

ICU Management of Trauma Patients

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Objectives: To describe the current state of the art regarding management of the critically ill trauma patient with an emphasis on initial management in the ICU.

Data Sources and Study Selection: A PubMed literature review was performed for relevant articles in English related to the management of adult humans with severe trauma. Specific topics included airway management, hemorrhagic shock, resuscitation, and specific injuries to the chest, abdomen, brain, and spinal cord.

Data Extraction and Data Synthesis: The basic principles of initial management of the critically ill trauma patients include rapid identification and management of life-threatening injuries with the goal of restoring tissue oxygenation and controlling hemorrhage as rapidly as possible. The initial assessment of the patient is often truncated for procedures to manage life-threatening injuries. Major, open surgical procedures have often been replaced by nonoperative or less-invasive approaches, even for critically ill patients. Consequently, much of the early management has been shifted to the ICU, where the goal is to continue resuscitation to restore homeostasis while completing the initial assessment of the patient and watching closely for failure of nonoperative management, complications of procedures, and missed injuries.

Conclusions: The initial management of critically ill trauma patients is complex. Multiple, sometimes competing, priorities need to be considered. Close collaboration between the intensivist and the surgical teams is critical for optimizing patient outcomes. (*Crit Care Med* 2018; 46:1991–1997)

Key Words: abdominal trauma; chest trauma; hemorrhagic shock; resuscitation; trauma; traumatic brain injury

Trauma remains the most common cause of mortality in the 1–45 years old age groups. The most frequent causes of death are hemorrhagic shock and traumatic brain injury (TBI). (1) As prehospital, emergency department, and

operative management have improved, more critically injured patients are surviving to admission to the ICU, presenting numerous, sometimes competing, challenges for management. This review focuses on the initial assessment and management of the critically ill trauma patient. Other issues that are germane for subsequent management of these patients, such as nutrition (2), stress ulcer prophylaxis (3, 4), and venous thromboembolism prophylaxis (5, 6), have been reviewed elsewhere.

INITIAL ASSESSMENT

The initial assessment of the trauma patient has become well standardized by the Advanced Trauma Life Support course of the American College of Surgeons (7). The assessment begins with the primary survey (airway, breathing, circulation, disability, exposure/environment) to identify and manage immediately life-threatening injuries. The secondary survey includes a more detailed head to toe examination of the patient along with appropriate radiographic studies. As this assessment may be truncated for management of immediately life-threatening injuries, the ICU team often needs to complete the assessment and document all interventions and findings. This “tertiary survey” is critical for avoiding missed injuries (8–11).

AIRWAY AND VENTILATOR MANAGEMENT

Airway management in trauma patients must take into account the patient’s injuries and physiologic state, potential for anatomic distortion, risk for cervical spine injury, a potentially full stomach, and premorbid status. The indications for securing an airway include airway injury, impaired gas exchange, shock, and depressed mental status with a decreased ability to protect the airway. Rapid sequence induction is standard. Use of video laryngoscopy may not improve outcomes compared with direct laryngoscopy (12–14). If orotracheal intubation is unsuccessful, a supraglottic airway (e.g., intubating laryngeal mask airway) may be a useful rescue device, although there are some circumstances, for example, severe facial trauma, in which the initial airway should be surgical, for example, cricothyrotomy or awake tracheostomy.

The potential harmful effects of mechanical ventilation, ventilator-induced lung injury, including barotrauma, volutrauma, and atelectrauma, need to be considered when applying mechanical ventilation to trauma patients. A lung-protective strategy, using tidal volumes of 6–8 mL/kg and maintaining plateau pressures under 30 torr, is beneficial for high risk patients

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and, likely, all patients. An “open lung” strategy using appropriate levels of positive end-expiratory pressure along with recruitment maneuvers remains controversial (15). “Permissive hypercapnia” can be used with the caveat that it can worsen intracranial hypertension in patients with severe TBI.

