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Jeff E. Schunk and Sara A. Schutzman

Pediatrics in Review 2012;33;398

DOI: 10.1542/pir.33-9-398

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<http://pedsinreview.aappublications.org/content/33/9/398>

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Pediatric Head Injury

Jeff E. Schunk, MD,* Sara A. Schutzman, MD[†]

Author Disclosure
 Drs Schunk and Schutzman have disclosed no financial relationships relevant to this article. This commentary does not contain a discussion of an unapproved/investigative use of a commercial product/device.

Educational Gap

Recent studies have provided updated guidelines for the diagnosis of head injury and the management of patients who experience concussions. A multidisciplinary panel has recently issued new guidelines for return to play after head injury.

Objectives

After reading this article, readers should be able to:

1. Understand the anatomy and pathophysiology relevant to pediatric head injuries.
2. Take an appropriate history, perform an appropriate physical examination, and decide what imaging, if any, is warranted in the case of a child with a head injury.
3. Know the characteristics of the various types of intracranial injuries.
4. Understand the proper management of both minor and severe head injuries in children.

Introduction

Pediatric head injury is extremely common. Although the vast majority of children with head trauma have minor injuries, a small number, even among well-appearing children, will have more serious injuries with the potential for deterioration and significant sequelae. The clinician is challenged to discern which few among the many injured are at high risk for intracranial complications. Clinical symptoms are neither completely sensitive nor specific for significant injury: vomiting may be associated with intracranial injury (ICI), but most children who experience vomiting do not have a complication. Computed tomography (CT) accurately identifies ICIs requiring intervention, but also identifies minor lesions with unclear clinical importance (ie, not requiring intervention) and exposes developing brains to ionizing radiation with the associated risks.

Although clinical decision rules determine which children are at highest risk and provide a useful clinical framework, they may not necessarily direct care. Additionally, in this era of reliance on imaging, it is important to remember what the clinical examination tells us regarding brain function, information that may or may not correlate with the structural information provided on head CT.

The purpose of this discussion is to review important aspects of pediatric head trauma. Sections on epidemiology, mechanisms of injury, and the pathophysiology of specific injuries will provide a backdrop for the discussion of clinical assessment and indications for imaging and admission. What follows is a discussion of concussion, postconcussion syndrome, and return-to-play recommendations.

Epidemiology

Childhood head injuries account for more than 600,000 emergency department (ED) visits per year and presumably a larger

Abbreviations

BSF:	basilar skull fracture
CSF:	cerebrospinal fluid
CT:	computed tomography
EDH:	epidural hemorrhage
GCS:	Glasgow Coma Scale
ICI:	intracranial injury
ICP:	intracranial pressure
PECARN:	Pediatric Emergency Care Applied Research Network
SAH:	subarachnoid hemorrhage
SDH:	subdural hemorrhage
TBI:	traumatic brain injury

*Professor, Division of Pediatric Emergency Medicine, Department of Pediatrics, University of Utah School of Medicine, Primary Children's Medical Center, Salt Lake City, UT.

[†]Assistant Professor, Department of Pediatrics, Harvard Medical School, Senior Associate Physician in Medicine, Division of Emergency Medicine, Department of Medicine, Children's Hospital Boston, Boston, MA.

number of visits and calls to primary care providers. Most pediatric head injuries are minor, including scalp and face contusions, abrasions, and lacerations that do not raise concern for significant underlying pathology; however, trauma is the leading cause of death in children older than 1 year, and among trauma patients, head injury is the leading cause of death and disability. Pediatric head trauma-related deaths in the United States are in excess of 3,000 per year.

Although children exhibit almost limitless creativity with regard to sustaining injury, most pediatric head trauma results from falls, motor vehicle collisions, auto versus pedestrian incidents, bicycle-related injuries, and sports. Younger children suffer more falls and are more often the victims of child abuse, whereas motor vehicle crashes and sports-related mechanisms play a greater role in older children. This discussion focuses on blunt head trauma rather than penetrating trauma (eg, gunshot wound) because penetrating injury is much less common and unlikely to present to the primary care clinician.

Although the approach to head injury should consider the potential for serious injury in all cases, some mechanisms can be regarded as relatively trivial and unlikely to be associated with serious injury. These injuries include low-velocity self-propelled contact into stationary objects (eg, the toddler runs into the door frame) and falls from standing or sitting height or lower. However, the presence of any symptoms of head trauma despite the history of an apparently benign mechanism would no longer qualify the head injury as trivial.

Rarely, minor mechanisms may create more serious injury in the presence of undiagnosed intracranial pathology (eg, hemorrhage into a brain tumor). The clinician also must be alert to more dangerous mechanisms that could be concealed, either because the child chooses not to disclose or because the injury was inflicted.

General Pathophysiology

Brain injury results from the blow to the head and the interplay of brain parenchyma, the brain's coverings, the brain's housing structure (the cranial vault), and the vascular supply. It is useful to consider the relevant anatomic structures as layers from outside to inside.

The scalp consists of five layers of soft tissue that cover the skull. Common injuries to the skin and subcutaneous tissue (the outer two layers) include lacerations, abrasions, and freely mobile contusions. Beneath lies the strong galea aponeurotica that also connects muscular tissue on the front and back of the skull. Underneath are the loosely applied areolar tissue layer and then the pericranium.

Hemorrhages may occur in the subgaleal region from direct blows or as a result of bleeding from a fracture. Cephalohematomas are hematomas caused by bleeding beneath the periosteum, a condition well known to those who care for newborn infants.

The skull can be divided into the calvarium or bony skullcap and the skull base. The skullcap is composed of the frontal, parietal, occipital, and temporal bones. The base of the skull is made up of the sphenoid, palatine, and maxillary bones and portions of the temporal and occipital bones. Injury to the calvarium results from direct forces, and fractures commonly are linear.

Less commonly, skull fractures may be depressed (intruded by more than the thickness of the bone), comminuted (consisting of multiple fragments), diastatic (widely split), or open (communicating with a laceration). Fractures involving the skull base, known as basilar skull fractures (BSFs), are more complicated because of adjacent anatomic structures (eg, cranial nerves, sinuses), their association with ICI, and the risk they pose for meningitis.

