated cluster, a single infected person admitted to a hospital in South Korea led to 186 cases and 36 deaths across multiple healthcare facilities.[78,79] This particular case highlights the consequences of substandard infection control practices and inadequate use of PPE, as the virus does not pass easily person to person unless there is close contact.[76] Healthcare workers must remain alert for the continued threat of MERS as global travel continues to accelerate.

EBOLA VIRUS DISEASE

Ebola virus disease (EVD) is a zoonotic disease affecting people and non-human primates. The presumptive enzootic reservoir is believed to be African fruit bats, although this cause is still not definitive. Ebola is an RNA *Filovirus* and a member of the *Filoviridae* family. There are five species of Ebola virus, of which four are pathogenic to humans. The most common and possessing the highest case fatality rate (60%-90%) is the *Zaire ebolavirus*. *Sudan ebolavirus, Taï Forest ebolavirus, Bundibugyo ebolavirus* also are pathogenic to humans, but

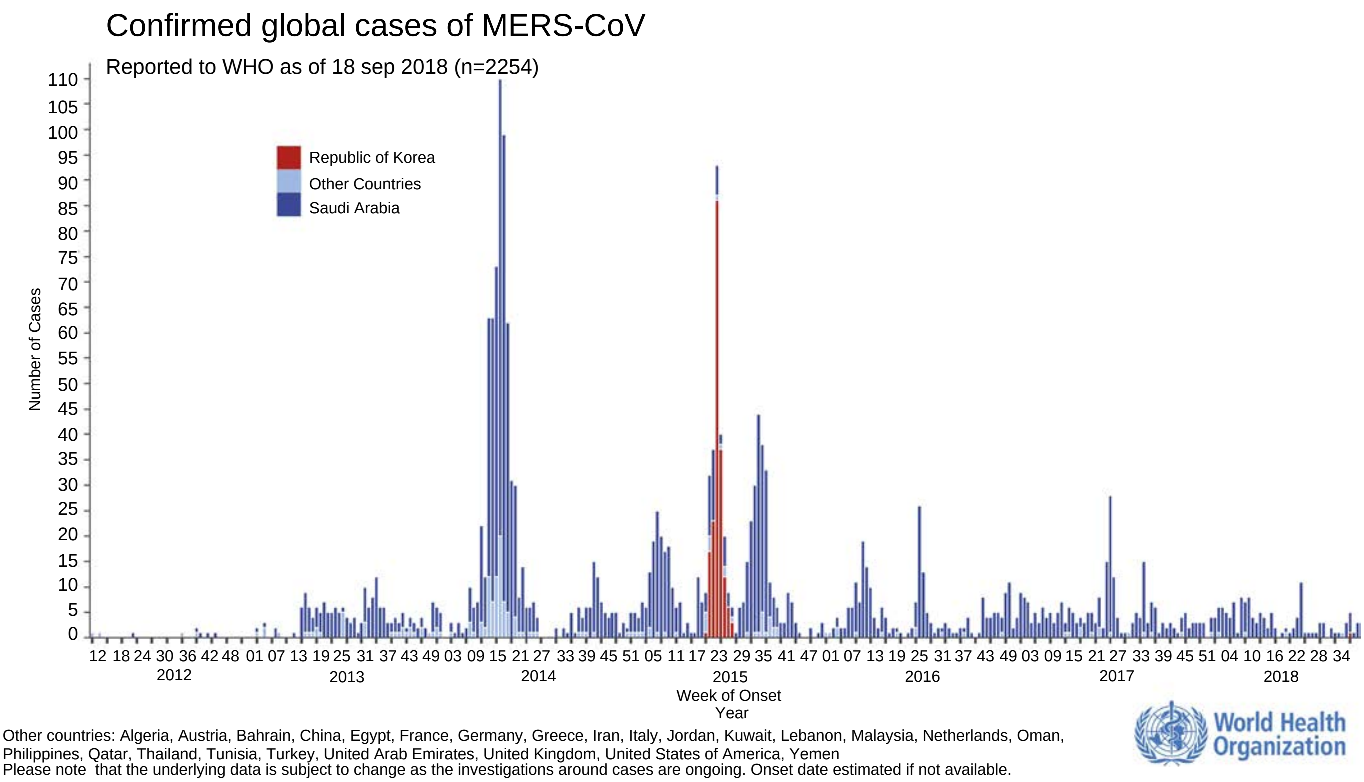

Fig. 68.18 Global distribution of Middle East Respiratory Syndrome. (Data from World Health Organization. Available from http://www.who.int/emergencies/mers-cov/epi-18-september-2018.png?ua=1.)

at lower fatality rates. The *Reston ebolavirus* while known to cause disease in nonhuman primates and swine, has not been indicted as a cause of human disease.[80] Marburg virus, the first known *Filovirus* was first described in 1967. Both viruses are known to cause viral hemorrhagic fever in humans with high fatality rates. The higher fatality rate of EVD makes it an important biothreat pathogen and, given the severity of recent outbreaks, worthy of more detailed discussion.[81,82]

Historically, the virus has been isolated to rural regions of Sub-Saharan Central Africa. EVD outbreaks have occurred relatively frequently over the past 30 years since the first outbreak in 1976 alongside the Ebola river in the village of Yambuku, Zaire, in what is now the northern Democratic Republic of Congo and South Sudan.[83] Ultimately, two genetically distinct species of virus were isolated bearing the names of their respective geographic areas.[81] In 2014, the beginnings of what would become the largest outbreak of EVD on record took hold in West Africa. On March 21, 2014, the WHO was notified of a growing outbreak in southeastern Guinea. The disease spread quickly to its capital, Conakry, and then to the neighboring countries of Liberia and Sierra Leone.[82] West Africa had never experienced an EVD outbreak and given the socio-economic conditions of these nations, was poorly equipped to respond. By the time the outbreak was declared over in June of 2016, there were 28,610 cases and 11,308 deaths for a mortality rate of 39%. To put things in perspective, from 1976 to 2014 there were fewer than 2500 cases reported worldwide (Table 68.9).[84]

The Ebola virus is transmitted through contact with infected secretions. Ebola has been identified in blood, saliva, vomit, feces, urine, sweat, nasal secretions, semen, and genital secretions. There is no evidence of airborne spread. Ebola enters the host through mucosal surfaces or

TABLE 68.9 Number of Cases and Deaths During Ebola Outbreaks Worldwide 1976-2014

Country	Year	Town	No. of Cases	No. of Deaths	Species
Democratic Republic of the Congo	2014	Multiple	66	49	*Zaire ebolavirus*
Uganda	2012	Luwero District	6*	3*	*Sudan ebolavirus*
Democratic Republic of the Congo	2012	Isiro Health Zone	36*	13*	*Bundibugyo ebolavirus*
Uganda	2012	Kibaale District	11*	4*	*Sudan ebolavirus*
Uganda	2011	Luwero District	1	1	*Sudan ebolavirus*
Democratic Republic of the Congo	2008	Luebo	32	15	*Zaire ebolavirus*
Uganda	2007	Bundibugyo	149	37	*Bundibugyo ebolavirus*
Democratic Republic of the Congo	2007	Luebo	264	187	*Zaire ebolavirus*
South Sudan†	2004	Yambio	17	7	*Zaire ebolavirus*
Republic of the Congo	2003	Mbomo	35	29	*Zaire ebolavirus*
Republic of the Congo	2002	Mbomo	143	128	*Zaire ebolavirus*
Republic of the Congo	2001	Not specified	57	43	*Zaire ebolavirus*
Gabon	2001	Libreville	65	53	*Zaire ebolavirus*
Uganda	2000	Gulu	425	224	*Sudan ebolavirus*
South Africa	1996	Johannesburg	2	1	*Zaire ebolavirus*
Gabon	1996	Booue	60	45	*Zaire ebolavirus*
Gabon	1996	Mayibout	37	21	*Zaire ebolavirus*
Democratic Republic of the Congo§	1995	Kikwit	315	250	*Zaire ebolavirus*
Côte d'Ivoire	1994	Tai Forest	1	0	*Taï Forest ebolavirus*
Gabon	1994	Mekouka	52	31	*Zaire ebolavirus*
South Sudan†	1979	Nzara	34	22	*Sudan ebolavirus*
Democratic Republic of the Congo§	1977	Tandala	1	1	*Zaire ebolavirus*
South Sudan†	1976	Nzara	284	151	*Sudan ebolavirus*
Democratic Republic of the Congo§	1976	Yambuku	318	280	*Zaire ebolavirus*

*Numbers reflect laboratory-confirmed cases only.
†Formerly part of Sudan.
§Formerly Zaire.
Table excludes 2014-2016 epidemic and 2017-2018 outbreaks in Democratic Republic of Congo.
Ebola, Ebola virus disease.
Source: CDC. Outbreaks chronology: Ebola virus disease. Atlanta, GA: CDC; 2015. http://www.cdc.gov/vhf/ebola/outbreaks/history/chronology.html
From Bell BP, Damon, IK, Jernigan, DB, et al. Overview, control strategies, and lessons learned in the CDC response to the 2014-2016 Ebola Epidemic. *MMWR Suppl*. 2016;65(3):4–11.

TABLE 68.10 Most common clinical findings and interventions for 27 patients with Ebola virus disease during hospitalization in the United States or Europe.

Finding or Intervention	Percentage of Patients
Diarrhea	100
Fever	93
Vomiting	74
Supplemental oxygen requirement	70
Central venous catheter	67
Indwelling urinary catheter	63
SIRS	59
Hypoxemia	52
Oozing from IV sites	52
Rectal tube	44
Pulmonary edema	44
EKG abnormalities	41
Respiratory failure	33

Uyeki TM, Mehta AK, Davey RT, etal. Clinical management of Ebola virus disease in the United States and Europe. *N Engl J Med.* 2016;374[7]:636-646.

breaks in the skin.[82] This route of transmission reinforces the necessity of appropriate PPE use for all healthcare workers. Once in the body, the incubation period is anywhere from 2 to 21 days. The mean incubation period in the 2014 epidemic was noted to be 11.4 days and did not vary by country.[85] The most common symptoms reported between symptom onset and case detection included fever (87.1%), fatigue (76.4%), loss of appetite (64.5%), vomiting (67.6%), diarrhea (65.6%), headache (53.4%), and abdominal pain (44.3%). The most prominent feature in the 2014 outbreak was the progression of the gastrointestinal symptoms and resultant dehydration, severe electrolyte disorders, acute kidney injury, hypoperfusion, systemic inflammatory response syndrome, and shock (Table 68.10).[83,85] Capillary leak syndrome becomes profound in the late stages of the disease and rarely can be associated with respiratory distress. Preterminal events include decreased level of consciousness, which can progress to seizure and coma, disseminated intravascular coagulation, hepatic necrosis, renal failure, and gastrointestinal hemorrhage. Nonsurvivors succumb to multiorgan failure refractory to supportive care.[82,83,85,86]

