Children with head injuries frequently present to emergency departments (EDs). Emergency medicine providers are often the first medical professionals to evaluate these children. The challenge for the provider is to determine which children have significant intracranial injuries that require intervention. Medical providers must rapidly stabilize injured patients and diagnose the primary injury while limiting secondary brain injury. There has been increased awareness of the damaging effects of even mild traumatic brain injuries, or concussions, leading to campaigns for prompt evaluation and education of health care providers, families, and athletic staff.

The authors have nothing to disclose.

* Corresponding author. Pediatric Emergency Medicine, UMass Memorial Medical Center, 55 Lake Avenue North, Worcester, MA 01655.

E-mail address: Catherine.james@umassmemorial.org

http://dx.doi.org/10.1016/j.emc.2013.05.007
It is also imperative to identify the children who are victims of abusive head trauma to protect them from future injury.

INTRODUCTION/EPIDEMIOLOGY

Head injuries are a common reason for ED visits for children in the United States. Although most children suffer only minor injury, there are almost 600,000 ED visits, 60,000 hospitalizations, and more than 6000 deaths per year for children ages 19 years and younger. Head injuries are the most common cause of injury-related pediatric hospitalizations and deaths, with a total cost of more than $1 billion in 2000. The causes of trauma vary based on the child’s age and developmental level. In patients 19 years and younger, the most common cause of head injuries is falls, followed by being struck by or against an object, other or unknown causes, motor vehicle accidents (occupant or pedestrian), and assaults. Falls are the most common cause of head injury for patients 0 to 4 years of age, whereas motor vehicle–related injuries are the most common cause of head injury in the 15 to 19-year age group. The rate of ED visits is higher for younger patients, but the hospitalization and death rates are higher for older children. Boys outnumber girls for both ED visits and deaths in all age groups. Motor vehicle accidents are the most common cause of head injury deaths for all age groups.  

Head injury in children differs from adults in several aspects. Clinical evaluation can be challenging in the young patient because they are unable to provide a history of the event and do not always cooperate with the physical examination. The developing anatomy and age-specific biomechanical properties of the head and neck result in different injuries in different age groups. Children’s heads are proportionally larger and heavier in relation to their bodies than adults, the occiput and forehead are more prominent, and the facial bones are proportionally smaller. The pediatric skull is more compliant than the adult, thus it can absorb more force without a fracture, but this also increases the shearing forces between the skull, dura, subdural vessels, and brain. Children also have relatively weaker necks than adults, which allows more movement of the head when forces are applied to the torso. The pediatric brain has higher water content and lesser degree of myelination, so it is less dense and may sustain more acceleration-deceleration injury than adults.

INJURY TYPES

The primary injury is the mechanical damage to the scalp, skull, and brain that occurs at the time of trauma. Many children evaluated for head injury have only superficial injuries, such as a scalp laceration or hematoma, or a nondepressed skull fracture without intracranial injury. Concussion is a type of primary brain injury that does not involve structural derangements and will be discussed in more detail in a later section. Depressed, open, and basilar skull fractures may be associated with intracranial injuries. Brain injuries can be extra-axial (epidural hematoma, subdural hematoma, subarachnoid hematoma, intraventricular hemorrhage) or intra-axial (cerebral contusion, intracerebral hematoma, diffuse axonal injury). More than one type of injury may occur simultaneously.

- **Epidural hematomas** develop from bleeding into the space between the skull and dura mater. Although classically resulting from shearing of the middle meningeal artery, they may also be caused by venous bleeding. They are less common in infants and young children than adults because the dura is more firmly adherent to the skull. They usually result from a fall or direct blow to the head and can
occur without skull fracture. Computed tomography (CT) scans demonstrate lens-shaped convexities that do not usually cross suture lines.

- **Subdural hematomas** are caused by injury to the bridging cortical veins between the dura and arachnoid membranes. The mechanism usually involves sudden acceleration/deceleration of the head. They are more common in younger children. They can also occur without skull fracture and may be bilateral. They are crescent-shaped on CT scans and can cross suture lines. Abusive head trauma is a common cause in infants and toddlers.

- **Subarachnoid hematomas** result from tearing of small pial vessels due to blunt trauma or shearing forces. They are often widely distributed and do not usually cause mass effect.

- **Intraventricular hemorrhages** may result from bleeding from an intracerebral hematoma, extension of a subarachnoid hematoma, or tearing of subependymal veins or periventricular structures. They are often associated with other injuries. Premature infants often suffer from spontaneous intraventricular bleeding.

- **Cerebral contusions** are localized areas of neuronal injury with associated bleeding. They are caused by blunt trauma to the head, which causes movement of the brain against the skull.

- **Intracerebral hematomas** result from tearing of intraparenchymal vessels. They may cause mass effect.

- **Diffuse axonal injury** is widespread damage to axons in multiple parts of the brain and usually results from acceleration/deceleration or rotational forces. It is often caused by motor vehicle collisions or abusive head trauma.

**Secondary injury** is further damage to neuronal cells that occurs as a consequence of hypoxia, hypoperfusion, metabolic derangements, and increased intracranial pressure (ICP). This process begins immediately after the injury and may progress over days to months, leading to impaired oxygen and glucose delivery to neurons, and eventual cell death. Types of secondary injury include brain herniation, cerebral edema, infarction, and hydrocephalus. Since the primary injury occurs before the patient presents for care, a major goal of the emergency provider is to prevent secondary injury.