Ventilator-associated pneumonia (VAP) is another risk of mechanical ventilation. Minimizing the length of time that an artificial airway is in place and that a patient requires mechanical ventilation is critical to the prevention of VAP. In addition, the Institute for Healthcare Improvement has promoted a ventilator bundle to decrease the rate of VAP. Components of the bundle include the following: 1) elevation of the head of the bed, 2) daily sedative interruption and daily assessment of readiness to extubate, 3) peptic ulcer disease prophylaxis, 4) deep venous thrombosis prophylaxis, and 5) daily oral care with chlorhexidine (16).

Critically ill trauma patients are at high risk for developing severe hypoxemia due to pulmonary contusions, aspiration, pneumonia, or the acute respiratory distress syndrome. Airway pressure release ventilation (APRV) is a mode of ventilation that can be used to improve oxygenation by maximizing mean airway pressure and subsequent alveolar recruitment while limiting barotrauma. Early use of APRV compared with low tidal volume ventilation seems safe, but clear benefit has not been demonstrated (17–19). For patients with refractory hypoxemia, rescue therapies may include prone positioning (20) and pulmonary vasodilators (epoprostenol or inhaled nitric oxide) (21). Extracorporeal membrane oxygenation (ECMO), originally thought to be contraindicated because of bleeding risk, can also be used safely in selected patients (22–24). Clear benefits of these interventions are difficult to demonstrate.

HEMORRHAGE, MASSIVE TRANSFUSIONS, AND COAGULOPATHY

The most common cause of hypotension in trauma patients is hemorrhage. Significant tissue trauma, however, adds to the hemorrhagic shock state by eliciting immunologic and inflammatory responses (25). Thus, there is more to “traumatic shock” than just blood loss and tissue ischemia. Nonetheless, biomarkers of tissue ischemia or “oxygen debt” such as lactate or base deficit are useful to trend. Elevation of these variables, or failure to normalize them, is associated with increased mortality (26).

Trauma patients with severe hemorrhagic shock often require a massive transfusion, usually defined by requiring at least 10 units of packed RBCs (PRBCs) within 24 hours. The optimal fluid for resuscitation of a trauma patient in severe hemorrhagic shock would likely be whole blood. In civilian practice, however, whole blood is not widely available. Consequently, PRBCs are used. Fresh frozen plasma (FFP), platelets, and cryoprecipitate can be administered to essentially reconstitute whole blood. Although the optimal ratios of these blood components remain unclear, early administration of FFP and platelets as part of “damage control resuscitation” seems to be beneficial (27). A ratio of 1:1:1 may improve outcomes in terms of achieving hemostasis and preventing

early death from exsanguination compared with higher ratios of PRBCs (28, 29). Small studies of banked whole blood are underway. Administration of crystalloids, colloids, or hypertonic solutions (in the absence of concomitant TBI) is to be minimized (30, 31).

For patients who have active hemorrhage, novel approaches include permissive hypotension, although optimal mean arterial pressure (MAP) has not been determined (32–35), and Resuscitative Endovascular Balloon Occlusion of the Aorta for selected patients with abdominal or pelvic hemorrhage (36–39).

Severe hemorrhage and resuscitation leads to a vicious cycle of deterioration from the “lethal triad” of coagulopathy, metabolic acidosis, and hypothermia. The coagulopathy of trauma includes dilution of clotting factors, consumption of factors at the site of bleeding, intravascular coagulation, fibrinolysis, hypothermia, acidosis, inflammation, and other factors (40). Although prothrombin time, activated partial thromboplastin time, and platelet counts are typically used to monitor coagulation status, many trauma centers have turned to thromboelastography or rotational thromboelastometry which may better represent overall clotting function and allow for more targeted therapy (41–43).

A number of adjuncts to blood product administration to decrease coagulopathy have been studied. Lyophilized plasma, for example, shows promise (44). Trauma patients often develop hypofibrinogenemia, which has typically been managed with cryoprecipitate, although fibrinogen products are available in some countries and may have benefits (45–48). Recombinant activated Factor VII initially showed promise but has mainly gone out of favor because of cost and lack of clear benefit (49). Prothrombin complex concentrates are sometimes used, although data on efficacy in the absence of warfarin-induced coagulopathy are variable (50–52).