During the West African Ebola outbreak, 11 people received treatment for EVD in the United States. A total of three patients brought from West Africa to the United States experienced multiorgan failure requiring mechanical ventilation and renal replacement therapy. One patient lived and two died. Care was provided in specialized biocontainment units (Fig. 68.19). Advanced life-sustaining therapy was managed at Emory University Hospital, Serious Communicable Disease Unit; at the University of Nebraska Medical Center—Nebraska Medicine, Nebraska Biocontainment Unit; and at Texas Health Presbyterian Hospital Dallas. All three patients were male and presented with severe gastrointestinal symptoms including severe diarrhea and multiorgan dysfunction notable for respiratory, cardiovascular, renal and hepatic insufficiency. Two patients were encephalopathic. All had extremely high viral loads. Of the two patients that died, the last 12 hours of life was characterized by refractory lactic acidosis and abdominal distension suggesting gastrointestinal perforation or ischemia.[51,87]

The 2014 West African Ebola outbreak is notable for the fact that approximately 27 patients with EVD have now been cared for in resource-rich settings. Signs and symptoms of EVD at the time of presentation to a hospital in the United States or Europe were similar to those presenting to Ebola treatment centers in West Africa. Mortality when care was provided in Europe or the United States was 18.5%. This is significantly better than mortality of patients in the more resource-challenged environment of the West African Ebola treatment units (37%-74%) and the historic mortality of 66%.[84,88] That said, EVD remains a fatal disease despite full access to advanced life support, modern diagnostic and laboratory support, and aggressive ICU care. Also notable is the fact that critically ill patients who have survived have presented early in the continuum of the disease. While no one can assume early support translates to better outcomes, the first-world experience with EVD suggests that early intervention may improve outcomes.[87]

PERSONAL PROTECTION

The logistics of caring for patients with disease due to highly contagious pathogens is complex and outside of the scope of this chapter as is a detailed discussion of PPE. That said, it is relevant to note some consistencies which are likely to reduce human-to-human transmission whether patient-to-patient or patient-to–healthcare worker. More important than the unique physical structure of the units located in the United States and Europe within which patients with highly communicable diseases receive care is the culture of safety and preparedness, including the critical importance of training healthcare workers to care not only for the patient, but for themselves as well.[89] As noted, the Ebola virus is a highly infectious pathogen. The infectious dose is fewer than 10 virus particles. Given that blood viral titers can exceed 10^8 virus particles per milliliter, it is not surprising that even highly trained healthcare workers have become infected.[88,90]

Those providers caring for patients on hospital wards, in the operating room, and in the ICU have the highest risk of contracting the disease themselves. This is reflected in the experience with the SARS-CoV, MERS-CoV, and, of course, the Ebola virus. In all situations, patients become infected and, in some cases, die as a result of lack of appropriate training and a lack of the critical knowledge specific to the pathophysiology of each disease as well as the logistics of caring for patients while protecting oneself.[89]

While adherence to standard infection control universal precautions is advisable when caring for all patients, anesthesia providers who have close contact to oral and respiratory secretions need to pay particular attention to elements of hospital protocols that apply to patients with respiratory infections or the risk thereof. In addition to precautions necessary to prevent contact routes of transmission vis-à-vis

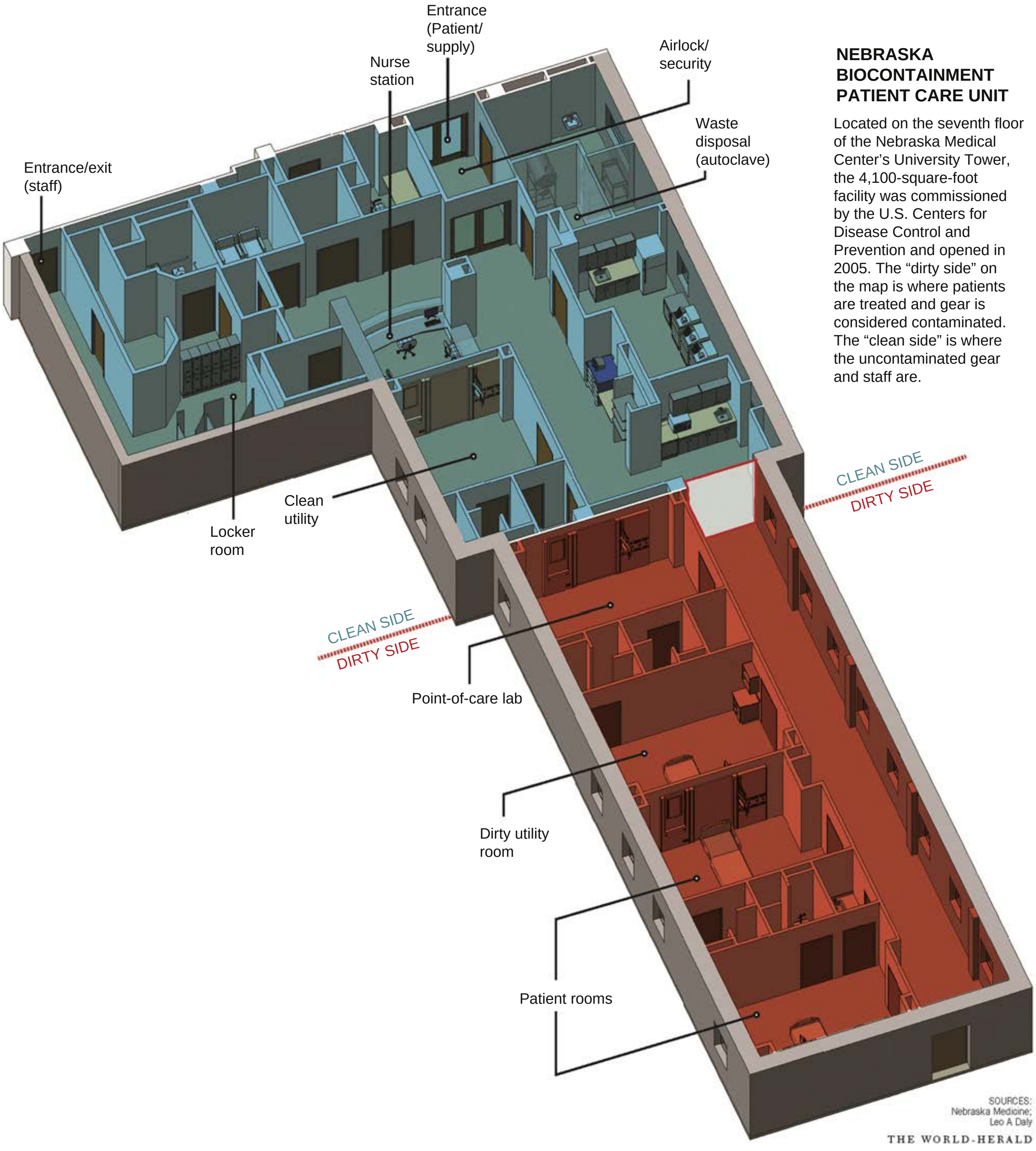

Fig. 68.19 Nebraska Biocontainment Unit, where three patients with Ebola virus disease received care from anesthesiologist-intensivists in 2014. (Courtesy Omaha World-Herald, Omaha, NB.)

hand hygiene, gloves, and gowns, droplet precautions should be in place for all patients with suspected or confirmed influenza, SARS, or MERS. At a minimum, providers should be wearing a facemask when entering the room of a patient with suspected influenza, including perioperative areas such as the preoperative holding area or postanesthesia care unit. When moving a suspected influenza patient on droplet precautions from one area of the hospital to another, having the patient wear a facemask during transport is prudent. This would of course include transport to and from the operating room if deemed medically safe and appropriate.[66]

Anesthesiologists routinely perform medical procedures that are aerosol-generating in patients with highly contagious pathogens such as discussed above. We should only perform procedures on patients with suspected or confirmed disease that are critical to optimizing care. As an example, for Ebola patients, centrally or peripherally inserted catheters should not be placed for hemodynamic monitoring, but rather only for critical vascular

Fig. 68.20 Anesthesiologist placing ultrasound guided central venous line in the Nebraska Biocontainment Unit.

access from which to obtain blood samples without the need for repeated needlesticks and to administer fluids, electrolytes and vasoactive medications. Similarly, when considering respiratory support, early intubation may be preferable despite the inherent risk of aerosolization when compared to noninvasive positive pressure ventilation (NIPPV) and the risk of vomiting, hematemesis, and or aspiration. NIPPV may also result in aerosolization of fluids containing viruses putting caregivers at excess risk not only from Ebola, but also from other highly contagious diseases. Arterial lines should be placed in the upper extremity whenever possible and avoid placement in a femoral artery site due to the potential risk of fecal soiling of the line (Fig. 68.20).[51,80] If intubation is necessary, having a carefully predetermined plan for semi-elective non-emergent intubation is essential. Some of the issues to address to minimize complications associated with intubation include ensuring availability of all medications that may be required and access to all likely equipment that might be needed to secure the airway. In addition, depending on the level of biologic precaution necessary, providers must accommodate for the time necessary to don appropriate PPE prior to entering the patient-care environment.

Finally, all healthcare staff must be instructed to review and understand the guidelines for personal protection appropriate for each pathogen. Certainly, when caring for Ebola patients, the minimum level of PPE should include disposable water-resistant suit or coverall, including a hood; an N95 mask; a disposable full-face shield; two sets of gloves (appropriately sealed), and impermeable foot and leg coverings. It is highly recommended that the addition of a powered air purifying respirator be included when providers are doing any medical procedure with high risk of aerosolization.[51,82] Doffing of PPE is perhaps the highest-risk moment for unrecognized contamination with Ebola virus. For this reason, the entire doffing process should be strictly supervised by a doffing partner. The doffing partner should have no other role besides supervising the healthcare worker who is removing PPE (Fig. 68.21).

Fig. 68.21 Strict supervision of the personal protective equipment doffing process by a dedicated doffing partner in the Nebraska Biocontainment Unit.

Acknowledgment

The editors and publisher would like to thank Dr. David J. Baker for contributing a chapter on this topic in the prior edition of this work. It has served as the inspiration for the current chapter.

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69 Anesthesia for Ophthalmic Surgery

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KEY POINTS

- Ophthalmic procedures are considered to be "low-risk." However, the patient population is higher risk because of the extremes of age involved and associated comorbidities.
- Some ophthalmic surgeries (cataract, glaucoma, simple vitrectomy) are short procedures, but high in volume. The demand for efficiency while maintaining patient safety is a challenge.
- The majority of ophthalmic procedures can be performed under topical anesthesia or orbital block in combination with monitored anesthesia care (MAC).
- A working knowledge of the anatomy and physiology of the eye is essential in providing safe anesthesia care. This includes the effect of anesthetic drugs and interventions on intraocular pressure and systemic effects of ophthalmologic medications.
- Orbital blocks, particularly retrobulbar blocks, can have severe complications, including retrobulbar hemorrhage, and retrograde spread of local anesthetic into the subarachnoid space causing brainstem anesthesia, loss of consciousness, and respiratory arrest.
- Routine preoperative laboratory testing is not necessary for cataract surgery and has not been shown to reduce adverse perioperative events.
- General anesthesia is required in 30% to 40% of ophthalmic surgeries. This includes pediatric ophthalmic surgery; complex procedures, some of which require muscle relaxation; and adults who cannot undergo MAC for a variety of reasons.
- Strabismus surgery is an independent risk factor for postoperative vomiting in pediatric patients.
- Succinylcholine can be used in unfasted patients who have an open-globe injury that requires emergent vision-saving surgery under general anesthesia.
- Prevention of prolonged postoperative anesthesia care unit stay and unanticipated hospital admission requires careful patient evaluation, optimization of underlying medical conditions, adequate pain control, prevention of postoperative nausea and vomiting, and maintenance of hemodynamic stability.

