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Pediatric Head Injury

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to this article.

Objectives After completing this article, readers should be able to:

1. Describe the clinical features and management of epidural hematoma.
2. Recognize that the syndrome of inappropriate antidiuretic hormone secretion is possible following head injury.
3. Know the association of cervical cord injury with head trauma.
4. Discuss the long-term cognitive/behavioral consequences of head trauma.

Epidemiology

Head injury is a leading cause of morbidity and mortality in childhood. More than 1.5 million head injuries occur annually in the United States, resulting in approximately 300,000 pediatric hospitalizations, with males twice as likely as females to sustain a head injury. Overall, up to 90% of injury-related deaths among children are associated with head trauma. Motor vehicle collisions are the most common cause of pediatric head injury, followed by falls. Football is the most common cause of sports-related head injury, with 75% of fatal head injuries occurring in high school students and 74% of football-related head injury fatalities associated with subdural hematomas. Head trauma injuries include scalp hematoma and laceration, skull fracture, intracranial hemorrhage, cerebral contusion, and diffuse axonal injury (DAI). Most children sustaining blunt head trauma have minor traumatic brain injury.

The American Association of Neurologic Surgeons defines traumatic brain injury (TBI) as a blow or jolt to the head or penetrating head injury that disrupts the normal function of the brain. Mild TBI may result in a brief change in mental state or consciousness; severe TBI may result in prolonged unconsciousness, coma, or death.

Anatomy

Any blow to the head can transfer energy from the skin, through the skull and meninges, to the brain. When evaluating head trauma, the clinician should remember the anatomic layers of the head that may be affected: the skin, galea aponeurotica, periosteum, cranial bone, epidural space, dura mater, subdural space, arachnoid mater, subarachnoid space, and brain (Fig. 1).

Scalp Hematomas and Lacerations

Head injuries frequently include scalp lacerations. Although the scalp is highly vascularized, it is uncommon for a child to lose enough blood from a scalp laceration to cause shock or hypovolemia. If a child presents with shock after head trauma, the clinician should ascertain that there are no other sources of bleeding, such as from intra-abdominal injuries. Bleeding beneath the periosteum results in a cephalohematoma; bleeding beneath the galea results in a subgaleal hematoma. Subgaleal hematomas may be caused by mechanisms such as hair braiding, hair pulling, vacuum extraction with vaginal delivery, and falls. Unlike cephalohematomas, which are restricted by suture lines, subgaleal hematomas can cross suture lines and lead to significant blood loss and hypovolemia.

Although a rare complication of head trauma, blood may accumulate behind the orbit, resulting in loss of vision and proptosis. If history and physical examination findings indicate an underlying hematologic abnormality, a complete blood count and coagulation

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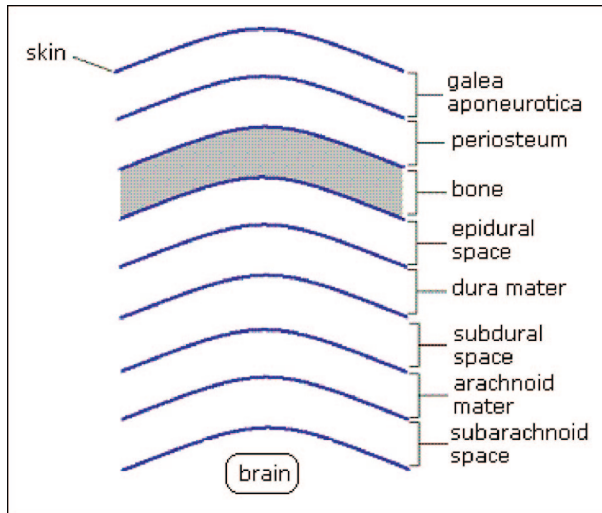


Figure 1. Layers of the scalp and meninges. Adapted from Atabaki SM. Prehospital evaluation and management of traumatic brain injury in children. *Clin Pediatr Emerg Med.* 2006; 7:94–104, with permission from Elsevier.

studies should be obtained to see if blood product replacement is warranted. Stable patients may be observed as outpatients and instructed to avoid using nonsteroidal anti-inflammatory drugs. All scalp wounds should be cleaned and examined to rule out underlying skull fractures.