**INITIAL EVALUATION OF THE HEAD-INJURED CHILD**

The initial evaluation and management of the head-injured child focuses on diagnosing the primary brain injury and stabilizing the patient to prevent or limit secondary brain injury. As for any injured patient, the airway must be stabilized, and breathing and circulation must be ensured while taking precautions to protect the cervical spine. Once these concerns have been addressed, full assessment of the child may be performed and the provider must determine whether imaging studies are needed.

**History**

The history should focus on the mechanism and timing of the injury as well as previous and current symptoms. If the child is conscious and mature enough, much of the history can be obtained directly from the patient. The parent and any witnesses to the event may also provide additional information. Details about the mechanism of injury should include the height and surface for falls; a description of the object that struck the head; and whether protective devices like seatbelts, car seats, and helmets were used. If the injury involved a motor vehicle, questions should include the approximate speed of the vehicle, amount of damage to the vehicle, and any injuries other occupants sustained. The provider should inquire about the presence...
and duration of loss of consciousness, seizures (time of onset, length, and focality), nausea/vomiting, headache, visual disturbances, amnesia, and confusion. In addition, the provider should obtain information about the progression of symptoms and concomitant injuries.

**Physical Examination**

The physical examination begins with a rapid assessment of airway, breathing, circulation, neurologic status and an evaluation for other life-threatening injuries. Abnormal vital signs, such as bradycardia, tachycardia, hypotension, or hypoxia should be noted and addressed. Examination of the head should be performed to look for scalp swelling, hematomas, abrasions, and lacerations; obvious fracture or deformity; fullness of the fontanel in infants; hemotympanum; Battle sign (bruising over the mastoid) or raccoon eyes (periorbital bruising); and bleeding or drainage from the ears or nose concerning for cerebrospinal fluid leak. The clinician should assess pupillary symmetry, size, and responsiveness, and perform a fundoscopic examination to look for retinal hemorrhages and papilledema. The cervical spine should be inspected and palpated. The remainder of the body should be assessed for associated injuries.

The neurologic examination includes evaluation of mental status, cranial nerves, and motor, sensory, and cerebellar functions. The mental status examination is one of the most important parts of the physical examination and should be serially monitored. The provider should ask the parent if the patient is acting at his or her baseline, especially if the child is preverbal. The Glasgow Coma Scale (GCS) is an important part of this assessment and has been modified for use in infants and young children. The remainder of the neurologic examination must be tailored to the child’s age and developmental stage.

**Pediatric GCS**

The GCS has long been used as a scoring system for serial evaluation of patients with head trauma; however, verbal and motor responses must be evaluated with respect to a child’s age. For example, the normal babbling of infants could be termed “incomprehensible sounds” and they are not able to understand commands. Even a fully conscious but frightened (or stubborn) toddler may not follow instructions. A modified GCS for infants and young children was therefore developed to allow assignment of a score to preverbal children. Eye opening is evaluated similarly to adults, but verbal and motor scores are modified (Table 1). The verbal component of the modified GCS score may be affected by the injured child’s fear or discomfort. Therefore, the GCS should be reassessed once the child has been calmed and has received pain medications.

<table>
<thead>
<tr>
<th>Eye Opening</th>
<th>Best Verbal Response</th>
<th>Best Motor Response</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spontaneous</td>
<td>Smiles, coos, babbles, interacts, oriented to sounds</td>
<td>Normal spontaneous movement</td>
</tr>
<tr>
<td>To speech</td>
<td>Cries but consolable</td>
<td>Withdraws to touch</td>
</tr>
<tr>
<td>To pain</td>
<td>Cries to pain</td>
<td>Withdraws to pain</td>
</tr>
<tr>
<td>None</td>
<td>Moans to pain</td>
<td>Abnormal flexion</td>
</tr>
<tr>
<td>None</td>
<td>Abnormal extension</td>
<td></td>
</tr>
</tbody>
</table>

Table 1: Pediatric Glasgow Coma Scale (GCS)
In pediatric trauma patients of all ages, the initial GCS has been found to correlate well with overall mortality, death on arrival to the ED, and major injury. When applied to children 2 years and younger, the pediatric GCS has been found to perform similarly to the standard GCS for children older than 2 years, especially regarding the need for acute intervention for children with blunt head trauma. The GCS is also an important component of the published pediatric head injury decision rules, as described in the next section.

**IMAGING STUDIES**

CT of the head provides rapid identification of most life-threatening injuries, including intracranial blood collections that require emergency evacuation. However, these injuries are uncommon and the risks of radiation to a developing brain, sedation of a pediatric patient, and the cost of the study are not negligible. The lifetime risk of fatal cancer from a single head CT has been estimated to be 1 in 1500 for a 1-year-old and 1 in 5000 for a 10-year-old. Other reports have also shown increased risks of both brain tumors and leukemia associated with CT scans in childhood and encourage physicians to weigh the risks and benefits before ordering these studies. In addition, children often require procedural sedation to obtain a CT scan because of fear, agitation, or young age and procedural sedation carries the risk of airway and hemodynamic compromise.

**Decision Rules for Head CT**

Young children are more difficult to assess than adults, and signs and symptoms of intracranial injury vary in different age groups. Therefore, many decision rules have been developed to help the emergency provider identify which head-injured pediatric patients require imaging studies. A systematic review of clinical decision rules for pediatric head injury was published in 2012. The 3 studies found to have the highest quality and accuracy were the CHALICE (Children’s Head Injury Algorithm for the Prediction of Important Clinical Events), CATCH (Canadian Assessment of Tomography for Childhood Head Injury), and the PECARN (Pediatric Emergency Care Applied Research Network) studies. They are all large multicenter studies and all perform with high sensitivity and negative predictive value.