Overview of Ophthalmic Anesthesia

Vision is one of the most important functions of the human body. Visual impairment and blindness limit a person's ability to function normally in daily living,[1-3] has a large economic impact in the United States, and results in significant loss of quality-adjusted life years.[4,5] Currently, about 4.2 million adults in the United States are visually impaired.[6] Early diagnosis and treatment, including surgery, can potentially reverse vision impairment, such as in cataract extraction, or delay and attenuate the pathophysiological process, as in glaucoma and diabetic retinopathy. Anesthesia providers specialized in ophthalmic anesthesia play an important role in helping patients undergo surgery comfortably and safely.[6]

Ophthalmic surgeries are the most common operations performed in the elderly.[7] They consist of a broad spectrum of procedures, ranging from cataract surgery with minimal sedation to more complex procedures such as orbital decompression, or combined corneal transplantation and retinal surgery that requires general anesthesia. The majority of ophthalmic surgeries are short in duration and primarily performed in ambulatory surgical centers. Patients who undergo ophthalmic surgery can be as young as preterm infants to nonagenarians. Ophthalmic procedures are generally considered low risk because they do not cause significant physiological perturbations, and do not involve a large volume of blood loss or significant postoperative pain.[8] However, ophthalmic surgery is often carried out in elderly patients who have a high prevalence of coexisting illnesses such as diabetes, hypertension, coronary artery disease, and chronic obstructive pulmonary disease. Adequate preoperative evaluation and optimization are important in the prevention of same day cancellation and unplanned hospital admission.

Patient safety is paramount in ophthalmic anesthesia. In the American Society of Anesthesiologists (ASA) Closed Claims Project from 1980 to 2000, the identifiable causes of severe eye injury include intraoperative patient movement, either under general anesthesia or monitored anesthesia care (MAC), and needle trauma related to an orbital block with or without patient movement.[9,10] In an ASA closed claims analysis of injury and liability associated with MAC for ophthalmic and nonophthalmic surgeries, the major mechanisms of injury included inadequate oxygenation or ventilation, cardiovascular event, equipment failure, inadequate anesthesia leading to patient movement, and oversedation leading to death or permanent brain damage.[11] Presently, there is little data available regarding postoperative complications associated with ophthalmic anesthesia. A retrospective study of prolonged postanesthesia care unit (PACU) stay following ophthalmic surgery found the incidence was about 0.6%,[12] with the major factors being hypotension, bradycardia, postoperative nausea and vomiting (PONV), and oversedation. A similar study found new onset cardiac dysrhythmia, pain, PONV, and pulmonary-related events (hypoxia and pulmonary aspiration) were the main reasons for unintended hospital admission with an incidence of 0.23%.[13] Overall, ophthalmic surgery is safe with a low incidence of anesthesia-related complications.

Fast-track surgery has been successfully implemented in ophthalmic surgery to facilitate early discharge home and to allow patients to resume normal activities of daily living.[14] Each patient's anesthetic plan should be personalized based on the type, duration, invasiveness of surgery, underlying comorbidities, degree of anxiety, expectation of anesthesia, and history of anesthetic complications with the goal of "fast-tracking" and minimizing potential adverse events postoperatively. Perioperative surgical home is a patient-centric, team-based model of care to improve population health, reduce costs, and satisfy patients.[15] It can improve outcome[16] and may be a useful model in ambulatory surgery settings, including ophthalmic surgery.

Patient satisfaction has become a standard indicator of value in health care, including anesthesia care.[17,18] For ophthalmic surgery, information, communication, pain control, comfort, and support are the most important predictors for patient satisfaction in the perioperative period.[19,20]

OCULAR ANATOMY

The eye is a delicate and complex organ that allows us to see images, color, and movement, and helps with depth perception and balance. The eye is a sphere measuring approximately 24 mm in diameter. It is situated in the pyramidal bony orbit. The wall of the globe has three layers: the sclera, the uveal tract, and the retina. Fig. 69.1 shows the individual components of the eye.

The sclera is the outermost layer. It is the tough, fibrous white of the eye, and it is continuous with the cornea anteriorly. The tissue where the cornea and sclera meet is the limbus, which contains stem cells responsible for regeneration of the epithelium. About 60% of the focus power of the eye is from the curvature of the cornea.

The middle layer, the uveal tract, has three structures: the choroid, the iris, and the ciliary body. The choroid is a layer of blood vessels located posteriorly. Bleeding in this layer is one cause of intraoperative expulsive hemorrhage. The pigmented iris controls light entry with muscle fibers that change the size of the pupil. Sympathetic stimulation dilates the pupil by causing iris dilator muscles to contract, whereas parasympathetic stimulation causes meiosis, or pupillary constriction by causing the iris sphincter muscles to contract. The ciliary body lies just behind the iris; it produces aqueous humor. Ciliary muscle fibers adjust the focus

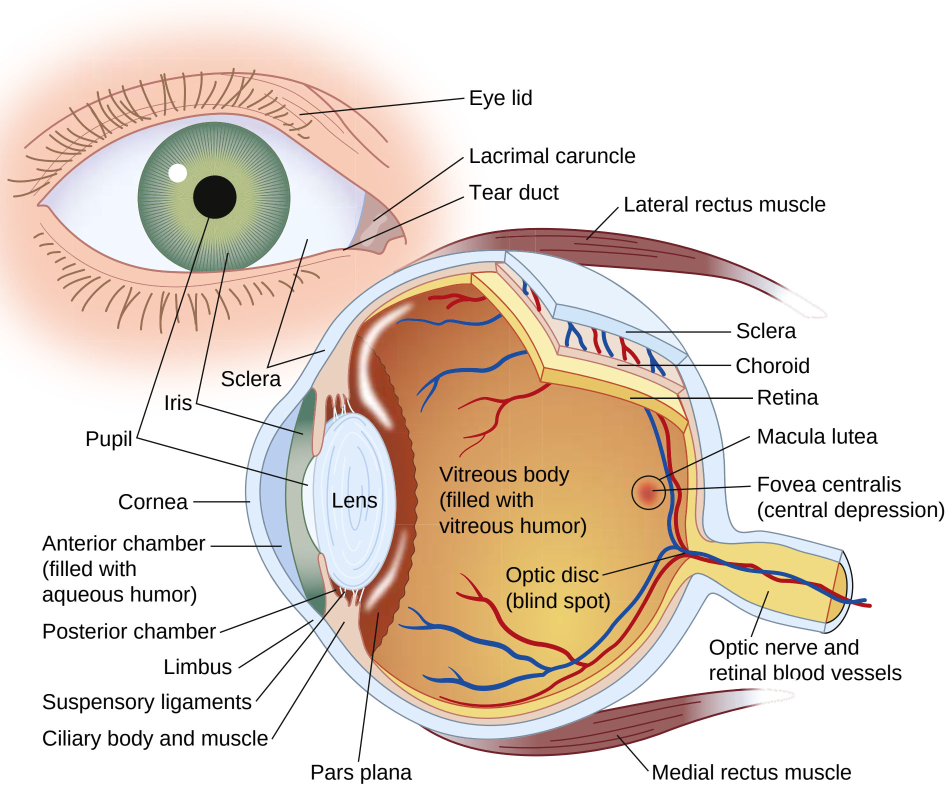

Fig. 69.1 Anatomy of the human eye.

by releasing tension on the suspensory fibers, or zonules, of the lens. Opacification of the lens causes a cataract. Uveitis is an inflammatory condition of these structures (iris, choroid, and ciliary body).

The innermost eye layer is the retina, which is a membrane consisting of highly specialized nerve tissue continuous with the optic nerve. Light stimulates retinal photoreceptors to produce neural signals that the optic nerve carries to the brain. There are no capillaries in the retina; the choroid layer provides oxygen and nourishment to the retina. Retinal detachment from the choroid layer compromises the retinal blood supply and is a major cause of vision loss. The retinal layer ends approximately 4 mm behind the iris. The area between the limbus of the cornea and the retina is called the *pars plana*. Because there is no retinal layer there, it is a safe entrance site for vitrectomy procedures.

The center of the eye is filled with vitreous gel. This thick fluid has attachments to blood vessels and the optic nerve. Traction of the vitreous on the retina is a cause of retinal detachment. Scarring, bleeding, or opacification of the vitreous is treated by vitrectomy.

The extraocular muscles move the globe within the orbit. They arise from a fibrous ring near the apex of the orbit and insert on the sclera. The six extraocular muscles lie within a cone behind the eye surrounding the optic nerve, ophthalmic artery and vein, and ciliary ganglion.

The eyelids have an outer layer of skin, a muscle layer, a tarsal plate of cartilage, and a layer of conjunctiva. The conjunctiva is a mucous membrane that lines the inner eyelids and covers the globe up to the corneal-scleral junction.

The lacrimal gland sits in the superior temporal orbit. It releases tears across the surface of the globe. Tears drain via the puncta near the medial canthus of the eyelids. Tears flow through the canaliculi to the lacrimal sac and duct, to drain into the nasopharynx.

The ophthalmic artery provides most of the blood supply to the orbital structures. It is a branch of the internal carotid artery, close to the circle of Willis. The superior and inferior ophthalmic veins drain directly into the cavernous sinus.

Cranial nerves (CN) innervate the ocular structures. The optic nerve (CN II) carries the neural signals from the retina. The oculomotor (CN III), trochlear (CN IV), and abducens (CN VI) control the extraocular muscles. Touch and pain sensations are carried via the trigeminal nerve (CN V). Sensation to the lower lid is via the maxillary nerve (CN V_2). Sensation to the upper lid is via the frontal branch of the ophthalmic nerve (CN V_1). The nasociliary branch of the ophthalmic nerve sends sensory fibers to the medial canthus, lacrimal sac, and ciliary ganglion.

The ciliary ganglion provides sensory innervation to the cornea, iris, and ciliary body. Parasympathetic fibers originate from the oculomotor nerve (CN III) and synapse in the ciliary ganglion before supplying the iris sphincter muscle. Sympathetic fibers originate from the carotid plexus and travel through the ciliary ganglion to innervate the dilator muscle of the iris. Local anesthetic blockade of the ciliary ganglion produces a fixed, mid-dilated pupil.

The facial nerve (CN VII) exits the base of the skull from the stylomastoid foramen. It supplies motor innervation to the orbicularis muscle via the zygomatic branch. Local anesthetic block of the facial nerve can prevent lid squeezing.

OCULOCARDIAC REFLEX

The oculocardiac reflex was first described by Aschner and Dagnini in 1908. It is also known as the *trigeminovagal reflex*. Traction on the extraocular muscles or pressure on the globe causes bradycardia, atrioventricular block, ventricular ectopy, or asystole. In particular, it is seen with traction on the medial rectus muscle, but it can occur with stimulation of any of the orbital contents, including the periosteum. The response fatigues with repeated stimulation.

