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Mild head injury is a common reason for emergency department (ED) visits and hospital admission in the pediatric population. Review of pertinent literature reveals that children with mild head injury comprise up to 93% of all head-injured children admitted to the hospital.

There are many controversies concerning the assessment and management of children after mild head injury.[1-6] The majority of these patients achieve a full recovery, even without medical intervention. However, some children with mild head injury may deteriorate because of intracranial complications such as brain edema or hematoma. Therefore, it is of utmost importance to identify factors that predict which child with mild head injury is at risk for subsequent deterioration.

The care of children with head injuries requires rapid and knowledgeable assessment, diagnosis, and treatment. The purpose of this article is to help ED physicians determine which children with mild head injuries require neuroimaging, can be sent home for observation by reliable caregivers, or require admission.

— The Editor

Current Concepts in the Management of Minor Closed Head Injury in Children

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Types of Head Injury

Scalp Injuries. The scalp is highly vascular and known to produce profuse bleeding when injured. An infant with an open or closed scalp injury can have rapid deterioration from blood loss alone and even develop hypovolemic shock.[6,7]

A subgaleal hematoma in an infant may be the only sign of intracranial injury.[8,9] The presence of a subgaleal hematoma has been shown as an objective indicator for epidural hematoma (EDH) in infants (< 2 years of age).[8] In the development of the Trauma Infant Neurologic Score (TINS), 72.7% (8 of 11) of infants with EDH had a significant subgaleal hematoma.[8] Due to variable psychomotor development and incomplete verbal skills, distinguishing which infants require head computed tomography (CT) scans using the various children's coma scales has been problematic. Under the TINS recommendations (see Table 1), a CT scan should be performed on all infants with a subgaleal hematoma secondary to trauma.[8]

Concussion. Traditionally, concussion refers to a brief alteration in consciousness (with or without loss of consciousness [LOC]) after a closed head injury and is accompanied by a flac-

cid motor state, followed by complete recovery.[6,10] More recently, experts have placed such mild head injury at the lower end of a spectrum of conditions caused by diffuse axonal injury, secondary to evidence which suggests disruption of axonal functioning after such injury.[11] Frequently, a brief period of vomiting, pallor, confusion, or amnesia (both retrograde and anterograde) follows this event.

The possibility of long-term sequelae in children with minor head injury is controversial. Numerous studies contend that some pediatric patients may experience a postconcussion syndrome, which is similar to conditions reported in adults after minor head injury.[5,6] This syndrome may present as a behavioral disability when the child is confronted by increasing intellectual, academic, and social demands of advancing age. These patients may need referral to a pediatric neurologist for evaluation. However, studies that screen for pre-injury impairments show less to no long-term neurobehavioral morbidity for children due to minor head injuries.[4,5] Sports-related concussions are discussed in a later section.

Skull Fractures. Skull fractures may be classified as linear, diastatic, depressed, compound, or basilar. Open skull fractures may be associated with dural lacerations and raise the possibility of infection. All of these children require a neurosurgical consultation.

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Linear skull fractures constitute 75-90% of skull fractures in children.[6] The most significant complication is intracranial hematoma, which is 10- to 20-fold more likely when a skull fracture is present.[12] The parietal bone is the most common site of skull fractures (70%), and usually is not indicative of abuse.[13] Diastatic fractures are the traumatic separation of cranial bones at the suture sites. Nonaccidental skull fractures are more likely to be bilateral, comminuted, depressed, wider than 1 mm, and associated with other injuries.[13,14] They also are more likely to involve nonparietal bone and cross suture lines.[13,14] *(See Table 2.)* When complex fractures are present, especially without a complete history consistent with the extent of the injury, practitioners should be suspicious of nonaccidental trauma.[15]

A leptomeningeal cyst, also known as a "growing" skull fracture, may occur when a dural tear allows the arachnoid membrane to emerge from a skull fracture.[5] Development of such a cyst occurs when fluid causes demineralization of the bone at the fracture site and the subarachnoid space fills with cerebrospinal fluid (CSF). Usually presenting in children younger than 3 years, leptomeningeal cysts are not as common as previously suspected.[4] All children with an acute linear or diastatic skull fractures require a head CT scan. Appropriate management for all depressed linear or diastatic skull fractures includes reevaluation of the fracture in 2-3 months; if it is still growing, surgery may be required. One report showed that of the small percentage of cysts that grow (3%), most fractures were diastatic.[4]

A depressed skull fracture involves disruption of the integrity of the skull. It is generally accepted that clinically significant depressed skull fractures include injuries in which the bone fragment is depressed below the inner table of the skull to a depth greater than the full thickness of the skull.[6] On physical examination, step-off regions adjacent to swollen pericranium may sometimes be mistaken for depressed skull fractures. If no break of the skin has occurred over the depressed fracture, it is termed a simple fracture. If the full thickness of the overlying scalp has been lacerated, then it is a compound, depressed skull fracture.[16] An example of a commonly overlooked compound fracture is one caused by penetration through the orbital roof.

The "ping-pong" skull fracture, which is a special variant of a depressed skull fracture, is seen more commonly in the newborn infant when the cranium is less well mineralized and more easily distorted. The diploë (spongy tissue) within the inner and outer tables of the skull has not yet developed and can be dented like a ping-pong ball or the fender of an automobile. A head CT scan is necessary for all children with potentially depressed skull fractures, and neurosurgical consultation is appropriate for definitive management. The depression will remain without surgical correction.

The skull base is a frequent site of fracture, accounting for approximately one-fifth of skull fractures.[17] Some characteristic signs and symptoms of a basilar skull fracture include CSF rhinorrhea, hemotympanum, ecchymosis behind the ear over the mastoid bone (Battle's sign) resulting from fracture of the temporal bone, and ecchymosis around both eyes (raccoon eyes), which is suggestive of an anterior basal skull fracture. Fracture of

Table 1. Trauma Infant Neurologic Score (TINS)

	MINIMUM/ MAXIMUM	0	1	2
Mechanism of trauma	1/2	—-	Fall < 1 m or mild blow	Fall > 1 m, penetrating injury
Intubated on admission	0/1	No	Yes	——
Alertness	0/2	Fully alert	Decreased but arousable	Unconscious
Motor deficit	0/2	None	Lateralizing signs	No movement
Pupils	0/2	Reactive bilaterally	Anisocoria or nonreactive pupil	Dilated and nonreactive
Scalp Injury	0/1	None	Subgaleal hematoma	——

Total score ranges from 1 to 10 points.
An infant with a history of trauma will get at least a TINS of 1 point.
TINS higher than 8 suggests poor prognosis.
TINS ≥ 2 indicates the need for a CT scan.