Both hyperfibrinolysis and fibrinolysis shutdown, at the time of presentation, compared with normal levels of fibrinolysis, are associated with worse outcomes from major trauma (53–55). Patients with hyperfibrinolysis may benefit from the use of tranexamic acid (56). Conversely, patients with physiologic levels of fibrinolysis or with fibrinolysis shutdown could be harmed (57). Monitoring fibrinolysis seems prudent (58–60).

With a better understanding of the coagulopathy of trauma and evidence that a more aggressive approach to normalizing hemostatic mechanisms can improve outcomes, the concept of damage control resuscitation has been established and recommended (61). For the moment, point of care viscoelastic testing seems to be the best way to guide therapy (62).

ENDPOINTS OF RESUSCITATION

The optimal single endpoint to determine the adequacy of resuscitation remains elusive. Normalization of vital signs and urine output are typical first steps, but these variables may miss ongoing, occult hypoperfusion. Markers of inadequate global oxygen delivery, such as base deficit, lactate level, or mixed venous oxygen saturation, should be used (26, 63). A recent Eastern Association

for the Surgery of Trauma guideline suggested that focused ultrasound and arterial pressure waveform analysis could be useful for predicting volume responsiveness and may predict complications and organ failures, but not mortality (64–67).

SPECIFIC INJURIES

Chest Trauma

Severe chest trauma can cause injuries to the airways, lungs, heart, great vessels, and esophagus. Ultrasound (the extended Focused Assessment by Sonography for Trauma) can rapidly identify immediately life-threatening injuries, whereas CT of the chest can further define these injuries and allow precise interventions.

Pneumo- and hemothoraces are typically managed with tube thoracostomy. Early interventions, such as video-assisted thoracic surgery to drain a retained hemothorax, may reduce long-term morbidity (68). Rib fractures can result in significant morbidity particularly in older patients. Adequate pain control is imperative to prevent splinting, atelectasis, and pneumonia. Regional analgesia (69) and rib fracture plating may be beneficial (70).

Pulmonary contusions can cause profound acute respiratory failure. Treatment is largely supportive with traditional mechanical ventilation, although advanced ventilator modes (e.g., APRV) and ECMO may be required in the most severe cases (17, 23, 24).

For patients with blunt aortic injury, the management has dramatically changed in recent years. The standard open repair for significant injuries has largely been replaced by endovascular repairs (71–74). For less significant injuries and for injuries in which repair is delayed due to competing priorities or patient physiology, strict blood pressure and heart rate control are imperative (74).

Abdominal Trauma

For patients with blunt abdominal trauma, solid organ (liver, spleen, kidney) injuries are most common. Many of these injuries can be successfully managed nonoperatively as long as the patient remains hemodynamically stable. Unexplained hemodynamic instability or physiologic deterioration early after injury in a patient who is being managed nonoperatively should prompt an investigation into whether the patient has “failed” nonoperative management and bled or if the patient has a missed injury. Hollow viscus injuries from blunt trauma may be missed during the initial assessment of the patient because of an altered mental status, distracting injuries, or falsely negative imaging studies. Changes in the patient’s abdominal examination or unexplained sepsis should prompt consideration of a hollow viscus injury and need for laparotomy.

Patients with penetrating trauma have traditionally required laparotomies more frequently than patients with blunt injuries. However, nonoperative management of patients with penetrating trauma who are hemodynamically stable with isolated solid organ injury and no peritonitis at the time of admission has also become standard (75–77).

For trauma patients in extremis who undergo a damage control laparotomy, during which major bleeding is controlled, contamination minimized, and the abdomen packed, the abdominal fascia is left open to prevent abdominal compartment syndrome and allow reexploration once homeostasis has been restored. Ideally, the abdomen is left open for no more than 72 hours (78). During this time, patients are usually sedated. Neuromuscular blockade is only used if absolutely necessary. Prior to reexploration and attempted closure, minimizing use of crystalloids and judicious fluid removal, either with diuretics or renal replacement therapy, may improve fascial closure rates (79)..

