Abstract:
The continued growth in emergency department (ED) use combined with limited inpatient bed availability often leads to boarding of patients needing inpatient or intensive care unit admission in the ED. Emergency department personnel are experienced in the rapid assessment of trauma patients but may be less prepared or comfortable with providing ongoing management of trauma patients, especially critically injured pediatric patients. This article reviews management principles of traumatic brain injury, mechanical ventilation, and shock in the pediatric trauma patient and is intended to guide ED management of these patients until they can be transferred to an appropriate level of inpatient care.

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When There Are No Inpatient Beds: Providing Pediatric Critical Care for Trauma Patients in the Emergency Department

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In an ideal world, the emergency department (ED) would be easily accessed by those truly needing emergency care. Seriously injured and ill patients would arrive and be cared for rarely and dispositioned in a timely fashion. Patients needing surgery or hospital admission would move through the ED expediently to their final destination. Unfortunately, that ideal rare, exists in today's ED. More than 100 million Americans, 30 million of them children, present to the ED each year.[1] A persistent rise in ED visits over the last several decades has led to an overcrowding crisis in many communities.[2,3] This increase is often attributed to overuse of the ED for minor illnesses, but there is also evidence that EDs are seeing steadily increasing numbers of patients with serious illness and injuries. Lack of available inpatient hospital beds, particularly intensive care unit (ICU) beds, also contributes to ED crowding

and extended ED length of stay.[1] Emergency department physicians and staff may be challenged not only with seeing large numbers of patients, but also with providing care for extended periods of time to seriously ill and injured patients awaiting inpatient or ICU admission.

Although ED care providers are trained to provide initial assessment and stabilization for acutely ill and injured patients, they may be underprepared, in terms of training and resources, to provide ongoing critical care management.[4] Delay in transfer of critically ill patients to the ICU has been associated with increased hospital length of stay and mortality rates.[5] In an ideal world, patients needing ICU level care would be quickly evaluated and transferred to an ICU. When a critically ill patient cannot be immediately transferred to an ICU, they must be provided with appropriate care in an ED or transport setting, in essence bringing the ICU to the patient. The purpose of this article is to review some of the more common elements of ICU level trauma care that may be required in the ED or transport setting.

TRAUMATIC BRAIN INJURY

Traumatic brain injury (TBI) is a leading cause of morbidity and mortality for pediatric patients in the United States, accounting for more than 400 000 ED visits and more than 2000 deaths annually.[6] Through the years, many therapies have been proposed for the treatment of TBI; few of these have been studied or proven in pediatric patients. In 2003, a multidisciplinary group convened a set of guidelines[7] for the management of pediatric patients with TBI.[4] A major focus of these guidelines is good supportive care of the critically injured patient, with particular attention to prevention and treatment of shock and respiratory failure. Recent literature continues to support these guidelines, with a growing body of evidence demonstrating that hypotension and hypoxia, especially if unrecognized and untreated, are independent predictors of poor outcome in TBI.[8-11]

Careful attention should be paid to the ability to maintain an airway and adequate oxygenation and ventilation in patients with TBI. Hypoxia has been shown to negatively affect morbidity and mortality in this group.[7,10] In cases of mild to moderate isolated TBI, patients may require only supplemental oxygen. If a patient's ability to maintain an adequate airway and control of ventilation is compromised, endotracheal intubation may be required. Ventilation should be provided to maintain a partial pressure of carbon dioxide (P_{CO_2})

within normal limits (35-45 mm Hg). Both hyper- and hypoventilation may be deleterious to patients with TBI. Hypoventilation may increase cerebral blood flow, leading to increased intracranial pressure (ICP) if cerebral autoregulation of blood flow is impaired by injury. Hyperventilation lowers P_{CO_2} and causes subsequent cerebral vasoconstriction, with the potential for ischemia and secondary insult to the already injured brain. Only in cases of persistently elevated ICP refractory to other medical management should consideration be given to maintaining a lower level of P_{CO_2} (30-35 mm Hg).[7] Further discussion of specific ventilation strategies will be covered later in this article.