Skull Fractures

Children who have head injuries often present with skull fractures, which are described as linear, depressed, or basilar. Any skull fracture may have an associated serious underlying intracranial injury. Because 50% of brain injuries occur in the absence of skull fractures, a computed tomography (CT) scan rather than a plain radiograph of the skull should be obtained whenever brain injury is suspected (eg, in the child who has an abnormal Glasgow Coma Scale [GCS] score or focal neurologic findings).

It is estimated that 75% of all skull fractures are linear. In most cases of isolated linear skull fractures, management is pain control and outpatient observation, but for children younger than 2 years of age, neurosurgical evaluation and follow-up is recommended. On rare occasions, a leptomeningeal cyst may develop from a dural tear at the time of the skull fracture. The leptomeningeal cyst, or even brain tissue, may extend through the skull fracture, interfering with fracture healing and leading to a “growing fracture” that requires surgical repair. (1)

Depressed skull fractures occur with higher impact forces. These fractures require neurosurgical evaluation



Figure 2. Patient who has both raccoon eyes and the Battle sign after blunt trauma to the head. Reprinted from Atabaki SM. Prehospital evaluation and management of traumatic brain injury in children. *Clin Pediatr Emerg Med.* 2006;7:94–104, with permission from Elsevier.

and possibly operative repair and elevation when the fragment is depressed greater than the thickness of the skull. Children who sustain depressed skull fractures are at higher risk for developing seizures and often are prescribed anticonvulsant medications prophylactically.

Patients who have basilar skull fractures may present with a Battle sign (ecchymoses behind the ear) or hemotympanum, resulting from fracture of the temporal bone and accumulation of blood in the mastoid air cells (Fig. 2). Basilar skull fractures also may lead to venous sinus drainage and raccoon eyes (periorbital ecchymoses) (Fig. 2). In addition, cerebrospinal fluid (CSF) may leak from the ears or nose. If clinical examination suggests a possible basilar fracture, a head CT scan should be performed because basilar skull fractures are very difficult to diagnose with plain radiographs. Because of possible complications, patients who suffer basilar skull fractures require close observation in the hospital.

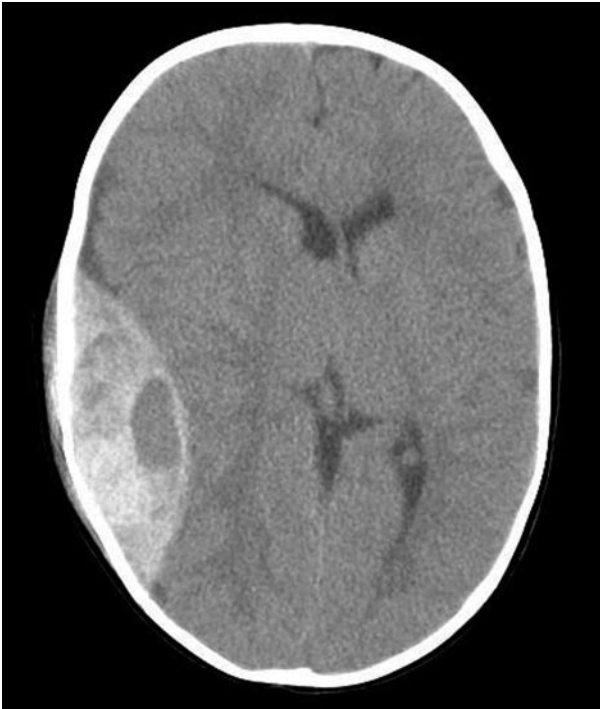


Figure 3. Noncontrast computed tomography scan of the head in a child who has head trauma. The convex shape of the hematoma is characteristic of an epidural hemorrhage. Midline shift indicates cerebral edema.