Within the skull are the intracranial contents, consisting of the brain and its covering membranes (the meninges), blood, and cerebrospinal fluid (CSF). The meninges play an important role in the genesis of serious ICI. The outermost meningeal layer, the dura mater, is attached tightly to the inner aspect of the skull. The epidural space is a potential space between the dura and the skull. Meningeal arteries course between two layers of the dura and may become more grooved into the skull as the skull matures, so that a skull fracture may injure these vessels and cause bleeding into the epidural space. Meningeal arteries are particularly vulnerable to injury because they run beneath the thinnest part of the skull. Channels exist within the dura for venous drainage and these dural sinuses also may be lacerated. Blood collecting in the epidural space is referred to as an epidural hematoma or epidural hemorrhage (EDH).

Beneath the dura lies the arachnoid mater, a thin tissue layer coursing close to the brain but not following the brain sulci. This membrane separates the CSF-containing subarachnoid space beneath it from the subdural space. Within the subdural space lie the bridging veins that return blood from the brain to the dural sinuses. These bridging veins are susceptible to shearing forces when there is rapid acceleration or deceleration that moves the brain within the skull. A hematoma in this space is termed a subdural hemorrhage (SDH).

The third, innermost meningeal layer is termed the pia mater and adheres to the underlying brain, coursing over all gyri and sulci. This layer contains many small vessels that can be injured from direct blow or shear forces, resulting in a subarachnoid hemorrhage (SAH).

Beneath the meninges lies the brain parenchyma, a semi-solid tissue that is not affixed to the skull and can move freely within it. The CSF that bathes the brain and the spinal cord provides some degree of cushioning for the brain.

It is useful to discuss brain injury as having two phases. The primary injury is mechanical damage sustained immediately at the time of trauma from direct impact (eg, brain impacts the inner aspect of the skull or a skull fragment moves into the brain) or from shear forces when the gray matter and white matter move at different speeds during deceleration or acceleration.

Secondary injury refers to ongoing derangement to neuronal cells not initially injured during the traumatic event. Ongoing injury results from processes initiated by the trauma, including hypoxia, hypoperfusion (local or systemic shock), metabolic derangements (eg, hypoglycemia), expanding mass and increased pressure, and edema. Because primary injury occurs at the moment of trauma, little can be done to mitigate it other than prevention, so treatment during trauma resuscitation focuses on preventing secondary injury.

When considering secondary injury, two additional concepts warrant further discussion. The first consideration relates to pressure and volume within the cranial vault. After infancy, the cranial vault is a relatively stiff, poorly compliant structure and the intracranial volume is relatively fixed. From a simplistic standpoint, the vault contains brain, blood, and CSF, and any increase in the volume of one component necessitates a relative decrease in another.

If volume compensation does not occur, intracranial pressure (ICP) will increase. With progressive increases in ICP, the patient will experience headache, vomiting, and depressed mental status, then posturing, and ultimately vital sign deterioration. Increasing ICP may lead to global ischemia through mechanisms discussed later in this section. Ultimately, increased ICP will lead to brain herniation (abnormal movement of the brain across skull structures).

Herniation can occur at several different anatomic locations (Fig 1). When a mass lesion is one-sided and supratentorial, uncal herniation may occur. This type of herniation involves movement of the innermost part of the temporal lobe, the uncus, over the tentorium, with resultant pressure on the midbrain and pressure on the third cranial nerve, impairing its parasympathetic fibers and leading to ipsilateral pupillary dilation.

Central herniation occurs when central brain structures, including the diencephalon and temporal lobes, move caudally through the tentorium cerebelli. Cingulate or subfalcine herniation occurs when the cingulate

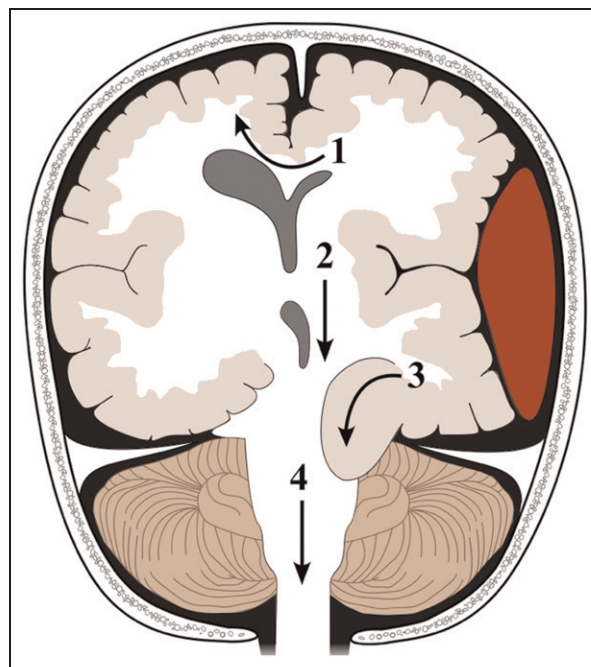


Figure 1. This figure depicts four types of brain herniation: (1) cingulate (subfalcine), (2) central, (3) uncal (transtentorial), and (4) tonsillar. (Figure is reproduced with permission from Kaye AH. Head Injuries. In: Smith JA, Tjandra JJ, Clunie GJ, Kaye AH, eds. *Textbook of Surgery*. 3rd ed. Oxford, UK: Wiley-Blackwell; 2006:445–453.)

gyrus is pushed across the midline under the falx cerebri. Although subfalcine herniation does not affect the midbrain directly, it can affect blood flow and can progress to central herniation.

In tonsillar herniation, the cerebellar tonsils move down through the foramen magnum with compression of the lower brainstem and upper cervical spinal cord. Compression of the brainstem may result in severe neurologic dysfunction, cardiovascular and respiratory instability, and death.

