Severe Traumatic Brain Injury in Children: An Evidence-Based Review of Emergency Department Management

Abstract

More than 1.7 million traumatic brain injuries occur in adults and children each year in the United States, with approximately 30% occurring in children aged <14 years. Traumatic brain injury is a significant cause of morbidity and mortality in pediatric trauma patients. Early identification and management of severe traumatic brain injury is crucial in decreasing the risk of secondary brain injury and optimizing outcome. The main focus for early management of severe traumatic brain injury is to mitigate and prevent secondary injury, specifically by avoiding hypotension and hypoxia, which have been associated with poorer outcomes. This issue discusses methods to maintain adequate oxygenation, maximize management of intracranial hypertension, and optimize blood pressure in the emergency department to improve neurologic outcomes following pediatric severe traumatic brain injury.

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Case Presentations

A 22-year-old mother of 3 brings her 3-month-old boy to the ED, stating that he has not been feeding well. She is vague in her description of the child’s symptoms. The nurse calls you into triage because she notes the child appears unresponsive. The mother denies any trauma. The infant’s vital signs are as follows: afebrile; heart rate, 160 beats/min; blood pressure, 70/40 mm Hg; respiratory rate, 30 breaths/min; and oxygen saturation, 93% on room air. You struggle to calculate a GCS score, as this patient is not yet verbal. On physical examination, the child is minimally responsive and has irregular and shallow respiration, so you prepare to intubate. During placement of an IV line, the child flexes his left arm in response to pain, but no spontaneous movement of the right arm or leg is noted. During the secondary survey, you note a bulging fontanelle and a dilated left pupil, with deviation of the left eye both downward and peripherally. You have the clerk page neurosurgery emergently. The respiratory therapist asks you if you would like to hyperventilate the patient. What should your target PaCO₂ level be? What medication(s) should be given immediately? Once stabilized, are there any other services or specialists that should be involved with this patient, based on the history?

You then get an EMS notification that they are bringing in a 17-year-old girl who was an unrestrained front passenger in a high-speed motor vehicle crash. When the patient arrives, you quickly calculate a GCS score of 7 (E1, M4, V2), and you page the trauma surgery team to the ED. The patient’s vital signs are as follows: afebrile; heart rate, 115 beats/min; blood pressure, 110/60 mm Hg; respiratory rate, 10 breaths/min; and oxygen saturation, 90% on room air. She has obvious right-sided head trauma and is in cervical spine immobilization. You immediately place a non-rebreather mask on the patient and call for rapid sequence intubation medications and equipment. You intubate the patient while maintaining cervical spine precautions. The neurosurgeon calls you back and states that he is on his way to the hospital. What initial steps should be taken to stabilize this patient? How will you determine the disposition for this patient?

Introduction

Accidental injury is the primary cause of death for children and adolescents in the United States, and severe traumatic brain injury (TBI) is an important contributor to morbidity and mortality in trauma. Approximately 30% of trauma-related deaths are attributed to TBI. It was estimated that, between 2002 and 2006, there were more than 1.7 million TBIs occurring in adults and children each year, with an overall mortality rate of 3%, or approximately 52,000 deaths per year. Thirty percent of TBIs occur in children aged 0 to 14 years, and 92% of this group will visit an emergency department (ED) for their injury. In 2006, the overall mortality rate for children aged < 14 years suffering from any TBI was 0.42%.

Approximately 40% of TBIs in children aged 0 to 14 years are due to falls, and 24% result from unintentional blunt trauma to the head, and the most likely mechanism is a motor vehicle crash. Assault, or nonaccidental trauma (NAT), is the third leading cause of TBI in children aged 0 to 4 years. While the most common cause of deadly TBI in children aged < 4 years is assault, the most common cause of death from TBI in young people aged 5 to 24 years is a motor vehicle crash. For patients who die secondary to TBI, risk factors include male sex and age < 4 years. The rate of ED visits is also highest for children aged < 4 years.

The total cost for all patients with TBI in 2010 was estimated at $76.5 billion, 90% of which was spent on patients who were hospitalized. For patients who do not die from the initial injury, approximately half are left with a severe disability. Today, there are 5.3 million people in the United States who are living with serious motor, cognitive, sensory, and/or emotional effects from TBI.

Severe TBI represents a spectrum of diseases, resulting from both blunt and penetrating trauma, and it is most simply defined by an initial Glasgow Coma Scale (GCS) score of < 9. Severe pediatric TBI can be thought of as 2 distinct, but related, diseases: primary injury and secondary injury. Primary injuries are either diffuse, focal, or a combination of the two. This is the injury with which the patient presents, over which the physician has no control. Half of all patients who die from severe TBI die in the first 2 hours after the primary injury. Following the primary injury, there may be edema, changes in cerebral blood flow and perfusion, and decreased respiration/hypoxia. All of these constitute and/or contribute to the complex environment that creates the patient’s secondary injury. The primary goal of emergency treatment of severe TBI is to mitigate and prevent secondary injury, specifically by avoiding hypotension and hypoxia.

Progress has been made in decreasing absolute mortality in pediatric TBI. From 2001 to 2010, there has been a 70% increase in the number of ED visits due to TBI, a slight increase in hospitalizations for patients with TBI (11%), and a decrease in mortality by 7%.

This issue of Pediatric Emergency Medicine Practice reviews the various types of primary injuries, outlines effective measures to optimize initial resuscitation and minimize secondary injuries, provides treatment strategies for herniation syndromes and elevated intracranial pressure (ICP), and discusses novel treatment strategies for pediatric patients with severe TBI.
Critical Appraisal Of The Literature

A literature search was performed in Ovid MEDLINE® and PubMed using the search terms severe pediatric traumatic brain injury and traumatic brain injury children. A total of 230 review articles, 184 clinical trials, and 98 systematic reviews from the year 1959 to the present were reviewed. The Cochrane Database of Systematic Reviews was searched using the term traumatic brain injury pediatric; 52 reviews were identified, with 6 of those pertinent to the scope of this article, related to management, outcome, or prevention of severe TBI in children. The National Guideline Clearinghouse document titled “Head Injury - Triage, Assessment, Investigation, and Early Management of Head Injury in Children, Young People, and Adults” was also reviewed. The following guidelines released by the Brain Trauma Foundation for pediatric and adult traumatic brain injuries were also reviewed: Guidelines for the Acute Medical Management of Severe Traumatic Brain Injury in Infants, Children, and Adolescents - Second Edition (2012); Guidelines for the Management of Severe Traumatic Brain Injury, Third Edition (2007); and Guidelines for the Pre-hospital Management of Severe Traumatic Brain Injury, Second Edition (2007). Information from government databases was reviewed, including the United States Centers for Disease Control and Prevention (www.cdc.gov).8-14

There are very few high-quality studies evaluating management of severe pediatric TBI. Using standard evidence-level scales, the majority of the recommendations discussed in this article fall into lower or intermediate levels of evidence, with few recommendations meeting criteria for a higher level of evidence. Other recommendations are based on consensus opinion extrapolated from adult studies or current standards of care. High-quality pediatric studies are still warranted, with many clinical questions related to pediatric TBI remaining unanswered.

Table 1. Types Of Primary Traumatic Brain Injuries

<table>
<thead>
<tr>
<th>Intra-axial Injury</th>
<th>Extra-axial Injury</th>
<th>Vascular Injury</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diffuse axonal injury</td>
<td>Epidural hematoma</td>
<td>Vascular dissection</td>
</tr>
<tr>
<td>Cortical contusion</td>
<td>Subdural hematoma</td>
<td>Carotid cavernous fistula</td>
</tr>
<tr>
<td>Intracerebral hematoma</td>
<td>Subarachnoid hemorrhage</td>
<td>Arteriovenous dural fistula</td>
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</table>

Etiology And Pathophysiology

TBI can be divided into 2 distinct categories: primary injury and secondary injury. Primary injury is directly related to the initial impact and external force.8 Primary injury may be due to rotational, angular, or linear forces, and may include intra-axial, extra-axial, and vascular injury. (See Table 1.)

Secondary injury is a complication of the primary injury that may develop over a period of hours to months and may be acute or chronic.8 Secondary injury may be exacerbated by physiologic and systemic complications such as hypoxia, hypotension, intracranial hypertension, edema, seizures, hypercapnia (hypventilation), glucose and electrolyte abnormalities, vasospasm, and hyperthermia.9

The factors contributing to secondary injury are potentially preventable, and are the main focus for early management for emergency clinicians. Other factors contributing to secondary injury include cellular and biochemical endogenous cascades causing inflammation and excitotoxicity, leading to axonal injury and neuronal cell damage and death.10,11 Although this is an area of active research, there are currently no specific treatments for these inflammatory cascades.11

The most common primary injuries include subdural hematoma, epidural hematoma, subarachnoid hemorrhage, cerebral contusions, diffuse axonal injury, intraventricular hemorrhage, and penetrating injury.