Adapted from: Beni-Adani L, Flores I, Spektor S, et al. Epidural hematoma in infants: A different entity? *J Trauma* 1999;46:306-311.

the parietal bone and squamous portion of the temporal bone may extend into the petrous portion of the temporal bone longitudinally. These fractures are associated with tears of the tympanic membrane, disruption of the auditory ossicles, and in some cases, facial nerve injury. Fractures may destroy the cochlear-vestibular apparatus and facial nerve, leading to permanent hearing loss and facial palsy. Other complications of a basilar skull fracture may include CSF fistulas. CSF can escape through fractures of the walls of the paranasal sinus associated with dural tears. Fractures of the petrous pyramid may be associated with leakage out of the ear through a tympanic membrane or leakage in the pharynx through the eustachian canal. Meningitis may result from an undetected fistula, and therefore, if suspected, the patient should be referred for further work-up, including a radionuclide study with a radioactive tracer to detect the path of the fistula. Antibiotic prophylaxis for basilar skull fracture is not recommended because it may lead to subsequent meningitis with antibiotic-resistant bacterial strains. However, if fever develops, a lumbar puncture should be performed and intravenous broad-spectrum antibiotic coverage should be started promptly. Antibiotic adjustments can then be performed when the organism's sensitivities become available.[6] Neuroimaging consult and referral is needed for all children with basilar skull fractures.

Brain Injuries

Contusion. A cerebral contusion occurs when the brain parenchyma is bruised or crushed, resulting in hemorrhage and edema. When brain injury occurs on the side of impact, a coup lesion results. Coup lesions classically result from abrupt acceleration of the head. Contrecoup lesions are caused by damage to the opposite side of the brain from the impact, and are most severe in rapid deceleration injuries. However, either coup or contrecoup lesion can be the result of an acceleration or a deceleration force.

Diffuse Axonal Injury. The shearing forces that accompany closed head injury can directly damage the axons in the CNS.[10] The importance of this mode of injury, called diffuse axonal injury (DAI), has come to be recognized in a wide spectrum of insults. Clinical experience has demonstrated that there are patients who suffer closed head injury and never recover consciousness, but nevertheless, lack large macroscopic intracranial lesions such as contusions, hematomas, or diffuse edema. Pathological studies have shown that these patients suffer shearing injuries of axons in the white matter as well as rents in the rostral brainstem and corpus callosum.[10] Pioneering work in microscopic examination of these lesions demonstrated that axons under these conditions underwent transection and characteristic formation of retraction balls. In the severest form, lesions in the brainstem and corpus callosum lead to permanent loss of consciousness.[10] Less severe forms, which do not involve the brainstem, lead to temporary alterations in consciousness and neurologic deficits. The least severe forms of DAI may correlate with concussions. DAI also may accompany contusions and subdural and intracerebral hematomas, such as those caused by angular or rotational deceleration in shaken baby syndrome.[18-20]

Intracranial Hematoma. The most life-threatening complications of mild head injury are intracranial hemorrhages. Types of intracranial hemorrhages include subarachnoid hemorrhages, subdural hematomas, epidural hematomas, and parenchymal contusions. Although the incidence of intracranial hemorrhage is lower with mild head injury than with head injuries resulting in a low coma score, hemorrhages occur in up to 12% patients with mild head injuries.[5,21] Prompt surgical evacuation of clinically significant intracranial hematomas maximizes neurological recovery. The primary rationale for neuroimaging is the detection of intracranial hemorrhages requiring surgical intervention.

Epidural hematomas collect between the bone and dura, forming a lens-shaped lesion on CT scan. In 60-80% of cases, there is an overlying fracture; they are usually arterial in origin, but may be venous, depending on the location.[12] Epidural hematomas result from brief linear contact forces that commonly occur in

unintentional falls.[22] Although not common, the classic pattern of events is for the child to be lucid between initial loss of consciousness and rapid neurologic deterioration and death. Many investigators have found that epidural hematomas may be subtle in presentation. Epidural hematoma may occur after relatively minor head trauma and in alert children with nonfocal neurologic examinations.[24] Epidural hematoma in infants younger than 2 years may be difficult to diagnose; infants often are brought to the ED fully alert and with no neurologic deficit.[8] As previously discussed, guidelines to perform CT as early as possible are helpful, because the prognosis is excellent with early treatment.[8,22] TINS was developed as a tool to facilitate the early diagnosis of epidural hematoma in infants.[8] Rapid CT scanning and surgical intervention have dramatically reduced morbidity and mortality due to this lesion.

A subdural hematoma is caused by the accumulation of blood between the dura and the arachnoid membranes. The mechanism of injury is commonly rapid deceleration/acceleration or vigorous shaking (as in shaken baby syndrome), resulting in torn bridging veins, or infrequently, bleeding from the cortex due to a damaged surface cerebral artery.[4,25,26] CT scan will shows crescent-shaped black lesion. Skull fractures occur in the minority of cases.[12] Recent studies point to continued poor outcomes in acute subdural hematoma, despite early surgical intervention.[27] The majority of subdural hemorrhages in children younger than 2 years are due to child abuse.[23,25,28-30] A study of 33 children presenting with subdural hematoma found that 85% of the children were younger than 1 year of age; 27% died, and 45% had profound disability. Eighty-two percent of the injuries were highly suggestive of child abuse.[25] A high index of suspicion for nonaccidental trauma is essential in young children presenting with a subdural hematoma.

Intracerebral hemorrhage is a vascular injury that occurs in the parenchyma itself and commonly is associated with some element of parenchymal damage. The smaller lesions usually do not require surgical intervention, although they may cause a focal mass effect. Larger lesions that represent a significant mass may require evacuation. Intracerebral hemorrhages may not be present or detectable on initial CT scans, but they may appear or enlarge within the next 24-48 hours. Patients with intracerebral hemorrhage, along with those who have subdural or epidural hematomas, require close observation in an intensive care unit, especially if the initial therapy does not include surgical intervention.[31]

Subarachnoid hemorrhage, common in pediatric trauma, results from the laceration of superficial vessels running in the subarachnoid space. Subarachnoid hemorrhage may lead to vasospasm and further ischemic injury to the brain.[32] Unless an obstructive or communicating hydrocephalus develops, surgical intervention rarely is required.[31] CT scan does not detect 10% of subarachnoid hemorrhages, requiring CSF studies to detect blood.[33] Treatment of subarachnoid hemorrhage is usually symptomatic; intravascular volume should be maintained to minimize ischemic damage from associated vasospasm.[31]

Table 2. Skull Fractures: Accidental vs. Non-Accidental

INFLICTED	ACCIDENTAL
History does not correlate with mechanism of injury or no history	History consistent with injuries
Associated with intracranial injury, especially subdural hematomas	Not associated with intracranial injury
Bilateral	Unilateral
Non-parietal	Parietal
Comminuted/Stellate	Linear
Depressed	Nondepressed
Wider than 1 mm	Narrower than 1 mm
Associated with other injuries	Involves only one body area
Crossing suture lines	

Adapted from: Sheridan C, Mellick LB, Sherwin T. Recognizing child abuse and neglect: The role of the emergency physician. *Emerg Med Reports* 1993;14:67-74.

Posttraumatic Seizure. The incidence of posttraumatic seizures (PTS) in all head trauma is 5-10% and in minor head trauma is 1-6% .[4, 5,12] PTS can be classified as immediate, early, and late.[12] Immediate PTS is thought to be due to depolarization of the cortex. They occur within seconds of the trauma, are usually generalized, and rarely reoccur.[12] Early PTS occurs in the first week, usually within 24 hours, and is secondary to an exacerbation of the insult, requiring prompt evaluation.[5,12] More commonly found in severe traumatic brain injury, dural lacerations, and intracranial hemorrhages, late PTS (more than 1 week from trauma) is attributed to scarring, vascular compromise, distortion, and mechanical irritation of the brain. Recurrent seizures are most likely in late PTS.[12] Studies have shown that the use of prophylactic antiepileptic therapy is not warranted in minor head injury.[5]

A recently published study suggests that children with isolated minor head injuries and simple posttraumatic seizures who recover fully in the emergency room; whose CT scans show no intracranial abnormalities; and who have no prior history of neurological disease, epilepsy or anticonvulsant use, are at low risk for recurrent seizures or neurological complications. They potentially could be sent home to a reliable caretaker and a stable home situation.[34] However, because of the limited sample size in this study, the statistical risk of a bad outcome may be as high as 9%. Therefore, much larger studies are needed before this becomes a standard policy.