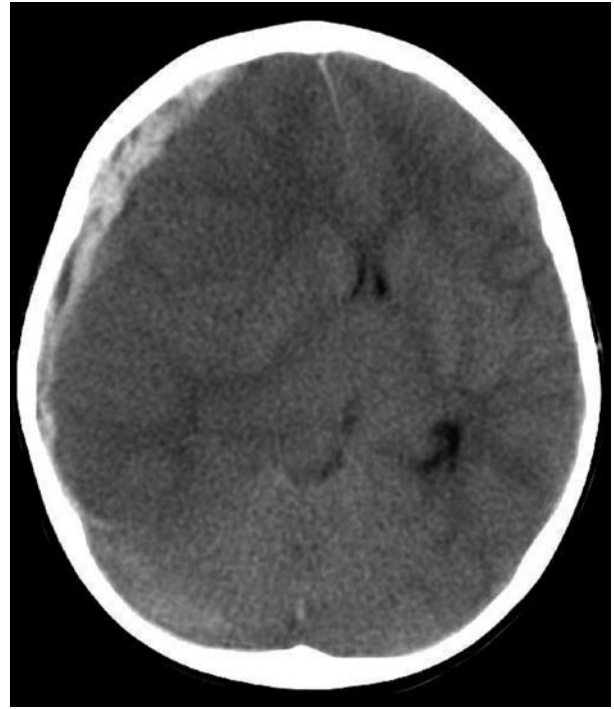


Figure 4. Subdural hematoma with associated midline shift in a child who sustained blunt head trauma and acceleration-deceleration injury after a motor vehicle collision. The concave shape of the hematoma is consistent with a subdural hemorrhage.

Intracranial Injuries

Studies report intracranial injuries in 6% to 30% of children who present with minor blunt trauma. Injuries include subdural, epidural, subarachnoid, intraventricular, and intraparenchymal hemorrhages; cerebral contusion; shearing injuries; and diffuse axonal injury. Epidural hematomas are rapid hemorrhages caused by tears of the meningeal arteries or veins and often are convex, as blood accumulates between the skull and dura mater (Fig. 3). Epidural hemorrhages often are associated with temporal bone fractures. Patients sustaining epidural hemorrhages may have a lucid period for several hours after the initial injury, followed by rapid deterioration in mental status. When clinically unrecognized, such hemorrhages may be fatal. Patients who have epidural hemorrhages require close observation and immediate neurosurgical consultation and evaluation for possible surgical evacuation. The prognosis of isolated epidural hemorrhage after surgical evacuation is very good because injuries causing epidural hematomas generally do not damage the cerebral cortex.

Subdural hematomas are more common in children experiencing head trauma and frequently are associated

with skull fractures. Subdural hematomas, due to tears of the parasagittal bridging veins, are classically concave, as the blood accumulates in the subdural space along the surface of the brain (Fig. 4). If a patient does not regain consciousness after head injury and has a subdural hematoma, immediate surgical intervention is required because there may be underlying brain injury.

Patients who undergo rapid acceleration or deceleration injuries to the head, as seen in motor vehicle accidents, falls, and abusive trauma (severe shaking), may develop DAI. DAI describes a widespread shearing injury of the white matter and should be suspected if a patient presents with diffuse subarachnoid bleeding and cerebral edema. Patients experiencing DAI frequently develop increased intracranial pressure (ICP). DAI also may occur after relatively minor head trauma to a person who recently had a first concussion (second impact syndrome).

Patients sustaining blunt head trauma often have cerebral contusions that occur primarily in cortical tissue, usually under the site of impact. Cerebral contusions may be small, causing relatively minor symptoms, or large and

accompanied by cerebral edema and increased ICP. Frequently, patients who present with intracranial hematomas or skull fractures have associated cerebral contusions.

A small percentage of patients may develop chronic subdural hematomas, which usually are caused by isolated venous hemorrhages and do not involve injury to the brain tissue. Affected patients typically present with chronic headache. Chronic subdural hematomas are more common in the elderly population than in children.

Of those patients who have intracranial injuries and skull fractures, 5% are hospitalized and 1% to 3% of all patients receive interventions such as ICP monitoring, osmotic therapy, evacuation of intracranial hematomas, and initiation of anticonvulsant therapy. Injury patterns after TBI can be bimodal; the primary insult occurs at the time of impact, and the secondary insult generally occurs 1 to 5 days later, usually resulting from hypotension and hypoxemia. Secondary insults are significant causes of morbidity after severe TBI. For patients whose TBI is moderate or severe, management includes aggressive maintenance of the patient's PaO₂ at greater than 100 mm Hg and systolic blood pressure at greater than the 5th percentile for age to prevent poor cerebral perfusion. To calculate the patient's 5th percentile blood pressure, 70 can be added to two times the patient's age in years.