Subdural Hematoma

A subdural hematoma is a collection of blood in the space between the arachnoid and dura mater, and may result from blunt force, inertial shearing, or rotational force. It typically results from tearing of the bridging cortical veins and has a crescentic appearance on computed tomography (CT) scan. (See Figure 1, page 4.) In children, subdural hematomas are often bilateral, and are associated with parenchymal injury. They are usually supratentorial and are often found in the falx and along the tentorium. Midline shift, cerebral compression, and increased ICP may be seen. Treatment options and early surgical intervention are often based on a combination of clinical status, neurologic examination, size of collection, and degree of midline shift.

Epidural Hematoma

Epidural hematomas result when bleeding occurs between the skull and the dura mater, and may occur in up to 3% of head injuries. (See Figure 2, page 4.) Epidural hematomas are less common in children aged < 2 years compared to adults due to increased adherence of the dura to the skull and increased mobility of the middle meningeal artery in children. Epidural hematomas may result from arterial bleeding (middle meningeal artery) or venous bleeding.
(dural sinus or diploic veins) and often reach peak size within 6 to 8 hours. CT scan of an acute epidural hematoma will demonstrate a hyperdense, lenticular, or convex shape collection. Rapid consultation with a pediatric neurosurgeon is critical, as emergency craniotomy and surgical decompression may improve neurologic outcome.

**Traumatic Subarachnoid Hemorrhage**

Traumatic subarachnoid hemorrhage may be caused by damage to small subarachnoid vessels or an intraventricular or subarachnoid rupture of either a contusion or a hemorrhage in the area under the arachnoid mater. It may lie next to or under an area of subdural hematoma and can also be seen with an epidural hematoma or intraventricular hemorrhage. On CT scan, a hyperdense collection may be seen extending into brain sulci. (See Figure 3, page 5.) Complications of subarachnoid hemorrhage include rebleeding, vasospasm, hydrocephalus, and intracranial hypertension. Studies have demonstrated that the presence of traumatic subarachnoid hemorrhage is independently associated with poorer outcome.

**Cerebral Contusion**

Cerebral contusion with or without intraparenchymal hemorrhage is caused by direct injury to the brain. It is a type of intra-axial hemorrhage that may result from a direct blow at the injury site (coup contusion) or at a location remote from the injury location (contrecoup contusion). (See Figure 4, page 5.) Coup and contrecoup contusions are uncommon in children aged < 4 years. Shortly after an initial contusion develops, the hemorrhage often expands or a new hemorrhage develops. This progression has historically been attributed to damaged microvessels, but coagulopathy is now thought to be a significant contributing factor. Approximately 30% of contusions exhibit mass effect during the subacute stage when edema is also expected to increase. Hemorrhagic contusions typically appear hyperdense on CT scan, but magnetic resonance imaging (MRI) is more sensitive for diagnosis of nonhemorrhagic contusions. Patients with a lower GCS score and with larger contusions are more likely to exhibit progression and are more likely to require surgical decompression.

**Diffuse Axonal Injury**

Diffuse axonal injury is irreversible damage caused by severe sudden acceleration, deceleration, and rotational force that results in shearing, axonal stretch, and neuronal death. Infants are more vulnerable to this type of injury than older children and adults due to their larger head sizes, weaker neck muscles, and wider cerebrospinal fluid (CSF) spaces. A clinical history of immediate loss of consciousness with possible decerebrate or decorticate posturing may be suggestive of diffuse axonal injury. CT scan results are usually normal but may include small petechial hemorrhage at the junction of subcortical white and gray matter, minimal intraventricular blood, or cerebral edema. (See Figure 5, page 6.)

**Figure 1. Subdural Hematoma On Computed Tomography**

Arrows point to areas of subdural hematoma. Image courtesy of William A. Knight, IV, MD.

**Figure 2. Epidural Hematoma On Computed Tomography**

Arrow points to area of epidural hematoma. Image courtesy of William A. Knight, IV, MD.
**Intraventricular Hemorrhage**

The tearing of subependymal veins, extension of a subarachnoid hemorrhage into the ventricle, or dissection of an intracerebral hematoma into the ventricle may cause intraventricular hemorrhage. Blood then fills the aqueduct and can result in an obstructive hydrocephalus. CT scan is useful for assessment of ventricle size and identification of blood in the ventricle.

**Penetrating Injury**

Penetrating head trauma occurs when an object pierces the skull and impacts brain tissue. This injury is uncommon in children, but, when present, it is responsible for significant morbidity and mortality. Children aged < 2 years are more likely to have penetration of the skull during trauma due to incomplete ossification of the bones.

**Elevated Intracranial Pressure**

ICP is the measurement of pressure within the confines of the rigid skull. The space within the skull is comprised of brain (80%), CSF (10%), and blood (10%). The Monro-Kellie doctrine states that once fontanelles and sutures are closed, the total volume within the cranial vault remains fixed, and changes in the volume of one component may occur only at the expense of another. The brain is able to compensate for small changes in pressure through adjustments in CSF and blood volume, after which point, minimal changes in volume lead to large changes in ICP.

Increased ICP immediately following head trauma may be due to a combination of bleeding, hematoma formation, and/or edema. Since direct ICP monitoring is typically not available during the early stages of resuscitation, one should assume that ICP is elevated following a severe TBI. Cerebral perfusion pressure is a calculated value that represents the perfusion gradient across the brain that is important in maintaining cerebral blood flow. It is calculated by using the formula CPP = MAP - ICP. (CPP, cerebral perfusion pressure; MAP, mean arterial blood pressure.) The relationship between cerebral perfusion pressure and cerebral blood flow is represented by the formula CBF = CPP/CVR. (CBF, cerebral blood flow; CVR, cerebrovascular resistance.)

Cerebral perfusion pressure must be maintained within normal limits or ischemic brain damage results. Because cerebral perfusion pressure is closely related to MAP, it is important to maintain systolic blood pressure (SBP) in appropriate ranges. Under normal physiologic conditions, cerebral autoregulation helps to maintain cerebral perfusion pressure within a narrow range. When autoregulation is intact, increases in MAP result in vasoconstriction, a decrease in cerebral blood volume, and a decrease in ICP. Autoregulation is impaired after TBI, and normal compensatory methods are no longer effective. With low blood pressures and low MAPs, vasodilation occurs in an attempt to maintain blood flow to the brain. With high blood pressure and impaired...
autoregulation pressures, cerebral blood flow is also increased and ICP becomes elevated.

Hypercapnia and hypocapnia also affect cerebral blood flow. Hypercapnia causes increased cerebral blood flow through cerebral vasodilation, whereas hypocapnia causes a decrease in blood flow. Hypoxia also results in vasodilation, increases cerebral blood flow, and raises ICP through volume changes and increased vasogenic edema. Edema following a TBI is secondary to a combination of vasogenic factors, disruption of the blood-brain barrier, and cytotoxic effects.\(^{18}\)

**The Glasgow Coma Scale And The Pediatric Glasgow Coma Scale**

The GCS was developed in 1974 as a method to rapidly identify an impaired level of consciousness in patients with acute brain injury. In young children, a low GCS score may not always accurately predict outcome, however.\(^{21}\) In 2005, Holmes et al compared a modified pediatric GCS (pGCS) to the adult GCS, taking into account the limitations in assessing verbal and motor responses in children aged < 2 years.\(^{22}\) (See Table 2, page 7.) Use of the pGCS is recommended in children in this age group.

**Differential Diagnosis**

In an unresponsive patient, other causes of altered mental status should always be considered. Seizures, ingestions, electrolyte abnormalities, or metabolic disorders should be part of the differential diagnosis. Stroke is less common in pediatric patients than in adults, but should also be considered. When there is no obvious history of preceding trauma, it is important to consider what caused the patient to sustain a head injury. A seizure may precipitate a head injury or the patient may have suffered a posttraumatic seizure following a direct impact. Intoxication may be a confounding factor in the neurological evaluation of adolescents and young adults. A differential diagnosis of causes of TBI in an infant may include accidental trauma, NAT, meningitis, birth trauma, bleeding disorders, vitamin K deficiency, osteogenesis imperfecta, and metabolic disorders.\(^{23}\)

**Prehospital Care**

The goals of prehospital emergency medical services (EMS) care are early recognition of the pediatric patient with severe TBI and safe and efficient transport to the nearest facility with available trauma surgery and pediatric neurosurgery. Initial resuscitation focuses on stabilizing the airway, breathing, and circulation. Patients with severe TBI should also have cervical spine immobilization placed, and an initial GCS or pGCS score should be calculated.