Patients with active hemorrhage and orthopedic injuries may not tolerate early, definitive fixation of fractures. A damage control orthopedics approach, focusing on hemorrhage control, restoration of perfusion, and minimization of contamination, similar to that described above for abdominal trauma, may be appropriate (80). Definitive repairs can be postponed.

TBI

For patients with severe TBI, there is little that can be done to treat the “primary injury” to the brain. Therefore, ICU management of the patient with TBI largely focuses on prevention and mitigation of “secondary insults” such as hypoxia, hypotension, cerebral edema, and ischemia. Patients may present to the ICU either postoperatively after evacuation of a mass lesion (such as a subdural or epidural hematoma), for close neuromonitoring in the setting of severe TBI, or with severe concomitant injuries in the setting of a mild or moderate TBI.

Comatose patients, typically defined as having a Glasgow Coma Scale score of less than 9, should have their airway secured. Rigorous prevention of hypoxia is essential. Maintaining adequate ventilation is also important as hypercarbia can increase intracranial pressure (ICP). Standard management should include adequate analgesia and sedation, initiation of posttraumatic seizure prophylaxis, maintenance of normothermia, initiation of enteral nutrition when possible, stress gastritis prophylaxis, and venous thromboembolism prophylaxis. Frequent neurologic tests including a pupillary examination are important to detect early evidence of worsening that may require neurosurgical intervention.

Intracranial pressure monitoring may decrease early mortality following severe TBI, although some studies have not demonstrated benefit (81–83). If ICP monitoring is employed, the current recommended threshold for treatment of elevated ICP is 22 mm Hg (84). Any unexplained elevation in ICP should be evaluated by a neurologic assessment and repeat CT to rule out recurrent or worsening intracranial hemorrhage. Management of elevated ICP is based on the principle of the Monro-Kellie doctrine that the intracranial space has three components: brain tissue, blood, and cerebrospinal fluid (CSF). Tiered treatment is targeted toward reduction in the volume of one of these components (85). First tier therapies generally include head of bed elevation to improve cerebral venous drainage, short acting sedation and analgesia to allow for reduction in cerebral metabolic rate, and subsequent

cerebral blood flow while maintaining ability to perform clinical neurologic assessments and drainage of CSF via an external ventricular drain. Second tier therapies include hyperosmolar therapy with either mannitol or hypertonic saline to reduce cerebral edema, mild hyperventilation inducing reflexive cerebral vasoconstriction, and, in some cases, neuromuscular blockade. Third tier therapies, such as decompressive craniectomy, therapeutic hypothermia, and barbiturate coma, are controversial (84, 86–89).

Maintenance of cerebral perfusion pressure (CPP = MAP – ICP) has become a cornerstone of therapy in patients with TBI. Through autoregulation, the normal cerebral vasculature maintains adequate blood flow across a wide range of systemic pressures. Cerebral autoregulation, however, is often abnormal in patients with severe TBI (90). With a loss of autoregulation, a rise in systemic pressure can increase ICP, whereas systemic hypotension can cause hypoperfusion and ischemia. The standard target CPP is 60–70 mm Hg depending on the patients' autoregulatory status (91–93).

Spinal Cord Injury

Spinal cord injury (SCI) is a particularly devastating sequela of trauma. Cervical spinal cord injuries account for over 50% of traumatic spinal cord injuries and are associated with much higher short- and long-term morbidity than injuries affecting the thoracic or lumbar cord (94). Given that little can be done for the acute primary injury to the spinal cord, the mainstay of treatment of all patients with SCI is largely supportive, focusing on minimizing secondary insults and complications.