Initial Evaluation

Identifying Mild Head Injury. The challenge to all health care professionals who evaluate patients with acute head trauma is to properly identify the severity and type of head injury. Although the term “minor head injury” is used commonly, it can be difficult and misleading to define. Traditionally, a previously neurological-

Table 3. Historic and Clinical Features Suggestive of Moderate to Severe Head Injury

HISTORIC FACTORS	CLINICAL FACTORS
Bleeding diatheses	Altered mental status/ hemodynamic status
Neurologic disease	Abnormal behavior
Substance abuse	Hypertension/hypotension
TINS of 2 or more	Evidence of hypoxemia
	Skull fracture
	GCS < 15
	+ focal neurologic/physical findings
	Multiple trauma

Adapted from: Woestman R, Perkin R, Serna T, et al. Mild head injury in children: Indentification, clinical evaluation, neuroimaging, and disposition. *J Pediatr Health Care* 1998;12:288-298.

TINS = Trauma Infant Neurologic Score *(See Table 1.)*
GCS = Glasgow Coma Score *(See Table 4.)*

ly healthy child who suffers an acute injury resulting in a Glasgow Coma Scale (GCS) score of 13-15, has no abnormal or focal neurologic (including fundoscopic) deficit, and no evidence of skull fracture (hemotympanum, Battle's sign, raccoon eyes, or palpable bone depression) was diagnosed with a minor head injury.[35] Some believe if any of the criteria in Table 3 apply, the patient must be evaluated for moderate to severe head injury.[6]

The GCS and, for younger children, the modified GCS or the children's coma scale are most often used to quantify the neurologic status.[36-38] *(See Table 4.)* The GCS measures three parameters: 1) the best eye opening; 2) the best verbal responses; and 3) the best motor response. The best eye opening and verbal responses are self-explanatory. Tests for motor responses should be performed and interpreted carefully. If painful stimuli are administered, they should be in the cutaneous distribution of a cranial nerve to elicit a central (brainstem) response; compressing the supraorbital nerve as it crosses the brow or pinching the earlobe are appropriate stimuli that can elicit strong central responses. Pinching or squeezing the limbs or torso are inappropriate stimuli because they may elicit spinal reflexes that may mimic a central response, even in a patient who is otherwise brain dead.

The GCS requires an adult level of development. Modifications of the GCS or other coma scales have been introduced and found to be valid for assessing children whose neurodevelopmental function is below 10 years.[37,38]

GCS scores range from 3 to 15 points. Patients with GCS scores of 13-15 are considered to have mild head injuries, patients with scores of 9-12 have moderate head injuries, and those with scores of 3-8 have severe head injuries. Monitoring intracranial pressure (ICP) is generally reserved for those patients with GCS scores less than 8. An even more important consideration is how the GCS score is changing with time. A worsening level of consciousness suggests rising ICP or an expanding intracranial mass lesion, whereas a lethargic or stuporous child whose condition is improving rapidly often has the postconcussive syndrome of childhood.[6] The GCS score should, therefore, be recorded in the initial medical record and should be repeated at regular intervals.

Available evidence indicates the risk of structural brain injury is inversely correlated with GCS in mild head injured patients. The reported incidence of cranial CT-documented structural brain injury in pediatric mild head injured patients with an initial GCS score of 13 or 14 ranges from 14% to 33%, with up to 12% requiring subsequent neurosurgical intervention.[5] Accordingly, pediatric mild head injured patients with an initial GCS of 13 or 14 warrant immediate neuroimaging.[5,6]

In addition to the GCS score, focal neurological deficits may suggest localized brain injury or a focal mass lesion. Both the pattern and temporal profile of neurological deficits may localize the injury or suggest a cerebral herniation syndrome. For example, a unilaterally dilated pupil (mydriasis) with an associated contralateral hemiparesis or motor posturing suggests transtentorial herniation, whereas paraparesis may reflect subfalcine herniation with compression of the anterior cerebral arteries.

Among children with minor closed head injury, LOC is uncommon, but is associated with an increased risk for intracranial injury.[35] There is no evidence that the sole criteria of length of LOC will suggest a difference in outcome.[35,39]

Studies performed since the advent of CT scanning suggest that children with LOC, or who demonstrate amnesia at the time of evaluation, or who have headache or vomiting at the time of evaluation, have a prevalance of intracranial injury detectable on CT that ranges from 0% to 7%.[5,21,35]

All patients with traumatic injury require a thorough primary and secondary survey. Initial mental status should be especially noted, with serial examinations. An alert patient who is talking may harbor an expanding intracranial hematoma and progress to coma within minutes.

Radiographic Evaluation. Patients with mild head trauma can be divided into two groups: those who can be sent home with little risk of adverse sequelae (the majority), and those who require medical and surgical observation and/or intervention. Many experts agree that these two groups cannot be distinguished reliably by clinical examination alone.[3,4,21,40] The last comment must be emphasized. Although some authors have suggested that clinical criteria such as a normal neurologic examination are sufficient in evaluating children, others have reported a high incidence (2.5-9%) of intracranial abnormalities, even with a normal neurologic examination. This leads to a general consensus that clinical criteria are insufficient to rule out intracranial injury.[4,21,40]

Skull Radiograph. Recent literature has emphasized the status of skull radiography as a poorer predictor of intracranial injury than neurologic examination.[41] The quintessential question to the evaluation of any head injury is whether there is intracranial injury. Intracranial injury cannot be ruled out if the radiograph is negative for fractures. Recent studies, limited to children, have evaluated the sensitivity and specificity of skull radiographs to show intracranial injury. The sensitivity was con-

cluded to be between 50% and 100%, with adolescents making up the majority of the higher figure.[35] The specificity (those with a normal radiograph and no intracranial injury) was reported as 53-97%.[35]

CT Scan. The use of CT scan has given the physician a necessary modality to evaluate a child who presents with vomiting, severe headache, lethargy, a history of possible LOC, or unwitnessed injury. By ruling out intracranial injury with CT scan, a child and parent may avoid the anxiety, inconvenience, and cost of a hospital admission, although close observation for the first 24-48 hours is still paramount.[5] The early detection CT scan allows for prompt medical and surgical intervention, significantly reducing morbidity and mortality.

A head CT does not identify all skull fractures, but associated intracranial injuries are visualized. Although CT scanning itself is safe, sedation may be required, and the risks should be considered. In addition, cost and resource allocations can be prohibitive.[35]

High-energy forces associated with impact or violent shaking result in a variety of central nervous system injuries that can be detected by modern neuroimaging techniques.[42] The evolution of these injuries, as well as processes developing secondary to the original insult, often are effectively displayed on serial imaging studies.