In many cases, the patient must be taken to the closest available ED for stabilization. These patients should then be transferred as quickly as possible to a center with pediatric neurosurgery and a pediatric intensive care unit (PICU). Prior to transfer, clinicians should obtain intravenous access and a controlled, patent airway. CT of the brain should be considered prior to any transport.\(^{24}\)

Prehospital guidelines have been standardized and have led to decreased morbidity and mortality in pediatric patients with TBI.\(^{6,25}\) The most clinically significant prehospital action is the prevention of hypoxia.

**Airway, Oxygenation, And Ventilation**

Oxygen saturation should be monitored continuously during prehospital care. Hypoxia (partial pressure of oxygen in arterial blood \([\text{PaO}_2] < 60 \text{ mm Hg}\)), hypocapnia (partial pressure of carbon dioxide in arterial blood \([\text{PaCO}_2] < 35 \text{ mm Hg}\)), and hypercapnia (\([\text{PaCO}_2] > 45 \text{ mm Hg}\)) should be avoided when end-tidal carbon dioxide (ETCO\(_2\)) monitoring is available. Nasotracheal intubations should be avoided due to possible concomitant facial trauma or basilar skull fracture. If available, cuffed tubes are preferred to minimize aspiration of stomach contents. Hypoxia is noted to be an independent risk factor for both increased morbidity and mortality after TBI.\(^{7}\)

Ventilation can be performed via bag-valve mask or via endotracheal intubation. There has been no demonstrated difference in morbidity or mortality between the 2 modalities.\(^{26}\) Patients should be oxygenated with 100% fraction of inspired oxygen (FiO\(_2\)). Retrospective data show that patients who have a \([\text{PaO}_2] > 300 \text{ mm Hg}\) at admission to the ED have a higher rate of survival. Survival to discharge is also increased in patients with a \([\text{PaCO}_2]\) between 36 and 45 mm Hg.\(^{27}\)

Prophylactic hyperventilation should not be...
performed in the prehospital setting, as there is a potential for a rebound increase in ICP. Transient mild hyperventilation (lowering the PaCO₂, to 30 mm Hg) can be used as a lifesaving technique, but it is only recommended if a patient shows signs of impending herniation. Hyperventilation should be halted when signs of herniation are no longer present.²⁸

**Blood Pressure And Perfusion Optimization**

Hypotension is another critical risk factor for increased morbidity and mortality. The best way to noninvasively estimate whether a patient has adequate cerebral perfusion pressure is to measure the patient’s blood pressure. Age-appropriate SBP increases as a person gets older. (See Table 3, page 8.) Vavilala et al published guidelines based on the minimum age-appropriate SBP.²⁹ They noted that patients with age-appropriate SBPs above the 75th percentile had significantly better outcomes than those below the 75th percentile.

Lactated Ringer’s or normal saline isotonic solutions are used in bolus form to treat hypotension. The use of isotonic solutions are thought to result in the lowest incidence of subsequent herniation. Boluses are calculated by patient weight, at 20 mL/kg.³⁰ Dextrose should not be given to the pediatric TBI patient unless it is to treat a glucose level < 70 mg/dL to maintain iso-osmolar blood concentrations of electrolytes.³¹

**Emergency Department Evaluation**

**Focused History And Physical Examination**

The history for a child with severe TBI is typically straightforward, and the presentation is dramatic, with the major exceptions being in the very young child or children who are victims of NAT. Important historical factors include age, comorbid medical conditions, medications and allergies, and the last meal eaten by the patient. Simultaneous drug or alcohol intoxication can be a confounder in older pediatric and adolescent populations. TBI should be considered if any of the following have occurred: motor vehicle crash, fall from significant height (> 5 ft for patients aged ≥ 2 years or > 3 ft for patients aged < 2 years), penetrating trauma to the skull, loss of consciousness, signs of basilar skull fracture, and/or persistent vomiting.³² In infants, NAT must be considered, as discussed in greater detail in the “Special Circumstances In Pediatrics” section, pages

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**Table 2. Comparison Of The Glasgow Coma Scale And Pediatric Glasgow Coma Scale**²²

<table>
<thead>
<tr>
<th>Glasgow Coma Scale (Ages ≥ 2)</th>
<th>Score</th>
<th>Pediatric Glasgow Coma Scale (Ages &lt; 2)</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Best response</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Eye Opening (E)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Spontaneous</td>
<td>4</td>
<td>Spontaneous</td>
<td>4</td>
</tr>
<tr>
<td>To speech</td>
<td>3</td>
<td>To speech Hypotensive Hypoxic</td>
<td>3</td>
</tr>
<tr>
<td>To pain</td>
<td>2</td>
<td>To pain</td>
<td>2</td>
</tr>
<tr>
<td>None</td>
<td>1</td>
<td>None</td>
<td>1</td>
</tr>
<tr>
<td><strong>Verbal (V)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Oriented, converses</td>
<td>5</td>
<td>Coos, babbles</td>
<td>5</td>
</tr>
<tr>
<td>Disoriented, converses</td>
<td>4</td>
<td>Irritable, cries</td>
<td>4</td>
</tr>
<tr>
<td>Inappropriate words</td>
<td>3</td>
<td>Cries to pain</td>
<td>3</td>
</tr>
<tr>
<td>Incomprehensible sounds</td>
<td>2</td>
<td>Moans to pain</td>
<td>2</td>
</tr>
<tr>
<td>None</td>
<td>1</td>
<td>None</td>
<td>1</td>
</tr>
<tr>
<td><strong>Motor (M)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal spontaneous movements</td>
<td>6</td>
<td>Normal spontaneous movements</td>
<td>6</td>
</tr>
<tr>
<td>Localizes pain</td>
<td>5</td>
<td>Withdrews to touch</td>
<td>5</td>
</tr>
<tr>
<td>Withdrews to pain</td>
<td>4</td>
<td>Withdrews to pain</td>
<td>4</td>
</tr>
<tr>
<td>Abnormal flexion (decorticate rigidity)</td>
<td>3</td>
<td>Abnormal flexion (decorticate rigidity)</td>
<td>3</td>
</tr>
<tr>
<td>Abnormal extension (decerebrate rigidity)</td>
<td>2</td>
<td>Abnormal extension (decerebrate rigidity)</td>
<td>2</td>
</tr>
<tr>
<td>None</td>
<td>1</td>
<td>None</td>
<td>1</td>
</tr>
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TBI may be complicated by multiple factors, including rapid deterioration, direct facial or tracheal trauma, and maintenance of cervical spine precautions. RSI with preoxygenation should be used in almost all patients because the sympathetic stimulus from endotracheal intubation can cause an increase in ICP. Airway adjuncts should be prepped at the bedside, including video laryngoscopy, jet insufflation, and surgical airway kits. The focus should be on strict avoidance of hypoxia, hypercapnia, and hypocapnia.

Pretreatment For Intubation

Atropine
Due to the risk for bradycardia during the peri-intubation period, atropine use may be considered for children aged < 1 year and children aged < 5 years receiving succinylcholine. The dose of atropine is 0.02 mg/kg, with a maximum dose of 0.5 mg and a minimum dose of 0.1 mg. This minimum dose can be used as a guideline, as minimum dosing for emergency intubations has been removed from the Pediatric Advanced Life Support (PALS) guidelines. Atropine can be given intramuscularly or intravenously. It is used to blunt the bradycardic reflex that is seen in children with the stimulation from endotracheal intubation.

Although atropine is still used routinely, the need for pretreatment with atropine has been questioned in recent literature. The American College of Emergency Physicians recently published a recommendation that atropine should be kept in reserve as an option for intubation pretreatment, but should no longer be considered mandatory for all pediatric intubations. These are limited studies and this is an area for future research. Disadvantages of atropine use include the loss of the pupillary reflex in further neurological examinations and the decreased ability to use bradycardia as a sign of impending herniation.

Lidocaine
Lidocaine has been given as pretreatment to pediatric patients to prevent a transient increase in ICP that is often seen when a patient is stimulated during endotracheal intubation. The dose of lidocaine is 1.5 mg/kg, with a maximum dose of 100 mg intravenously. Lidocaine should be given 2 to 3 minutes prior to intubation. It suppresses the cough reflex and blunts the increase in ICP. Lidocaine is a class IB antidyssrhythmic, has side effects, and should be used judiciously in patients with a history of bradycardia or heart block. Emergent intubation should not be delayed to wait for the administration of lidocaine. Evidence to support lidocaine pretreatment is not strong, and current trends in pediatric intubations show a decrease in the use of premedication.