The other important concept in considering secondary injury involves cerebral perfusion. Cerebral perfusion pressure is the difference between the mean arterial blood pressure and ICP. In health, cerebral blood flow is maintained despite variable blood pressures by autoregulatory changes in cerebral vascular resistance. When severe injuries occur, this ability to autoregulate may be impaired, so that cerebral blood flow will be dependent on cerebral perfusion pressure.

In the absence of appropriate autoregulation, cerebral perfusion will diminish with elevated ICP or with systemic hypotension. In either instance, resultant ischemia, neuronal death, and subsequent edema all contribute to secondary injury.

General Management Considerations

Management focuses on prevention of secondary injury, so initial attention is directed to the ABCs of trauma resuscitation, focusing on maintaining adequate airway, breathing (ventilation and oxygenation), and circulation (blood pressure and perfusion). Cervical spine precautions are taken when head injury is present because head injury may be associated with cervical spine injury. Oxygen is applied, ventilation is supported as necessary to provide normocarbica (partial pressure of carbon dioxide at 35–45 mm Hg), and circulatory concerns are addressed.

Hyperventilation is no longer the standard of care, although there is still a limited role for acutely lowering increased ICP in the intensive care unit or operating room. Patients with Glasgow Coma Scale (GCS) <8 (see below) typically are intubated during trauma management.

The role of pharmacotherapy in head injury may include drugs for rapid sequence intubation, cardiovascular support, anticonvulsants when seizures occur, and medications to decrease ICP. In the most severely injured patients and those with herniation syndromes, mannitol (0.5–1.0 g/kg intravenously) and hypertonic (3%) saline can be used to promote osmotic withdrawal of water from the brain into the intravascular space in an effort to reduce ICP. Corticosteroid medications have no role in the treatment of acute brain injury; however, their use in treatment of spinal cord injury is controversial.

General Assessment

Assuming that concerns regarding airway, breathing, and circulation have been addressed, or arrangements for transfer or emergency care are being made, a general assessment should be performed, with specific emphasis on historical features and physical findings that might be indicators of potential complications from the trauma.

Historical elements should focus on details of the injury mechanism and timing of symptoms. Attention is placed on loss of consciousness, amnesia, confusion, seizure, vomiting, headache, and general behavior. Injury mechanism details should include height and surface for falls (eg, 2 feet or 2 stories; onto dirt, carpet, or concrete), use of restraining devices (eg, in car seat and car seat did not move), action of victim (eg, rolled up on car hood or thrown 20 feet), speed for crashes (eg, 10 mile per hour car crash, downhill on bike “really fast”), helmet use for sports, or velocity of object if child is struck (eg, golf club swung by teen versus 2-year-old). Other activities around the time of injury are important. Head injury can occur as the result of a medical condition (eg, seizure leads to fall with resultant head injury), and head

injury in teens may occur in the presence of intoxication and drug use.

Physical examination includes examination of the head for evidence of abrasions, lacerations, or scalp hematomas (location, size, and character: boggy or firm); draining or bleeding from the ears or nose; blood behind the tympanic membrane (hemotympanum); apparent bruising behind the ear over the mastoid (Battle sign); blood accumulating in periorbital tissues (raccoon eyes); depression in the skull; and continuity of the skull within lacerations.

Drainage or blood from the ears and nose, hemotympanum, Battle sign, and raccoon eyes are signs of BSF, frequently associated with ICI, and they are detailed in the basilar skull fracture section. Neurologic assessment is performed and although focal findings are very uncommon, they should be sought.

Assessment of mental status is of utmost importance and serves as a common triage branch point to identify patients at risk for trauma complication. This evaluation usually is done by using the GCS, which is used in most head injury research (Table 1) and provides a starting point for following the patient’s progress. For the very young child, there is a modified coma scale for infants (Table 2). It is important to judge the best response, and to follow the coma scale serially for deterioration.

Specific Injuries

Concussion

Although variably defined, in general a concussion is a head injury that results in alteration of mental status,

Table 1. Glasgow Coma Scale (Best Score Is 15)

Activity	Best Response	Score
Eye opening	Spontaneous	4
	To verbal stimuli	3
	To pain	2
	None	1
Verbal	Oriented	5
	Confused	4
	Inappropriate words	3
	Nonspecific sounds	2
	None	1
Motor	Normal spontaneous	6
	Localizes pain	5
	Withdraws to pain	4
	Abnormal flexion	3
	Abnormal extension	2
	None	1

Table 2. Modified Coma Scale for Infants (Best Score Is 15)

Activity	Best Response	Score
Eye opening	Spontaneous	4
	To speech	3
	To pain	2
	None	1
Verbal	Coos, babbles	5
	Irritable, cries	4
	Cries to pain	3
	Moans to pain	2
	None	1
Motor	Normal spontaneous	6
	Withdraws to touch	5
	Withdraws to pain	4
	Abnormal flexion	3
	Abnormal extension	2
	None	1

with or without loss of consciousness. From a practical standpoint, concussion often is used to refer to more minor head injury when the GCS is 14 to 15, the patient has some symptoms (eg, headache, dizziness, vomiting, amnesia, or confusion), there is no evidence of a fracture, and there are no focal neurologic deficits. A more detailed discussion of concussion is below.

Skull Fractures

The main importance of skull fractures is that they are markers for significant impact to the head that increases the risk of ICI significantly; however, it is important to note that ICI also occurs in the absence of fractures, and many fractures are not associated with ICI. Rarely, the fracture itself may lead to a complication (more common with basilar or depressed skull fracture). Before the advent of CT, skull radiography was an important modality to identify children at risk for complications; however, because plain radiographs give no direct information about ICI, currently they are of very limited utility. Skull fractures now are diagnosed most commonly when a CT scan is obtained.

An exception to the lack of utility of skull radiographs occurs when child abuse is suspected. When child maltreatment is suspected, the presence of a skull fracture, old or new, with or without ICI, has important implications; so skull radiographs, with their higher sensitivity for fracture, are included as part of a more comprehensive skeletal survey. Skull fractures in children younger than 2 years in the absence of a history of appropriate mechanism should prompt a more thorough evaluation for inflicted

trauma (including skeletal survey) and appropriate reporting and referrals.