There are several differences among the 3 studies. Slightly different age groups were included in each study. CHALICE included patients with any severity of injury without specified time after the injury, whereas both CATCH and PECARN include only children with minor head injuries evaluated within the first 24 hours of injury. CATCH and PECARN both include observation as a strategy to determine which children require imaging. The CATCH and CHALICE rules were derived to identify which head-injured children needed a head CT, whereas the PECARN rule was derived to identify which children did not. For these reasons, the 3 studies cannot be directly compared. The review’s investigators suggest that the 3 decision rules should be compared and validated in a single population. This comparison has not been published at the time of this writing; however, the PECARN study is the largest of the 3 and includes a separate algorithm for children younger than 2 years. At this time, the PECARN rule is the only one of the three that has been validated.

**CHALICE**

The CHALICE decision rule was derived by the UK Emergency Medicine Research Group and was published in 2006. The study enrolled 22,722 children younger than 16 years. The goal of the CHALICE study was to identify which head-injured children are at high risk of clinically significant intracranial injury, defined as death as a result of
head injury, need for neurosurgical intervention, or marked abnormality on CT scan. This rule had a sensitivity of 98% and specificity of 87% for clinically significant intracranial injury. The CHALICE rule recommends a head CT scan for a head-injured child with the following (CT scan rate of 14%):

- Witnessed loss of consciousness (LOC) greater than 5 minutes
- Amnesia of more than 5 minutes
- Abnormal drowsiness
- 3 or more episodes of emesis
- Suspicion of nonaccidental trauma
- Seizure in a patient without epilepsy
- An injury mechanism of high-speed motor vehicle collision (MVC), fall of more than 3 m, or head struck by a high-velocity object
- GCS less than 14 (or <15 if younger than 1 year)
- Penetrating or depressed skull injury
- Bulging fontanel
- Signs of basilar skull fracture
- Abnormal neurologic examination
- Scalp swelling, bruising, or laceration larger than than 5 cm if younger than 1 year

**CATCH**
The CATCH decision rule was derived by the Pediatric Emergency Research Canada (PERC) Head Injury Study Group and published in 2010. The study enrolled 3866 children age 16 years and younger. The goal of the CATCH study was to determine which children with minor head injury within the past 24 hours needed neurologic intervention. Minor head injury was defined as blunt trauma to the head with GCS of 13 to 15 with witnessed loss of consciousness, amnesia, disorientation, persistent vomiting, or irritability. The rule was 100% sensitive and 70% specific in determining the need for surgical intervention in the “high-risk” group. It was 98% sensitive and 50% specific for identifying visible brain injury on CT in the medium-risk group.

Head-injured children were considered “high risk” if they had any of the following (CT scan rate of 30%):

- GCS less than 15 at 2 hours after injury
- Suspected open or depressed skull fracture
- Worsening headache
- Irritability

They were considered “medium risk” if they had any of the following (CT scan rate of 52%):

- Signs of basilar skull fracture
- A large, boggy scalp hematoma
- A dangerous mechanism of injury (fall >3 feet or 5 stairs, MVC, fall from a bicycle without helmet)

**PECARN**
The PECARN decision rule was developed by the Pediatric Emergency Care Applied Research Network in the United States and was published in 2009. The goal of the PECARN study was to identify which head-injured children were at very low risk of clinically important traumatic brain injuries and therefore did not require head CT scans. They defined a clinically important traumatic brain injury (ciTBI) as an injury that resulted in death, neurosurgical intervention, intubation for more than 24 hours
for brain injury, or hospital admission of 2 nights or more associated with TBI on CT scan. Skull fractures were not considered a ciTBI unless the fracture was depressed by more than the width of the skull.

The PECARN investigators performed a prospective cohort study of head-injured patients younger than 18 years with GCS of 14 to 15 who presented within 24 hours of injury. They analyzed more than 42,000 patients with blunt head trauma in the derivation and validation phases of the study. Preverbal children (younger than 2 years) were analyzed separately from children older than 2 years. The Pediatric GCS was used for preverbal children. Overall, ciTBI occurred in 376 children and 60 children required neurosurgery. In the validation group of children younger than 2 years, the prediction rule (Table 2) had a sensitivity of 100% for ciTBI and a negative predictive value of 100% if none of the predictors were present. In the validation group of children 2 years and older, the prediction rule had a sensitivity of 96.8% for ciTBI and a negative predictive value of 99.95% if none of the predictors were present.

In the PECARN study, altered mental status was defined as agitation, somnolence, repetitive questioning, or slow response to verbal communication. Severe mechanisms of injury included MVC with ejection, death of another passenger or rollover, unhelmeted bicyclist or pedestrian struck by a motorized vehicle, a fall of more than 3 feet in a child younger than 2 years or more than 5 feet in a child 2 years or older, or head struck by a high-velocity object.

A secondary analysis of the PECARN data found that children with only a severe mechanism of injury and no other predictors had a lower risk of ciTBI than children with more than one predictor (relative risk 0.07 for children <2 years and 0.11 for children ≥2 years).17 The risk was even lower if they did not have other signs of trauma, seizures, abnormal neurologic examination, scalp hematoma, loss of consciousness, headache, or amnesia. Another analysis of the PECARN data found that children with ventricular shunts did not have higher risk of ciTBI than children without shunts, although they were scanned more often.18 Because these children will likely have many head CT scans in their lifetime, it would be beneficial to avoid unnecessary scans. Children with bleeding disorders have traditionally been thought to be at high risk for