Critical Care

Table 3. Age-Appropriate Minimum Systolic Blood Pressure

<table>
<thead>
<tr>
<th>Age</th>
<th>Systolic Blood Pressure (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-28 days</td>
<td>&gt; 60</td>
</tr>
<tr>
<td>1-12 months</td>
<td>&gt; 70</td>
</tr>
<tr>
<td>1-10 years</td>
<td>&gt;70 + (2 x age in years)</td>
</tr>
<tr>
<td>&gt; 10 years</td>
<td>&gt; 90</td>
</tr>
</tbody>
</table>
**Fentanyl**
There is currently no strong evidence in children that opioid pretreatment improves outcome or increases the success of intubation.\(^45\) Fentanyl pretreatment may be used for sedation and pain control. If used, it should be given at 1-3 mcg/kg intravenously approximately 3 minutes prior to intubation attempt. Emergency intubation should not be delayed to allow for fentanyl administration. It is proposed that, with pain control, the noxious stimulation of the endotracheal intubation procedure is mitigated, preventing increases in ICP. Fentanyl may also decrease the respiratory drive of the patient and may contribute to both hypoxia and hypotension.

**Induction And Sedation**
Prior to endotracheal intubation, the patient must be sedated and induced into a state where intubation is easily accomplished. Typically, etomidate, ketamine, or midazolam are used.

**Etomidate**
Etomidate is a commonly used agent for sedation and induction. It carries a lower risk of cardiovascular depression than benzodiazepines and opioids, and is potentially neuroprotective.\(^46\) The dose is 0.3 mg/kg intravenously. The major side effect of etomidate is concern for transient adrenal suppression. Theoretically, this could have severe consequences, though current research has not shown any increase in morbidity or mortality with a single dose of etomidate for RSI induction.\(^47\)

**Ketamine**
Ketamine offers both amnestic and analgesic effects. Ketamine has the known positive effects of bronchodilation and also does not cause hemodynamic instability.\(^48\) Ketamine is used at a dose of 1-2 mg/kg intravenously, or 3-7 mg/kg intramuscularly. For many years, there was concern that ketamine increased ICP in patients. This deleterious effect on ICP has now been questioned,\(^49\) and some studies theorize that ketamine causes a decrease in ICP.\(^50\) The use of ketamine is still controversial and is discussed more thoroughly in the “Controversies And Cutting Edge” section, page 16.

**Midazolam**
Midazolam is no longer widely used in induction for RSI in pediatric patients with severe TBI. This is due to the fact that it has a slower onset of action (2-3 minutes) and has more cardiovascular depression associated with its use. Midazolam is used at a dose of 0.2-0.3 mg/kg intravenously, with a maximum dose of 2 mg. Midazolam has antiepileptic properties that may be potentially useful in a patient with a severe TBI.

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**Paralysis**
Neuromuscular blockade makes the conditions surrounding RSI easier, creating a higher likelihood of successful intubation. However, neuromuscular blockade should be considered on an individual basis because it can also make it very difficult, or impossible, to maintain oxygenation through a difficult intubation that requires several attempts.\(^48\) It has been theorized that neuromuscular blockade is crucial in infants and toddlers, as they are noted to have increased heart variability.\(^51\) Defasciculating doses of paralytics are no longer in use in current practice.

**Rocuronium**
Rocuronium is often used for neuromuscular blockade in the pediatric TBI patient, and is favored by physicians who primarily treat pediatric populations. The dose is 1 mg/kg intravenously.\(^52\) Rocuronium is a nondepolarizing neuromuscular blockade agent. It has a variable onset, depending on the age of the patient, at 30 seconds to 1 minute in young children, and up to 1 to 2 minutes in adolescent children. It has a moderate half-life, and its effect lasts approximately 30 minutes in children and up to 40 minutes in infants. In multiple studies, rocuronium was shown to produce paralysis similar to succinylcholine with the same rapid onset. The most negative aspect of rocuronium is the duration of paralysis.\(^52,53\)

**Succinylcholine**
Succinylcholine is the only depolarizing neuromuscular blockade agent, and it is widely used for RSI in pediatric patients with severe TBI. In children, it is dosed at 1-2 mg/kg intravenously. It can also be given intramuscularly at 3-5 mg/kg. In the trauma patient, the major contraindication for the use of succinylcholine is concomitant hyperkalemia. In patients with severe TBI who also have suffered a crush injury, another agent should be used. Succinylcholine should also be used with caution in populations with myasthenia gravis and muscular dystrophy. If used in myasthenia gravis patients, higher doses may be necessary due to the decrease in acetylcholine receptors. Malignant hyperthermia is the most dangerous side effect of succinylcholine use and should be monitored in patients who become hyperthermic after its administration.\(^48\)

Succinylcholine has a very rapid onset of action, and flaccid paralysis is achieved in < 60 seconds. The duration of paralysis after a single intravenous dose is between 4 and 6 minutes. This is desirable when considering paralysis for what may be a difficult airway. Repeat doses may be used, but increase the risk for bradycardia and hypotension. For years, succinylcholine was thought to increase ICP, and, therefore, was not used on patients with TBI. The evidence for this has been largely refuted, and, today, succinylcholine is routinely used without concern about its effect on ICP.\(^24,54\)
Clinical Pathway For Treatment Of Pediatric Patients With Severe Traumatic Brain Injury

**Pediatric patient with GCS score < 9**

- Immobilize cervical spine
- Assess airway
- Assess respiration
- Assess circulation (heart rate, blood pressure)
- Place IV
- Perform glucose point-of-care
- Avoid hypoxia and hypotension

**AIRWAY PATENT**

- Assess disability
- Undress the patient, check for other traumatic injuries
- Oxygenate with FiO\(_2\) 100%

**AIRWAY NOT CONTROLLED**

- Intubate using RSI (unless patient has critical vital signs, or has no muscle tone) with or without pretreatment

**Signs of active or pending herniation?**

- Decrease in mental status
- Pupil irregularity
- Decorticate or decerebrate posturing
- Bradycardia and hypertension

**IF HYPOTENSIVE**

- Initiate treatment for herniation:
  - Administer 3% hypertonic saline 6.5-10 mL/kg IV (Class II) or mannitol 1 g/kg IV (Class III)
  - RSI, if not already intubated
  - Consider hyperventilation to PaCO\(_2\) 30-35 mm Hg (Class III)
  - Emergent neurosurgery consultation

**IF HYPOXIC**

- Monitor heart rate and blood pressure closely

**SBP > 75\(^{th}\) percentile of age-appropriate SBP?**

- YES
  - Is the patient stable for transport to CT?

**NO**

- Initiate bolus intravenous fluids treatment with 0.9% normal saline, 20 mL/kg
- Maintain SBP

**Maintain PaO\(_2\) as high as possible with 100% FiO\(_2\) (Class III)**

- Maintain normal PaCO\(_2\) (35-45 mm Hg) (Class III)

**Is the patient stable for transport to CT?**

- YES
  - Intracranial injury on CT?

**NO**

- Signs of herniation?

**YES**

- Midline shift, mass effect, or herniation?

**NO**

- Intracranial injury on CT?

**YES**

- PICU admission or transfer to appropriate trauma center
- Neurosurgery consultation

**Emergency neurosurgical decompression or transfer to a trauma center with neurosurgery availability**

**Abbreviations:** CT, computed tomography; FiO\(_2\), fraction of inspired oxygen; IV, intravenous; PaCO\(_2\), partial pressure of carbon dioxide in arterial blood; PaO\(_2\), partial pressure of oxygen in arterial blood; RSI, rapid sequence intubation; SBP, systolic blood pressure.

See page 11 for Class of Evidence definitions.
Ventilation Management

Maintaining adequate ventilation and preventing hypoxia are primary goals in the care of the pediatric TBI patient. Hypoxia (PaO₂ of < 60 mm Hg or an oxygen saturation of < 90%) is one of the primary parameters associated with discharge survival; the higher the initial partial pressure of oxygen is for the patient, the higher the rate of long-term survival.27 All patients should receive supplemental oxygen via endotracheal tube or via nonrebreather mask. Oxygenation of all pediatric TBI patients must be monitored in the ED via continuous pulse oximetry.