Concussion and Outpatient Management

Concussion was defined more than 30 years ago in the neurosurgical literature as trauma-induced alteration in mental status with or without loss of consciousness. In 2001, the First International Conference on Concussion in Sport (2) described concussion as a pathophysiologic process resulting in the self-limited impairment of neurologic function that has an associated set of clinical symptoms (Table 1). The symptoms usually are acute in onset and the result of functional rather than structural disturbances. Concussion can appear as a graded set of clinical syndromes, and patients may have one or any combination of the symptoms and signs. Individuals have described disturbance of vigilance, distractibility, lack of coherent thought, and inability to complete goal-directed tasks. Patients suffering concussions generally do not have structural damage to the brain; results of neuroimaging studies usually are normal.

It is estimated that up to 25% of patients suffering minor head trauma develop a concussion. Children who have minor head trauma and are discharged from the emergency department or hospital with no signs or symptoms of intracranial injury often are referred to their

Table 1. Symptoms and Signs of Concussion

- Headache
- Dizziness
- Depression
- Confusion
- Nausea/vomiting
- Sensitivity to light or noise
- Lethargy
- Slow response to questions
- Decreased energy
- Irritability
- Blurred or double vision
- Poor concentration
- Poor balance
- Insomnia
- Anxiety
- Poor memory

Data from Atabaki SM. Prehospital evaluation and management of traumatic brain injury in children. *Clin Pediatr Emerg Med.* 2006;7:94–104.

primary care physicians for follow-up. Outpatient clinicians should be aware that such children may have had a concussion and, therefore, are at risk for possible concussion sequelae, which is of particular concern if the child participates in contact sports.

Postconcussion syndrome, which may occur even after minor head injury, is characterized by headaches, depression, anxiety, behavioral problems, dizziness, amnesia, irritability, hyperactivity, and sleep difficulties. Prior studies of patients older than 16 years of age who had head injury described headache, mood changes, depression, and problems with memory lasting several months after the original injury.

Parents often are eager to know when to allow their child who has a concussion to return to playing sports. Table 2 outlines common recommendations for return to a sport after a concussion that attempt to prevent second impact syndrome. Although rare, second impact syndrome develops when a patient who is still symptomatic from an initial concussion receives a second concussion. The second concussion predisposes the patient to develop DAI, diffuse cerebral edema, brain herniation, coma, and possible death. Children should not return to contact sports if they demonstrate symptoms of mild-to-moderate concussion after head trauma both at rest and on exertion. Ideally, they should wait until they are symptom-free for 1 week prior to returning to sports (Table 2). For children who have suffered a severe con-

Table 2. Recommendations for Return to Sports after Head Injury Based on Grade of Concussion

Severity	Symptoms	Management
Grade 1 (Mild)	No LOC, ringing, headache, dizziness, or memory loss	<ul style="list-style-type: none"> • Observation • May not return to competition until symptom-free upon exertion
Grade 2 (Moderate)	LOC <5 min or PTA >30 min	<ul style="list-style-type: none"> • Observation • May not return to competition for 1 wk after symptom-free upon exertion
Grade 3 (Severe)	LOC >5 min or PTA >24 h	<ul style="list-style-type: none"> • Admit • Refer for neurocognitive testing prior to resumption of contact sports

LOC=loss of consciousness; PTA=posttraumatic amnesia
Data from Cantu RC. Guidelines for return to contact sports of a cerebral concussion. *Physician Sportsmed.* 1987;14:76–79.

cussion, return to sports may be allowed after a 1-week symptom-free period that begins at least 1 month after the day of the head injury.

Triage of Head Injury

Pediatricians often face the difficult task of deciding how to triage, by phone, the child who has had a head injury. Important questions to ask during the initial contact phone call are:

- Did the child cry immediately after injury? (No crying may indicate loss of consciousness.)
- Is there a “goose egg” or scalp hematoma present? (Young children who have parietal hematomas are more likely to have an underlying skull fracture.)
- Was there bleeding or fluid draining from the nose or ears? (A “yes” answer may indicate the presence of a basilar skull fracture.)
- Was the fall greater than 3 feet? (A “yes” answer indicates increased risk of skull fracture in infants.)
- How old is the child? (Infants are at increased risk for skull fracture and intracranial injury.)
- Has the child had a recent head injury? (A second head injury to a now symptomatic child may result in increased morbidity.)