The CT without intravenous contrast should be performed as part of the initial evaluation for suspected inflicted head injury. It has a high sensitivity and specificity for diagnosing acute intraparenchymal, subarachnoid, subdural, and epidural hemorrhage.[42] Abnormalities that require emergency surgical intervention generally are well-demonstrated.

Among children with a normal cranial CT scan after mild head injury, delayed intracranial sequelae requiring intervention are extremely uncommon.[5,43-45] In otherwise stable patients, a normal cranial CT scan can identify patients who may be safely discharged from the ED.

As discussed earlier, even with an excellent history and physical examination, children with intracranial injury are difficult to distinguish from those without. This is especially true for infants.[46,47] For this reason, most authors are hesitant to make recommendations about which children do not need CT imaging after head injury. In very general terms, children with a normal neurologic exam, GCS of 15, a fall of fewer than 3 feet, no history of neurologic symptoms, and no scalp abnormalities are unlikely to have intracranial injuries and may not require CT imaging—particularly if the child is older than 12 months.[47]

Magnetic Resonance Imaging (MRI). CT scanning usually is chosen over MRI due to speed, convenience, and cost. CT scanning also is more sensitive in detecting acute intracranial hemorrhage, especially subarachnoid hemorrhage.[42] However, MRI is advantageous in detecting and characterizing small extra-axial hemorrhages in infants with equivocal CT findings.[29,48]

The MRI is the best modality to fully assess intracranial injury, including extra-axial collections, intraparenchymal hemorrhages, contusions, shear injuries, brain swelling, or edema.[42] Imaging should be performed with T1 and T2 weighting with proton-density or inversion-recovery sequences to differentiate CSF collections from other water-containing lesions. Gradient echo sequences should be included to detect hemorrhage or mineralization not demonstrable by other MRI techniques. Although the specific type and order of pulse sequences may vary, imaging must be performed at least in the axial and coronal planes. Because MRI may fail to detect acute subarachnoid or subdural hemorrhage, its use should be delayed in acutely ill children.[42] Abused infants may not demonstrate neurologic signs and symptoms, despite significant central nervous system injury. Multiplanar MRI is probably the most important technique for assessing the pattern, extent, and timing of the injury, particularly in the absence of findings on CT scan.[48] In these cases, the MRI should be done as soon after the presentation as the child's condition allows. MRI also offers the highest sensitivity and specificity for diagnosing subacute and chronic injury and should be considered whenever typical skeletal injuries associated with shaking or impact injuries are identified.[42,48]

Infants Younger than 2 Years

The most difficult pediatric population in which to decide a course of therapy is infants younger than 2 years. Numerous

Table 4. Glasgow Coma Scale (GCS) and Modified GCS

Glasgow Coma Scale		Modified GCS for children younger than 2 years of age	
SIGN	SCORE	SIGN	SCORE
EYE OPENING		EYE OPENING	
Spontaneous	4	Spontaneous	4
To verbal command	3	To speech	3
To pain	2	To pain	2
No response	1	No response	1
BEST MOTOR RESPONSE		BEST MOTOR RESPONSE	
Obeys verbal commands	6	Normal spontaneous movements	6
Localizes pain	5	Withdraws to touch	5
Withdraws to pain	4	Withdraws to pain	4
Flexion response to pain	3	Flexion response to pain	3
Extension response to pain	2	Extension response to pain	2
No response	1	No response	1
BEST VERBAL RESPONSE		BEST VERBAL RESPONSE	
Oriented	5	Smiles, listens, follows objects	5
Confused	4	Irritable cry, consolable	4
Inappropriate words	3	Inappropriate, persistent cry	3
Nonspecific sounds	2	Agitated, restless	2
No response	1	No response	1

studies have found clinical symptoms and signs of brain injury to be insensitive indicators of intracranial injury in infants.[3,8,9,46,47] These studies indicate that up to 48% of infants with intracranial injury are asymptomatic.[8] Parents frequently delay seeking medical attention for their infants due to underestimating an apparently minor head injury (e.g., a fall less than 1 meter).[5] The infant initially appears in good neurologic condition but can quickly or insidiously deteriorate. Infants also are more likely than older children and adults to have a lag time between ED admission and surgical therapy because infants cannot complain, and the various coma scales may be difficult to interpret.[6] As previously stated, the TINS was developed to aid in determining evaluation guidelines and prognosis.[8] An infant with a TINS of 2 or higher should have a CT scan.

Previous studies have indicated liberal use of radiographic imaging when evaluating head-injured infants. Masters et al recommended that age younger than 2 years in itself be considered a moderate risk factor for intracranial injury after head trauma.[49]

Data indicate that even among children in the first two years of life, younger age is a somewhat useful criterion for distinguishing patients at high risk for intracranial injury. Patients in the first three months of life appear to be at especially high risk of intracranial injury.[8,9] Clinicians should have a low threshold for radiographic imaging in head-injured infants younger than 3 months of age.

Data also show a relationship between mechanism of injury and likelihood of intracranial injury, with direct falls from heights greater than 3 feet and stair falls being more likely than falls of fewer than 3 feet to result in intracranial injury.[9,47]

As previously mentioned, the presence of a scalp hematoma is probably the most useful clinical indicator of intracranial injury in children younger than 2 years of age.[8,9,46,47] Asymptomatic infants older than 3 months of age who do not have significant scalp hematomas may be managed safely without radiographic imaging.[9]

The Special Case of Child Abuse

Inflicted head injury is the most common cause of traumatic death in infancy. Accidental blunt head injury in children younger than 2 years of age is very uncommon.[13,14] On average, among children hospitalized for blunt trauma, those injured by abuse sustain more severe injuries, use more medical services, and have worse survival and functional outcome than children with unintentional injuries.[26,50,51] A retrospective study showed that in infants younger than 1 year of age, 64% of head injuries and 95% of severe intracranial injuries were the results of child abuse.[4] The mechanisms and patterns of injury differ between accidental and nonaccidental head injury and may help distinguish one from the other.[6] The location of bruises on the body also can help in discerning accidental from inflicted injuries.[52,53]

Few injuries appear to result when children fall from low heights, such as from beds or couches or down stairs.[18,54-58] However, some of the most severe infant head injuries occur when an adult falls on the stairs while carrying an infant. The relative severity of these injuries is probably the result of two different injury mechanisms: fall from a height and fall down stairs. Two studies showed a 46% occurrence of skull fracture in infants who fell with their caregivers while being carried down stairs.[57,58] After a free fall, the severity of injury is directly proportional to the vertical distance involved in the fall.[58] Most falls from a short distance produce mainly a translational (linear) force to the head, which may cause simple fractures but is only of clinical significance when an epidural hematoma is produced.[18,59]

What should be clear to health care professionals is that falls from short heights do not produce multiple or visceral injuries, and that clinically significant head injury is uncommon. It follows that when children who are said to have had minor falls are found to have multiple, severe, or life-threatening injuries, the reliability of the history should be seriously questioned.[59]

The single most important element in the diagnosis of child abuse is the history.[13] The interview should be conducted in a supportive manner and without confrontation. The initial presentation is frequently in the ED, where the most untainted information may be collected from a remorseful caregiver. Thorough, complete, and accurate documentation, including direct quotes, is essential. If child abuse is suspected, a standard work-up, including head CT scan, should be performed, as well as any specific studies pertaining to the history and physical exam.