### Table 2
PECARN head injury decision rule

<table>
<thead>
<tr>
<th>Children Under 2 years of Age</th>
<th>Children 2 years and Older</th>
</tr>
</thead>
<tbody>
<tr>
<td>Head CT scan recommended if the following features are present (CT scan rate 14%):</td>
<td>GCS = 14</td>
</tr>
<tr>
<td>Pediatric GCS = 14</td>
<td>GCS = 14</td>
</tr>
<tr>
<td>Other signs of altered mental status</td>
<td>Other signs of altered mental status</td>
</tr>
<tr>
<td>Palpable skull fracture</td>
<td>Signs of basilar skull fracture</td>
</tr>
<tr>
<td>If none of the above are present, head CT vs a period of observation recommended if any of the following features are present (about 30% of patients):</td>
<td></td>
</tr>
<tr>
<td>Nonfrontal scalp hematoma</td>
<td>History of loss of consciousness</td>
</tr>
<tr>
<td>History of loss of consciousness of 5 sec or more</td>
<td>History of vomiting</td>
</tr>
<tr>
<td>Severe mechanism of injury</td>
<td>Severe mechanism of injury</td>
</tr>
<tr>
<td>Are not acting normally per parents</td>
<td>Severe headache</td>
</tr>
<tr>
<td>If none of the above are present, head CT is not recommended</td>
<td></td>
</tr>
</tbody>
</table>

intracranial hemorrhage after minor head injuries. An analysis of the PECARN data found that only 1% of head-injured children with bleeding disorders in their study population had intracranial hemorrhage and that these children had symptoms that would recommend a CT scan based on the PECARN decision rule.19

**Observation** The PECARN investigators recommend observation as a possible alternative to CT scan depending on other clinical factors, including age younger than 3 months, the presence of single versus multiple findings, worsening signs or symptoms, clinician experience, and parental preference.16 Observing a head-injured child in the ED instead of obtaining an immediate CT scan allows time to see whether their symptoms improve or worsen. Children who clinically deteriorate (which may be due to an intracranial hemorrhage or cerebral edema) during that time should undergo a CT scan. The provider must keep a high index of suspicion for ciTBI in infants younger than 3 months because clinical signs are more difficult to evaluate in this age group. A subanalysis of the PECARN data investigated the effect of observation on head CT use and outcomes.20 The investigators found that the rate of head CT scan use was lower among children who were observed without a higher rate of ciTBI, especially if their symptoms improved during the observation period. The length of the observation period is not specified.

A recent study found that parents of children with minor head injuries (as determined by triage evaluation) were slightly more likely to prefer observation to immediate CT (57% vs 40%) when they were given information about the risks and benefits of CT scan for head-injured children.21 Most parents in this study preferred to be allowed to choose from all management options (89%) rather than have the physician decide the management plan (9%).

A recent retrospective study of the incidence of delayed diagnosis of intracranial hemorrhage in children with uncomplicated minor head injuries found this to be a rare event.22 Investigators reviewed approximately 18,000 cases of head-injured children younger than 14 years who had a GCS of 15, normal neurologic examination, no loss of consciousness of more than 1 minute, and no amnesia. Ten children had diagnosis of intracranial hemorrhage not apparent until at least 6 hours after the time of injury. Three of these children presented more than 6 hours after the injury and the others had been discharged after an evaluation performed less than 6 hours after the injury. These data suggest that an observation period of 6 hours is sufficient for most children with minor head injuries.

**Other Imaging Studies**

Magnetic resonance imaging (MRI) is usually impractical from the ED as the initial study in the acutely injured patient due to lack of availability, time required to perform the study, need for sedation, and difficulty monitoring an unstable patient in the MRI scanner. Although they expose the child to less radiation than a CT scan, skull x-rays have fallen out of favor (except as part of the skeletal survey for suspected child abuse) because they do not provide information about intracranial injury, as infants may have intracranial hemorrhage without fracture. Bedside ED ultrasound may prove to be useful to diagnose skull fractures.23,24 It is rapid, noninvasive, and does not require sedation or movement of the patient out of the ED; however, it cannot be used to diagnose intracranial injury in patients without open fontanelts.

**MANAGEMENT**

Management of a head-injured child focuses on limiting or preventing secondary injury. If imaging studies reveal an intracranial lesion that requires neurosurgical
evacuation, the emergency provider must continue to stabilize the patient until the patient can be taken to the operating room or transferred to a higher level of care. Other patients with severe injuries that do not require surgical management may still require admission for further monitoring and treatment. The provider should perform serial examinations to evaluate for signs of deterioration. Important aspects of management are discussed as follows.

**Airway and Breathing**

Hypoxia is a leading cause of secondary brain injury and must be promptly recognized and treated. Cervical spine precautions must be maintained while the airway is opened and during endotracheal intubation. If a head-injured pediatric patient requires intubation, medication selection should balance prevention of intracranial hypertension with preservation of systemic blood pressure. Rapid-sequence intubation with sedation and paralysis should be performed. Atropine has been used to blunt the vagal response to laryngoscopy, but recent data suggest that it may not be useful.25,26 The use of lidocaine to blunt ICP response to laryngoscopy is also controversial.27–29 Etomidate may be considered as a sedative because it has been shown to reduce ICP without significantly reducing systemic blood pressure, which improves cerebral perfusion pressure.30,31 It has a rapid onset and short duration of action, which allows for serial neurologic assessments. Ketamine has been found to reduce ICP in mechanically ventilated head-injured children in an ICU setting, and may also be useful in the ED setting.31,32 It has a short duration of action and maintains systemic blood pressure. Thiopental may also be considered to decrease ICP,31 but should not be used in hypotensive patients. Rocuronium or succinylcholine may be used as paralytic agents.