Examination of the child is warranted if an answer to any of these questions confirms a risk for significant head injury. If answers to the questions do not indicate a risk for a significant head injury, the parents should be counseled to supervise the child closely up to 24 hours after the time of injury and to contact the physician if there is any change in the child’s mental status or any seizures, persistent or increasing headache, or protracted vomiting (more than two to three episodes).

Prehospital and Hospital Care of Significant Head Injury

The initial management of the head-injured child follows the ABCDE approach of assessing **A**irway, **B**reathing, **C**irculation, **D**isability, and **E**xposure/Environment. Head injury may lead to respiratory failure due to central nervous system damage, decreased level of consciousness, and obstruction of the upper airway by the tongue. Hypoventilation and shallow breathing also may result from direct trauma to the medullary respiratory center. During pediatric resuscitation following head trauma, the cervical spine always should be immobilized because the mechanism of injury leading to head trauma also may place the child at risk for cervical cord injury.

The patient who has a GCS score of less than 9 should be intubated immediately via rapid sequence intubation (RSI). An individual whose GCS score is less than 9 is presumed to be unable to maintain his or her own airway and is at high risk for loss of airway protective mechanisms such as the gag reflex. Administering anesthetic agents such as ketamine that may increase ICP during RSI is not recommended. Most intubation protocols include administering etomidate as a sedative plus high-dose vecuronium or rocuronium for neuromuscular blockade.

Although succinylcholine theoretically can increase ICP, its use remains widespread for RSI after trauma because succinylcholine has a rapid onset of action. Pre-medication with lidocaine (1.5 mg/kg intravenously), given 2 minutes before intubation, is recommended in all cases of RSI after head trauma to decrease the rise in ICP associated with laryngoscopy and intubation. Following intubation, an *orogastric* tube should be placed to decompress the stomach. Placing a *nasogastric* tube is con-

traindicated because a nasogastric tube theoretically could penetrate the base of the skull if an undetected basilar skull fracture is present.

Recommendations for the acute management of the patient who has sustained a TBI have changed over the years. Prior teaching was to hyperventilate the patient who has severe TBI, but hyperventilation now is recommended only as a life-saving maneuver for suspected cerebral herniation. In all other instances of patient intubation and ventilation, ventilation should be normal to keep the endotracheal tube concentration of CO₂ at 35 mm Hg to prevent vasoconstriction and to maintain adequate cerebral perfusion.

Fluid management is critically important in patients sustaining moderate or severe TBI. Maintaining systolic blood pressure allows adequate perfusion of all organs and ensures adequate cerebral perfusion pressure. Many clinicians are hesitant to hydrate the patient who has TBI aggressively for fear of increasing ICP. Several studies have demonstrated that hypotension is a sensitive predictor of morbidity and mortality. In fact, hypotension may lead to a twofold increase in mortality. (3) Current recommendations for managing severe head trauma include maintaining systolic blood pressure in the normal range by infusing 20 mL/kg of isotonic crystalloid in boluses to prevent hypotension. If the blood pressure is normal, but increased ICP as well as uncal herniation are suspected, mannitol (0.5 to 1 g/kg) should be administered. Mannitol decreases ICP acutely. Hypertonic saline has been used successfully to decrease ICP and provide fluid resuscitation after TBI. (4)(5)

Evaluation of disability and assessment of neurologic response also are important. In the 1970s, Teasdale and Jennett (6) developed the GCS to assess neurologic status after head injury. Table 3 is a version of the GCS adapted for pediatric use. According to this scoring system, minor TBI is defined as a GCS score of 13 to 15, moderate as a score of 9 to 12, and severe as a score of 3 to 8. As noted before, a patient who has a GCS score less than 9 warrants immediate intubation. There is some disagreement among experts as to the definition of minor head trauma, which can vary from a GCS score of 13 to 15 to a GCS score of 14 to 15.