The afferent of the reflex limb arises from the ophthalmic division of the trigeminal nerve and continues to the Gasserian ganglion and the sensory nucleus of the trigeminal nerve near the fourth ventricle. Here, the afferent limb synapses with the motor nucleus of the vagus nerve. The efferent impulses travel to the heart via the vagus nerve leading to decreases in both heart rate and contractility of the heart.

The reflex may be seen more often with procedures performed with topical anesthesia, and it occurs commonly in pediatric patients undergoing strabismus surgery. Retrobulbar block is not uniformly effective at preventing the reflex. Orbital injections can also trigger the response. The response is exacerbated by hypercapnia or hypoxemia.[21,22] In the event of arrhythmia, the anesthesiologist should first ask the surgeon to stop manipulations. Any condition that may exaggerate the reflex such as hypoxia, hypercapnia, and an inadequate depth of anesthesia should be assessed and treated. If significant bradycardia persists or recurs, intravenous glycopyrrolate or atropine can be given. Rarely, epinepherine is given to treat severe bradycardia or asystole. Rarely, epinephrine is given to treat severe bradycardia or asystole.

INTRAOCULAR PRESSURE

Normal intraocular pressure (IOP) is 16 ± 5 mm Hg and a value in excess of 25 mm Hg is considered pathological. Normal IOP is necessary to maintain cornea curvature and a proper refracting index of the eye. The intraocular perfusion pressure, defined as the difference between the mean arterial pressure and the IOP, is part of the system regulating blood perfusion to the eye's internal structures. High IOP impairs the blood supply, leading to a loss of optic nerve function.

The globe is a relatively noncompliant compartment. The volume of the internal structures is fixed except for aqueous fluid and choroidal blood volume. The quantity of these two factors regulates IOP. 80% to 90% of aqueous formation occurs through active secretion by the ciliary body mediated by Na-K ATPase and carbonic anhydrase enzymes. The remainder is from passive filtration and ultrafiltration across the ciliary epithelium. It then enters the angle of the anterior chamber to flow through the trabecular meshwork into the Schlemm canal and the episcleral veins. IOP is primarily regulated by the resistance at the trabecular meshwork.[23]

Impairment of aqueous drainage at any point can elevate the IOP. Sclerosis of the trabecular meshwork is believed to cause the chronic pressure elevation in open-angle glaucoma. Closed-angle glaucoma occurs when there is an obstruction to aqueous drainage from closure of the anterior chamber angle. This happens from peripheral iris swelling or anterior displacement. Patients with a preexisting narrow angle may be predisposed to this condition. The acute increase in pressure causes severe pain and is an ophthalmologic emergency.

Anesthetic drugs and the conduct of anesthesia have important effects on IOP. All volatile anesthetic drugs cause a decrease in IOP.[24-26] Nitrous oxide (N_2O) has no effect on IOP. The commonly used intravenous induction anesthetics such as propofol, thiopental, and etomidate all reduce IOP, with propofol causing a moderate reduction in IOP even when used as low-dose intravenous sedation.[27] Ketamine does not increase IOP (see the section Anesthesia for Pediatric Ophthalmology). The mechanism by which anesthetic drugs reduce IOP is not fully elucidated but involves depression of central nervous system ocular centers, resulting in relaxation of extraocular muscle tone.[27] Short-acting opioids such as fentanyl, alfentanil, and remifentanil decrease IOP on induction of anesthesia.[28,29] Midazolam has little effect on IOP,[30] and sedation with midazolam has been used to facilitate the measurement of IOP in children.[31] Nondepolarizing neuromuscular blocking agents (NMBAs) such as rocuronium, vecuronium, and atracurium have little effect on IOP. Succinylcholine increases IOP by about 8 to 10 mm Hg[32] due to a variety of postulated mechanisms including reduced aqueous humor outflow, increased choroidal blood volume, and increased central venous pressure.[33,34] IOP increases after reversal of nondepolarizing NMBA with neostigmine and atropine but remains unchanged with sugammadex.[35]

The most significant rise in IOP during general anesthesia occurs at laryngoscopy and emergence; the increase in IOP is even greater with repeat laryngoscopy.[36] Video-guided laryngoscopy causes a smaller increase in IOP than direct laryngoscopy.[37,38] Insertion of a laryngeal mask airway results in little or no increase in IOP.[39,40] Other anesthetic interventions that increase IOP include compression of the eyes by ventilation, hypoxia, hypercapnia, and hypertension.[34] Coughing, straining, or vomiting can increase IOP 30 to 40 mm Hg. A poorly placed anesthesia mask can put enough pressure on the eye to reduce blood flow to zero. Ocular blocks initially increase IOP by 5 to 10 mm Hg, but this falls to below baseline values within 5 minutes. Peribulbar blocks cause the greatest increase in IOP, likely due to the greater volume of local anesthetic that is injected.[41]

Physiological factors that increase IOP include being placed in supine, prone, or Trendelenburg positions. A normal blink increases the IOP by 10 mm Hg, while a forceful lid squeeze can increase IOP to more than 70 mm Hg.[42]

OPHTHALMIC DRUGS

Ophthalmic medications can be delivered by topical, ocular (intravitreal, subconjunctival, retrobulbar, and intracameral), and systemic methods. Topical eye drops may be absorbed systemically; significant routes of systemic absorption occur at conjunctival capillaries and nasal mucosa. Less significant absorption can occur through the lacrimal drainage system, pharynx, gastrointestinal tract, skin at the cheek and eyelids, and at the aqueous humor and inner ocular tissues.[43] When systemic therapy is required, the blood ocular barrier may inhibit passage of lipophilic drugs; however, this barrier may be impaired by ocular inflammation, intraocular surgery and trauma, or ocular diseases.[44] The population at risk of systemic side effects of eye drops are children and the elderly because of anatomical differences, risk of toxicity, the presence of concomitant diseases, and polypharmacy. Most drugs have negligible excretion into breastmilk, with the exception of Timolol eye drops, which can potentially cause adverse effects in the nursing infant.[45]

The total dose of a drug delivered by topical eye drop can be significant. Consider that one drop (typically 50 μL) of 10% phenylephrine contains 5 mg of drug, which is appreciably more than the intravenous dose of phenylephrine (0.05-0.1 mg). Adverse reactions such as hypertension, arrhythmias, and adverse cardiac events have been reported.[46]

Systemically administered drugs such as acetazolamide and mannitol can produce side effects such as fluid or electrolyte disturbance that can affect anesthetic management of these patients. Table 69.1 lists the systemic effects of ophthalmic medications.

OPHTHALMIC PROCEDURES

1. CATARACT EXTRACTION
   Cataracts are opacities of the crystalline lens of the eye. Cataract extraction is performed under topical or regional block. General anesthesia is rarely used.
   a. Phacoemulsification is the technique of choice for cataract surgery and refers to the use of ultrasonic vibration of fragments of the lens with simultaneous irrigation and aspiration. This technique allows for very small incisions. Femtosecond laser is a relatively new technique where laser energy is used for corneal incision, capsulotomy, and fragmenting the lens. Supplemental oxygen is contraindicated during its use because of fire risk.
   b. Extracapsular cataract extraction refers to the removal of the lens, while leaving the posterior lens capsule and zonules intact to allow implantation of intraocular lens.
   c. Intracapsular cataract extraction is the total removal of the opaque lens with the lens capsule, and it is rarely performed due to large incision and a relatively high rate of complications.
2 GLAUCOMA PROCEDURES
   d. Trabeculectomy is a glaucoma surgery that creates a transscleral fistula to allow aqueous humor to drain into the subconjunctival space. Mitomycin C or 5-flurouracil (5-FU) is usually used to prevent scarring of the flap.
   e. Baerveldt and Ahmed devices are drainage implantable devices that shunt aqueous humor under the conjunctiva.
   f. Minimally invasive glaucoma surgery (MIGS) includes procedures such as CyPass, iStents, and trabectome.
3. CORNEAL PROCEDURES
   a. Penetrating keratoplasty is a full thickness corneal transplant.

TABLE 69.1 Systemic Side Effects of Ophthalmic Drugs

Drug	Mechanism	Use	Side Effects
Acetylcholine	Cholinergic agonist	Meiosis	Bradycardia, bronchospasm, hypotension
Acetazolamide (PO, IV, IM)	Carbonic anhydrase inhibitor	Decrease IOP, Glaucoma	Confusion, drowsiness, hypokalemia, hyponatremia, metabolic acidosis, abnormal hepatic function tests, polyuria, renal failure
Anti-VEGF, e.g., ranibizumab, aflibercept	Inhibits vascular endothelial growth factor	Inhibits neovascularization	Conjunctival hemorrhage, eye pain, endophthalmitis, uveitis, stroke (in high-risk patients)
Atropine	Anticholinergic	Mydriasis	Dry mouth, dry skin, fever, agitation (central anticholinergic syndrome)
Cyclopentolate	Anticholinergic	Mydriasis, cycloplegia	Central anticholinergic syndrome (see atropine)
Echothiopate	Irreversible cholinesterase inhibitor	Glaucoma	Total body inhibition of plasma cholinesterase Prolongs effects of succinylcholine
Epinephrine	α-, β agonist	Mydriasis, decrease IOP	Hypertension, tachycardia, ventricular arrhythmias
Mannitol	Osmotic diuretic	Decrease IOP	Initial increase in circulating blood volume; congestive heart failure in patients with poor ventricular function
Phenylephrine	α Adrenergic agonist	Mydriasis, vasoconstriction	Hypertension
Pilocarpine	Cholinergic	Constrict pupil	Bradycardia, bronchospasm
Scopolamine	Anticholinergic	Mydriasis, cycloplegia	Central anticholinergic syndrome (see atropine)
Tamsulosin	α_1 antagonist	Benign prostatic hyperplasia	Floppy iris syndrome
Timolol	β_1 and β_2 antagonist	Glaucoma	Bradycardia, bronchospasm, exacerbate congestive heart failure

IM, Intramuscular; *IOP*, intraocular pressure; *IV*, intravenous; *PO*, per os, by mouth; *VEGF*, vascular endothelial growth factor.

b. Lamellar keratoplasty—In Descemet stripping endothelial keratoplasty (DSEK) and Descemet membrane endothelial keratoplasty (DMEK) procedures, only the diseased endothelium is removed and replaced with a corneal graft.

c. Pterygium excision—A pterygium is an abnormal growth of fleshy tissue on the conjunctiva. Excision is performed when it impinges on the cornea, affecting vision, or for cosmesis.

4. VITREORETINAL SURGERY

a. Vitrectomy is the surgical removal of the vitreous humor and its replacement with a physiologic solution. A posterior vitrectomy is indicated for the removal of foreign bodies such as "dropped lens" through the torn posterior capsule during cataract surgery, to repair retinal detachments, to remove membranes and media opacities, and to alleviate vitreous traction on the retina.

b. Scleral buckle is a procedure used for the treatment of retinal detachment. A silicone band is placed around the globe within the extraocular muscles under the conjunctiva to push the sclera toward the detached retina. It can be used as the sole treatment or in combination with vitrectomy to treat retinal detachment.

c. Radioactive plaque implantation is done for the treatment of choroidal melanoma. It is frequently combined with prophylactic vitrectomy with silicone oil injection to prevent retinal detachment from radiation.