**Circulation**

Once the airway has been secured and ventilation established, adequate circulating volume must be ensured. Systemic hypotension contributes to secondary brain injury because cerebral perfusion pressure (CPP) is the difference between mean arterial pressure (MAP) and ICP (ie, CPP = MAP – ICP). Initial resuscitation usually begins with isotonic fluids. Hypotonic fluids should be avoided; normal saline has a higher sodium content than lactated Ringer solution. Hypertonic saline may prove to be useful for fluid resuscitation in the head-injured patient.31,33 Blood products may be needed if there are associated injuries.

**Hyperosmolar Therapy**

Increased ICP decreases cerebral perfusion pressure and contributes to secondary brain injury. Hyperosmolar therapy has long been used to reduce ICP in head-injured patients. The 2 most commonly used agents are mannitol and hypertonic saline. Current pediatric guidelines recommend the use of hypertonic saline rather than mannitol for head-injured pediatric patients with intracranial hypertension.31 Hypertonic saline increases serum osmolarity directly and has been found to be more effective in reducing ICP than mannitol.34 Concentrations of 3.0%, 7.5% and 23.4% are available; though a central line is preferred, 3% saline can be given safely through a peripheral intravenous line at a dose of 6 ml/kg over 15 minutes or as a continuous infusion of 0.1–1 ml/kg/hour to maintain ICP <20 mmhg.34–36 In contrast, mannitol increases serum osmolarity by acting as an osmotic diuretic, and risks of its use include dehydration, electrolyte abnormalities, and renal failure if euvoledema is not maintained.36 It may be used if hypertonic saline is not available. Mannitol is given at a dose of 0.5 to 1.0 mg/kg. These therapies should be avoided until the patient is adequately volume-resuscitated unless there are clear signs of herniation.
Antiseizure Prophylaxis

Posttraumatic seizures increase metabolic demand on the brain and may contribute to secondary brain injury. They occur in about 10% of children with TBI and are usually focal. Early posttraumatic seizures occur within 7 days of injury and late seizures occur more than 7 days after the injury. Risk factors for early posttraumatic seizures include age younger than 2 years, GCS less than 8, and nonaccidental trauma. Adult consensus guidelines recommend phenytoin prophylaxis for the first 7 days after severe brain injuries to lower the rate of early posttraumatic seizures, though it has not been found to affect the occurrence of late seizures. Although there are no consensus guidelines for children, antiseizure prophylaxis with phenytoin or fosphenytoin may be considered for children with severe brain injuries.

Antiemetics

Many head-injured patients present to the ED with vomiting; it is one of the predictors in the CHALICE and PECARN rules and one of the inclusion criteria for CATCH. Patients with known intracranial injuries often receive antiemetics, but providers may be concerned about masking serious injuries if antiemetics are given to patients who have not undergone a head CT or to patients with planned discharge home. A recent retrospective study showed a reduction in repeat ED visits within 72 hours for head-injured patients with negative head CT scans who received ondansetron for nausea or vomiting while in the ED. The likelihood of hospital admission was not affected. A small number of patients in this study received ondansetron but did not have a head CT performed. None of the patients who returned to the ED within 72 hours had a missed diagnosis of ciTBI. However, because the number of patients in this study was small, this practice cannot be definitively recommended at this time.

Other Therapies

For a severely head-injured child, the head of the bed should be elevated to 30° and the head kept midline to promote venous drainage. There are limited data on the use of therapeutic hypothermia to prevent secondary brain injury in severely head-injured children. The rationale for use of therapeutic hypothermia is to reduce cerebral metabolic demands, but so far, it has not been shown to improve neurologic outcomes or mortality rates. Although corticosteroids are beneficial for reducing cerebral edema in children with brain tumors, they have not been found to reduce ICP or improve outcomes for children with brain injuries and are therefore not recommended. Hyperventilation to PaCO₂ lower than 30 reduces ICP by causing reduction of cerebral blood flow, but it may induce brain ischemia and decrease cerebral oxygenation; therefore, it is not recommended.

DISPOSITION AND FOLLOW-UP OF HEAD-INJURED CHILDREN

Children with severe head injuries that require neurosurgical intervention or have respiratory and/or hemodynamic compromise require hospital admission. Additional indications for admission include other traumatic injuries requiring intervention, evaluation for child abuse, or intractable vomiting. Children with isolated minor head injuries (GCS 14 and 15) and normal head CT results may be safely discharged because the risk for neurologic deterioration is very low. Children who did not undergo a head CT but remained asymptomatic after a period of observation may also be discharged.

All children who sustain head injuries should be discharged with written instructions and with a responsible adult who will remain with the child for the next 24 hours.
Indications to return to the ED include persistent vomiting, worsening headache, or worsening neurologic symptoms. In the absence of neurologic decline, repeat CT imaging is not indicated for patients with an initial negative scan. Children should be instructed not to return to gym class or sports until all of their symptoms have resolved and they have been reevaluated by a physician. They should be given instructions to follow-up with their primary care provider or in a specialized head injury clinic within the next few days for reevaluation.

**CONCUSSION**

Concussion, a common complaint in the pediatric ED, represents a subclassification of the mild TBI group. Traditionally, concussion has been defined in various ways and grading scales that relied mainly on loss of consciousness and amnesia to dictate physician management of concussions. In 2008, a consensus group of international experts met in Zurich to update concussion guidelines based on the most current evidence. Concussion was defined as a "complex pathophysiologic process affecting the brain, induced by traumatic biomechanical forces." Several common features used to further define a concussive head injury include a "rapid onset of short-lived impairment of neurologic function that resolves spontaneously" and the requirement that "no abnormality on standard structural neuroimaging studies is seen." The American Academy of Neurology's (AAN) 2013 concussion guidelines define concussion as a "clinical syndrome of biomechanically induced alteration of brain function, typically affecting memory and orientation, which may involve loss of consciousness."