The Advanced Trauma Life Support guidelines (7) recommend a gross assessment of neurologic status by evaluating the patient's response to stimuli following the "AVPU" mnemonic: A=Alert, V=Verbal, P=Painful, U=Unresponsive. However, these guidelines are not as sensitive as the GCS in predicting neurologic outcome. A late sign of increased ICP is Cushing triad of hypertension, bradycardia, and irregular breathing. Abnormal

Table 3. Modified Pediatric Glasgow Coma Scale*

Eye Opening

- 4 Spontaneously
- 3 To voice
- 2 To pain
- 1 No response

Motor Response

- 6 Obeys
- 5 Localizes pain
- 4 Flexion withdrawal
- 3 Flexion abnormal (decorticate posturing)
- 2 Extension (decerebrate posturing)
- 1 No response

Verbal Response

- 5 Appropriate words, spontaneous cooing
- 4 Inappropriate words
- 3 Cries
- 2 Incomprehensible sounds, grunts
- 1 No response

*The Glasgow Coma Scale score is the sum of the best scores obtained from each column.

Data from American College of Emergency Physicians and American Academy of Pediatrics. *Advanced Pediatric Life Support-The Pediatric Emergency Medicine Course*. 1998.

pupillary response also may indicate increased ICP, brainstem herniation, and compression of the third cranial nerve. Papilledema can develop later in the course of intracranial hypertension, and its absence does not rule out the presence of increased ICP.

In the assessment of environment/exposure, the clinician should look for signs of external injuries and basilar skull fracture: hemotympanum, CSF otorrhea, CSF rhinorrhea, raccoon eyes (periorbital bruise), and Battle sign (postauricular bruise). Of note, external signs of trauma might be absent despite the presence of severe internal brain injury.

The clinician should assess the child for signs or symptoms of intoxication because this condition can place the child at higher risk for head injury. A bedside blood glucose determination should be obtained to rule out hypoglycemia (glucose <80 mg/dL [4.4 mmol/L]), which when present, should be treated.

For seizure prophylaxis, anticonvulsant therapy is administered for 1 week for patients who present with intracranial hemorrhage. Anticonvulsant therapy is continued after 1 week if the patient has a late seizure (ie, any seizure not immediately associated with the acute head trauma).

Syndrome of Inappropriate Secretion of Antidiuretic Hormone

Occasionally, children who suffer TBI develop the syndrome of inappropriate secretion of antidiuretic hormone (SIADH), especially if subdural or subarachnoid hemorrhage is present. In SIADH, the patient develops decreased urine output, hyponatremia, low serum osmolality, and high urine osmolality that may lead to lethargy, altered mental status, seizures, and even coma. Therefore, monitoring electrolytes is necessary in any child suspected of having intracranial injury. SIADH treatment includes fluid restriction to two thirds of maintenance requirements and close monitoring of serum electrolytes and osmolality. In severe cases, when the serum sodium concentration is less than 120 mEq/L (120 mmol/L) or the patient is exhibiting clinical signs of hyponatremia, rapid correction of the hyponatremia with 3% saline is necessary.

Diagnostic Evaluation and Neuroimaging

Since the 1970s, CT has had a significant impact on the management of closed head injury because CT can detect intracranial injuries quickly. Head CT scans are ordered in most emergency departments for many children who suffer head trauma because evidence suggests that such patients can be discharged safely if the head CT appears normal. More than 90% of CT scans obtained in the alert child after minor head injuries are negative, suggesting that this modality is being overused.

The 1990s witnessed a doubling of the rate of neuroimaging for head trauma, with approximately 600,000 pediatric cranial CT scans currently performed annually in the United States. Radiation from head CT is 300 times that of a chest radiograph, and early exposure to radiation poses a significant risk for cancer. In fact, cancer risk from radiation is as high as 1 in 2,000 in CT-exposed children. (8) Furthermore, several cost-benefit analyses of the liberal use of CT in evaluating pediatric head trauma demonstrate additional costs and risks for the pediatric population undergoing head CT, especially because of the need for sedation.