Fracture of the calvarium is more common than fracture of the base of the skull. Most fractures are linear and, when considered in isolation (ie, not associated with ICI), of little consequence. No specific therapy need be directed to the fracture except pain management. Follow-up with primary care is appropriate to detect the exceedingly rare late complication of a growing fracture. Depressed skull fractures (those intruded more than the thickness of the bone) carry increased risk of primary injury to the brain because of intrusion of the fragment and, depending on the location, may have significant cosmetic sequelae (Fig 2). Neurosurgical consultation is necessary for all depressed skull fractures, even in the absence of more serious ICI.

Basilar Skull Fractures

BSF requires special consideration for several reasons. They can have unique clinical presentations providing clinical clues that often are readily apparent. Hemotympanum or blood draining from the ear, are the most common signs of a BSF. CSF draining from the ear or draining from the nose (attributable to a cribriform plate fracture), Battle sign, and raccoon eyes also are signs of BSF. Persistent

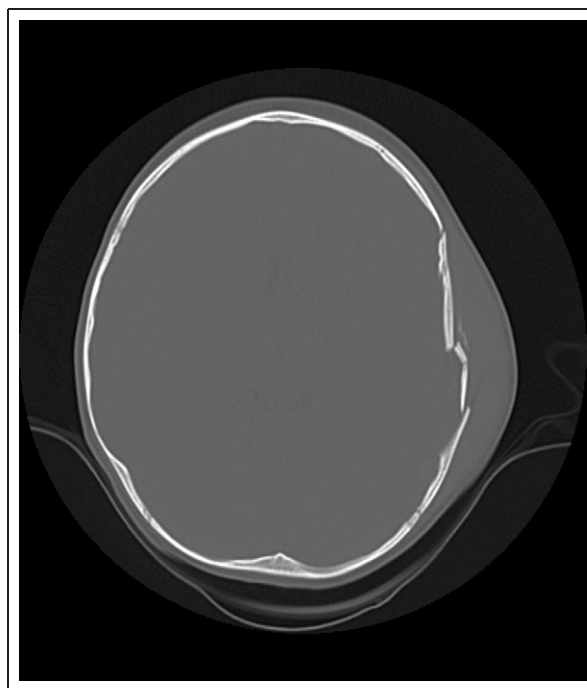


Figure 2. This toddler fell from a horse and CT scan shows depressed and comminuted parietal skull fracture.

clear drainage from the nose after head trauma should alert the clinician to the possibility of a BSF. BSF also can occur in the absence of these clinical findings and be apparent only on CT; conversely, CT scan may not detect all such fractures.

BSFs are important because they are associated with ICI and have a higher incidence of complications from the fracture itself, owing to the unique anatomic location. ICI occurs in about 20% of BSF patients who have a normal neurologic examination and GCS of 15. (1) Therefore, when signs of a BSF are noted, CT scanning is necessary.

BSF is associated also with an increased risk of meningitis. Fractures adjacent to the paranasal or sphenoid sinuses can lead to meningitis if bacteria from these areas enter the normally sterile subarachnoid space. The overall risk of developing meningitis after sustaining a BSF is probably less than a few percent, but the risk is increased if there is CSF rhinorrhea or otorrhea.

Use of prophylactic antibiotics is controversial. If there is ongoing CSF leakage, neurosurgical intervention may be needed to facilitate healing of the dural tear. Anatomic adjacency of the base of the skull to cranial nerve pathways means that BSF may cause hearing loss, facial paralysis, and a decreased sense of smell, as well as other cranial nerve dysfunction. Conductive hearing loss also may occur from blood in the middle ear.

General Intracranial Injuries

Perhaps the most important issue for the clinician evaluating a head-injured child is determining if there is an ICI. With improved CT images and current neurosurgical practice, however, detecting an ICI does not equate to a need for neurosurgery. Visible lesions on CT scan may or may not be associated with functional issues or serious morbidity. The two broad classifications of ICIs include *focal* hemorrhage (EDH, SDH, SAH, intracerebral hemorrhage, and cerebral contusion), which typically are visible on initial imaging, and *diffuse* injury (cerebral edema, diffuse axonal injury), which tends to progress and may become more visible on subsequent imaging.

Epidural Hemorrhage

When bleeding occurs between the skull and the dura mater, the patient is said to have EDH. The bleeding source is arterial in ~30%, fewer are clearly identified as venous, and in the remainder the source is unclear. EDH is caused most commonly by a blunt trauma mechanism, with falls most frequent. Often there is an overlying fracture (60% to 80%), and the EDH has a lens-shaped appearance on CT (Fig 3). Typically, the underlying brain parenchyma is not injured. Classic teaching suggested

that patients with EDH had LOC, then a lucid interval, and then deteriorated. However, that clinical presentation is rare; only ~20% of children with an EDH even experience LOC. Some children may present with marked lethargy or focal neurologic findings and progress to more frank signs of herniation.

However, presentation with more subtle signs, such as persistent vomiting and headache, is more common, and more than 30% of patients who have EDH are alert with normal neurologic findings at the time of diagnosis. Although some small epidurals may produce minimal or no symptoms, they have the potential to expand, which can result in cerebral herniation and death. Fear of missing an expanding EDH, with its high potential for mortality, has, in part, fueled the marked increase in use of CT scanning occurring in recent decades.