The ideal PaCO₂ in pediatric TBI patients is between 35 and 45 mm Hg, or normocapnia. Both hypocapnia and hypercapnia should be avoided. A retrospective cohort analysis showed that 60% of pediatric TBI patients have an episode of hyperventilation with a PaCO₂ of < 35 mm Hg during the first 48 hours after injury, and that this is associated with worse outcome.35 Hypercapnia increases ICP and is never desired in the pediatric patient with TBI.

Ventilator settings should provide adequate oxygenation combined with maintenance of normocapnia. If possible, tidal volumes should be set at approximately 6 mL/kg. Multisystem trauma patients may require higher volumes to achieve adequate oxygenation. Increased tidal volumes put patients at risk for acute respiratory distress syndrome. Positive end-expiratory pressure should also be limited, with most experts suggesting a positive end-expiratory pressure of 2 cm H₂O above the lower inflection point.56 It should be noted that almost all of the current pediatric recommendations are extrapolated from adult data from the Acute Respiratory Distress Syndrome Network (www.ardsnet.org).57

While not commonly performed in the ED, high-frequency oscillatory ventilation can be used for patients when traditional ventilation fails.58 The chest is the most common site of concomitant injury in the pediatric patient with TBI, and the lung is the most frequently injured organ. In these cases, high-frequency oscillatory ventilation is a viable alternative to traditional ventilation.35

Circulation Management

Hypotension is the single most powerful prognosticator of poor outcomes in pediatric patients with TBI. Even transient hypotension decreases the discharge survival rate 4-fold.59 In a retrospective study, children who had an SBP > the 75th percentile for age had improved outcomes.29 The timing of hypotensive episodes is also important. In another retrospective study, Samant et al showed that a pediatric patient is most sensitive to hypotension in the first 6 hours after a severe TBI.60

Blood pressure is initially supported with crystalloid intravenous fluids at 20 mL/kg. If blood pressure does not improve with a total of 60 mL/kg of crystalloid infusion and the patient has no need for blood transfusion from primary traumatic injury, vasopressors may be indicated. Norepinephrine, epinephrine, dopamine, and phenylephrine are the most commonly used agents. There is no strong evidence for any agent being superior, though a single trial cited that norepinephrine produced a higher cerebral perfusion pressure.51

If the patient has a need for blood products due to a profusely bleeding scalp wound or from concomitant traumatic blood loss, then colloid transfusion is indicated. Patients with an initial hemoglobin of < 7 mg/dL should be transfused.62 If a patient is hypertensive, blood pressure should be monitored, but not treated pharmacologically, as cerebral autoregulation is often grossly impaired after severe TBI, and lowering the blood pressure can cause a decrease in cerebral perfusion pressure.

Diagnostic Studies

Laboratory Tests

Initial laboratory tests should be ordered on an individual basis and guided by presentation and suspected injuries. A hematocrit and type and screen should be obtained for any serious trauma patient, including those with severe TBI, particularly if the patient might require surgical intervention. Coagu-
Imaging

Noncontrast Computed Tomographic Scan Of The Head
For a patient who has sustained severe head trauma, the goal of neuroimaging is to obtain rapid, sensitive, and specific diagnostic imaging. Noncontrast head CT remains the initial study of choice for rapid evaluation and diagnosis.

Computed Tomographic Angiography
Computed tomographic angiography (CTA) is used to evaluate for cerebrovascular injury. Little is known about the risk factors for vascular injury, and indications for CTA may be underrecognized.

Magnetic Resonance Imaging
MRI is a highly sensitive, multiplanar imaging technique that can provide information on the evolution and age of a TBI and hemorrhage. Additional advantages include increased sensitivity for parenchymal injuries, improved visualization of the posterior fossa, and utilization of nonionizing radiation. Disadvantages of use in the ED include increased time of examination, higher cost, limited availability, and lower sensitivity for detecting skull fracture.

Radiography - Plain Skull Films
Skull radiographs are fast and easy to obtain, but are of limited use during the initial trauma evaluation. Plain films of the skull may detect skull fractures, but are of limited use in prediction of intracranial injury. However, plain films may be of use in particular circumstances, such as during a skeletal survey for suspected NAT.

Brain Ultrasound
Ultrasound may be useful in the neonatal period, when fontanelles are open, to identify some intracranial bleeds. Benefits are decreased radiation exposure, widespread availability, and potentially decreased cost. Similar to other ultrasound studies, the quality and accuracy of the ultrasound depends on appropriate probe selection, machine settings, and examiner experience. Ultrasound should not be relied upon to fully rule out the presence of an intracranial hemorrhage.

Optic Nerve Sheath Diameter Measurement
Ultrasound optic nerve sheath diameter measurement has shown good diagnostic accuracy in adults for detection of elevated ICP in TBI with intracranial hemorrhage. Although optic nerve sheath diameter measurement in pediatrics has been useful in case reports, in a larger study with pediatric emergency physicians, it was not shown to have adequate test characteristics and was not useful in detection of ventriculoperitoneal shunt failure in children.

Management

Management Of Herniation Syndromes

Physical Examination Findings Concerning For Herniation
Herniation occurs when ICP reaches a level that causes translocation of the brain parenchyma and brainstem across the rigid structures of the skull. The surrounding CSF can no longer flow freely around the brain and spinal cord, creating additional pressure gradients. This can occur at multiple levels of the brain, and different types of herniation present with different physical examination findings.

Subfalcine Herniation
This is the most common type of herniation in pediatric patients, usually resulting from an acute focal injury to one side of the brain, causing herniation...
across the falk cerebri. (See Figure 6.) This is noted as midline shift on head CT. Because of the superior location of the falk, the brainstem of the patient is not compressed, which leads to less predictable symptoms, sometimes including unilateral motor compromise in the lower extremities and bladder incontinence. Eventually, the anterior cerebral artery may have compromised flow, causing stroke-like symptoms such as same-sided motor and sensory deficits and problems with speech.73

Central Herniation
This type of herniation occurs when the temporal lobe(s) is(are) translocated across the tentorium cerebelli. (See Figure 6.) This causes the patient to have a forced downward gaze with dilated, unreactive pupils. This type of herniation can cause a Duret hemorrhage from pontine artery disruption and is quickly lethal to the patient.74

Transtentorial Herniation
Also called an uncal herniation, this is another common type of herniation in pediatric patients, in which the supratentorial section of the brain descends toward the infratentorial region across the tentorium cerebelli. (See Figure 6.) This type of herniation results from diffuse or focal injuries to the supratentorial portion of the brain. The uncus puts pressure on the brainstem and can cause compression of the midbrain and cranial nerve III. This causes dilation of the pupil on the side of the injury combined with deviation of the eye downward and peripherally. Midbrain compression causes contralateral hemiparesis. The corticospinal tract of the brain can be affected, and this can cause ipsilateral hemiparesis. This can mislead the emergency clinician as to the site of the original injury and is called the Kernohan notch phenomenon.73,74

Tonsillar Herniation
Tonsillar herniation occurs when contents of the posterior fossa are translocated across the foramen magnum, compressing both the spinal cord and the brain stem. (See Figure 6.) The most significant physical findings are a gradual decrease in the level of consciousness followed by respiratory failure and paralysis with a flaccid tone.73

Management Of Intracranial Pressure
Normal ICP increases with age, though the range is small; 6 mm Hg in infants and up to 15 mm Hg in adults.75 Normally, the body uses autoregulation to change the vascular resistance in the cranium in order to adjust the blood flow to the brain. As any single component of the skull changes in volume, there must be a decrease in volume of the other 2 components to maintain ICP.76 In the setting of severe head injury, autoregulation fails, leading to increased ICP, decreased cerebral perfusion pressure, ischemia, herniation, and death.

ICP can be managed in the ED initially with positioning, medication, and ventilation. All of these interventions can be performed without invasive ICP monitoring. These therapies are initiated based on the deterioration of the clinical neurological status of the patient. When ICP can be measured, the current standard is maintenance at < 20 mm Hg. The goal for cerebral perfusion pressure varies with age,

Table 4. Cerebral Herniation Syndromes And Signs

<table>
<thead>
<tr>
<th>Herniation Subtype</th>
<th>Physical Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subfalcine</td>
<td>• Early unilateral motor deficits of lower extremities</td>
</tr>
<tr>
<td></td>
<td>• Bladder incontinence</td>
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<tr>
<td></td>
<td>• Late same-sided motor and sensory deficit</td>
</tr>
<tr>
<td></td>
<td>• Late speech difficulty</td>
</tr>
<tr>
<td>Central</td>
<td>• Forced downward gaze</td>
</tr>
<tr>
<td></td>
<td>• Dilated, unreactive pupils</td>
</tr>
<tr>
<td>Transtentorial (uncal)</td>
<td>• Dilation of pupil ipsilateral to injury</td>
</tr>
<tr>
<td>herniation)</td>
<td>• Eye deviation downward and peripherally</td>
</tr>
<tr>
<td></td>
<td>• Contralateral or ipsilateral (Kernohan notch phenomenon) hemiparesis</td>
</tr>
<tr>
<td>Tonsillar</td>
<td>• Early gradual decrease in level of consciousness</td>
</tr>
<tr>
<td></td>
<td>• Late respiratory failure</td>
</tr>
<tr>
<td></td>
<td>• Late flaccid paralysis</td>
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</tbody>
</table>
but should be kept > 45 mm Hg, even in the youngest of children. It has been shown that if ICP cannot be controlled, then it is almost universally fatal for the patient.