Clinical criteria for neuroimaging after minor head trauma in children remain unclear, and there is substantial variation regarding patient selection for head CT. In the adult literature, recommendations range from obtaining head CT in every patient who sustains loss of consciousness (LOC) and amnesia for the traumatic event to imaging only those patients who have focal neurologic deficits with LOC of more than 5 minutes. In its practice parameter for the management of minor closed head injury, the American Academy of Pediatrics

suggests observing the child who has had no LOC and either observing or conducting head CT imaging for those experiencing LOC. (9)

Recently, several evidence-based recommendations for neuroimaging after pediatric head trauma have appeared. Palchak and associates (10) recommended cranial CT in the presence of abnormal mental status, clinical signs of skull fracture, scalp hematoma in a child who is younger than 2 years of age, vomiting, or headache. Haydel and Shembakar (11) found that any of the following variables is a sensitive indicator for intracranial injury or depressed skull fracture in children whose head injury is nontrivial and who has sustained LOC: headache, emesis, intoxication, seizure, short-term memory deficits, or physical evidence of trauma above the clavicles.

Special Considerations

Children Younger than 2 Years of Age

Children younger than 2 years of age are at higher risk for intracranial injury after blunt head trauma, and infants are at even higher risk for intracranial injury and skull fractures because they have thinner, more pliable skulls. Based on their research, Greenes and Schutzman (12) found that for children younger than 2 years of age, parietal and temporal scalp hematomas were highly associated with skull fracture and intracranial injury, but frontal hematomas were not. The authors also noted a trend toward higher rates of skull fracture in younger patients. Infants also are at greater risk for nonaccidental trauma.

Nonaccidental Trauma

Nonaccidental trauma, also known as inflicted head trauma, should be suspected if the head injury is unwitnessed and no clear, appropriate mechanism explains the head injury. All children who are suspected of sustaining nonaccidental trauma should undergo head CT to determine new and old intracranial injuries. An ophthalmology consultation should be obtained to determine the presence of any retinal hemorrhages, which have a high correlation with inflicted head trauma. Patients younger than 2 years of age who are suspected of being abused also should undergo a complete skeletal survey to uncover possible concomitant bone injuries suggestive of physical abuse, such as spiral fractures of the long bones, rib fractures, metaphyseal fractures, and fractures in different stages of healing. Child Protective Services should be notified immediately if abuse is suspected, and siblings at risk should be removed from the home, if indicated.

Cervical Spinal Cord Injury

Cervical spine injuries include contusions, hemorrhage, fracture, ligamentous sprain, “stingers,” and muscular strains. Trauma to the top of the head when the neck is held in flexion, an injury that occurs frequently during contact sports and in motor vehicle and bicycle collisions, is the most common mechanism resulting in cervical spine injury. The clinician should be aware that this same mechanism may cause head injuries. Stingers are common, self-limited football injuries due to traction of the brachial plexus that can cause a transient burning sensation, paresthesia, and weakness in the arm.

Consequences of Head Injury

The long-term neurocognitive and behavioral consequences of head injury vary widely among patients based on the type and location of injury. Patients may suffer from chronic headache, depression, anxiety, difficulties with expressive language and working memory, behavioral changes, and attention-deficit/hyperactivity disorder. Overall, children have a better prognosis than adults after head trauma, and improvement in neurocognitive function may continue for years after their injury.

Those patients whose initial GCS scores are more than 8 have very good long-term outcomes; those whose initial GCS scores are less than 3 have very high mortality and morbidity. Any child whose GCS score is less than 14 on presentation should be referred for neurocognitive assessment. Some children who suffer head trauma develop problems with short-term memory and executive function and may benefit from speech and occupational therapy. In cases of severe head injury, the duration of coma appears to be the most sensitive indicator of neurocognitive outcome. Children whose coma lasts less than 2 weeks have considerably better neurocognitive outcomes and fewer developmental and behavioral sequelae than do children whose coma lasts more than 2 weeks.

Head Injury Prevention

Because the consequences of head injury can be severe, parents should be counseled about the preventive strategies. Children always should be supervised and should use safety equipment during activities that could result in head injury. Safety equipment includes car seats, seat belts, and bicycle helmets and other protective headgear during such sports as football, baseball (during batting), hockey, horseback riding, skateboarding, wrestling, and skiing.

Summary

Pediatric head trauma is common and can range from minor to severe. Management is directed toward detecting and treating possible brain injury and is based on the extent of the head injury. Most pediatric head injuries are minor. Despite advances in the care of the child who has experienced TBI, the treatment varies greatly. Pediatricians often are the first responders to the head-injured child and must assess and triage the children quickly. Early decisions include whether to refer to an emergency department and whether neuroimaging is needed.