Patients with EDH require emergent neurosurgical consultation and close monitoring. Patients with larger EDH, midline shift, or significant symptoms are treated with emergent craniotomy and evacuation of the hematoma. Because some small EDHs do not expand significantly, relatively asymptomatic patients who have small epidurals may be managed expectantly, at the

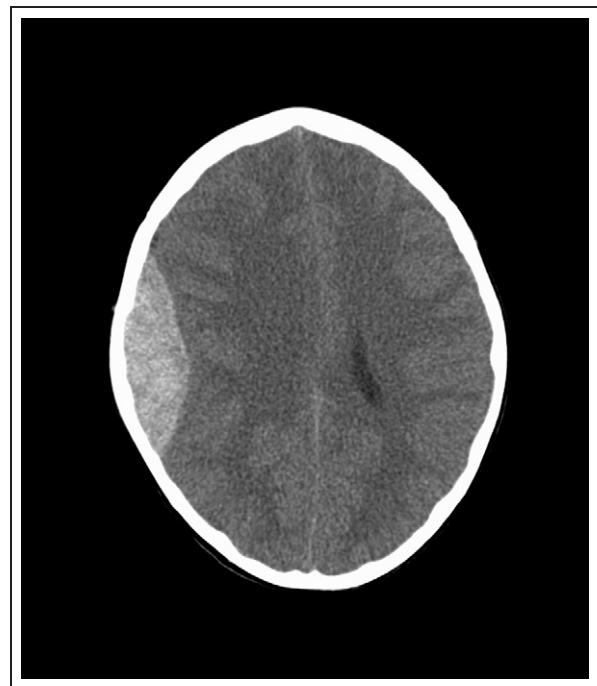


Figure 3. This CT scan demonstrates a right epidural hematoma with typical lens shape. The mass effect has caused effacement of the lateral ventricle and shift of the midline to the patient's left.

neurosurgeon's discretion, with admission and very close monitoring. Patients who have EDH successfully drained emergently have a good long-term outcome in more than 80% of cases.

Subdural Hemorrhage

When bleeding occurs between the dura and the arachnoid membrane, an SDH results. Usually, tearing of the bridging veins is the source of the bleeding and results from a direct blow, falls from significant height, or from inflicted head trauma, as seen in child abuse. SDH is not usually associated with an overlying fracture and has a crescent shape on CT (Fig 4).

Unlike the EDH, an SDH usually is associated with underlying brain injury and the hemorrhages may be bilateral. Children may present with LOC, altered mental status, seizures, irritability, vomiting, lethargy, or signs or symptoms of increased ICP (eg, bulging fontanel, decreased responsiveness). In about half the instances of SDH, the children present in coma or significantly depressed GCS.

Mortality of patients presenting with acute SDH is high and ranges from 10% to 20%. SDH in infants is associated with child abuse but is not diagnostic that abuse has occurred. Child abuse should be suspected highly when there



Figure 4. CT scan demonstrates subdural hemorrhage left posterior and left lateral that resulted from child abuse.

is no explanation for the injury, when the mechanism of injury does not match the degree of injury, or in instances in which there appears to be evidence of SDH with both new and old blood.

A chronic SDH may occur in children with coagulopathies, but usually results from child abuse, and may present with subtle findings, including macrocephaly, full fontanel, fussiness, seizures, and vomiting.

SDH requires emergency neurosurgical consultation. Patients who have an acute large SDH with evidence of mass effect within the cranium and altered level of consciousness are candidates for surgical drainage. Smaller SDH and more chronic forms may be managed without surgical decompression. Children with SDH often have significant long-term morbidity that may include developmental delay and seizures. These adverse, persistent neurologic sequelae are more likely to occur in patients who present with coma, or when CT scan demonstrates underlying brain injury.

Subarachnoid Hemorrhage

In more severely injured patients, SAH occurs about 25% of the time. This lesion results from tearing of the small vessels of the pia mater secondary to significant blunt trauma and associated shearing forces. Because the bleeding is in a space that communicates with other CSF-containing spaces (within the brain, around the brain and spinal cord), problems related to the mass effect that is seen with EDH and SDH rarely occur. SAH often is seen in association with other ICIs, so presentation is variable, but SAHs occurring in isolation may present with LOC, headache, or signs of meningeal irritation (eg, vomiting, photophobia, nuchal rigidity).

Cerebral Contusion

A cerebral contusion is essentially a brain bruise caused by a well-localized area of neuronal injury with bleeding (Fig 5). This injury results from movement of the brain against the skull. Blunt trauma to the head may cause a cerebral contusion near the site of impact (a “coup” lesion) or may cause a cerebral contusion opposite the site of impact (a “contrecoup” lesion). Typical signs may be subtle, and can include vomiting, headache, LOC, or, less commonly, a focal neurologic finding or a seizure. In most instances, small contusions have little acute or long-term sequelae.

Diffuse Axonal Injury

Diffuse axonal injury involves injury to the white matter tracts within the brain and is likely caused by shear forces. This type of injury is caused by severe acceleration,



Figure 5. This 12-year-old was struck by a softball in the occiput. CT scan demonstrates cerebral contusion left posterior parietal region with small area of surrounding edema.

deceleration, or rotational forces, occurring most commonly in motor vehicle crashes. The injury often is at the gray-white matter junction but may occur deeper within the corpus callosum, brainstem, or cerebellum. These children usually are in coma at presentation, although occasionally the child will have only concussion-type symptoms. The CT scan shows small areas of hemorrhage located near the gray-white interface that do not expand. Management of diffuse axonal injury is supportive, mortality is 10% to 15%, and persistent neurologic dysfunction occurs in 30% to 40%.

Diffuse Brain Swelling

This condition is seen almost exclusively in children who experience severe head trauma and the mechanism appears to be a reaction to cellular injury. Diffuse brain swelling may not be apparent on initial imaging; subsequent CT scans demonstrate findings of progressive edema. The cellular insults may be varied, and cytotoxic edema, vasogenic edema, and autoregulatory dysfunction all may play a role. The children present with marked depression or deterioration of the GCS, and the main threat is the associated increase in ICP.

Who Needs Computed Tomography?

The clinician's goal is to identify patients who develop clinically important ICI so as to prevent deterioration and secondary brain injury (eg, from expanding EDH), while limiting unnecessary radiographic imaging. Unfortunately, defining sensitive and specific clinical predictors for identifying high-risk patients who require a head CT has been challenging.