A complete physical examination is equally as important as a careful history of the cause of trauma. Often, the severity of the injury is not proportionate to the description of the "accident." Skull fractures are the second most frequently encountered fracture in the abused child; long bone shaft fractures are the most common fractures in child abuse.[13] As was stated previously, linear parietal skull fractures are most likely accidental. Head injury in infants commonly presents as shaken baby syndrome.[20] Altered level of consciousness, coma, seizure, listlessness/lethargy, irritability, respiratory difficulty, and poor feeding can be associated with shaken baby syndrome.[13,14] Although not pathognomonic for child abuse, retinal hemorrhages occur in approximately 80% of these cases and frequently correspond with subdural or subarachnoid bleeding.[13,23] Skeletal trauma, such as sternal fractures and posterior rib fractures, frequently are seen in shaken baby syndrome due to the placement of the perpetrator's hands.[13] Acceleration/deceleration forces are created as the head whips forward, stopping as the chin strikes the chest, then backward until the occiput strikes the back, as well as when the baby is thrown on a sofa or bed.[14] Shearing injuries to the intracranial bridging veins and incompletely myelinated cortical nerves are sustained.[14,23] CT scanning is a mainstay of the diagnosis of nonaccidental head injury by detecting subdural and subarachnoid hemorrhages, especially when associated with retinal hemorrhages. Laboratory studies may show bloody CSF, low serum hemoglobin, and elevated coagulation studies.[14] If abuse is suspected, taking measures to prevent further abuse from occurring is vital.

Sports-Related Head Injuries

Sports should contribute to the physical and mental health of children. Unfortunately, head injuries occur in sports and can cause permanent, irreversible damage to the central nervous sys-

tem.[60] As many as 20% of pediatric head injuries are sports-related, with 10% of those children requiring hospitalization.[61] It is estimated that 20% of high school football players and 40% of college football players will sustain a head injury at some point in their careers. Because of their lifestyle, children who sustain a sports-related head injury are 2-4 times more likely to have a similar repeated injury.[59]

Most acute head injuries are caused by a combination of rotational (angular) and translational (linear) acceleration, as well as the force of impact to the head. Rotational acceleration of the brain (caused by rapid turning of the head) will result in diffuse shearing forces deep in the brain causing axonal injury (concussions). Translational acceleration will cause tensile (pulling apart) and compressive forces, which usually result in focal brain injury. Impact forces will exert stress at the point of contact, resulting in skull fractures and injury to the adjacent structures (blood vessels, brain parenchyma, dura). Brain acceleration can occur without impact to the head; 35% of concussions in college football involved no impact to the head.[60] Because adolescents are larger, faster, stronger, and more skilled at delivering a forceful impact than their younger counterparts, the incidence of head injury is higher in adolescents.

Acute brain injuries can be divided into two categories: 1) focal brain injuries, including epidural and subdural hematomas as well as cerebral contusions; and 2) diffuse axonal damage, ranging from concussions to diffuse axonal injuries.[60] Recognition of focal brain injuries is crucial because they are potentially the most life threatening and require emergency transport and neurosurgical monitoring. The most common focal brain injury and etiology of sports-related fatality (87% of all head-related football fatalities) is the subdural hematoma.[60]

Diffuse axonal damage is more common than focal injury and represents a spectrum of injuries from momentary confusion to a prolonged coma.

The most common sports-related head injury is the concussion, the syndrome characterized by immediate and transient impairment of neural function due to mechanical force. A concussion can occur even if there is no LOC. The most common signs and symptoms include confusion, memory loss (antegrade and retrograde), decreased level of consciousness, headache, slowed information processing, diminished attention span, and decreased coordination immediately or within minutes of the head injury.

Table 5. Diagnostic Grading Scales for Sports-Related Concussion

	Severity of Grade		
GUIDELINE	1	2	3
Practice Parameter American Academy of Pediatrics[35]	No LOC, amnesia, or confusion	No LOC Confusion and amnesia >15 min	Any LOC
Cantu[65] guidelines	No LOC Amnesia < 30 min.	LOC < 5 min **OR** Amnesia > 30 min.	LOC > 5 min **OR** Amnesia > 24 h
Colorado[66] Medical Society	No LOC Confusion *without* amnesia	No LOC Confusion *with* amnesia	Any LOC
Practice Parameter American Academy of Neurology[67]	No LOC Mental status changes < 15 min	No LOC Mental status changes >15 min.	Any LOC

LOC = Loss of consciousness

Adapted from: Collins MN, Lovell MR, McKeag DB. Current issues in managing sports-related concussion. *JAMA* 1999;282:2283-2285; and American Academy of Pediatrics Section of Radiology. Diagnostic imaging of child abuse. *Pediatrics* 2000;105:1345-1348.

Repeat head injury is of special concern because the effects of repeated concussion are now being recognized as cumulative in nature.[39,60,62-64] Second impact syndrome (SIS) is when a second concussive injury occurs prior to the resolution of symptoms from the first head injury.[39,60] The mechanism of SIS is believed to be the loss of vascular autoregulation causing an increase in ICP, thus leading to herniation.[39] At least 17 deaths have been associated with SIS.[39] Cognitive impairment and diminished neuropsychological functioning can result from repeated concussions.

Many guidelines have been recommended in an attempt to provide a uniform evaluation and recommendation on how long an athlete must wait before continuation of the sport, and at what level of injury the athlete should be excluded from competition.[39,60,65-68] Table 5 shows the most common diagnostic grading scales for sports-related head injuries. Once the grade of the concussion is decided, choosing the level of precaution before returning the athlete to play can be variable, as seen in Table 6. Since current concussion management guidelines are not evidence-based, categorizing the severity of the injury and appropriate management can prove difficult.[39]

Management Decisions

The management of a patient with minor head injury can be a challenge, since a full spectrum of complications may occur. Using an evidence-based approach, the American Academy of Pediatrics (AAP) and its Committee on Quality Improvement in collaboration with the American Academy of Family Physicians (AAFP) and its Commission on Clinical Policies and Research performed an extensive review of the literature to provide practice parameters for children with minor closed head injuries.[35]

Table 6. Time Recommended to Rest Before Resuming Sport after First Concussion[39,42]

GUIDELINE	Severity of Grade 1	2	3
Practice Parameter American Academy of Pediatrics[35]	20 min	1 week	1 month
Cantu[65] guidelines	1 day	2 weeks	1 month
Colorado[66] Medical Society	20 min	1 week	2 weeks Initially evaluated by a physician
Practice Parameter American Academy of Neurology[67]	15 min	1 week	1 week if LOC was brief 2 weeks if LOC was prolonged Initially hospitalize

LOC = Loss of consciousness

Adapted from: Collins MN, Lovell MR, McKeag DB. Current issues in managing sports-related concussion. *JAMA* 1999;282:2283-2285; and American Academy of Pediatrics Section of Radiology. Diagnostic imaging of child abuse. *Pediatrics* 2000;105:1345-1348.