5. OCULOPLASTIC SURGERY

a. Eyelid procedures. Eyelid procedures include the correction of ectropion (eyelid turning outward), entropion (eyelid turning inward), ptosis (drooping of the upper eyelid), and blepharoplasty (to remove redundant tissue of the eyelid).

b. Dacryocystorhinostomy refers to the surgical reopening of the obstructed channel between the lacrimal sac and the nasal cavity caused by a congenital defect or chronic infection.

c. Orbital surgery includes repair of blowout fractures, drainage of an orbital abscess, decompression for exophthalmos caused by hyperthyroidism, or tumor excision in the orbit or optic nerve.

d. Evisceration, enucleation, and exenteration. Evisceration refers to the removal of the contents of the eye. Enucleation refers to removal of the eye itself including the globe, but leaving orbital contents such as bone, extraocular muscles, and fat in place. Exenteration refers to the removal of the entire contents of the orbit including the lacrimal gland, optic nerve, and orbital bones.

e. Tarsorrhaphy is the partial or total suturing together of the eyelids.

Preoperative Evaluation

Exclusion criteria for ambulatory ophthalmic surgery under general anesthesia include noncompensated congenital cardiac and pulmonary disease, and some syndromes with multiple system involvement in pediatric patients. Severe cardiomyopathy, pulmonary hypertension, home-oxygen dependence, and super-morbid obesity (body mass index >50) are exclusion criteria in adult patients. Known difficult airway in either group may preclude outpatient surgery.

LABORATORY STUDIES

Previous investigations have shown that preoperative laboratory tests are typically normal before low-risk ambulatory surgery,[47,48] and the ASA recommends that routine preoperative laboratory testing before cataract surgery is not necessary in ASA physical status classification I and II patients.[49] Routine preoperative testing has not been shown to decrease cancellation rate and adverse events, or to improve outcomes.[50] While this is not extensively studied in other types of ophthalmic surgical procedures, a study examining preoperative medical testing for patients undergoing vitreoretinal surgery showed that preoperative testing did not influence rates of postoperative systemic complications.[51] Any laboratory testing, including an electrocardiography, should be based on the patients' history and physical findings rather than the indiscriminate ordering of a battery of tests.

CARDIOVASCULAR EVALUATION

The American Heart Association and American College of Cardiology have published guidelines for perioperative cardiovascular evaluation for noncardiac surgery.[52] Ophthalmic procedures, such as cataract extraction, are specifically identified as low-risk procedures. For these procedures, evaluation is focused on patients with major clinical predictors of risk.

ANTICOAGULATION

Many elderly patients undergoing eye surgery are on antiplatelet or warfarin therapy. Perioperative management of anticoagulants involves weighing the risks of thrombosis versus hemorrhagic complications. In a study of more than 19,000 cataract procedures, the incidence of hemorrhagic and thrombotic complications was low.[53] The risk of hemorrhagic complications depends on the degree of anticoagulation and the hemorrhagic potential of the surgical procedure. Serious hemorrhagic complications are most probable in orbital and oculoplastic surgery; of intermediate probability in vitreoretinal, glaucoma, and corneal transplant surgery; and least likely in cataract surgery.

It is safe to proceed with cataract surgery without stopping antiplatelet or warfarin therapy provided that the international normalized ratio level is within the therapeutic range.[54] Ocular blocks are rarely associated with severe hemorrhagic complications. For intermediate risk surgery such as oculoplastic and glaucoma surgery, warfarin and antiplatelet drugs may increase the risk of intraoperative or postoperative bleeding.[55,56] MIGS are associated with a 100% rate of hyphema, and the effects of anticoagulation therapy in patients undergoing these procedures is unclear.[57]

There is limited data on the risk of hemorrhagic complications during eye surgery in patients who are taking novel oral anticoagulants (NOACs). A recent systematic review and meta-analysis found that NOACs reduced the risk of intraocular bleeding by onefifth compared with warfarin.[58] However, another study comparing NOACs with antithrombotic drugs found no difference in the risk of substantial intraocular bleed, but the number of adverse events was scarce.[59] In the authors' institution, the ophthalmologist requests patients to stop NOAC therapy 2 days before intermediate-risk surgery.

If an interruption of anticoagulation treatment is needed, an individualized approach is recommended to minimize the risk of perioperative bleeding. Bridging therapy should be considered in patients with a high risk for thromboembolic events.

Orbital Blocks

Intraocular surgery can be performed under local, regional, or general anesthesia. Regional anesthesia is standard for most ophthalmic procedures such as cataract, glaucoma, cornea, and vitreoretinal surgeries.[60] Regional anesthesia provides akinesia (immobility) and anesthesia of the eye. The requirement for akinesia will vary by type of surgery and surgeon preference. Facial nerve block may be required to obtain akinesia of the orbicularis oculi muscle of the lid, as its motor innervation is located outside the cone.

Regional anesthesia techniques are generally reliable, safe, and provide good postoperative analgesia. Compared with general anesthesia, regional anesthesia is less likely to be associated with PONV. Patients are often able to bypass Phase 1 recovery with a quicker discharge home. Patients often view eye blocks with anxiety and apprehension, and may have fears of being aware and awake during their procedure. Some patients may not be able to cooperate, to lay flat, or lie still during surgery, and therefore may not be able to tolerate surgery under regional anesthesia alone.

RETROBULBAR BLOCK

Retrobulbar block has long been considered the gold standard of regional techniques for ophthalmic surgery until the 1990s with the introduction of peribulbar block and sub-Tenon block. Retrobulbar block is achieved by injecting local anesthetic inside the muscular cone (Fig. 69.2).

With the globe in primary gaze, a 3-cm, 23- to 27-gauge needle with its bevel opening faced toward the globe is placed at the junction of the inferior and lateral walls of the orbit just above the inferior orbital rim.[61] The needle is advanced parallel to the orbit floor (with a 10-degree elevation from the transverse plane) for approximately 15 mm until it is past the equator of the eye. The needle is turned medially and slight upward to aim toward an imaginary point behind the globe on the axis formed by the pupil and the macula where 2 to 5 mL of local anesthetic solution is injected. The needle tip approaches but does not pass the midsagittal plane of the globe. Some intorsion on downgaze is expected because the superior oblique muscle is outside the muscle cone and may not be blocked. Many modifications to the classical retrobulbar anesthetic injection have been described to minimize complications of the block. Traditionally, blunt-tipped needles were advocated because they were thought to protect against ocular trauma and because they allowed more accurate definition of tissue planes. However, studies have shown that blunt needles are as likely as sharp needles to cause globe penetration and optic nerve injury. Fine cutting needles produce minimal tissue distortion with little pain.[62] Compared with peribulbar block, retrobulbar block has a quicker onset and is associated with less chemosis.

Retrobulbar hemorrhage complicates about 1% of retrobulbar injections.[63] Venous hemorrhage spreads slowly and does not usually result in long-term visual problems. Arterial hemorrhage can produce rapid orbital swelling, marked proptosis with immobility of the globe, inability to close the lids, and massive blood staining of the lids and conjunctiva. Compressive hematoma can threaten retinal perfusion leading to severe visual loss. Ophthalmoscopic examination should be performed to evaluate for ischemic damage to the optic nerve or retina. IOP should be measured and a lateral canthotomy may need to be performed to decompress the orbit.

Intravascular injection can occur despite a negative aspiration test. The total dose of local anesthetic used is small, and systemic effects are unlikely, even if the total dose is given intravenously. Accidental intraarterial injection can cause central nervous system excitation and seizures. This occurs because of retrograde passage of local anesthetic solution from the ophthalmic artery to the internal carotid artery and delivery to the thalamus and other midbrain structures. Injection of local anesthetic or retrograde tracking of local anesthetic along the optic nerve sheath into the subdural or subarachnoid space causes partial or total progressive brainstem anesthesia. Wide ranging symptoms occur, from aphasia, confusion, and dysphagia to apnea, cardiac arrest, loss of consciousness, and seizures.[64,65] Management is supportive and should result in total recovery within several hours.

Optic nerve damage, or globe perforation with retinal detachment and vitreous hemorrhage, are devastating complications of retrobulbar block. Risk factors include physician inexperience and a highly myopic eye (axial length longer than 25 mm). This complication is associated with a poor prognosis, particularly in cases of delayed diagnosis.[66]

PERIBULBAR BLOCK

Peribulbar block was first reported in 1986.[67] The needle is introduced outside the cone, thus preventing retrobulbar hemorrhage and reducing the risk of optic nerve injury (see Fig. 69.2). Many modifications of this block exist; the classic technique involves two injections—one inferiorly and temporally and the second superiorly and nasally. Peribulbar anesthesia may also be administered with one injection; a 3 cm, 23-gauge Atkinson needle is placed at the junction of the middle and lateral thirds of the lower lid just above the inferior orbital rim. The needle should be directed vertically backward, parallel to the floor of the orbit. The needle

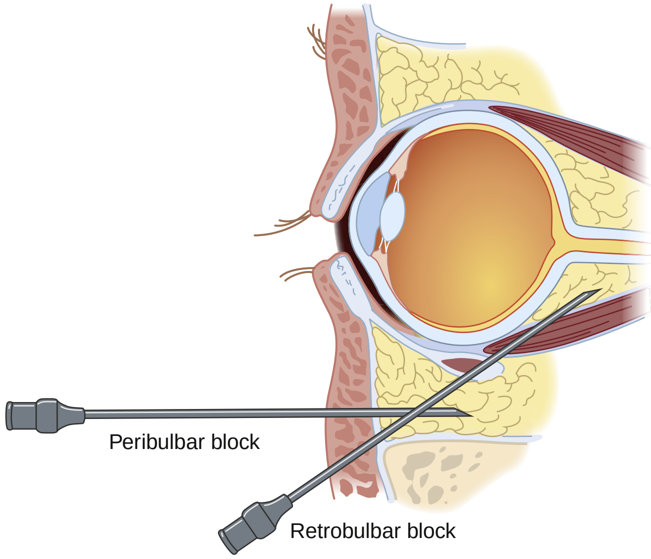

Fig. 69.2 Position of needles for retrobulbar and peribulbar blocks. (Redrawn from Spaeth GL. *Ophthalmic Surgery: Principles and Practice*. Philadelphia: WB Saunders; 1982.)

insertion depth should be less than 25 mm. If contact is made with bone, the needle should be redirected slightly upward then 5 to 10 mL of local anesthetic is injected.[68] Studies support the use of peribulbar block in providing equivalent patient analgesia and operating conditions as retrobulbar block for a variety of ophthalmic procedures.[69,70]

SUB-TENON BLOCK

To avoid the complications of sharp needles, a technique was developed using a blunt cannula under the fascia of Tenon.[71] Various lengths of cannulas have been used. Using topical anesthesia with sedation, a speculum is placed to retract the lids. A 2- to 3-mm spot of cautery can be made 5 mm from the limbus in the inferonasal or inferolateral quadrant. A 2-mm incision is made in the conjunctiva with blunt dissection through the fascia. A blunt cannula is directed posteriorly, but not beyond the equator of the globe, with injection of 1 to 3 mL of local anesthetic. A small degree of conjunctival edema is often seen. Analgesia is usually excellent.