Up to 3.8 million recreation-related and sport-related concussions occur annually in the United States. This number could certainly be an underestimation because of underreporting and the lack of injury surveillance systems in youth sports. Overall, the incidence of concussion is greater for males than females because of the greater number of male participants in sports. However, concussion risk is higher for female athletes participating in soccer and basketball than for male athletes participating in these sports. Sports that present the highest risk of concussion are American football and ice hockey (AAN guidelines).

**Pathophysiology**

The biomechanics and pathophysiology of the brain tissue damage in concussion have been investigated in animal models in the laboratory setting; however, it is still unclear whether these results can be applied to clinical concussions. It is hypothesized that concussion results from acceleration-deceleration and rotational forces on the brain, causing deformation of the brain through compressive, tensile, or shearing forces. This transient deformation may alter the function in astrocytes and neurons through various proposed mechanisms, including abrupt neuronal depolarization, changes in glucose metabolism, and alterations in cerebral blood flow, which allow for initiation of biochemical pathways leading to cell death within hours to days. In addition, it has been proposed that a hypometabolic state may persist for up to 4 weeks after the injury, which may account for prolonged symptoms in some patients.

**Signs and Symptoms of Concussion**

The diagnosis of concussion involves the assessment of a range of domains, including somatic and cognitive symptoms, physical signs, emotional and behavioral changes, and sleep disturbances (Table 3). Headache is the most common presenting symptom. Brief loss of consciousness occurs in fewer than 10% of concussions.
Patients may initially be asymptomatic and then develop symptoms several hours after the time of concussion. An important part of the evaluation of a patient with a possible concussion is discussion with eyewitnesses to the injury and parents or coaches to determine whether the patient has improved or deteriorated since the time of injury. Worsening symptoms or failure to improve are indications for imaging.

Several versions of standardized symptom tools and sideline assessment tools exist to aid physicians and trainers in objectively evaluating postconcussive cognitive deficits. The Standardized Assessment of Concussion (SAC), Sports Concussion Assessment Tool 2 (SCAT2), and the Sports Concussion Office Assessment Tool (SCOAT) which can easily be found online, can be used for baseline evaluation and to monitor progress over time. These tools provide a means to assess memory and concentration, which has proven to be more reliable than routine orientation questions (eg, time, location). Neuropsychological testing of cognitive processing speed, memory performance, and reaction time has proven useful in identifying the presence of concussion in adolescents (sensitivity 71%–88%), but is impractical for use in the acute ED setting. At this time, there is no clearly superior tool, as all require further validation, especially in grade-school athletes. In addition, these screening tools cannot be used alone to diagnose concussion, but should be used as an adjunct to the comprehensive evaluation of a patient with a suspected concussion.

As in all patients with a head injury, the patient with a possible concussion should undergo a careful physical examination, including examination of the head for hematomas or skull fractures, assessment of mental status, and a complete neurologic examination. Although the neurologic examination may be normal, careful attention to balance may reveal subtle deficits. Studies have shown that postural stability deficits may last 72 hours after concussion. Imaging studies should not be used to diagnose concussion, but should be considered to rule out more significant TBI if suspected.

Management

Treatment for patients with concussion is centered on physical and cognitive rest, symptom management, and education of the patient, family, and other significant contacts (eg, coaches, teachers, employers). After a suspected concussion, patients should be withdrawn from physical activities immediately. The phrase, “When in doubt,
“sit them out!” is crucial in the management of possible pediatric concussions. Children should be prohibited from returning to play until fully evaluated, as discussed previously, and the clinician should ensure that there are no indications of a more serious head injury. RTP guidelines have been developed to provide an individualized course to allow patients adequate recovery time before resuming physical activity to minimize the exacerbation of postconcussive impairments. This graduated protocol follows a stepwise increase in activity level as outlined in Table 4. Patients should start at the first stage when they are asymptomatic and not taking any medications that may mask or modify the symptoms of concussion. Once the patient remains asymptomatic at the current stage for 24 hours, he or she may progress to the next stage. If concussive symptoms occur, the patient should return to the last asymptomatic step and attempt progression again after an additional 24-hour period without symptoms. As each stage takes 24 hours to complete, patients take approximately 1 week to proceed through the entire rehabilitation protocol. Probable risk factors for prolonged return to play include young age, early posttraumatic headache, fatigue/fogginess and early amnesia, alteration in mental status, or disorientation.

Compared with younger patients, RTP management in adults may be more rapid, with some athletes being allowed to RTP on the same day as the concussion. However, collegiate and high school level athletes allowed to RTP on the same day may demonstrate neuropsychological deficits after injury that may not be evident on the sidelines and are more likely to have delayed onset of concussive symptoms. Therefore, same day RTP is not advised for adolescent or pediatric patients under any circumstances.

Numerous studies have shown that children and adolescents have a longer recovery period, up to 7 to 10 days longer, when compared with college-aged or professional athletes. Interestingly, at the time of this writing, 40 states have passed legislation to further emphasize the need for a conservative approach to RTP for these young patients. Many states now require that student athletes are removed from play if they are suspected to have a concussion and are required to obtain written medical authorization before returning to physical activity. In addition, many also require concussion education for trainers, coaches, parents, and athletes.