Several issues contribute to the challenge of evaluating head-injured children:

- Although patients with ICI often have symptoms or functional derangements, many patients with these same symptoms have no ICI.
- Patients with normal neurologic examinations who exhibit symptoms as common as vomiting or headache may harbor an ICI that has the potential to become life-threatening. Repeated examination of the fundi is prudent because papilledema may not be present initially but may develop later in the course of intracranial hypertension.
- Many intracranial lesions detected by CT are only rarely associated with significant morbidity (eg, small cerebral contusion or small SAH).
- Although CT can effectively identify clinically important ICI, this imaging modality carries the risks of radiation, including the long-term sequelae of radiation-induced malignancy.

Investigators have identified several clear predictors of ICI:

- GCS \leq 14 or altered mental status.
- Focal neurologic abnormalities.
- Skull fracture.

Patients who have any of these findings should undergo CT imaging.

However, most patients have none of these findings (ie, they have a GCS of 15, nonfocal neurologic examination, and no obvious skull fracture); yet, patients who lack these features account for a large proportion of patients who actually have ICI. Within this group, the incidence of ICI is about 5% and the need for neurosurgery $<$ 1%. Identifying reliably sensitive and specific clinical indicators has been difficult, however, because studies have found conflicting evidence regarding the significance of LOC, vomiting, seizures, and headache.

Children younger than 2 years should be considered separately. Younger children are more difficult to assess clinically, are more easily injured even from short falls, have a higher incidence of asymptomatic or occult

injuries, and more often are victims of inflicted injury. In addition to the predictors of ICI found in older children, nonfrontal scalp hematomas (surrogate markers for skull fracture) were found to be predictors of ICI, with larger hematomas in younger children of greater concern. (2)

In all age groups, because of the variability of clinical predictors in identifying ICI and concern for missing ICI, clinicians have adopted a very liberal approach to the use of CT scans. ED-based studies have shown that this group with mild head injury undergoes CT scanning from 35% to 55% of the time. Head CT for pediatric minor head injury increased in Canada from 15% in 1995 to 53% in 2005 for head-injured children. In the United States, use has increased dramatically in the face of relative stability of serious injury, implying that more and more normal CT scans are being obtained.

Risk of Head Computed Tomography Scanning

Widespread imaging has increased concerns regarding safety, specifically related to sedation and radiation risk. Concern for adverse events from sedation is justified, but with increasing speed of scanners, the need for sedation should decrease. Clinical experience and research in pediatric sedation has blossomed, and overall hospital practices in this regard have become safer, so that sedation-related adverse events are less of a concern.

The potential for ill effects from ionizing radiation cannot be overlooked. Evidence for this risk assessment comes primarily from information on radiation exposure following nuclear bomb detonation and data derived from therapeutic use of radiation. It is estimated that CT scanning will induce a new malignancy at a rate of ~ 1 in 5,000 CT scans. It appears that the greatest lifetime risk occurs in the youngest patients (both because of life-years remaining and susceptibility of tissues), and overall risk decreases as age increases. From the standpoint of an individual or individual clinician, this rate does not seem high, but when one considers the tens of thousands of normal head CT scans being performed each year, the public health impact may not be trivial.

Recent Investigations

Recent investigations (3)(4) have better identified more meaningful predictors by using multicenter design, including large numbers of head-injured children, focusing on groups at relatively low risk, and determining decision rules to aid clinicians determining the need for CT. Some of these large studies also altered the primary outcome measure from “presence of an ICI,” as previous studies

had done, to “clinically important” traumatic brain injury (TBI). In a study through the Pediatric Emergency Care Applied Research Network (PECARN) involving more than 42,000 pediatric patients at 23 centers, a clinically important TBI was defined as death, need for neurosurgery, intubation >24 hours, or hospitalization for ≥ 2 nights. (3)

Decision rules are developed to guide the clinicians in a more thoughtful approach to CT scanning so as to avoid overuse while still identifying clinically important ICI. No rules eliminate all risk unless all patients are scanned, but they provide a needed framework for risk assignment. The clinician who appropriately elects not to scan should understand the risk of serious ICI.

In the PECARN study, (3) when only children with GCS of 14 to 15 are considered, high-risk criteria ($\sim 4\%$ incidence of clinically important ICI) were GCS = 14, other signs of altered mental status, and palpable skull fracture (if age <2 years) or signs of BSF (if age >2 years) for which CT was recommended.

Other risk factors ($\sim 1\%$ incidence of clinically important ICI) for age >2 years: loss of consciousness, severe injury mechanism, vomiting, and severe headache; and for age <2 years: loss of consciousness, severe injury mechanism, nonfrontal scalp hematoma, and not acting normally per parents. Recommendations were for either CT imaging or observation. If none of these risk factors was present, the incidence of clinically important ICI was $<0.05\%$ and CT was not recommended.

In a multicentered Canadian study involving 3,866 children with GCS of 13 to 15, high-risk factors (associated with need for neurologic intervention) were GCS <15 at 2 hours after injury, suspected open or depressed skull fracture, worsening headache, and irritability. (4) Medium-risk factors (associated with presence of brain injury on CT) were signs of BSF, large boggy scalp hematoma, and dangerous mechanism.

Indications for Head Computed Tomography Scanning

Table 3 lists the situations in which head CT scanning is recommended. This list represents a compilation of the recent multicenter studies, as well as recent reviews. This table pertains to all age groups, and these clinical factors indicate a relatively high risk of detecting a clinically important TBI that would make obtaining a head CT justifiable. Table 4 (age >2 years) and Table 5 (age <2 years) outline patients who generally have a risk of ICI of $\sim 3\%$ to 5% , and have a risk for clinically important TBI of $<1\%$, and assumes that findings in Table 3 are not present. These patients could be considered for imaging, but observation of

Table 3. Emergent Head Computed Tomography Scan Is Recommended

Penetrating injury
 Glasgow Coma Scale (GCS) ≤ 14 or other evidence of altered mental status
 Focal neurologic abnormalities
 Signs of depressed or basilar skull fracture
 Worsening headache
 Prolonged loss of consciousness (LOC) (more than a few minutes)
 Clinical deterioration during observation or significant worsening of symptoms
 Seizure (other than impact seizure) or any prolonged seizure
 Pre-existing condition that places child at increased risk for intracranial hemorrhage (eg, bleeding disorder)
 In addition for children < 2 years old:
 Concerns for inflicted injury
 Seizure
 Irritability
 Bulging fontanel
 Persistent vomiting
 Large, boggy, nonfrontal scalp hematomas in children < 1 year old
 Definite (more than brief) LOC