FACIAL NERVE BLOCK

A facial nerve block is performed to prevent squeezing of the eyelids, which interferes with surgery and increases IOP. Akinesia of the orbicularis muscle, which is supplied by the facial nerve, can be achieved by blocking the nerve at its terminal branches (Van Lint block) or proximal trunk (O'Brien or Nadbath-Rehman block).

1. Van Lint block: the needle is placed 1 cm lateral to the orbital rim, and 2 to 4 mL of anesthetic is injected along the superolateral and inferolateral orbital rims. The modified Van Lint block is a common variant achieved by inserting the needle 1 cm more lateral than the original insertion point to avoid lid edema. The disadvantages of this block include patient discomfort, proximity to the eye, and postoperative ecchymoses(Figure 69.3A).

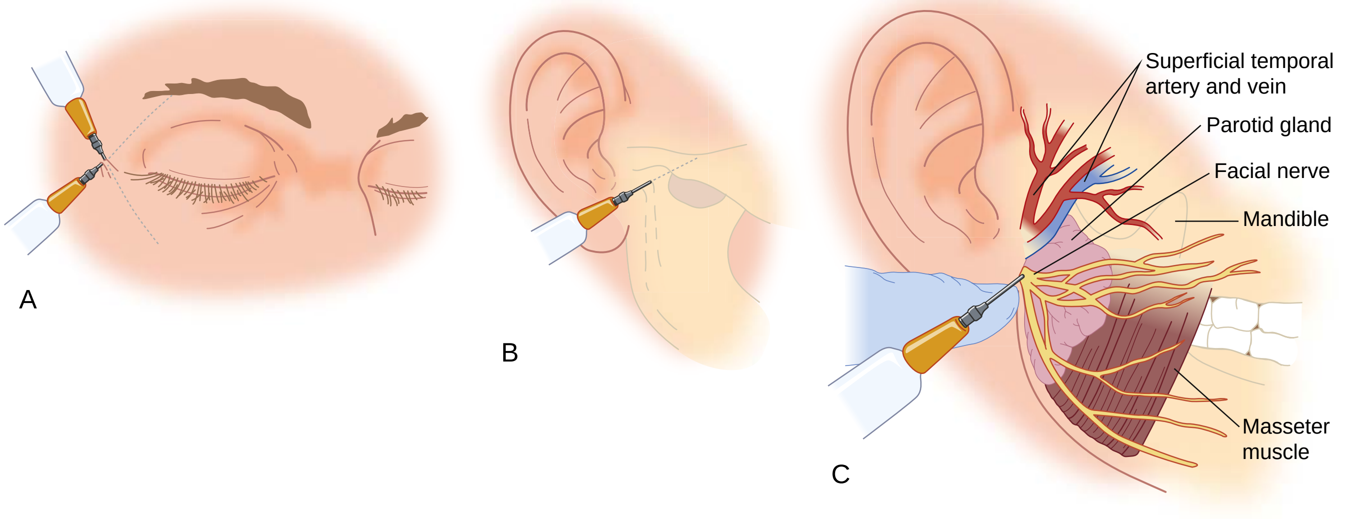

Fig. 69.3 Facial Nerve Blocks. (A) Van Lint; (B) O'Brien; (C) Nadbath-Rehman. (Redrawn from Spaeth GL. *Ophthalmic Surgery: Principles and Practice.* Philadelphia: WB Saunders; 1982.)

2. O'Brien block: the mandibular condyle is palpated inferior to the posterior zygomatic process and anterior to the tragus of the ear as the patient opens and closes the jaw. The needle is inserted perpendicular to the skin approximately 1 cm to the periosteum. As the needle is withdrawn, 3 mL of anesthetic is injected (Figure 69.3B).
3. Nadbath-Rehman block: a 12-mm, 25-gauge needle is inserted perpendicular to the skin between the mastoid process and the posterior border of the mandible. The needle is advanced its full length, and after careful aspiration, 3 mL of anesthetic is injected as the needle is withdrawn. This blocks the entire trunk of the facial nerve. The patient should be told to expect a lower facial droop for several hours postoperatively. The major disadvantage to this block is the proximity of the injection to important structures, such as the carotid artery and the glossopharyngeal nerve (Figure 69.3C).

LOCAL ANESTHETIC SOLUTIONS FOR REGIONAL TECHNIQUES

For local anesthesia, we use a 1:1 ratio of bupivacaine 0.75% and lidocaine 2% without epinephrine. Hyaluronidase is added to speed tissue penetration. Hyaluronidase can also be important in preventing anesthetic-related damage to the extraocular muscles. A human recombinant brand (Hylenex) is commercially available.

Monitored Anesthesia Care

Approximately 60% to 70% of ophthalmic procedures can be accomplished by a combination of orbital blocks and MAC. In the last 2 decades, the dramatic improvement in surgical techniques in ophthalmic surgery has had significant impact on anesthesia management rendering many ophthalmic procedures less invasive and shorter in duration.

MAC with regional anesthesia provides many advantages over general anesthesia in lowering postoperative morbidity, mortality, and pulmonary complications.[72,73] There is better postoperative pain control,[74] a lower incidence of intraoperative hypotension, faster postoperative recovery,[75] less PONV, and less unplanned hospital admission.[76]

Other than cataract surgery, most ophthalmic procedures require orbital blocks that are quick to perform but painful for the patient. Intravenous sedation and analgesia is provided to prevent pain and anxiety.[77] Patient movement during orbital blocks, either from pain, anxiety, under sedation, or over sedation, has been linked to complications leading to blindness. Other unique intraoperative conditions make ophthalmic surgery challenging. Many elderly patients are anxious[20] and cognitively impaired,[78] making it difficult for them to lie flat and still during surgery. Covering the face with surgical drapes and rotation of the bed 90 or 180 degrees away from the anesthesia provider also increases difficulty by limiting access to the airway.

The technique for MAC is highly variable and depends on the training and experience of the anesthesia provider, the environment of practice, availability of drugs, and the surgeons' and patients' expectation for sedation. There is no universal regimen for MAC.[79]

Intravenous sedation is a continuum ranging from mild and moderate sedation to deep sedation that overlaps with general anesthesia.[80] The depth of sedation can quickly increase to a deeper level depending on age, weight, cardiac function, genetics, and general health. Some patients have a high tolerance to sedatives while others may be more sensitive. This variability presents a challenge for the fast-track practice of ophthalmic anesthesia. The newly published "Practice Guidelines for Moderate Procedural Sedation and Analgesia 2018"[81] provides evidence-based recommendations for MAC that are also applicable in ophthalmic anesthesia.

TABLE 69.2 Observer's Assessment of Alertness/Sedation Scale

Subscore	Responsiveness	Speech	Facial Expression	Eyes
5	Responds to name spoken in normal tone	Normal	Normal	Clear
4	Lethargic response to name spoken in normal tone	Mild slowing or thickening	Mild relaxation	Glazed mild ptosis
3	Responds only after name spoken loudly or repeatedly	Slurring or slowing	Marked relaxation	Glazed marked ptosis
2	Responds after mild prodding or shaking	Few recognized words		
1	Does not respond to mild prodding or shaking			

From Chernik DA, Gillings D, Laine H, et al. Validity and reliability of the Observer's Assessment of Alertness/Sedation Scale: study with intravenous midazolam. *J Clin Psychopharmacol*. 1990;10(4):244–251.

The challenge of MAC in ophthalmic surgery is to quickly and safely sedate a patient adequately for an ocular block, and still maintain operating room efficiency. Ideally, the metrics of intravenous sedation in ophthalmic surgery should be quantitatively measurable, including the time required to reach the targeted level of sedation and analgesia, and the efficacy of pain control while avoiding respiratory depression or apnea. It is not necessary to use loss of consciousness as an endpoint of sedation. To do so can lead to apnea, hypoxia, involuntary movement in reaction to pain, and hemodynamic instability. During orbital blocks and surgery, patients should be sedated to a moderate level such as subscore 3 of the Observer's Assessment of Alertness/Sedation Scale (Table 69.2). The objective is to have a patient who is comfortable, free of pain and anxiety, yet able to follow commands in order to prevent movement that can lead to ocular trauma. Treating complications that occur during orbital blocks and being prepared to convert to general anesthesia in the event that MAC is inadequate, or the airway compromised are important responsibilities of the anesthesia provider.

MONITORED ANESTHESIA CARE FOR CATARACT SURGERY UNDER TOPICAL ANESTHESIA

Topical anesthesia has become popular due to its ease of administration and avoidance of complications of orbital blocks. Topical anesthesia requires patient cooperation because of the lack of ocular akinesia. Patients undergoing cataract surgery with topical anesthesia may experience some discomfort and require intravenous analgesic medication in small doses.[82] Most patients undergoing cataract surgery want sedation, which can be given either intravenously or orally.[83] Local anesthetic eye drops, such as 0.5% tetracaine, 0.75% bupivacaine, and 2% lidocaine, are very effective in producing analgesia although their effects can dissipate with constant irrigation.

With use of femtosecond laser, it may take up to 5 minutes to appropriately dock and position the patient, however it takes only 30 seconds for laser delivery. Discomfort during the procedure is managed with topical anesthesia. Ideally, sedation should be avoided to allow patient cooperation. If necessary, a small dose of a sedative or analgesic can be given.

Midazolam is the primary sedative used for anxiolysis during cataract surgery with topical anesthesia. Its side effects of over sedation, paradoxical reaction, postoperative neurocognitive disorders, and memory deficit are concerning in the elderly patient. Low-dose propofol, short-acting narcotics, or both are reasonable alternatives. Over sedation during cataract surgery should be avoided to prevent sudden awakening under surgery that may lead to undesired movement secondary to disorientation. Dexmedetomidine used as a sole medication is not appropriate for use in cataract surgery due to its slow onset, inadequate analgesic effect, and long elimination half-life.[83-85] Ketamine alone is also not a good choice due to its dysphoric and hallucinogenic properties. Intraoperative handholding is helpful to provide reassurance and reduce anxiety.

MONITORED ANESTHESIA CARE FOR OPHTHALMIC SURGERY WITH AN ORBITAL BLOCK

Orbital blocks, such as retrobulbar, peribulbar, and sub-Tenon blocks, are commonly performed nerve blocks for ophthalmic procedures. Orbital blocks are performed by either the ophthalmologist or the anesthesia provider. In an ASA Closed Claim Project analysis of complications associated with eye and peripheral nerve blocks, the risk of adverse events was increased when anesthesia providers performed both the orbital block and MAC.[10]

Many sedatives and analgesics, either alone or in combination, are used during the performance of an orbital block.[81] The use of sedatives alone such as midazolam, propofol, sodium thiopental, and dexmedetomidine can provide an adequate level of sedation, but their lack of analgesic effect may lead to involuntary head movement. The sole use of narcotics such as alfentanil, fentanyl, remifentanil, and sufentanil can provide profound analgesia, but inadequate sedation and increased nausea and vomiting.[86]

Propofol is an excellent drug for providing titratable sedation. Its lack of analgesic effect makes it insufficient for orbital blocks as evidenced by a high rate of involuntary movement during injection.[87] When ketamine is combined with propofol, up to onethird of patients who receive the combination will have undesired movement during the block.