Careful attention to volume status and perfusion is important in the management of TBI. Medical personnel sometimes worry about giving intravenous (IV) fluids to TBI patients; there is a myth that the administration of any IV volume may worsen cerebral edema. Adequate blood pressure is required to maintain cerebral perfusion, and ensuring adequate intravascular volume is important for maintaining blood pressure and perfusion to the brain and other vital organs. Cerebral perfusion pressure (CPP) can be estimated by subtracting ICP from the mean arterial pressure (MAP). Ideal CPP in infants and children has not been well established, but targeting a range between 40 (infants) and 65 mm Hg (adults) seems reasonable.[12] A normal MAP is age dependent and can be estimated by the formula (50 + 2× age in years) for any child older than 1 year.[13] Often, ICP monitoring is not immediately available in the ED. It is therefore advisable to attempt to maintain normal to slightly high MAPs in patients with TBI. If ICP monitoring is available, CPP should be targeted to stay in the range of 40 to 65 mm Hg. Hypotension, if present, should initially be treated with fluid resuscitation. If blood pressure remains low or low-normal in the setting of persistently elevated ICP, vasopressor agents such as dopamine or norepinephrine may be needed to maintain a normal to high-normal MAP and adequate CPP.

In addition to ensuring adequate oxygenation, ventilation, and blood pressure, a few other basic principles should be observed in managing TBI patients. The patient's head should be kept midline and elevated to 30° if possible because this promotes venous return and may help control ICP. One caveat to remember is that patients with TBI may have associated spinal injuries, and any positioning of the head must be done while maintaining strict spinal precautions until an injury of the spine is excluded, but slight angulation of the entire bed, if possible, may be helpful. Other management strategies in

treating patients with TBI involve decreasing cerebral metabolic demands to help manage the elevated ICP, which often accompanies TBI. One of these strategies is to maintain adequate analgesia and sedation, particularly in patients with concomitant injuries or those requiring mechanical ventilation. Although the ability to monitor and follow a patient's neurologic examination is important, it must be balanced with the benefits of ensuring adequate analgesia and sedation. In cases of persistently elevated ICP, consideration should be given to deeper sedation, such as pentobarbital coma and even the use of neuromuscular paralysis.[7] Maintaining a normal body temperature is also important in the management of TBI. Hyperthermia may increase cerebral metabolic demands and lead to increased ICP. Although some studies support the use of mild hypothermia in the management of TBI, there is currently no strong evidence to support its routine use.[14-16] Finally, hyperosmolar fluid therapy may be used to manage elevated ICP. Both mannitol and hypertonic saline have been shown to be effective in this regard.[16,17-19] These agents work by altering the osmotic gradient across the blood-brain barrier, in effect, pulling fluid from the edematous brain.[7,12] There have been no definitive comparison studies of the 2 agents, and the choice of which to use may be based on availability or physician preference. Hypertonic (3%) saline may be administered in 5 to 10 mL/kg aliquots as needed until a serum sodium of 170 mEq/dL or a serum osmolarity of 360 mOsm has been reached.[19] Mannitol should be given in 0.5 to 1 g/kg aliquots as needed until a maximum serum osmolarity of 320 mOsm is reached.[12]

VENTILATOR MANAGEMENT

Many pediatric trauma patients may be managed without intubation and mechanical ventilation. Intubation may be required for airway protection in cases of craniofacial injury or head injury with altered mental status, to ensure oxygenation and ventilation with thoracic injuries, or to enable adequate sedation and analgesia and to decrease metabolic demands for patients with severe or multisystem trauma. Although multiple models of mechanical ventilators exist, with multiple modalities for delivering mechanical ventilation, the most important considerations in the mechanical ventilation of pediatric patients is close attention to initial choice of ventilator settings and close monitoring of the patient to ensure adequate oxygenation and ventilation.