Table 4. For Age > 2 Years, Moderate Risk for Intracranial Injury

The following group of patients should be considered for emergent imaging but observation for 4–6 hours can be considered as an alternative (estimated risk of clinically important traumatic brain injury $\sim 1\%$). In general, if more than one of the following are present, the clinician should perform head imaging:

Loss of consciousness
 Seizure (brief and impact)
 Severe headache
 Vomiting
 High-risk mechanism (fall greater than 5 feet in older children, struck by high-impact object, ejection from motor vehicle, motor vehicle crash with death of another or auto pedestrian, auto-bike without helmet)

Additional clinical constellations that should prompt imaging:

Behavioral change that is both significant and prolonged (especially more than a few hours)
 Multiple episodes of vomiting or onset delayed several hours after injury

at least 4 to 6 hours from the time of injury is a reasonable alternative.

It is clear from a subanalysis of the PECARN study that clinicians sometimes use observation before deciding to obtain a CT. When observation is chosen, the appearance of additional new symptoms, evidence of worsening symptoms, or clinical deterioration should prompt imaging. In instances in which multiple risk factors are present or symptoms are more severe, imaging probably is favored. Other factors that may influence the decision to image include quality of observation (eg, caretaker reliability, time of day), the ability to return for worsening symptoms, physician experience, and parental preference. Table 6 lists criteria typical of patients for whom imaging is not necessary.

Disposition

In general, patients with depressed skull fracture or any ICI should be hospitalized with emergent neurosurgical consultation for their lesions; however, some small cerebral contusions or SAHs may have little short- or long-term clinical significance, and deterioration is rare. Patients with normal CT scans and resolution of symptoms typically

do not require hospitalization. Patients with persistent symptoms (despite normal CT scans) who would not be

Table 5. For Age < 2 Years, Moderate Risk for Intracranial Injury

Patients with the following signs or symptoms should be considered for emergent imaging, but observation for 4–6 hours can be considered as an alternative (estimated risk of clinically important TBI $\sim 1\%$). In general, if more than one of the following are present, the clinician should perform head imaging:

Occipital, parietal, or temporal scalp hematoma
 Behavioral change per caregiver
 Nonacute (more than 24 hours) skull fracture
 High-risk mechanism (fall greater than 3 feet, struck by high-impact object, ejection from motor vehicle, motor vehicle crash or auto pedestrian with death of another, auto-bike without helmet)

Additional clinical constellations that should prompt imaging:

Multiple episodes of vomiting or onset delayed several hours after injury

TBI=traumatic brain injury

Table 6. Criteria for Patients Who Can Reliably Forego Computed Tomography Imaging

Normal neurologic examination
 Normal mental status
 Normal behavior per caregiver
 No loss of consciousness
 No vomiting
 No severe headache
 No evidence of skull fracture (for children <2 years, no nonfrontal scalp hematoma)
 No signs of basilar skull fracture
 No high-risk mechanism (fall greater than 3 feet in children <2, fall greater than 5 feet in older children, struck by high-impact object, ejection from motor vehicle, motor vehicle crash or auto pedestrian with death of another, auto-bike without helmet)
 No concern for inflicted injury

managed easily at home (persistent vomiting, severe headache, abnormal mental status) also should be admitted. For any child deemed stable for discharge (both those with and without imaging), symptoms concerning for ICI should be reviewed with reliable caretakers who are able to return to the ED should concerns arise.

Home Management of Minor Head Injury

Calls to practitioners from caregivers regarding pediatric head injury are frequent. In many instances, ongoing observation at home without an ED or office visit is reasonable, if there is a reliable caretaker with the means to seek additional care if needed, if there is no concern for inflicted injury, and if there are no underlying conditions that would predispose the child to an ICI. In cases in which there is a low-risk mechanism (typically a ground level fall from child's own height) and there are no other injuries, no LOC or mental status changes, no vomiting (one episode shortly after injury is of less concern), no significant headache, and no nonfrontal scalp hematomas (for children <2 years), ongoing home observation can be pursued. It is not necessary to prevent a child from napping as the child would normally, but the child should be checked periodically for clinical deterioration. Indications for seeking medical care should be reviewed with the caretaker.

Concussion and Mild Traumatic Brain Injury

Traditional classification of TBI grouped patients by degree of functional impairment using the GCS. This classification scheme is independent of CT findings, but the

likelihood of abnormal CT findings increase in frequency as the severity of the TBI increases (ie, as GCS decreases).

Concussion has been defined variably based on clinical findings and represents a subset of the mild TBI group (GCS 13–15). In a simple definition from the American Academy of Neurology, concussion is an alteration in neurologic function or mental status after head injury that may (or may not) involve LOC. Concussion typically has a rapid onset of impairment of neurologic function that is short-lived and resolves spontaneously. This condition represents a functional problem and, by convention, patients do not have abnormalities on CT imaging (if CT was obtained).

In addition to the common signs of headache, vomiting, or dizziness, signs in patients who experience concussion might also include a vacant stare or confused expression, difficulty focusing attention, disorientation, slurred or incoherent speech, delayed response to questions, emotional response that is out of proportion to the situation, repetitive questioning, coordination problems, or memory problems. Patients typically improve over minutes to a few hours. Failure to improve or worsening symptoms are an indication for imaging, if not performed previously. Repeated concussions probably have a cumulative effect and have been implicated in long-term cognitive impairment and neuropsychiatric problems in professional athletes.

Second-Impact Syndrome

Second-impact syndrome refers to a very rare but usually fatal diffuse cerebral edema as a consequence of mild head injury. This term is applied typically when an athlete develops diffuse cerebral edema from a second head injury while still symptomatic from a first concussion. The understanding of this condition is insufficient, based mostly on a limited number of case reports, and there is controversy surrounding this complication. Even the time period of risk is debatable, but probably is less than a couple of weeks.