Factors that must be considered include: 1) how frequently intracranial injury occurs with a mechanism of injury; 2) the need for medical and/or surgical intervention; and 3) the detrimental factors to outcome if delay to diagnosis or intervention would occur. There are no data showing a difference in outcome between immediate neuroimaging and careful observation in healthy children presenting with no neurological deficit from acute minor head injury.[35] In children with minor closed head injury and brief LOC there is up to a 7% chance of CT scan detecting an intracranial injury. Of those with intracranial injury, an estimated 2-5% may require neurosurgical intervention.[35]

The majority of head injuries in children are mild and without risk for serious sequelae. However, for children with more serious head injuries, or patients with a high-risk history (such as hematologic or oncologic disease) prompt diagnosis and treatment is the key to meaningful survival. Use of age-appropriate trauma scores in a child older than 2 years, with mild head injury, and especially an accurate history and physical examination, can be beneficial in determining the need for CT scans. *(See Figure.)* Thus, it is recommended that any child presenting with a history of altered level of consciousness (i.e., GCS < 15, LOC, lethargy, confusion); physical abnormalities (pupillary abnormalities, bradycardia, ; or symptoms such as vomiting, headache, or amnesia should receive a CT scan to evaluate for intracranial injury.

When considering a skull radiograph, remember that a CT scan will offer more necessary information. With the increasing prevalence of injuries inflicted on children, the ED physician should have a greater suspicion of nonaccidental trauma when the reported mechanism of injury and physical findings of a child with a head injury do not correlate.

Inpatient observation is required for a child with abnormal findings on a CT scan. It has been demonstrated that a normal CT scan successfully identifies children who can be safely discharged home to a reliable observer and that these children remain stable once discharged.[43-45] Discharge instructions should include close observation by a competent caregiver with appropriate instruction. Close observation without CT scanning is a possible option in minor closed head injury, especially in minor closed head injuries without LOC. *(See Figure.)*

Infants younger than 2 years present an especially difficult challenge to diagnosis. Infants may present with minimal history (fell from less than 1 m, GCS 15) and minor physical signs (subgaleal hematoma, yet no neurological deficits) and have intracranial injury. With early detection and prompt medical and surgical management, a patient with an intracranial lesion may have an excellent prognosis. Thus a head CT scan should be performed on any infant in whom the TINS is 2 or higher. Further research is required to create better clinical and prognostic tools for evaluation of infants.

Research on evaluating and caring for sports-related mild closed-head injury is ongoing, supporting the need for a physician to evaluate every athlete with a concussion on an individualized basis. Although LOC, even for seconds, has been an important indicator of concussion severity, other variables, such as vomiting, amnesia, difficulty concentrating, headache, or postural instability, are being shown to be critical in the evaluation and treatment of an athlete without LOC. Thus, no athlete should return to the game with any signs or symptoms of a concussion. Repeat assessments in the first 24 hours after the injury should be conducted to detect deterioration in cognitive function in which a higher level of care may be required urgently. The use of appropriate protective gear and the practice of safe technique in sports must be reinforced.

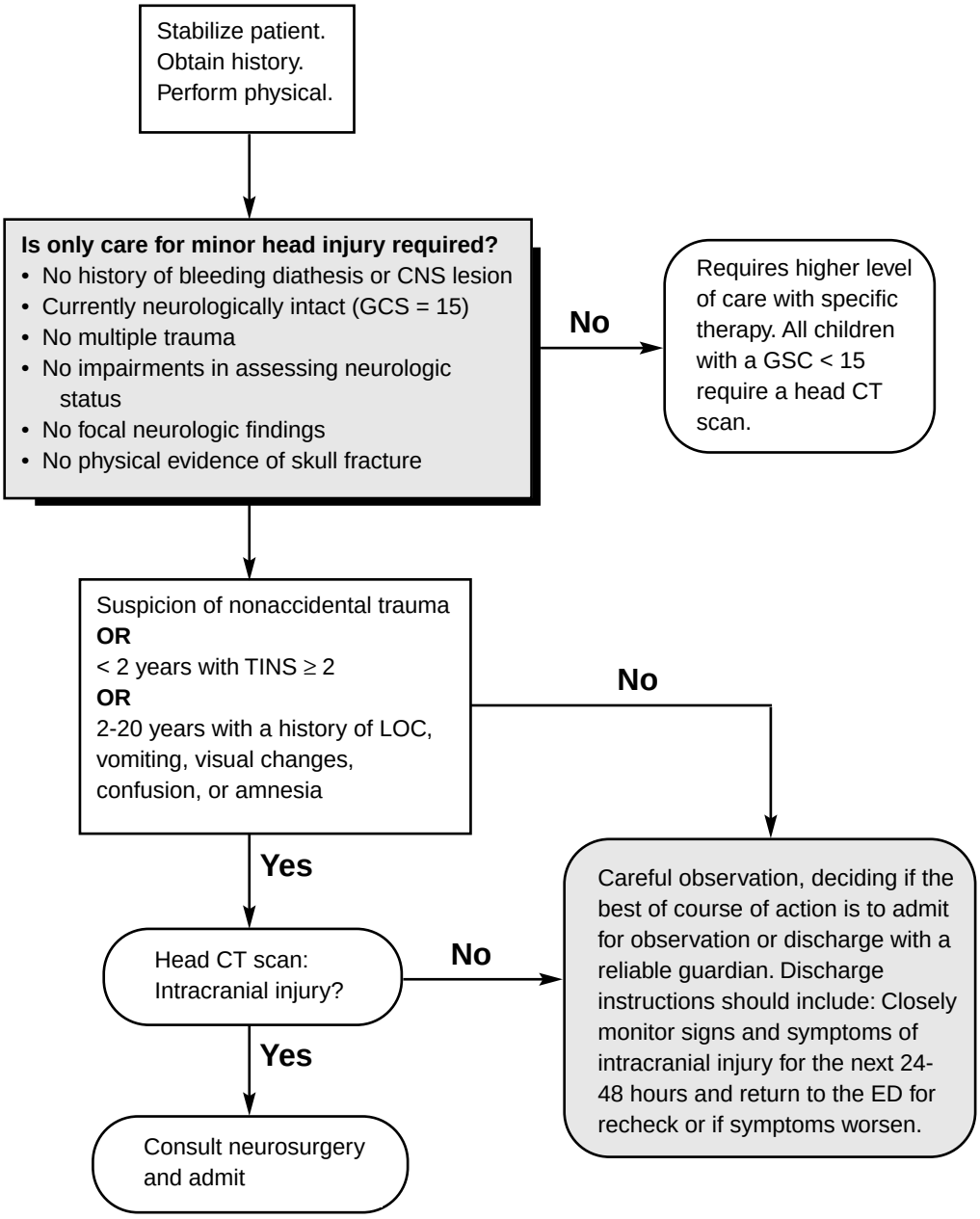
Conclusion

Although many questions regarding the management of pediatric mild head injury remain, available evidence supports certain management recommendations. All mild head injury patients with an abnormal mental status, focal neurological deficits, or a GCS score of 13 or 14 warrant emergent neuroimaging. Patients with a GCS score of 15 and LOC have a 0-7% incidence of a lesion requiring neurosurgical intervention. These patients also deserve

strong consideration for neuroimaging. Mild head injury patients with a GCS score of 15, no LOC, and a normal mental status are generally at very low risk for complications. In these patients, neuroimaging can be reserved for cases with additional risk factors. One should lean toward scanning in children whose mechanism of injury involves high energy, young age, significant scalp hematoma or other evidence of surface impacts, or whose parents or caretakers think they are "not quite right."