Most patients with severe cervical SCI are initially endotracheally intubated. The development of respiratory dysfunction correlates with the level of injury and severity of SCI as measured by the American Spinal Injury Association Impairment Scale (95). In addition to causing a secondary insult (89), hypoxemia can cause severe bradycardia and even asystole in patients with high cervical SCI due to unopposed vagal stimulation (96–99).

The respiratory management of the patient with SCI should include a combination of chest physiotherapy, secretion clearance, bronchodilators, mucolytics, respiratory muscle training, assisted breathing, and assisted coughing devices and techniques (100). Early aggressive pulmonary care, ventilator “bundles,” and dedicated weaning protocols are associated with improved survival, reduced frequency of pulmonary complications (including pneumonia), and decreased need for long-term ventilatory support (97, 100, 101). Early tracheostomy in high-risk patients may reduce ICU length of stay and duration of mechanical ventilation (102), plus improve patient comfort, ease of secretion clearance, ability to perform safer weaning trials, and ability to communicate more effectively. Patients with high cervical SCI (above the C3 level) can also be liberated from mechanical ventilation with technologies such as laparoscopically implanted diaphragm pacing systems (103).

Neurogenic shock from loss of sympathetic nervous system stimulation at the T6 level or above is a form of distributive shock, which can last for 1–3 weeks (104). Unopposed

parasympathetic activity and vagal stimulation can cause profound bradycardia and atrioventricular nodal block in addition to hypotension. The higher and more complete the injury, the more severe and refractory the neurogenic shock. First-line therapy is fluid resuscitation to maintain euvolemia. Second-line treatment includes vasopressors (particularly norepinephrine), inotropes, or a combination (98, 105).

Aggressive prevention of hypotension may improve neurologic outcome (106). Current recommendations are to maintain MAP above 85–90 mm Hg for the first 7 days following acute cervical SCI (98, 105). Treatment of bradycardia, for example, β -agonist therapy with enteral albuterol, is typically reserved for symptomatic patients (107).

Brain Death and Organ Donation

Brain death, defined as the “irreversible cessation of all functions of the entire brain, including the brain stem,” is an unfortunate but frequent sequela of severe TBI (108). Increasing ICP initiates a Cushing reflex characterized by systemic arterial hypertension and bradycardia. Rostral to caudal ischemia of the brain stem occurs, resulting in initial sympathetic stimulation with severe vasoconstriction leading to end-organ dysfunction followed by profound brainstem ischemia and hypothalamic-pituitary failure, resulting in severe hypotension. When ICP exceeds MAP, blood flow to the brain ceases.

The American Academy of Neurology guidelines for determining brain death include the following: 1) Establishment of an irreversible and proximate cause of coma without confounding factors, such as hypothermia, hypotension, drugs, and severe metabolic or electrolyte imbalances. 2) Clinical evaluation demonstrates the absence of all brainstem reflexes and no spontaneous breathing. 3) Ancillary testing, such as electroencephalography, cerebral angiography, nuclear scan, and transcranial Doppler, can be used if portions of the clinical examination cannot be completed. 4) Documentation of the official time of death in the medical record (109).

Death following TBI is a significant source of organs for donation. The hemodynamic changes that occur surrounding brain death can significantly impact extracerebral organs (110, 111). Other systemic effects of brain death include hypothermia, coagulopathy, and diabetes insipidus. A strategy that includes hemodynamic support, low tidal volume ventilation, aggressive normalization of electrolytes, aggressive treatment of hyperglycemia, and hormonal replacement therapy (vasopressin, corticosteroids, and thyroid hormone) for persistent vasodilatory shock, along with intensivist-led management, can decrease the risk for organ dysfunction (112–115).

CONCLUSIONS

Management of critically ill trauma patients continues to evolve, although the basic principles of rapid identification and management of life-threatening injuries remain the same. Major, open surgical procedures have often been replaced by nonoperative or less-invasive approaches, shifting much of the early management to the ICU, where the goal is to restore homeostasis while watching closely for failure of nonoperative

management, complications of procedures, and missed injuries. Close collaboration between the intensivist and the surgical teams is critical for optimizing patient outcomes.

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