Concerns for this lethal condition, however, and the concerns over cumulative adverse effects from repeated head injury, have prompted guidelines for return to play following a concussion.

Return to Play

To aid return-to-play decisions, several guidelines based on acute symptoms have been published (eg, Colorado Medical Society, the American Academy of Neurology); these guidelines are not based on detailed clinical evidence, and they have not been compared in clinical

Table 7. **Graduated Return to Play**

Stage	Activity	Stage Objective
No activity	Complete physical and cognitive rest	Recovery
Light aerobic exercise	Walking, swimming, stationary cycling, low–moderate intensity	Increase heart rate
Sport-specific exercise	Skating drills ice hockey, running drills, no impact	Add movement
Noncontact training	More complex training drills (eg, passing drills), may start progressive resistance training	Exercise, coordination, cognitive effort
Full-contact practice	Normal training activities after medical clearance	Assess skills by coaches; restore confidence
Return to play	Normal game play	

In general, the athlete who has sustained a concussion should proceed to the next level if without symptoms at the current level. Each step generally takes 24 hours. If symptoms recur, then the patient drops back to previous asymptomatic level. (Adapted from Table in Consensus statement on concussion in sport. *J Clin Neuroscience*. 2008;16:755–763, with permission.)

studies. Both sets of guidelines share some common recommendations.

Athletes suspected of having a concussion should be removed from participation immediately and they should not return while signs or symptoms are present. Athletes symptomatic for >15 minutes should not return to play until they are asymptomatic for 1 week.

Most recently, a multidisciplinary panel published a consensus guideline advocating abandoning acute grading scales in favor of clinical measures of recovery. (5) Return to play should be based on resolution of symptoms and normalization of neurocognitive function for the individual, rather than based on a predetermined amount of time. Recommendations are for physical and cognitive rest until asymptomatic, followed by a graduated, monitored return to play. This graduated return to play guideline from this consensus paper is presented in Table 7.

Postconcussion Syndrome

Postconcussive symptoms develop within a few days of the initial concussion and can last anywhere from a few days to a few months. Typical symptoms include headache, fatigue, dizziness, cognitive impairment (particularly concentration), and neuropsychiatric symptoms. Some children and teens may experience long-term behavioral and cognitive problems temporally related to experiencing a concussion.

About 80% of high school athletes who experience sports-related concussions have resolution of symptoms

- Based on strong research evidence, a thorough history and physical examination with emphasis on neurologic status and signs of skull fracture (including size, character, and location of scalp hematomas in infants) provide the clues necessary to assess the relative risk of serious intracranial injury (ICI). (1)(3)(4)(6)
- Based on strong research evidence, computed tomography (CT) scan remains a highly useful adjunct in evaluation of the head-injured child but should be used selectively for children at higher risk for ICI. (2)(3)(4)
- Based on research evidence or consensus opinion, observation can be used selectively in lieu of CT scanning for patients who are not at higher risk for ICI, minimizing the risks of CT-associated ionizing radiation. Any concerns during the observation period should prompt CT imaging. (3)(7)(8)
- Concussion from head injury in athletics has recently received increased public attention. Based on consensus opinion, premature return to play may confer increased risk to the athlete. (9)(10)

within 1 week, and fewer than 2% are symptomatic longer than 1 month. For patients whose symptoms persist beyond a few weeks, referral to a pediatric neurologist, neuropsychologist, sports medicine physician, or other specialist with expertise in head injury probably is indicated. Investigations into understanding postconcussion syndrome risk and effectiveness of interventions is limited.

Summary

- Pediatric head injury is very common and usually minor but can result in serious morbidity and is the most common cause of lethal trauma.

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1. A 5-year-old boy was in a motor vehicle collision as a restrained back seat passenger. He has a large laceration on his right forehead. On arrival to the emergency department, he opens his eyes with painful stimuli, but does not open his eyes when his name is called. He is mumbling, but is not using words. He withdraws to pain. His brain injury is best described as a
 - A. mild traumatic brain injury
 - B. moderate concussion
 - C. moderate traumatic brain injury
 - D. severe concussion
 - E. severe traumatic brain injury

2. A 15-month-old girl is seen for fussiness, crying, and poor oral intake. On physical examination, she is fussy, but consolable. Her vital signs are stable. She is well hydrated. She has unilateral hemotympanum. The most appropriate initial intervention at this time is
 - A. computed tomography (CT) scan of the brain
 - B. magnetic resonance imaging of the brain
 - C. prescription for antibiotics
 - D. skull radiographic films
 - E. skeletal survey

3. You see a 6-month-old girl with history of vomiting who presents with lethargy and irregular respirations. After intubating and stabilizing her, you obtain CT imaging of the brain that shows a parietal skull fracture and a crescent-shaped intracranial hemorrhage underlying the fracture. This radiographic finding is most consistent with a
 - A. cerebral contusion
 - B. diffuse axonal injury
 - C. epidural hematoma
 - D. subarachnoid hemorrhage
 - E. subdural hemorrhage

4. A 15-year-old boy was the unrestrained passenger in a motor vehicle collision. He was ejected from the vehicle and was found unconscious 20 feet from the vehicle. A CT scan shows areas of hemorrhage at the gray-white junction. His clinical presentation and radiographic findings are most consistent with
 - A. cerebral contusion
 - B. diffuse axonal injury
 - C. epidural hematoma
 - D. subarachnoid hemorrhage
 - E. subdural hemorrhage

5. You are evaluating a 6-year-old boy who sustained a head injury when he fell out of a tree. Clear fluid is noted to be draining from his nose. His parents deny any recent respiratory infection or history of nasal allergy. You order a CT scan. The most likely abnormality to show up on the scan will be
 - A. basilar skull fracture
 - B. cerebral contusion
 - C. depressed skull fracture
 - D. epidural hemorrhage
 - E. subdural hemorrhage

Pediatric Head Injury
Jeff E. Schunk and Sara A. Schutzman
Pediatrics in Review 2012;33;398
DOI: 10.1542/pir.33-9-398

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