Above all, even if there are no focal neurologic findings, think seriously about scanning or rescanning any child who seems to be getting worse. Neuroimaging criteria for children age 2 years and younger remain undefined. In the absence of intracranial injury on neuroimaging, antiepileptic prophylaxis in children with mild head injury is not warranted. Selective outpatient management of children with mild head injury can be done safely. Specifically, patients with a normal CT scan and a normal neurological examination can be managed as outpatients.

Figure. Evaluation and Treatment of Children with Minor Head Trauma



LOC = Loss of consciousness
GCS = Glasgow Coma Scale
TINS = Trauma Infant Neurologic Scale *(See Table 1.)*

References

1. Mandera M, Wencel T, Bazowski P, et al. How should we manage children after mild head injury? *Child's Nerv Syst* 2000;16:156-160.
2. Hahn YS, McLone DG. Risk factors in the outcome of children with minor head injury. *Pediatr Neurosurg* 1993;19:135-142.
3. Keskil IS, Baykaner MK, Ceviker N, et al. Assessment of mortality associated with mild head injury in the pediatric age group. *Child's Nerv Syst* 1995;11:467-473.
4. Quayle KS. Minor head injury in the pediatric patient. *Pediatr Clin North Am* 1999;46:1189-1199.
5. Savitsky EA, Votey SR. Current controversies in the management of minor pediatric head injuries. *Am J Emerg Med* 2000;18:96-101.
6. Woestman R, Perkin R, Serna T, et al. Mild head injury in children: Indentification, clinical evaluation, neuroimaging, and disposition. *J Pediatr Health Care* 1998;12:288-298.
7. Meyer P, Legros C, Orliaguet G. Critical care amnagement of neurotrauma in children: New trends and perspectives. *Child's Nerv Syst* 1999;15: 732-739.
8. Beni-Adani L, Flores I, Spektor S, et al. Epidural hematoma in infants: A different entity? *J Trauma* 1999;46:306-311.
9. Greenes DS, Schutzman SA. Clinical indicators of intracranial injury in head-injured infants. *Pediatrics* 1999;104:861-867.
10. Bernardete EA, Jallo GI. Closed head injury: Pathophysiology, diagnosis and management. *Trauma Quarterly* 1998;13:397-430.
11. Mittl RL, Grossman RI, Hichle JF, et al. Prevalence of MR evidence of diffuse

axonal injury in patients with mild traumatic brain injury and normal head CT findings. *Am J Neuroradiol* 1994;15:1583-1589.

12. Schutzman SA. Injury-Head. In: Fleisher GR, Ludwig SW, eds. *Textbook of Pediatric Emergency Medicine*. Philadelphia: Lippincott Williams & Wilkins; 2000:331-338.
13. Sheridan C, Mellick LB, Sherwin T. Recognizing child abuse and neglect: The role of the emergency physician. *Emerg Med Reports* 1993;14:67-74.
14. Jain AM. Emergency department evaluation of child abuse. *Emerg Med Clin North Am* 1999;17:575-593.
15. Stewart G, Meert K, Rosenburg N. Trauma in infants less than three months of age. *Pediatr Emerg Care* 1993;9:199-201.
16. Ward JD. Craniocerebral Injuries. In: Buntain W, ed. *Management of Pediatric Trauma.* Philadelphia: W.B. Saunders; 1995:177-187.
17. Koonsman M, Dunn E, Hughes K. et al. How much monitoring is needed for basilar skull fractures? *Am J Surg* 1992;164:487-490.
18. Duhaime AC, Alario AJ, Lewander WJ. et al. Head injury in very young children: Mechanisms, injury types and ophthalmologic findings in 100 hospitalized patients younger than 2 years of age. *Pediatrics* 1992;90:179-185.
19. Haseler LJ, Arcinue E, Danielsen ER, et al. Evidence from proton magnetic resonance spectroscopy for a metabolic cascade of neuronal damage in shaken baby syndrome. *Pediatrics* 1997;99:4-14.
20. Duhaime AC, Christian CW, Rorke LB, et al. Nonaccidental head injury in infants – the "shaken-baby syndrome." *N Engl J Med* 1998;338:1822-1829.
21. Dietrich AM, Bowman MJ, Ginn-Pease ME, et al. Pediatric head injuries: Can clinical factors reliably predict an abnormality on computed tomography? *Ann Emerg Med* 1993;22:1535-1540.
22. Shugerman RP, Paez A, Grossman DC, et al. Epidural hemorrhage: Is it abuse? *Pediatrics* 1996;97:664-668.
23. Morris MW, Smith S, Cressman J, et al. Evaluation of infants with subdural hematoma who lack external evidence of abuse. *Pediatrics* 2000;105: 549-553.
24. Schutzman SA, Barnes PD, Mantello M, et al. Epidural hematoma in children. *Ann Emerg Med* 1993;22:535-541.
25. Jayawant S, Rawlinson A, Gibbon F, et al. Subdural hemorrhages in infants: Population based study. *BMJ* 1998;317:1558-1561.
26. Ewing-Cobbs L, Kramer L, Prasad M, et al. Neuroimaging, physical and developmental findings after inflicted and noninflicted traumatic brain injury in young children. *Pediatrics* 1998;102:300-307.
27. Zumkeller M, Behrman R, Egmont-Heissler H, et al. Computed tomographic criteria and survival rate for patients with acute subdural hematoma. *Neurosurgery* 1996;39:708-712.
28. Dashti SR, Decker DD, Razzaq A, et al. Current patterns of inflicted head injury in children. *Pediatr Neurosurg* 1999;31:301-306.
29. Barlow KM, Gibson RJ, McPhillips M, et al. Magnetic resonance imaging in acute non-accidental head injury. *Acta Paediatr* 1999;88:734-740.
30. Young AER. Monitoring and management of the paediatric neurosurgical patient. *Curr Opin Anaesthesiol* 1999;12:517-521.
31. Dias MS, Borchers J, Hernan LJ, et al. Evaluation and management of pediatric head trauma. In: Fuhrman BP, Zimmerman JJ, eds. *Pediatric Critical Care.* St. Louis: Mosby-Year Book; 1998:1221–1248.
32. Perkin RM, Moynihan JA, McCleary M. Current concepts in the emergency management of severe traumatic brain injury in children. *Trauma Reports* 2000;1:1-16.
33. Edlow JA, Caplan LR. Avoiding pitfalls in the diagnosis of subarachnoid hemorrhage. *N Engl J Med* 2000;342:29-36.
34. Dias MS, Carnevale F, Li V. Immediate posttraumatic seizures: Is routine hospitalization necessary? *Pediatr Neurosurg* 1999;30:232-238.
35. American Academy of Pediatrics. The management of minor closed head injury in children. *Pediatrics* 1999;104:1407-1415.
36. Sternbach GL. The Glasgow Coma Scale. *J Emerg Med* 2000;19:67-71.
37. Gemke RJBJ, Tasker RL. Clinical assessment of acute coma in children. *Lancet* 1998;351:926-927.
38. Tatman A, Warren A, Williams A, et al. Development of a modified paediatric coma scale in intensive care clinical practice. *Arch Dis Child* 1997;77: 519-521.
39. Collins MN, Lovell MR, McKeag DB. Current issues in managing sports-related concussion. *JAMA* 1999;282:2283-2285.
40. Cheung DS, Kharasch M. Evaluation of the patient with closed head trauma: An evidence based approach. *Emerg Med Clin North Am* 1999;17:9-23.
41. Grasso SN, Keller MS. Diagnostic imaging in pediatric trauma. *Curr Opin Pediatr* 1998;10:299-302.
42. American Academy of Pediatrics Section of Radiology. Diagnostic imaging of child abuse. *Pediatrics* 2000;105:1345-1348.
43. Roddy SP, Cohn SM, Moller BA, et al. Minimal head trauma in children revisited: Is routine hospitalization required? *Pediatrics* 1998;101:575-577.
44. Davis RL, Hughes M, Gubler KD, et al. The use of cranial CT scans in the triage of pediatric patients with mild head injury. *Pediatrics* 1995;95: 345-349.
45. Schunk JE, Rodgerson JD, Woodward GA. The utility of head computed tomographic scanning in pediatric patients with normal neurologic examination in the emergency department. *Pediatr Emerg Care* 1996;12:160-165.
46. Greenes DS, Schulzman SA. Occult intracranial injury in infants. *Ann Emerg Med* 1998;32:680-686.
47. Gruskin KD, Schulzman SA. Head trauma in children younger than 2 years: Are there predictors for complications? *Arch Pediatr Adolesc Med* 1999; 153:15-20.
48. Barnes PD, Robson CD. CT findings in hyperacute nonaccidental brain injury. *Pediatr Radiol* 2000;30:74-81.
49. Masters S, McClean P, Arcarese J, et al. Skull radiograph examinations after head trauma: Recommendations by a multidisciplinary panel and validation study. *N Engl J Med* 1987;316:84-91.
50. DiScala C, Sege R, Li G, et al. Child abuse and unintentional injuries. *Arch Pediatr Adolesc Med* 2000;154:16-22.
51. Ewing-Cobbs L, Prasad M, Kramer L, et al. Acute neuroradiologic findings in young children with inflicted or noninflicted traumatic brain injury. *Child's Nerv Syst* 2000;16:25-34.
52. Carpenter RF. The prevalence and distribution of bruising in babies. *Arch Dis Child* 1999;80:363-366.
53. Sugar NF, Taylor JA, Feldman KW, et al. Bruises in infants and toddlers: Those who don't cruise rarely bruise. *Arch Pediatr Adolesc Med* 1999;153: 399-403.
54. Helfer RE, Sloris TL, Black M. Injuries resulting when small children fall out of bed. *Pediatrics* 1977;60:533-535.
55. Hymel KP, Bandak FA, Portington MD, et al. Abusive head trauma? A biomechanics-based approach. *Child Maltreat* 1998;3:116-128.
56. Reece RM, Sege R. Childhood head injuries: Accidental or inflicted? *Arch Pediatr Adolesc Med* 2000;154:11-15.
57. Chiavello CT, Christoph RA, Bond GR. Stairway related injuries in children. *Pediatrics* 1994;94:679-681.
58. Jofee M, Ludwig S. Stairway related injuries in children. *Pediatrics*