Previously healthy trauma patients without thoracic or lung injury should have fairly compliant lungs and be able to be maintained on relatively low ventilator settings. Initial ventilator settings are based on normal physiologic parameters for a healthy child of similar weight and age. A positive end-expiratory pressure (PEEP) of 5 cm is a good starting point. Many ventilators are designed to deliver both pressure and volume-control modes of ventilation. Either may be used, with the goal of delivering a tidal volume (TV) of 6 to 8 mL/kg. Target respiratory rate varies with patient age. Good starting points are a rate of 30 for infants, 20 for children, and 16 for older children and teenagers. Inspiratory time (Ti) should be set between 0.5 and 1 second to target an inspiratory/expiratory ratio of 1:3 and allow adequate time in the exhalation phase of the respiratory cycle for carbon dioxide elimination. Using these guidelines, initial ventilator settings for a previously healthy 5-year-old patient weighing 20 kg should be TV of 160 mL (8 mL/kg), PEEP of 5 cm, rate of 20, and inspiratory time of 1 second. Patients undergoing mechanical ventilation should be monitored with continuous pulse oximetry. A blood gas measurement should be obtained shortly after instituting mechanical ventilation and the pH and P_{CO_2} values used to gauge the effectiveness of ventilation. After this measurement, end-tidal carbon dioxide monitoring, if available, augmented with periodic blood gas measurements, may be used to monitor and adjust ventilation. Capillary or venous blood gas measurements may be adequate for monitoring pH and P_{CO_2} in some patients, but placement of an arterial line may also be necessary for frequent blood sampling and blood pressure monitoring in critically ill patients.

Ventilator adjustment may be required to correct difficulties with oxygenation or ventilation. Ventilation difficulties require an increase in minute ventilation to remove carbon dioxide. Minute ventilation (MV) is defined as TV times respiratory rate ($MV = TV \times RR$) and can be changed by manipulating either of these parameters. Tidal volume may be adjusted by increasing the TV setting in volume-control mode or increasing the peak inspiratory pressure in pressure control mode. An important point to remember is that the TV delivered to the patient may differ from that set on the ventilator if there is a large air leak around the endotracheal tube. Another important consideration is the potential for secondary lung injury from positive-pressure ventilation. Although patients with TBI may benefit from keeping P_{CO_2} levels in a low-normal range for ICP control, trauma patients without TBI may be managed with a strategy of "permissive hypercapnea" in which

P_{CO_2} levels are allowed to remain above normal as long as an acceptable pH level (generally considered pH >7.2) is maintained.[20]

Ventilator adjustment may also be necessary to improve oxygenation. The easiest parameter to manipulate is the fraction of inspired oxygen (F_{IO_2}). Delivery of F_{IO_2} greater than 60% for prolonged periods has been associated with free radical formation and secondary lung injury. In patients requiring more than 60% F_{IO_2}, or in those difficult to oxygenate on higher levels of F_{IO_2}, consideration should be given to increasing the PEEP delivered by the ventilator. Increasing PEEP increases the functional residual capacity of the lungs and may serve to recruit additional alveoli and improve oxygenation. However, increasing PEEP may also have the deleterious effect of decreasing venous return to the heart and decreasing systemic blood pressure. This effect can often be overcome by the provision of additional intravascular volume in the form of isotonic fluid or blood product administration. A final ventilator adjustment that may be considered to improve oxygenation is lengthening the inspiratory time (Ti). In doing so, care must be taken to allow adequate time in the respiratory cycle for expiration. Failure to do this may compromise ventilation and lead to the development of respiratory acidosis from carbon dioxide retention.