The ideal imaging modality for assessing head injury is noncontrast CT, which can detect both intracranial injury and skull fracture reliably. However, pediatricians should be aware of the long-term risks associated with radiation exposure from CT. The child who has normal neurologic findings after isolated head trauma and a negative CT scan is at very low risk for developing intracranial abnormalities later and may be discharged from the hospital with close observation.

Children who have suffered mild-to-moderate concussions should not return to contact sports until they are completely symptom-free, both at rest and on exertion, for 1 week. Any child who has experienced head injury with symptoms lasting for longer than 1 week should be referred for neurocognitive evaluation.

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PIR Quiz

Quiz also available online at www.pedsinreview.org.

10. A 17-year-old boy sustained a head injury while playing hockey. He was unresponsive to verbal stimuli for approximately 2 minutes. After recovering consciousness, he could not recall the events of the past few days, appeared confused, and complained of headache and dizziness for the next hour. Over the next 2 to 3 hours, he appears to have recovered completely, having no symptoms. Results of the neurologic examination are normal. He has had no previous history of head injury. He wants to resume playing hockey as soon as possible. Of the following, the *most* appropriate recommendation is that he:
 - A. Can play after 1 week of remaining symptom-free on exertion.
 - B. Can play as long as he continues to be symptom-free on exertion.
 - C. Can play if his neurocognitive testing results are normal.
 - D. Can resume playing now if CT of the head appears normal.
 - E. Should refrain from playing contact sports in the future.

11. A 3-year-old unresponsive girl is brought to the emergency department. She had fallen 3 hours earlier from a playground monkey bar that was approximately 6 feet high. She had cried immediately and appeared to recover well. One hour later, she started complaining of headache. She subsequently became lethargic, vomited twice, and was difficult to arouse. On examination, her heart rate is 70 beats/min, blood pressure is 130/90 mm Hg, and respirations are 18 breaths/min. Her Glasgow Coma Scale score is 6. Her right pupil is dilated and reacts sluggishly to light. A diffuse 6×6-cm swelling anterior and superior to her right ear and right hemotympanum are noted. After intubation and manual ventilation, CT of the head is obtained, which shows blood accumulation along the right lateral inner table of the skull, with a sharp convex margin and shift of the midline to the left. Which of the following is *most* likely to be associated with this injury?
 - A. Cavernous sinus thrombosis.
 - B. Diffuse axonal injury.
 - C. Ethmoid bone fracture.
 - D. Rupture of the bridging veins.
 - E. Tear of meningeal artery.

12. After receiving a helmet-to-helmet tackle while playing football, a 15-year-old boy was unresponsive to verbal stimuli for approximately 10 minutes. After he regained consciousness, it took him 4 hours to recall the events of the past few days. A CT scan of the head showed no abnormalities. Over the next 24 hours, he had a headache and ringing in his ears. One week after the event, he is completely symptom-free and says that he feels fine and wants to play football again. Allowing him to play football at this time would put him at increased risk of which of the following if he suffers a similar injury?
 - A. Diffuse cerebral edema.
 - B. Intracerebral hematoma.
 - C. Intraventricular hemorrhage.
 - D. Rupture of bridging veins.
 - E. Tear of meningeal artery.

13. A 5-year-old girl is brought to the emergency department after being struck by an automobile while crossing the street. At presentation, her Glasgow Coma Scale score is 9. Vital signs are: respirations, 20 breaths/min; heart rate, 140 beats/min; and blood pressure, 75/48 mm Hg. She has bruising and swelling of her right frontal and parietal area as well as a scalp laceration. Her pupils are normal and reactive to light. Abrasion and swelling are noted over her left leg. She is intubated orotracheally and ventilated manually. Of the following, the *most* appropriate plan for fluid management is:
 - A. Administration of half-normal saline with 5% dextrose (20 mL/kg) over 30 minutes.
 - B. Administration of normal saline (20 mL/kg) over 30 minutes.
 - C. Administration of 5% dextrose (20 mL/kg) over 30 minutes.
 - D. Administration of 20% mannitol (1 g/kg) over 30 minutes.
 - E. Restriction of fluid to two-thirds maintenance.

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