A simple preliminary action is to elevate the head of the bed to 30°. This has been shown to decrease ICP in adults, although the data are not conclusive in the pediatric population. The next step is to pharmacologically manage the patient with sedation and analgesia. This can be accomplished with a number of agents, including etomidate, ketamine, propofol, opioids, barbiturates, and benzodiazepines. There is no direct evidence that one particular agent is more effective than any other in the pediatric population. Similarly, a 2011 literature review in adults did not show superiority of any particular agent. It is also important to manage pain in the intubated patient, as noxious stimuli can lead to increased ICP.

If sedation and pain control together do not decrease ICP, paralysis is a secondary option. This is not routinely initiated in the ED, but it can be used to control decorticating or decerebrate posturing, to decrease the metabolic demand of the brain, or to eliminate shivering. Additionally, neuromuscular blockade can decrease the positive end-expiratory pressure needed for adequate ventilation. Currently, there are inadequate studies to make conclusions regarding the effect of neuromuscular blockade on clinical outcome.

ICP monitoring in the pediatric patient with severe TBI is considered the standard of care, though there is no good evidence to show that this impacts morbidity or mortality. Typically, ICP monitoring is not initiated in the ED. Patients who need ICP monitoring should be identified and admitted to the PICU or sent to the operating room. These patients include those with a persistently low GCS score, low motor score on the GCS, abnormal head CT, and diffuse cerebral swelling. The gold standard for ICP monitoring is the interventricular shunt. This allows for both direct measurement of ICP as well as withdrawal of CSF, as needed.

Management Of Neurologic Deterioration
Accurate and efficient recognition of neurologic deterioration in the patient with severe TBI is vital to prevent secondary brain injury.

Medical And Noninvasive Treatment Of Suspected Herniation
Osmotic Therapy
The use of an osmotic agent to decrease ICP has been well studied, and it is the most well-tolerated medical technique available. During and after the use of osmotic agents, serum osmolarity should be maintained < 360 mOsm/L. The 2 most widely used agents for osmotic therapy are hypertonic saline and mannitol.

Hypertonic Saline
Hypertonic saline (HTS) is currently used to acutely lower a patient’s ICP. The typical ED dose is 3% HTS at 6.5-10 mL/kg given intravenously over 5 to 10 minutes. While prophylactic HTS administration does not seem to improve outcome in adults, acute treatment with HTS is efficacious in decreasing ICP, increasing cerebral perfusion pressure, and improving outcome in adults and pediatric patients. HTS acts as an intravascular volume expander, drawing fluid from the brain tissue into the vasculature. This expansion also helps maintain adequate blood pressure and cardiac output, and this is one of the main benefits of HTS administration in the critically ill pediatric patient with severe TBI.

The major side effects of HTS therapy are seen in prolonged treatment and include elevation of serum osmolality, hypernatremia, acute kidney injury, central pontine demyelination, and a rebound increase in ICP after therapy is terminated. Resulting hypernatremia must be carefully monitored and slowly lowered to baseline once HTS is no longer being actively administered.

New research is examining whether higher concentrations of HTS are safe in pediatric patients. While the new information is promising, currently, only case reports using 23.4% saline in children with severe TBI are available. More research is needed before higher concentrations of HTS can be utilized reliably in the ED.

Mannitol
Used since the 1960s, 20% mannitol is given in intravenous bolus doses at 0.25-1 g/kg every 4 to 6 hours. Mannitol works in 2 different ways: (1) it creates an osmotic gradient between the brain tissue and the vasculature, drawing intracellular fluid into the cerebral blood vessels, and (2) it has a rheologic effect, which creates an actual decrease in the viscosity of the blood, thereby increasing the flow of blood to the brain tissues and causing reflexive vasoconstriction via autoregulation. However, this rheologic effect may be aborted by the fact that the traumatized brain has defective autoregulation.

The most concerning side effects are hypotension and hypovolemia. Normal saline should be used to counteract these reactions. Mannitol also causes an increase in blood osmolality, and all electrolytes need to be closely monitored during and after administration.

Hyperventilation
Prophylactic hyperventilation in the ED is not recommended. If a patient is showing signs of continued deterioration, moderate hyperventilation may be used as a technique to acutely lower ICP for a short duration. Hyperventilation can lead to cerebral ischemia and worsening of secondary brain injury.
The patient should be mildly hyperventilated to a PaCO₂ of 30 to 35 mm Hg. The only current use of this technique is to temporize a patient with refractory and critically elevated ICP while he is being readied for definitive neurosurgical intervention.

**Barbiturate Therapy**
If a patient has intractable elevations in ICP despite all medical, ventilatory, and surgical management, a treatment of last resort is to place the patient in a barbiturate coma. Pentobarbital is the most commonly used agent. Barbiturate coma is not a first-line treatment because of the detrimental effects that barbiturates have on blood pressure and circulatory status. It is theorized that barbiturates decrease the cerebral metabolic rate, which decreases ICP. The evidence for barbiturate coma is older, and is mixed as to whether or not this treatment is efficacious. This may be because patients may or may not respond to treatment for reasons that are currently unknown.

**Predictors And Factors Associated With Poor Outcome**
Factors that lead to poor clinical outcome and high rates of mortality include low initial GCS score, younger age at injury, abnormal pupillary response, hypothermia, NAT, and concurrent basilar skull fracture.

In 2015, Fulkerson et al published a novel review of predictive variables for survival in pediatric patients. They analyzed 67 patients with an initial GCS score of 3 or 4, noting a 55% mortality rate. Interestingly, patients with a normal pupillary reaction in both eyes had an 87% survival rate, while patients with even a single abnormal pupil were noted to have a survival rate of 23%. If a patient had bilateral reactive pupils and the mechanism of injury was accidental, there was 100% survival.

**Special Circumstances In Pediatrics**

**Neonatal Patients**
Neonatal patients are more likely to have a diffuse, as opposed to a focal, injury. These injuries are more difficult to diagnose based on initial head CT and have fewer options for definitive treatment. In animal studies, younger animals have increased cytotoxic inflammatory cascades and decreased cerebral blood flow when compared to older animals. This may explain why very young children have more-severe brain injury and poorer clinical outcomes. GCS score is not a reliable predictor of outcome for neonatal patients and the physical examination of the very young child is limited. Finally, younger age at the time of injury is independently predictive of poor outcome.

**Nonaccidental Trauma And Abusive Head Trauma**
NAT leading to severe TBI in the pediatric population, particularly in patients aged < 1 year, can be serious and devastating. Injury may be caused by blunt or penetrating trauma, and may be from mechanisms such as shaking or jerking. The emergency clinician must maintain suspicion for NAT and abusive head trauma in order to accurately diagnose, treat, and prevent further trauma. Nearly 80% of the fatalities resulting from trauma in patients aged < 3 years are directly attributed to NAT.

The term *shaken-baby syndrome* has been replaced with the term *abusive head trauma*. The incidence of abusive head trauma is difficult to determine, largely due to underreporting and missed diagnoses. When survey research is performed, parents report 40 times more abuse than is noted in official reports.

The vast majority of pediatric victims of abusive head trauma for whom medical attention is sought present to the ED. The history may be incongruous with the patient injury, misleading, and, in general, unreliable. There may be no obvious signs of head trauma on physical examination. Presenting complaints may include nonspecific severe symptoms such as poor feeding, irritability, seizures, lethargy, coma, and apnea.