MANAGEMENT AND RECOGNITION OF SHOCK

Shock is a state of inadequate delivery of oxygen and substrate to tissues. Any serious injury or illness can cause a state of shock if circulatory function is significantly impaired. In compensated shock, autonomic reflex mechanisms are activated to maintain vital organ perfusion. These include massive catecholamine release, leading to increased heart rate and systemic vascular resistance. These compensatory mechanisms are particularly active in previously healthy children and young adults and may make early phases of shock difficult to recognize in this population. If unrecognized and untreated, these compensatory mechanisms are overwhelmed, cellular function deteriorates, and a state of progressive organ dysfunction and metabolic acidosis heralds the development of uncompensated shock. Finally, terminal or irreversible shock implies organ damage to a degree that death is inevitable.[21-24]

Shock may be broadly categorized as hypovolemic, distributive, cardiogenic, or obstructive. In the trauma patient, the most common cause is hypovolemic shock in which acute blood loss leads to an inadequate circulating intravascular volume. Trauma patients may also experience obstructive shock in which cardiac output is mechanically obstructed by tension pneumothorax or by hemopericardium leading to pericardial tamponade. Distributive shock, characterized by systemic vasodilation leading to functional or relative hypovolemia, may be seen after spinal cord injuries and is sometimes termed spinal shock. Finally, myocardial contusion may cause myocardial dysfunction and cause cardiogenic shock.

Rapid recognition of shock, especially early or compensated shock, is crucial to limiting morbidity and mortality after trauma. Careful and repeated physical examinations may give valuable information as to the nature and cause of shock. The physical examination should start with an observation of the patient's mental status and responsiveness to the surrounding environment. Agitation, restlessness, and inability to be consoled by known caregivers may be an early sign of shock in infants and children. Even more concerning is the quiet, withdrawn child that does not make eye contact or respond to painful stimuli. Close attention should next be paid to airway and breathing. Effortless tachypnea is an early sign of shock as the patient attempts to compensate for an increasing metabolic acidosis through respiratory elimination of carbon dioxide.[13,21,22]

The next step in the rapid assessment of patients in shock is to evaluate the circulatory status by assessing skin perfusion, temperature, and capillary refill time. Healthy patients in a warm environment should have pink, warm skin with brisk (<2 second) capillary refill time. An early sign of hypovolemic and cardiogenic shock is the presence of cool distal extremities and prolonged capillary refill time. Conversely, patients with early distributive shock may have flushed skin and brisk capillary refill. Heart rate and pulse quality are other important elements of the cardiovascular assessment. Tachycardia is one of the earliest signs of shock and must also be interpreted in context to age-specific normal values. Hypovolemic or cardiogenic shock leads to narrow pulse pressure and weak "thready" pulses. In contrast, patients in early distributive shock may have widened pulse pressure with readily palpated "bounding" pulses.[18,19,21,22] Urine output is a sensitive indicator of renal perfusion and should be monitored closely as an indicator of intravascular volume status. Diminished urine output may be an early sign of intravascular volume depletion and may progress to a state of complete anuria in patients with severe shock.[18,21-25]

Blood pressure should also be measured as part of the cardiovascular assessment. Many references differentiate compensated vs uncompensated shock by the presence or absence of hypotension. Overreliance on blood pressure measurement, however, may lead to missed cases of shock. This is especially true in previously healthy children and young adults with hypovolemic shock, in whom arterial blood pressure may be normal or even slightly elevated during early stages of shock due to strong compensatory responses. With acute hemorrhage, blood pressure may be maintained in a normal range until approximately 30% of the circulating blood volume has been lost, at which point uncompensated shock ensues and may progress rapidly to terminal shock unresponsive to therapy.[13,25] Health care providers must therefore realize that hypotension is a late and ominous sign of shock in pediatric patients, and every effort should be made to recognize and treat shock states before such decompensation occurs.[13,21,22,25]

Certain principles apply regardless of the etiology of shock and should be instituted immediately for all patients presenting with signs of shock. Attention should first be directed toward airway and breathing. All patients should be placed on supplemental oxygen, preferably by high flow, non-rebreather mask. Patients with a patent airway and spontaneous respirations may still benefit from early intubation to reduce metabolic demand and assure adequate oxygenation and ventilation, especially in cases of severe or decompensated shock.[26-28]