1988;82:457-461.
59. Lyons TJ, Oates RK. Falling out of bed: A relatively benign occurrence. *Pediatrics* 1993;92:125-127.
60. Chorley JN. Sports-related head injuries. *Curr Opin Pediatr* 1998;10: 350-355.
61. American Academy of Pediatrics, Section on Sports Medicine and Fitness. Guidelines for Pediatricians: Head injuries. February 2000.
62. Perriello VA, Barth JT. Sports concussions: Coming to the right conclusions. *Contemporary Pediatrics* 2000;17:132-140.
63. Bernhardt DT. Football: A case-based approach to mild traumatic brain injury. *Pediatric Annals* 2000;29:172-176.
64. Genuardi FJ, King WD. Inappropriate discharge instructions for youth athletes hospitalized for concussion. *Pediatrics* 1995;95: 216-218.
65. Cantu RC. Guidelines for return to contact sports after a cerebral concussion. *Physician Sportsmed* 1986;14:75-83.
66. Colorado Medical Society. Report of the Sports Medicine Committee: Guidelines for the management of concussion in sports. Denver: Colorado Medical Society; 1991.
67. American Academy of Neurology, Quality Standards Subcommittee. The management of concussion in sports practice parameter. *Neurology* 1997;48:581-585.
68. Roos R. Guidelines for managing concussion in sports. *Physician Sportsmed* 1996;24:67-74.

Physician CME Questions

To earn CME credit for this issue of Trauma Reports, *please refer to the enclosed Scantron form for directions on taking the test and submitting your answers.*

1. Which one of the following statements concerning mild head injury in children is true?
   A. Skull x-rays are essential in making a decision for most pediatric patients with mild head injury.
   B. Children who are alert and have a normal neurological exam are at no risk of having an intracranial mass lesion.
   C. Among children with mild head injury, loss of consciousness is uncommon but is associated with an increased risk for intracranial injury.
   D. CT scanning is mandatory in all patients arriving to the ED with mild head injury.

2. Which of the following characteristics of a skull fracture is more indicative of accidental trauma than non-accidental trauma?
   A. Bilateral
   B. Linear
   C. Crossing suture lines
   D. Non-parietal bone location
   E. Comminuted

3. Which one of the following patients is safe to discharge home from the ED?
   A. A football player with chief complaints of confusion and vomiting lasting 10 minutes and a normal head CT scan
   B. A 2-year-old with bruises to the thighs, trunk, and ear and a depressed skull fracture
   C. A lethargic toddler with a normal head CT scan
   D. A healthy child with an unreliable guardian
   E. An alert and active child with a small epidural hematoma

4. What is the Traumatic Infant Neurologic Score (TINS) used for?
   A. Evaluating if sexual abuse occurred
   B. Evaluating if skull roentgenography is required
   C. Predicting long-term outcome of an infant after trauma
   D. Scoring the developmental age of an infant due to trauma
   E. Evaluating if head CT scanning is indicated

5. Which one of the following statements concerning nonaccidental (inflicted) head injury is true?
   A. External evidence (i.e., bruising) of trauma is found always.
   B. Nonaccidental head injury is difficult to diagnose because symptoms are often nonspecific.
   C. Subdural hematoma is rarely, if ever, seen in children with nonaccidental head trauma.
   D. Epidural hematoma is pathognomonic of child abuse.

6. Which one of the following signs or symptoms should raise suspicion of nonaccidental (inflicted) trauma?
   A. Acute subdural hematoma in a child who reportedly fell out of bed.
   B. Bruising on the forehead of a 14-month-old infant who has just started walking.
   C. Linear skull fracture in a child who fell down stairs.
   D. Basilar skull fracture in a child who fell from a second floor balcony.

7. While serving as the team physician for your local high school football team, you witness a player knocked unconscious for at least 10 minutes. This type of concussion is graded as:
   A. Grade 1.
   B. Grade 2.
   C. Grade 3.
   D. Grade 4.

8 The player in Question 7 comes to you on follow-up and requests to return to full participation. How long after this injury should you make him wait, according to AAP guidelines?
   A. One week
   B. Two weeks
   C. Three weeks
   D. One month

In Future Issues: Abdominal Trauma