The most common and sensitive physical examination finding indicative of abusive head trauma is retinal hemorrhage on a dilated funduscopic examination (see Figure 7), though this is difficult to

![Figure 7. Severe Retinal Hemorrhage](image)
Numerous severe retinal hemorrhages surround the optic nerve (*). Virtually no normal retina is visible due to the severity of the hemorrhages.

assess in the ED. Retinal hemorrhages are found in 83% of pediatric patients with abusive head trauma. Other concerning findings for NAT are bruising of the neck, torso, and ear found in children aged < 4 years, and any bruising or fractures in children aged < 4 months. Finally, stereotypical injuries consistent with NAT should also lead emergency clinicians to consider abusive head trauma including (but not limited to) linear marks, suspicious burns, posterior rib fractures, and long bone fractures.

The most common radiologic finding in patients with abusive head trauma is the subdural hematoma, which occurs in > 70% of patients. Contusions, diffuse axonal injury, and other intracranial injuries are also possible, along with spinal cord injuries. Note that approximately 25% of infants delivered vaginally have small subdural hematomas that resolve by 4 to 6 weeks of life. It is critical to remember that the incidence of pediatric NAT is not related to race or nationality, but it is affected by socioeconomic factors.

Severe Traumatic Brain Injury In Sports
Most TBIs associated with sporting activities are mild or concussive. While not common, severe TBI is possible. Certain sports have a higher incidence of severe TBI, including American football, hockey, motocross, and horseback riding. In hockey, 22% of all injuries are to the head and neck. Both hockey and football are collision sports, and most injuries result from collisions with other players, the surrounding areas, or the puck or ball. Both of these sports require players to wear helmets, decreasing the severity of injury to the brain when a participant does get injured. Motocross also has a high incidence of head injury, and some of these injuries can be devastating.

Controversies And Cutting Edge
Prophylactic Antiepileptic Medications
Posttraumatic seizures may be classified as either early (within 7 days of trauma) or late (7 or more days after trauma). Posttraumatic seizures may contribute to secondary brain injury by increasing ICP, cerebral metabolic demand, and hypoxia. Overall incidence of posttraumatic seizure is approximately 10%. It is recommended that prophylactic treatment with phenytoin may be considered to reduce early posttraumatic seizures. This is a level III recommendation based on 1 retrospective review demonstrating that children treated with phenytoin had lower incidence of posttraumatic seizure. Although levetiracetam may also be used as seizure prophylaxis, the research is extremely limited. In a pediatric prospective study, there was no decrease in the prevalence of early posttraumatic seizures in patients treated with levetiracetam.

Corticosteroids
Recently, the placebo-controlled CRASH (Corticosteroid Randomisation After Significant Head injury) trial in patients aged > 16 years concluded that patients who received corticosteroids after brain injury had an increased risk of mortality. The Brain Trauma Foundation guidelines currently advise against the use of corticosteroids to improve outcome or decrease ICP in severe TBI (level II recommendation).

Progesterone
Progesterone has shown promising neuroprotective properties in experimental and animal models, including decreased edema, improved neuronal survival, and mediation of secondary injury cascades (such as neuroinflammation, oxidative stress, and excitotoxicity). Despite small initial studies demonstrating no harm and potential benefits of progesterone for acute TBI, a large 2014 double-blind multicenter study with the primary outcome of functional recovery at 6 months did not demonstrate benefit in adult patients with TBI. There are currently no clinical trials of progesterone use in pediatric TBI.

Ketamine
There is evidence that ketamine may have neuroprotective properties by reducing neuronal apoptosis and modulating the inflammatory response. Based on initial data from small studies and case reports from the 1970s, ketamine was traditionally avoided in patients with head trauma for concerns that it would increase ICP. A meta-analysis of the adult and pediatric literature evaluating ICP levels within 24 hours of ketamine use suggested that ketamine does not increase ICP when compared with opioids. In pediatrics, data from ketamine use in mechanically ventilated and sedated patients suggest that ketamine might actually lower ICP. Three teenagers were included in another clinical trial of patients with TBI, and it was demonstrated that ketamine use in sedated and mechanically ventilated patients on a propofol drip resulted in lowered ICP. There are no known studies evaluating the use of ketamine in RSI for head trauma, although it may be considered due to its favorable hemodynamic profile.

Hypothermia
Early case series and initial randomized controlled trials in children suggest that hypothermia may be a safe and potentially beneficial treatment in children following TBI. The goal of hypothermia is to reduce the risk of secondary injury by decreasing metabolic demands, mediating inflammation, and decreasing cell death and seizures. Therapeutic hypothermia may decrease ICP in refractory intracranial hypertension. The Brain Trauma Foundation guidelines have a level II recommendation support-
The use of moderate hypothermia (32°C-33°C) for 48 hours, followed by slow rewarming, in pediatric patients with severe TBI with refractory intracranial hemorrhage, if the injury occurred within 8 hours.28

Although initial therapeutic hypothermia may improve refractory intracranial hemorrhage, recent studies have demonstrated conflicting results on outcome.125,127 A meta-analysis including 7 randomized controlled trials found no evidence of benefit from therapeutic hypothermia in children following TBI.128 The phase 3 Cool Kids randomized controlled trial assessing whether or not therapeutic hypothermia for 48 to 72 hours with slow rewarming would improve mortality in children after brain injury was terminated early due to futility.129

The Brain Trauma Foundation has a level III recommendation for consideration of moderate hypothermia (32°C-33°C) for 48 hours beginning early after severe TBI, with these guidelines released prior to publication of the most recent Cool Kids trial.28 The details of induction and rewarming of hypothermia are important.27 At this time, therapeutic hypothermia should be limited to use in clinical trials.

### Antifibrinolytics

Tranexamic acid (TXA) has been widely used to reduce surgical blood loss. The Clinical Randomisation of an Antifibrinolytic in Significant Haemorrhage 2 (CRASH-2) trial demonstrated that TXA reduced the risk of death in adult trauma patients if given within 3 hours following injury.130 The Clinical Randomisation of an Antifibrinolytic in Significant Head injury 3 (CRASH-3) trial is ongoing to determine the effect of TXA on outcomes following TBI.130 The Pediatric Trauma and Tranexamic Acid (PED-TRAX) trial is a retrospective review of all pediatric trauma patients (including head trauma, with a mean GCS score of 12 receiving TXA in a combat setting); it concluded that TXA use was associated with decreased mortality.131 There are no pediatric clinical trials evaluating use of TXA in isolated severe traumatic head injury.

### Hyperosmolar Therapy: Hypertonic Saline Versus Mannitol

For many years, mannitol was the preferred osmotic agent, but over the past few decades, HTS has become a popular alternative due to the potential decrease in adverse effects, particularly in patients with hemodynamic instability. The Brain Trauma Foundation pediatric guidelines provide a level II recommendation to consider use of HTS as a treatment for intracranial hypertension in severe TBI.28 A meta-analysis of adults with TBI demonstrated no statistical or clinical difference in the decrease of ICP when mannitol was compared to HTS, although the studies individually showed a trend towards HTS being more effective.132,133

Current guidelines are based largely on a 1992 double-blind randomized controlled trial, with the HTS group showing lower ICP.134 Another class II randomized controlled trial showed fewer interventions to maintain ICP < 15 mm Hg in a group receiving HTS.135 Currently, adequate clinical trials comparing mannitol to placebo or other treatment options in children do not exist.

### Biomarkers

Research over the past decade has focused on biomarkers and their possible role in diagnostic, prognostic, and therapeutic intervention for pediatric TBI.136 A systematic review concluded that the most commonly studied biomarkers in pediatric TBI included S100B, neuron-specific enolase, interleukin-6, myelin basic protein, and interleukin-8. The alpha II spectrin N-terminal fragment protein marker has been shown to be elevated in the blood following mild TBI and sports-related concussion, and may be useful for detection of diffuse axonal injury.137,138 The utilization of biomarkers in severe TBI is not practical for acute management in the ED, but it may play a role in guidance of treatment in the future.

### Time-And Cost-Effective Strategies

- Severe pediatric TBI has an enormous monetary and emotional cost to society. In the United States, 80 children per 100,000 children aged < 4 years old are hospitalized for TBI each year.139 The best way to mitigate the cost and severity of TBI is prevention.