Physical restrictions should be broad to prevent symptom recurrence and to avoid prolonged recovery. Restrictions should include sports activities, physical education classes, weight training, and even leisure activities, such as bike-riding and skateboarding. Complete cognitive rest should include a break from academic

<table>
<thead>
<tr>
<th>Stepwise return to play</th>
<th>Activity Level</th>
<th>Objective</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. No activity</td>
<td>Complete physical and cognitive rest</td>
<td>Recovery</td>
</tr>
<tr>
<td>2. Light aerobic activity</td>
<td>Walking, cycling, swimming at 70% maximum heart rate. No resistance training</td>
<td>Increase heart rate</td>
</tr>
<tr>
<td>3. Sport-specific exercise</td>
<td>Specific drills, but no head impact</td>
<td>Add movement</td>
</tr>
<tr>
<td>4. Noncontact training drills</td>
<td>Specific complex drills. May start light resistance training</td>
<td>Exercise, coordination, cognitive effort</td>
</tr>
<tr>
<td>5. Full-contact practice</td>
<td>Normal training (after medical clearance)</td>
<td>Assess skills by coaches; restore confidence</td>
</tr>
<tr>
<td>6. Return to play</td>
<td>Normal game play</td>
<td></td>
</tr>
</tbody>
</table>

studies, as well as any other activities that require attention and concentration, such as videogames, text messaging, computer use, and television viewing. Depending on symptom severity, patients may need school accommodations, such as a temporary leave of absence, shortened school day, reduction in workload, or more time to complete assignments. These will be determined by follow-up with the PCP or head injury specialist.

In the acute period, headache after concussion should be managed with acetaminophen. Aspirin and other nonsteroidal anti-inflammatory drugs are typically avoided so as to lessen the theoretical risk of inducing or exacerbating intracranial hemorrhage. However, no controlled trials have demonstrated this risk. Narcotics should be avoided to allow careful serial evaluations of mental status. In addition, prolonged use of narcotics and nonsteroidal anti-inflammatory drugs can lead to rebound headaches, which can further complicate the recovery process.

The decision to admit a patient with a concussion should be based on the same criteria as any patient with an acute head injury. Most patients with a concussion, however, can be discharged home. Patients and families must be educated about the importance of monitoring for potential neurologic deterioration in the following hours and days. Clear instructions and guidelines about what concerns necessitate a return to the ED should be discussed and provided in written form. These symptoms include persistent vomiting, sleepiness, increased confusion, change in behavior, worsening headache, or seizure. There is debate over the recommendation for periodic wakenings of the concussed patient to evaluate for signs of intracranial bleeding. No documented evidence suggests what severity of injury requires this approach, but some recommend it for patients who have experienced loss of consciousness, prolonged amnesia, or are who continue to experience severe concussive symptoms.

The patient should be in the care of a responsible adult for at least 24 hours. The patient should be instructed to follow-up with his or her primary care provider for a thorough history and neurologic examination before return to full play. Referral to a pediatric neurologist, neuropsychologist, sports medicine physician, or other specialist with expertise in head injury should be considered for complex or atypical concussions, prolonged symptoms, or for patients who have suffered multiple concussions.

**Complications**

Most patients with a concussion will have a spontaneous, sequential resolution of their symptoms within 7 to 10 days. Some patients have a prolonged recovery with sustained symptoms known as *postconcussive syndrome* (PCS). Although there is no clear definition of PCS, some define it as persistent headache, dizziness, cognitive impairment, and psychological symptoms lasting from 6 weeks to 3 months after a concussion. The symptomatic treatment of the myriad of symptoms of PCS is quite challenging. Currently, there is no evidence-based pharmacologic treatment to offer the concussed athlete. Subgroups of patients with prolonged recoveries or significant impact on quality of life may benefit from medical therapies. Such treatments should be considered by an experienced practitioner and include tricyclic antidepressants and/or antiepileptic drugs for postconcussive headache and selective serotonin reuptake inhibitors for postconcussive depression. Patients presenting to the ED with signs and symptoms of PCS should be referred to a pediatric neurologist, neuropsychologist, sports medicine physician, or other specialist with expertise in head injury.

**Second impact syndrome (SIS)** is a rare but feared complication of concussion. SIS occurs when a patient who has sustained an initial head injury sustains a second impact before the symptoms and pathophysiological changes from the first injury
have fully cleared. It is postulated that disordered cerebral autoregulation following cumulative brain injury leads to diffuse cerebral swelling and herniation, ultimately resulting in a 50% to 100% mortality rate.\textsuperscript{96,97} Although there is debate over whether SIS is truly due to a second impact or whether it is a catastrophic complication of a single head trauma, practitioners should be aware that pediatric and adolescent patients seem to be at the highest risk of this condition.\textsuperscript{98} These patients should be monitored closely for deterioration of neurologic state after one or more concussive events. Strict adherence to RTP guidelines will ensure that patients are not at risk for a second impact during the initial vulnerable period of initial brain injury.

The long-term effects of single or multiple concussions in patients of all ages have been the recent focus of medical literature and general media. \textit{Chronic traumatic encephalopathy (CTE)} is a progressive neurodegenerative disease caused by repetitive head trauma.\textsuperscript{99} It is characterized by widespread distribution of hyperphosphorylated tau protein as neurofibrillary tangles in the brain. Originally reported in 1928 with vague “punch drunk” symptoms, recent studies have provided pathologic staging of autopsy specimens with stages of clinical symptomatology.\textsuperscript{100} Manifestations include mood disturbances, parkinsonism, ataxia, dysarthric speech, poor concentration, attention and memory loss, and behavioral outbursts.\textsuperscript{101} Many popular professional contact sport athletes who have suffered numerous concussions have later struggled with depression, substance abuse, anger, and suicide.\textsuperscript{102} Autopsy results from these athletes suggest a link between these manifestations and CTE.\textsuperscript{103} Although the magnitude and frequency of head impact needed to cause the neurodegeneration associated with CTE is unclear, it has been neuropathologically diagnosed in an asymptomatic 18-year-old high school football player with a history of concussion.\textsuperscript{104,105} More research is needed to further investigate CTE and other long-term effects of concussions, particularly in the young, developing brain.