The combination of a short-acting opioid such as alfentanil or fentanyl, and propofol, or a triple combination of propofol-midazolam-alfentanil produces a profound synergism of hypnosis and analgesia.[88,89] When opioids are administered in combination with midazolam,[90-92] propofol,[93] sodium thiopental,[94] ketamine,[95] and dexmedetomidine,[96] their sedative effects improved significantly.[91,93]

The use of a mixture of propofol, alfentanil (500 μg/mL), and lidocaine 1% in a 6:2:2 ratio in volume[97] is a novel approach in "balanced anesthesia" during MAC. It contains both a sedative and an analgesic in a single syringe that is easy to titrate based on the patient's age and weight (or adjusted weight in the obese). The addition of lidocaine attenuates the pain from propofol. Within 30 to 90 seconds, most patients reach an OAA/S subscore 3 sedation-level and are ready for the ocular block. This mixture provides excellent analgesia and sedation, as well as hemodynamic stability, with minimal need for airway support. While infusing the mixture bolus, assessing the patient's response to questions allows for detection of the target sedation level. Significant sedation may continue after the block for several minutes, however, conversing with and instructing the patient to breathe deeply will prevent hypoventilation and desaturation.

An orbital block provides analgesia for 2 to 3 hours, which will be sufficient for the duration of most ophthalmic procedures. For more complex procedures such as enucleation, dacryocystorhinostomy, orbital decompression, scleral buckle, and radioactive plaque implantation, the analgesic effect of a retrobulbar block may not be adequate to block all sensation. The addition of intravenous sedation with propofol and narcotics is sufficient for most patients. Low-dose ketamine and dexmedetomidine can be added to the sedation regimen in patients who continue to experience discomfort.

Intranasal dexmedetomidine is well absorbed without the profound bradycardia or hypotension associated with intravenous administration. When given as an adjunct agent, the dose should be reduced, 0.5 to 0.7 μg/kg, administered intranasally as a drop from a tuberculin (TB) syringe. The elimination half-life of dexmedetomidine is about 2 to 3 hours and even longer in elderly patients.[98] Giving a single bolus toward the beginning of the procedure allows the drug to be eliminated during surgery and prevents adverse effects in the PACU. Intranasal dexmedetomidine can also be used in highly anxious patients who are undergoing complex procedures under MAC.

The following are key points of anesthetic management for ophthalmic procedures under MAC.

1. Cataract surgery can be performed under topical anesthesia or an orbital block. Most patients require mild sedation (anxiolysis) for surgery performed under topical anesthesia. Moderate sedation is required for the performance of an orbital block, after which, mild sedation is adequate.
2. Dacryocystorhinostomy: the success of the anesthetic management depends on an adequate initial dose of narcotics and propofol to prevent pain associated with the injection of local anesthetics near the tear duct and inside the nose. A continuous infusion of low-dose propofol and intermittent doses of narcotics, such as fentanyl, are usually necessary to provide adequate analgesia. The patient must be awake enough to protect their airway from aspiration of blood that enters the oropharynx from the nasolacrimal system. Adding intravenous low-dose ketamine or intranasal dexmedetomidine can provide additional analgesia if necessary. If the procedure is bilateral or a reoperation, general anesthesia is likely a better choice.
3. Orbitotomy: the procedure can be done under MAC if performed primarily for debulking of soft tissue without bony involvement.
4. Radioactive plaque implantation: the procedure requires incision of the scleral layer and suturing the plaque between the extraocular muscles. Significant manipulation during the procedure can cause discomfort. Even a well-executed retrobulbar block may not provide complete analgesia; a moderate to deep level of sedation is usually required. Many institutions perform this surgery under general anesthesia, but at the authors' institution, MAC is routinely used. Continuous analgesia with narcotics, and a moderately dosed propofol infusion is highly effective. Low-dose ketamine or dexmedetomidine may be added for those patients who require additional analgesia.
5. Cornea transplant: partial-thickness corneal transplant and penetrating keratoplasty can be carried out under retrobulbar block. Some surgeons also perform a facial nerve block to prevent eyelid squeezing. Intraoperatively, sedation with continuous infusion of propofol and titration of narcotics to achieve a moderate level of sedation is the goal. The patient must be awake enough that they will not move inadvertently especially during penetrating keratoplasty.
6. Enucleation: this procedure can be performed with MAC in appropriate patients. In addition to a retrobulbar block, infiltration of the orbital area with bupivacaine at the end of the procedure can be helpful for postoperative analgesia. The anesthetic management is similar to that of radioactive plaque implantation discussed earlier.
7. Ruptured globe: ruptured globe injury can be repaired under an orbital block and MAC depending on the degree of injury.[99] It is crucial to minimize elevation of IOP during the block and surgery.

SPECIAL CONSIDERATIONS IN MONITORED ANESTHESIA CARE

1. Morbid obesity/obstructive sleep apnea (OSA). Morbid obesity has been identified as an independent predictor of sedation-related cardiopulmonary complications.[100] In addition to obesity-associated comorbidities, such as OSA, restrictive lung disease, diabetes mellitus, and coronary artery disease, obese patients are at increased risk of airway closure and hypoxia during sedation.[101] The pharmacokinetics of drugs are altered in the morbidly obese. Many drugs, including narcotics, should be dosed based on ideal body weight (IBW). When both propofol and alfentanil were given based on adjusted body weight, IBW + 30% of the difference between the patient's total body weight—IBW, the sedation level, analgesic effect, and incidence of airway events were comparable to normal weight patients.[97] Administration of 100% oxygen for several minutes, in addition to supplemental oxygen via nasal cannula before sedation, may alleviate hypoxemia during the orbital block.
2. Elderly patients have increased sensitivity to all sedatives and narcotics,[79,102] as well as increased risk of complications associated with MAC.[11] In addition to a reduction in drug dose requirement, the onset of action will be slower compared with younger patients.[79,97]

General Anesthesia

General anesthesia is indicated in approximately 30% of ophthalmic procedures, primarily for pediatric patients and in adults undergoing complex or invasive procedures whose anesthetic requirement cannot be met by orbital blocks and MAC. Adults with cognitive impairment, hearing loss, language barriers, claustrophobia, or who have a contraindication for an orbital block may require general anesthesia. The special considerations for general anesthesia in ophthalmic surgery should focus on the prevention of intraoperative patient movement that can lead to complications, such as coughing and bucking which can dramatically increase IOP, and the risk of periorbital hemorrhage after procedures such as orbital decompression or enucleation. PONV can cause elevation of IOP, prolonged PACU stay, and unanticipated hospital admission.

Fluid shifts are generally minimal in ophthalmic procedures. Significant blood loss is uncommon except for procedures such as orbital decompression, enucleation and evisceration, and lacrimal duct procedures. The main contributors to hemodynamic instability during general anesthesia in these patients are the patient's underlying comorbidity, such as advanced age, diabetes mellitus, and hypertension. Fluid deficits caused by fasting, autonomic dysfunction due to vascular disease, diabetes mellitus, and Parkinson disease, exaggerate the anesthetic effects on the cardiovascular system.

The laryngeal mask airway (LMA) is widely used in both adult and pediatric patients undergoing ophthalmic procedures that do not require paralysis. Those procedures include strabismus repair, scleral buckle, orbitotomy, enucleation, glaucoma, and cataract surgery in adults. The advantages of the LMA over an endotracheal tube include less hemodynamic changes upon insertion, minimal effect on IOP,[103,104] and the lack of requirement for muscle relaxants or intubating equipment.[105] A disadvantage of the LMA in ophthalmic surgery is that if there is difficulty with ventilation after the surgery is underway, the airway is difficult to access because it is close to the surgical field and often the patient's head is turned 90 degrees away from the anesthesia provider. If it becomes necessary to adjust the LMA or perform endotracheal intubation, the sterility of the surgical field can become compromised during those maneuvers.

Endotracheal intubation is required for penetrating keratoplasty, deep anterior lamellar keratoplasty, combined corneal transplantation and vitrectomy, and some vitreoretinal procedures, in order to provide muscle relaxation. Prevention of patient movement is critical during penetrating keratoplasty. In addition to ensuring an adequate depth of anesthesia, profound muscle relaxation must be ensured by the use of a nerve stimulator where 0/4 twitches and minimal posttetanic count are present while the diseased cornea has been removed and the eye is completely open. Movement at this time could result in extrusion of ocular contents or choroidal hemorrhage. Profound relaxation is required until the donor cornea is secured with eight sutures, when ocular contents are no longer open to the atmosphere. Sudden movement during vitreoretinal procedures where there are instruments inside the eye can also lead to injury that must be prevented. Profound levels of muscle relaxation can be reversed with sugammadex.

Intraoperative hypotension in elderly patients must be treated aggressively. Multiple studies have shown intraoperative hypotension (variously defined as systolic blood pressure <90 mm Hg or mean arterial pressure <50-55 mm Hg, depending on the study) to be associated with acute kidney injury, myocardial injury, stroke and mortality.[106-111] Intraoperative hypotension should be managed with a continuous infusion of phenylephrine instead of repeated phenylephrine boluses to maintain hemodynamic stability. In this situation, decreasing anesthetic depth to correct hypotension could potentially result in patient movement if there is inadequate muscle relaxation.

The prevention of coughing and bucking during emergence is another important goal in ophthalmic surgery because they both dramatically increase IOP,[34] and the incidence of hemorrhage in both intraocular and extraocular surgery. If there is no contraindication, a "deep" endotracheal extubation can be performed in patients who are breathing adequately at a deep level of anesthesia.[112,113] A number of other strategies are also useful in preventing coughing, such as the use of an LMA where appropriate. Total intravenous anesthesia (TIVA) with propofol is superior to sevoflurane, especially in smokers,[114] but it can delay emergence and PACU discharge. Propofol and sevoflurane are additive pharmacodynamically.[88] A combination of 0.5 minimum alveolar concentration of sevoflurane and infusion of propofol at 50 to 100 μg/kg/min is a reasonable approach to prevent coughing and postoperative delirium without delaying PACU discharge. In intubated patients, topical intratracheal lidocaine works slightly better than intravenous lidocaine. However, in cases lasting greater than 2 hours, the benefits of intratracheal lidocaine diminish.[115,116] If a "deep" endotracheal extubation is contraindicated, the patient should be suctioned while at a deep level of anesthesia, intravenous lidocaine given, and extubation performed as soon as safely possible.