- Helmets can be used to help protect the brain when a child is performing activities that put them at risk for a severe head injury. In a review of helmet use in cyclists, helmets reduced the risk of severe TBI by 63% to 83% in all types of bicycle accidents.140 Many children do not wear helmets while cycling or during other potentially hazardous activities. Barriers to helmet use include negative perception of the helmet by the child, working parents, improper helmet fit, and parents who do not use helmets. Helmet use is increased when there is education provided to both parents and children, when free helmets are available, and when there is legislation requiring the use of the helmets. In some states, police temporarily impound the bike of any child who is riding a bicycle without a helmet, and this has been shown to increase the rate of helmet use in both children and adolescents.141

- The cost of severe TBI is more than $69 million each year in the United States. It is estimated that the average cost of an ED visit for abusive head trauma is $2612 and the cost of a hospital admission is $31,901.142 This has led to specific public health strategies to determine what puts young children at risk for abusive head trauma.
1. “My teenage patient smelled like alcohol and he appeared intoxicated, but I did not suspect head trauma.”
Severe TBI is often complicated by alcohol or drug intoxication in teens and young adults. Intoxication may be a confounding factor and can result in a less reliable physical examination. A high level of suspicion should be maintained for TBI in an intoxicated patient with a history of trauma.

2. “I assumed TBI was ruled out after the patient had a normal head CT scan.”
A normal initial head CT scan does not rule out TBI. Diffuse axonal injury may present with either normal or subtle findings on CT scan, but patients are at significant risk for secondary injury. A repeat CT scan or MRI may be indicated with suspected TBI, particularly if there is clinical deterioration.

3. “The patient came in after a seizure and was noted to have an occipital hematoma. I assumed she fell down after she started seizing.”
It can be difficult to determine whether the seizure or the fall happened first. It is important to consider that the seizure may have occurred secondary to potential TBI. It is crucial to obtain a detailed history and thorough physical examination. Maintain a high level suspicion for TBI when a patient does not return to neurologic baseline following a seizure.

4. “The mother told me that her baby fell out of his crib and that seemed like a plausible mechanism for a severe TBI.”
Very few children with accidental head injury have severe TBI. A simple fall or accidental blunt trauma rarely results in the force necessary to cause severe TBI. Make sure that the story makes sense with the current developmental stage of the child. Any bruising or injury to a child aged < 4 months or that is inconsistent with the history should be concerning for NAT.

5. “I was worried about a herniation syndrome, but I wanted to get the CT before I started to treat the ICP.”
Elevated ICP and signs of imminent brain herniation require emergent therapy and should occur prior to imaging. Signs of herniation may include a decline in mental status, an acute decrease in the GCS score, Cushing triad, pupillary defects, or posturing.

6. “The child briefly lost consciousness after he was hit in the head with a baseball. He was totally fine when he got here, so I assumed that his injury was not severe. While he was waiting to be seen, he quickly decompensated.”
A patient with an epidural hematoma may present with a brief loss of consciousness, followed by a lucid period, and then subsequent deterioration. This classic description of an epidural hematoma is less common in pediatric patients.

7. “I was busy assessing the patient’s abdominal bruising and large open femur fracture; I didn’t notice the large scalp injury until the patient started decompensating.”
In a patient with multiple injuries, it is critical to have a high suspicion for head injury. Do not allow distracting injuries to delay diagnosis of a TBI. Always perform a careful physical and neurologic examination in every trauma patient. Always look for injury under the cervical collar.

8. “I didn’t see any sign of trauma on the head of the infant, so I only looked for cardiovascular and pulmonary reasons for the patient’s decompensated status.”
Infants who present with increased ICP can present with depressed mental status, abnormal respiratory rates, bradycardia, and respiratory failure. TBI should always be on the differential for an unstable child, even if there is no reported trauma. Children with abusive head trauma may have a normal physical examination.

9. “I saw the bulging fontanelle in the neonate and I was worried about meningitis, so I didn’t even have abusive head trauma or NAT on my differential.”
A bulging or full fontanelle may be the only sign that a child has suffered an intracranial injury. Children who have been victims of shaking or jerking are at high risk for intracranial injuries, but they may have no external signs of injury on examination. The majority of children who are victims of abusive head trauma will have retinal hemorrhages on a dilated ophthalmic examination.

10. “I did the full physical examination and didn’t see any signs of head trauma. I was surprised to see an occipital injury on the CT.”
It is easy to miss the physical signs of trauma when the trauma is underneath the cervical collar. Always remove the collar, while maintaining cervical spine precautions, to investigate the neck and the base of the skull.
is a strong association between abusive head trauma and early crying, prolonged crying, and age of 2 to 4 months. This association provides an opportunity for both prevention and early intervention. Educational programs can be incorporated into the hospital stay during the birth of the child to educate parents about abusive head trauma and to give them tools to prevent it.143

Disposition

The pediatric patient with severe TBI presents a complex medical problem requiring the integrated practice of emergency clinicians, trauma surgery, neurosurgery, and pediatric intensive care. Patients who arrive in a hospital that is not a trauma center should be stabilized and transferred, preferentially, to a pediatric trauma center. Ideally, patients should be transferred to hospitals that have the PICU and neurosurgical capacity to definitively treat patients with severe TBI.

Summary

The goal of the emergency clinician in management of severe TBI is rapid identification and aggressive intervention, as this may decrease the risk and severity of secondary injury and improve long-term outcome. Initial resuscitation should emphasize maintaining normal physiologic parameters and avoiding hypotension, hypoxia, and intracranial hypertension. It is critical to recognize deterioration syndromes and initiate appropriate interventions for intracranial hypertension. Current research continues to search for treatment options to decrease mortality and improve outcomes. Public health efforts should continue to focus on the importance of prevention strategies.

Case Conclusions

The 3-month-old boy was successfully intubated. His PaCO₂ was kept within the target range of 35 to 45 mm Hg. You decided to start 3% hypertonic saline at 6.5 mL/kg, giving it intravenously over 10 minutes. You saw an almost immediate increase in muscle tone and a systolic blood pressure increase to 80 mm Hg. You quickly sedated the patient and gave him pain medication. Both pupils were then reactive, and the left eye was no longer deviated. You quickly took the patient to CT. On initial films, you saw bilateral subdural hematomas. The neurosurgeon arrived and placed an interventricular catheter. You called Child Protective Services to alert them of your suspicion for abuse. After an extensive PICU stay, the child remained ventilator-dependent. Child Protective Services removed the child from the custody of the parents; the long-term neurological status of the child is unknown.

The 17-year-old girl was intubated, and you quickly sedated her and treated her pain. Two large-bore IV lines were placed and she received 1 L of normal saline. Her vital signs were as follows: temperature, 38.5°C; heart rate, 95 beats/min; blood pressure, 120/70 mm Hg; respirations set at 14 breaths/min; and oxygen saturation, 98%. She was given rectal acetaminophen for the fever, and you had your fellow take her to CT for pan-scanning. She was found to have a large right-sided epidural hematoma with midline shift and an overlying skull fracture. She also had multiple right-sided anterior rib fractures. The patient was admitted to the PICU, but taken directly up to the operating room for evacuation of the hematoma. After a 4-day PICU stay, she began to regain consciousness. After a 1-month intensive rehabilitation program, the patient had only mild deficits in her speech.

References

Evidence-based medicine requires a critical appraisal of the literature based upon study methodology and number of subjects. Not all references are equally robust. The findings of a large, prospective, randomized, and blinded trial should carry more weight than a case report.

To help the reader judge the strength of each reference, pertinent information about the study, such as the type of study and the number of patients in the study is included in bold type following the references, where available. The most informative references cited in this paper, as determined by the authors, are noted by an asterisk (*) next to the number of the reference.


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5. Parents report that their 3-month-old baby rolled off the changing table earlier today. The baby has been acting “funny” since this happened about 12 hours ago. On examination, the baby opens her eyes to pain, moans to pain, and withdraws to pain. What is her pediatric GCS score?
   a. 10
   b. 9
   c. 8
   d. 7

6. In considering a step-wise approach to the trauma patient, which of the following steps should be performed last?
   a. Cervical spine immobilization
   b. Calculation of the GCS/disability
   c. Verification of patent airway
   d. Verification of adequate circulation

7. What is the strongest predictor of poor clinical outcomes in patients who have severe TBI?
   a. Hypercapnia
   b. Hypotension
   c. Hypoxia
   d. Inadequate ventilation

8. Which of the following recommendations for severe TBI treatment is not supported by class II or III evidence?
   a. 3% hypertonic saline for increased ICP
   b. 20% mannitol for increased ICP
   c. Elevation of the head of the bed 30°
   d. Maintenance of systolic blood pressure ≥ 70 mm Hg (2 x the age in years)

9. Which of the following has been shown to be a reliable physical examination finding concerning for abusive head trauma?
   a. Retinal hemorrhage
   b. Lethargy
   c. Irritability
   d. Seizures

10. The Brain Trauma Foundation guidelines for medical management of severe pediatric TBI provide level II evidence for the use of which of the following treatments?
    a. Corticosteroids
    b. Mannitol
    c. Hypertonic saline
    d. Progesterone
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