Establishing vascular access is another early priority in the management of shock. This is best accomplished through the placement of as large a caliber peripheral IV catheter as is possible for the patient's size. Severely injured patients should ideally have at least 2 functioning IVs. The maximum rate of flow through any given catheter is proportional to the diameter and inversely proportional to the length; therefore, short, large-caliber catheters are preferred over long central venous lines for initial resuscitation.[24,25] When IV access cannot be quickly established, consideration should be given to placing an intraosseous (IO) access device.[13,25] Historically, IO access was limited to infants and young children. Newer IO drill devices allow this route to be used in older children and adults.[29,30] Fluid therapy should be initiated immediately after access is established. Initial fluid therapy should consist of a 20 mL/kg bolus of isotonic crystalloid fluid given as quickly as possible. If heart rate, level of consciousness, and capillary refill do not improve, a second 20 mL/kg bolus should be rapidly administered. If system-

ic perfusion does not respond to administration of 40 to 60 mL/kg of crystalloid in patients with suspected hemorrhagic shock, 10 to 15 mL/kg of packed red blood cells (PRBCs) should be transfused and repeated as needed. Type-specific cross-matched blood is preferred; however, type O negative blood may be used in emergency circumstances until cross-matched blood is available. Patients exhibiting signs of shock should have emergent consultation by a trauma surgeon because they may require exploration to identify and correct ongoing hemorrhage.[13,25]

Treatment of obstructive shock requires identification and specific therapy for the type of obstruction. Pericardial tamponade may present with muffled heart sounds, diminished pulses, and distended neck veins. Chest radiograph and bedside ultrasound, when available, may be helpful in making the diagnosis. If time permits, pericardial drainage under ultrasound guidance is the preferred treatment. In patients with severe shock or cardiovascular collapse, emergent pericardiocentesis may be life saving and should be performed without delay. Tension pneumothorax is a common cause of obstructive shock in trauma patients and may present with hypoxia, hypotension, diminished pulses, diminished or absent breath sounds on the affected side, and distended neck veins and/or tracheal deviation. Chest radiographs may be helpful in making the diagnosis but should not delay treatment in critically ill trauma patients. These patients should have immediate decompression of the pneumothorax by placement of an over-the-needle catheter in the second intercostal space in the midclavicular line followed by tube thoracostomy.[13,24,25,31]

Distributive shock may be seen in acute spinal cord injuries when loss of systemic vascular tone creates a state of relative vascular volume depletion. Initial treatment of distributive shock is similar to that of hypovolemic shock. Vascular access should be obtained and crystalloid boluses of 20 mL/kg should be delivered until systemic perfusion improves. If systemic perfusion does not improve after 2 to 3 such boluses and occult hemorrhage has been excluded, vasoactive medications such as dopamine or norepinephrine may be needed. The α-adrenergic properties of these medications cause systemic vasoconstriction and may improve perfusion in cases of distributive shock. These infusions are ideally given through a central venous catheter because extravasation may cause significant tissue necrosis.

Ongoing management of trauma patients involves frequent reassessment to gauge the adequacy of resuscitation and to recognize any need for further intervention. Some patients may respond to initial

volume resuscitation with improvement of tachycardia, capillary refill, or blood pressure, only to return to an unstable shock state if they are experiencing ongoing hemorrhage. The often mentioned "lethal triad" of trauma refers to the development of hypothermia, metabolic acidosis, and coagulopathy that may develop in seriously injured patients. Pediatric patients are particularly susceptible to hypothermia given their relatively larger surface to body mass ratio, as compared with adults. Warming measures such as heated blankets, removal of wet clothing and bedding, and warming lights should be instituted and body temperature closely monitored. Serial measures of serum hemoglobin and hematocrit may aid in recognizing ongoing hemorrhage and identifying patients needing emergent surgical intervention. Patients requiring massive or ongoing volume resuscitation may develop coagulopathy from consumption and dilution of clotting factors. This may manifest externally as mucosal bleeding or oozing from skin sites such as needlesticks and cutaneous wounds. These patients may require transfusion of fresh frozen plasma and platelets in addition to PRBCs. Traditionally, trauma patients were transfused with PRBCs alone until coagulopathy became manifest as either excessive bleeding or abnormalities in laboratory values for platelet levels, prothrombin time, and activated partial thromboplastin time. Recent literature suggests that patients requiring massive transfusion, usually defined as more than 10 U of PRBCs for adult patients, should receive closer to a 1:1:1 ratio of red blood cells, plasma, and platelets.[32-34] Evidence-based pediatric guidelines for massive transfusion have not been well established, but it seems prudent to provide plasma and platelet replenishment in addition to PRBCs to any patient requiring massive transfusion.