Prevention of IOP fluctuation, especially in an open eye, glaucoma, and retinal detachment, can potentially reduce the risk of further eye injury. Video-guided laryngoscopy can significantly decrease the rise of IOP compared with direct laryngoscopy, as well as attenuate hemodynamic alterations.[37,117]

Prevention of PONV in ophthalmic surgery is important to improve patient satisfaction[118] and to prevent increases in IOP that are potentially harmful in relation to the procedure just performed. The treatment for the prevention of PONV should be determined by the Consensus Guidelines for the Management of PONV by Gan and colleagues.[119]

N_2O should be discontinued 15 to 20 minutes prior to the injection of sulphur hexafluoride (SF_6), or octafluoropropane (C_3F_8) used for retinal tamponade. N_2O is avoided for 3 weeks after injection of SF_6, or 8 weeks after C_3F_8 as it may increase the size of the gas bubble through diffusion. Patients are also advised to avoid air travel or high altitude because the reduced atmospheric pressure will lead to expansion of the intraocular gas bubble. Silicone oil is used for retinal tamponade in patients who need longer duration of tamponade, have difficulty in positioning, or cannot avoid air travel after surgery. Unlike gases, silicone oil is permanent and remains in the eye until surgically removed.[120]

Anesthesia for Pediatric Ophthalmology

Anesthesia for pediatric ophthalmology encompasses a diverse group of patients and procedures. Patients range from the premature infant with all the attendant comorbidities, to children with congenital syndromes, to healthy children and adolescents. Many ophthalmic procedures that are performed in adults under MAC require general anesthesia for a pediatric patient.

GLAUCOMA

Measurement of IOP and a thorough ocular examination in the awake child is often difficult due to lack of cooperation and therefore frequently requires general anesthesia. Anesthetic drugs and interventions, such as laryngoscopy, affect IOP. While accurate IOP measurement is important to the diagnosis and treatment of pediatric glaucoma, it is one of numerous diagnostic criteria. Examination of the optic nerve, pachymetry to measure corneal thickness, and gonioscopy of the iridocorneal angle are all important components of the examination. Trends in measurement of the IOP in the same patient over time, under the same conditions, are also useful in management.

An inhalation induction with sevoflurane is commonly performed in pediatric patients and is associated with a reduction in IOP within minutes. Premedication with oral midazolam facilitates a smooth induction, as agitation and crying will falsely elevate IOP. Oral midazolam appears to have no significant effect on IOP.[31] Oberacher and colleagues [31] studied the effect of oral midazolam in a group of young children and found no significant difference between the IOPs measured when awake versus sedated. Ketamine, which is thought to have minimal impact on IOP, has been used for sedation and IOP measurement in children. However, agitation and hallucinations during recovery have led to limited usefulness.

The ophthalmologist in coordination with the anesthesiologist is able to measure the IOP before a deep level of anesthesia is achieved. The ophthalmologist should be in the room with instruments ready prior to induction. The inhalational mask is positioned so that the ophthalmologist has unobstructed access to the eye. If necessary, the mask can be removed briefly to allow IOP measurement and then replaced. Unlike adults with glaucoma who are most often managed medically, pediatric patients are primarily treated with surgical interventions such as goniotomy, trabeculectomy, and pressure lowering devices.

About 10% of primary congenital glaucoma is inherited. Secondary glaucoma can be the result of a systemic condition such as neurofibromatosis, rubella, or Sturge-Weber syndrome (congenital capillary hemangiomatosis). However, most cases of pediatric glaucoma have no identifiable cause. These children require frequent examinations under anesthesia to monitor therapy. It is essential to establish a good rapport with the patient and family and to consider the use of premedication with midazolam and/or parental presence in order to achieve a smooth induction of anesthesia. An LMA is suitable for examinations under anesthesia, except for the very young infant where endotracheal intubation is preferred. If a surgical intervention is required, the LMA may be changed to an endotracheal tube at the discretion of the anesthesia provider.

STRABISMUS

Strabismus surgery is the most common type of ophthalmic surgery performed in children. It has historically been associated with an incidence of postoperative vomiting (POV) of more than 50% in numerous studies. Eberhart et al. identified strabismus surgery as an independent predictor for POV in children.[121] Additional independent predictors include age over 3 years, duration of surgery greater than 30 minutes, and a history of POV or PONV in the patient, sibling, or parent. When 2, 3, or 4 risk factors are present, the risk of POV is 30%, 55%, and 70%, respectively. With the development of risk scores and the Consensus Guidelines by Gan and colleagues[119] to predict and manage POV in children and PONV in adults, the incidence of these events has been dramatically reduced.

For children at high risk for POV (more than 2 risk factors), as those having strabismus surgery are, a combination of a 5-HT3 antagonist and a steroid should be administered prophylactically. Dexamethasone in a dose of 0.1 to 0.2 mg/kg at the beginning of the procedure, and ondansetron 0.1 mg/kg toward the end of the case is the recommended strategy. A propofol infusion at a subhypnotic dose in combination with other classes of antiemetics also reduces the incidence of POV.[122] Those children with all four risk factors should avoid receiving N_2O and inhalational anesthetics. TIVA with propofol should be considered in such cases. Droperidol is an effective antiemetic; however, the FDA “black box” warning has resulted in a dramatic decrease in its use.

The oculocardiac reflex is frequently elicited during strabismus repair by traction on the extraocular muscles (see the Oculocardiac Reflex section). When the response is profound, removal of the stimulus by the surgeon is the first step in resolution. The reflex usually accommodates over time and is rarely hemodynamically significant in healthy children. If, however, the bradycardia persists, it may be treated with an anticholinergic agent, either atropine or glycopyrrolate. A lesser known reflex, the oculorespiratory reflex leads to bradypnea and respiratory pauses.[123] The afferent limb is the same as for the oculocardiac reflex, the efferent limb has not been clearly established. It is not responsive to anticholinergics. The oculorespiratory reflex may be less appreciated because of the use of assisted modes of ventilation commonly used during strabismus surgery. Airway management is typically with an LMA unless a contraindication is present.

RETINOPATHY OF PREMATURITY

Retinopathy of prematurity (ROP) is a major cause of blindness and other visual disabilities in children throughout the world. Improved survival of extremely premature babies has led to an increased incidence. ROP was first linked with liberal oxygen use in premature infants in the 1940s. Setting target ranges of oxygen saturation in the 91% to 95% range has decreased the impact of oxygen as a risk factor.[124] Gestational age and low birth weight are major risk factors for the development of ROP. Other factors such as anemia, sepsis, and bronchopulmonary dysplasia (BPD) are also important predictors. Screening guidelines for ROP include a dilated fundus examination for all premature infants less than 30 weeks gestational age or 1500 grams at birth. Most of these initial screening exams are done in the neonatal intensive care unit without intervention from an anesthesia provider. Older infants may need to come to the operating room for surgery or laser treatments.

Care of the ex-premature infant is complex and starts with a thorough history with attention to gestational age, birth weight, neonatal intensive care unit (NICU) course, duration of intubation and ventilation, history of apnea and bradycardia, the presence BPD, and any congenital anomalies. Abnormalities of pulmonary function are common in children with BPD and many have increased airway reactivity.[125] This is an important consideration when any formerly premature child who has had BPD undergoes a surgical procedure. As with ROP, younger infants are surviving with BPD and the long-term sequelae are not completely known.

Although it has been more than 30 years since the initial recommendation for monitoring the formerly preterm infant following general or regional anesthesia, those recommendations are still current.[126] There is interinstitutional variation with the most conservative centers recommending monitoring for apnea and bradycardia for at least 12 hours until 60 weeks postconceptual age (PCA). Some infants who have had a complicated NICU course and continue to be oxygen dependent at home, have anemia, or continue to have episodes of apnea and bradycardia would not be suitable candidates to have surgery performed in an outpatient setting despite being greater than 60 weeks PCA.

General anesthesia is required for infants and children having laser treatments and other surgical interventions for ROP. The need for a motionless field is best ensured by endotracheal intubation and the use of muscle relaxants. Some infants may have been recently extubated, and it may be difficult to extubate at the end of the surgical procedure. They may experience periodic breathing upon emergence from anesthesia, in which case, the patient should be returned to the NICU and extubated when fully recovered.

Postoperative Considerations in Ophthalmic Surgery

It is commonly assumed that ophthalmic surgery is associated with little postoperative pain. While this may be true of cataract surgery,[127] significant postoperative pain is associated with posterior segment surgery, corneal surgery, ocular muscle surgery, and enucleations.[128] Yet, pain in ophthalmic surgery is often underrecognized, and postoperative pain management in complex ophthalmic surgery is frequently inadequate.[129] Postoperative pain after ophthalmic surgery is preferably treated with multimodal analgesia such as acetaminophen (oral or intravenous), nonsteroidal antiinflammatory medications, gabapentin, and regional block. Opioid use should be limited, as opioid prescription after short-stay surgery is a risk factor for chronic opioid use 1 year after discharge.[130]

Postoperative pain after cataract surgery is usually associated with ocular dryness, stinging, burning associated with topical medications, photophobia, or corneal abrasion as a result of the eyelid speculum being inserted or removed or corneal surface desiccation during surgery. Pain after cataract surgery is usually short-lived. Persistent pain may indicate complications such as choroidal effusion, suprachoroidal hemorrhage, and aqueous misdirection syndrome.[131] In the PACU, it is additionally important to consider elevated IOP as a potential cause of postoperative pain and PONV. Vasovagal syncope may also occur in the PACU especially in patients who have undergone strabismus surgery, and in those who have preexisting autonomic dysfunction. It is also seen in patients who sit up too quickly after being in a head-down position for an hour or two following insertion of a gas bubble during vitreoretinal surgery. Treatment is supportive and should include administration of oxygen, intravenous fluids, and anticholinergic drugs. The patient should be positioned supine with the head lower than the level of the heart.

Visual impairment is associated with an increased risk of falls, which is a leading cause of death and significant morbidity in the elderly.[132] Special care must be extended to minimize the risk of falls in elderly patients who have undergone ophthalmic surgery and have a patched eye; this is even more important in those with poor vision in the nonsurgical eye.

Ophthalmic Emergencies

Most urgent ophthalmic procedures do not need to be performed on an emergent basis. This is important because the anesthetic plan must consider the fasting status and general medical condition of the patient. In true eye emergencies, therapy should be started within minutes to hours of their recognition.

True emergencies such as ocular burns and central retinal artery occlusion require immediate intervention to prevent loss of sight. **Urgent situations** include open-globe injuries, endophthalmitis, acute narrow-angle glaucoma, acute retinal detachment, corneal foreign body, and lid laceration. Semiurgent conditions include ocular tumors, blowout fractures of the orbit, congenital cataract, and chronic retinal detachment. Treatment for these conditions should be started within days, but in some cases can be safely scheduled within weeks if necessary.

OPEN GLOBE AND FULL STOMACH

In a patient with a penetrating eye injury who requires emergent or urgent surgery, the anesthesia provider must protect the patient from pulmonary aspiration of stomach contents while protecting the eye from acute changes in IOP. Succinylcholine, used as part of a rapid-sequence induction of anesthesia to permit rapid intubation and airway protection, causes a modest increase in IOP of 8 to 10 mm Hg. There has been longstanding controversy surrounding the use of succinylcholine based primarily on anecdotal reports of vitreous loss during open eye surgery.[133] There have been a number of studies where no extrusion of ocular contents has occurred following succinylcholine use.[134-136]