\textbf{ABUSIVE HEAD TRAUMA}

An important consideration in the evaluation of an infant or young child with head trauma is whether he or she may have sustained an inflicted injury. Up to 30 per 100,000 infants younger than 1 year sustain severe or fatal inflicted brain injuries annually, which makes abusive head trauma (AHT) the most common cause of child abuse deaths.\textsuperscript{106,107} Victims of AHT tend to be younger than those with accidental head injuries and have more severe injuries. These injuries may occur as a consequence of shaking, blunt trauma, or a combination of both.\textsuperscript{108,109} The diagnosis can be challenging to make because many infants and young children with inflicted head trauma have nonspecific symptoms and, because they are nonverbal, cannot give a history. Missed diagnosis of AHT is common, placing the child at risk for future injury and even death.\textsuperscript{110–112} The diagnosis is more likely to be missed in younger infants, those from 2-parent households, nonminority children, and those with nonspecific symptoms,\textsuperscript{110} so the clinician must maintain a high level of suspicion when evaluating any head-injured child.

\textbf{History}

The clinician must take a thorough, detailed history of any head-injured child. A history that is inconsistent with the injury is a commonly noted “red flag” for nonaccidental trauma (eg, lack of an explanation for injury, an injury discordant with the proposed mechanism, or a history incompatible with the child’s developmental stage).\textsuperscript{113} Other factors that have been found to predict inflicted injury are changing histories and injuries blamed on siblings or home resuscitative efforts.\textsuperscript{114} Other historical factors
more common in victims of abusive head trauma are apnea, respiratory distress, seizures, vomiting, and lethargy. These symptoms may be mistaken for accidental head trauma or for another illness altogether. These findings highlight the importance of considering AHT in nonambulatory infants with severe head injuries and in infants with nonspecific symptoms.

**Physical Examination**

On physical examination, the provider should look for other signs of inflicted injury, such as skin findings (eg, bruising, bites, burns, or other patterned markings), abdominal injury, or fractures, although patients with AHT may not have external signs of trauma. Retinal hemorrhages, rib fractures, and long bone and metaphyseal fractures are other findings that should raise suspicion for inflicted injury.

**Imaging Studies**

The initial brain imaging study for an infant or child suspected to have AHT is a non-contrast CT scan. MRI can later be used to fully assess intracranial injuries of patients with a positive CT scans or patients with negative CT scans but strong clinical concerns. MRI can also be used for infants without symptoms of intracranial injury but with skeletal injuries consistent with shaking, because of its high sensitivity for diagnosing acute and chronic intracranial injury. Shaking without impact does not cause injury to the skull, therefore skull radiographs are not included in the initial evaluation of the head-injured child because they do not identify intracranial injuries. All studies should be reviewed by radiologists experienced in pediatric imaging.

Certain physical and neuroradiological features are helpful in distinguishing abusive head trauma from accidental head trauma (Table 5). Forceful shaking of an infant can stretch and tear bridging cortical veins and axons, leading to subdural hematomas and diffuse axonal injury. In contrast, accidental head trauma is usually caused by blunt trauma, so skull fractures and external injuries are more common. Mixed-density subdural hematomas are more common in abusive head trauma, but can also be seen in accidental trauma, so this finding does not necessarily indicate repeated trauma.

**Management of Abusive Head Trauma**

The initial management of head injury in suspected AHT is similar to the management of other head injuries: the primary priority is to stabilize and treat life-threatening

<table>
<thead>
<tr>
<th>Table 5</th>
<th>Radiographic and physical characteristics of accidental and abusive head trauma</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>More Common in Abusive Head Trauma</strong></td>
<td><strong>More Common in Accidental Head Trauma</strong></td>
</tr>
<tr>
<td>Subdural hematoma</td>
<td>Epidural hematoma</td>
</tr>
<tr>
<td>Interhemispheric hemorrhage</td>
<td>Isolated skull fracture</td>
</tr>
<tr>
<td>Cerebral edema</td>
<td>Scalp swelling or bruising</td>
</tr>
</tbody>
</table>
injuries. If there is any suspicion of abusive head trauma, a full workup for nonaccidental trauma should be performed, but this evaluation does not take priority over the management of acute medical issues. The workup includes a careful physical examination, complete blood count, urinalysis, liver and pancreatic enzyme tests, skeletal survey for children younger than 2 years, and fundoscopic examination. Appropriate specialists should be consulted, including child abuse pediatricians, ophthalmologists, and social workers. The history and physical examination should be carefully documented and photos taken as needed. State child protective services should be contacted according to local laws. The patient may need to be admitted to the hospital for management of their acute injuries or to ensure the child’s safety while further investigation is performed.

**SUMMARY**

Children with head injuries frequently present to EDs. Even though most of these children have minor injuries, head injury is the most common cause of traumatic deaths in pediatric patients. The provider must be vigilant to diagnose those who have life-threatening intracranial injuries or are victims of abusive head trauma. The pediatric GCS and decision rules for obtaining head CT imaging help the provider evaluate head-injured infants and children. The goal of the emergency physician is to diagnose and treat the consequences of the primary injury and to limit or prevent secondary injury and to assure proper followup for pediatric patients with minor head injury and concussion.

**REFERENCES**