SUMMARY AND RECOMMENDATIONS

Optimal early intervention has been shown to improve patient outcomes in many medical conditions including trauma.[28] Unfortunately, EDs are often overcrowded and understaffed, and inpatient and intensive care beds are often in short or limited supply. As a consequence, increasing numbers of critically ill patients are boarded in EDs while awaiting inpatient bed availability.[2,3,5,31]

Seriously injured pediatric trauma patients must be carefully monitored and frequently assessed, whether in the ED, the radiology department, the pediatric ICU, or in-transit between locations. Careful attention must be paid to the airway, breathing, and adequacy of oxygenation and ventilation. Caregivers should be comfortable with bag-mask ventilation, tracheal intubation, and even ventilator management for patients who may remain in the ED awaiting an ICU bed or transfer to a tertiary care center.

All patients should have adequate vascular access. Often, IV access can be difficult to obtain in infants and small children. Equipment for intraosseous access should be readily available and caregivers familiar with their use. Central venous catheter placement may also be needed, especially in patients requiring vasopressor infusion or administration of multiple medications. The most commonly used site for central line insertion in pediatric patients is the femoral vein. This site is often chosen due to relative ease of access and because placement does not require removal of the cervical collar in trauma patients or interfere with airway manipulation. However, in patients with intra-abdominal hemorrhage, a femoral line may not be the best choice; in these patients, a subclavian line may be a more optimal choice.

Patients must be carefully monitored for the subtle early signs of shock and every effort made to reverse shock before compensatory mechanisms are overwhelmed. Placement of an arterial line may be helpful for both blood pressure monitoring and frequent laboratory draws; especially in small children in whom central access is not established. Placement should be considered for any patient who is on vasopressors, has an ICP monitor in place, or has persistent hypotension or other signs of clinical instability. If an appropriately sized arterial line kit is not available, a 24- or 22-gauge catheter may be placed in the radial, dorsalis pedis, or posterior tibial artery. A single-lumen, 3 French, 5- or 8-cm-long central venous catheter may also be placed in the femoral artery of infants or children.

Maintaining airway, breathing, and circulation are always top priorities in the management of trauma patients. Control of pain and anxiety is another important component of trauma care that may be overlooked in the critically injured pediatric patient. Small doses of opiates and/or benzodiazepines may be given and repeated as needed, with constant monitoring for the depression of level of consciousness, respiratory drive, and blood pressure that may occur with these medications. If the child is intubated, ensuring adequate sedation is paramount to maintaining control of the airway. Inadequate sedation may lead to a host of secondary issues from airway edema to aspiration and may increase ICP in patients with TBI. Intubated patients may benefit from continuous low-dose infusions of narcotics and/ or benzodiazepines to maintain adequate levels of

sedation. On occasion, it may also be necessary to use neuromuscular blocking agents (paralytics) to assist ventilation or control ICP. These medications may be given as either intermittent doses or continuous infusions. It is vitally important to maintain adequate sedation in patients receiving neuromuscular blockade. Close monitoring of blood pressure and heart rate, especially changes in response to positioning, suctioning, or other noxious stimuli, may provide valuable information about the patient's level of sedation.

While awaiting transfer to an appropriate ICU setting, every effort should be made to "bring the ICU to the patient" by providing close monitoring, frequent reassessment, and rapid correction of problems as they arise. Emergency department personnel should also keep in mind that consultation with colleagues in critical care medicine or anesthesia is often available to help guide patient management, even if an ICU bed is not physically available for a critically injured patient. ✚

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