Chapter 138: Head Injury in Infants and Children

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INTRODUCTION AND EPIDEMIOLOGY

Minor head injury in children is responsible for almost 400,000 ED visits each year,[1] with children 0 to 4 years of age most commonly affected. Of all children with minor head injury coming to the ED, it is estimated that about 5% have intracranial injury,[2] and <1% of those with intracranial injury require neurosurgical intervention.[3,4]

Given the rarity with which head-injured children require intervention, the diagnostic challenge is to distinguish the small subset of seriously injured children, while minimizing evaluation of those at low risk of significant intracranial injury. Therefore, careful risk stratification of children with minor head injury is important for safe and efficient medical care.

This chapter focuses on evaluation of children with minor head injury and concussion, as well as the treatment of children in whom significant intracranial injuries are identified.

DEFINITION

The definition of minor head injury varies in the literature. **The American Academy of Pediatrics defines children with minor head injury as "those who have normal mental status at the initial examination, who have no abnormal or focal findings on neurologic (including funduscopic) examination, and who have no physical evidence of skull fracture."**[5] The Glasgow Coma Scale (GCS; Table 138-1), or its derivative for younger, preverbal infants and toddlers, is often used to determine the severity of head injury. **Head injuries resulting in a GCS score of ≤8 are severe, those with scores of 9 to 13 are moderate, and those with scores of 14 or 15 are mild.**

TABLE 138-1

Glasgow Coma Scale Score for Adults and Infants

Response	Adults	Infants	Score
Eye opening	Spontaneous To voice To pain No response	Spontaneous To voice To pain No response	4 3 2 1
Verbal response	Oriented Disoriented Inappropriate words Incomprehensible No response	Coos, babbles Irritable Cries to pain Moans to pain No response	5 4 3 2 1
Motor response	Obeys commands Localizes pain Withdraws to pain Decorticate posture Decerebrate posture No response	Makes normal spontaneous movements Withdraws to touch Withdraws to pain Decorticate posture Decerebrate posture No response	6 5 4 3 2 1

PATHOPHYSIOLOGY

Head trauma is classified as blunt or penetrating based on the mechanism of injury. In children, the vast majority of head trauma is caused by blunt force with the underlying mechanism varying according to the age of the patient. In younger children, the most common causes of head trauma are falls and assaults/child abuse.[6] In fact, in children under 2 years of age, nonaccidental trauma is the leading cause of death due to

head trauma. In older children, falls, sports and recreation, assault, and, increasingly, motor vehicle collisions are more common. Penetrating injuries are most frequently related to dog bites in infants or gunshot wounds in older children. In the largest study of pediatric minor head injury to date, including over 42,000 patients, radiographically documented intracranial injuries were found in approximately 1.8% of children with minor head injury who presented to the ED for evaluation, and only 0.9% required intervention.[4] This is a lower number than cited in prior studies, likely due to the inclusion and clinical follow-up of lower-risk children not undergoing CT. The true incidence of intracranial injuries is probably even lower, because many children with mild injuries do not present to the ED.

The pattern of injury is different in children compared to adults. In children, diffuse injuries are proportionally more common, and in adults, focal injuries such as epidural and subdural hematomas and cerebral contusions are more common.[6]

The differences in brain development and pediatric anatomy explain the specific pattern of childhood injuries compared to adults. In blunt head injury, rotation of the brain around its center of gravity leads to more diffuse injuries (diffuse axonal injury and subdural hematoma), whereas linear forces are generally less damaging to the brain and cause local (coup and contrecoup injury) rather than diffuse injury. The type and severity of the injury are determined both by the type of deceleration and its magnitude.[7] Younger age is a significant risk factor for intracranial injury. Even among infants, the greatest risk for intracranial injury is in children under 3 months of age.[2]

More details of physiology are discussed in chapter 257, "Head Trauma."

SKULL FRACTURES

Skull fractures can involve the calvarium or the base of the skull. The presence of skull fractures is a risk factor for underlying brain injury in infants under 2 years of age (**Figures 138-1 and 138-2**).[4]

FIGURE 138-1.
Linear fracture seen on CT. *Arrow* indicates skull fracture, and *asterisks* indicate normal cranial suture lines. [Image used with permission of Joseph Piatt, Jr., MD, Division of Neurosurgery, A. I. duPont Hospital for Children, Wilmington, Delaware; Departments of Neurological Surgery and Pediatrics, Thomas Jefferson University, Philadelphia, Pennsylvania.]

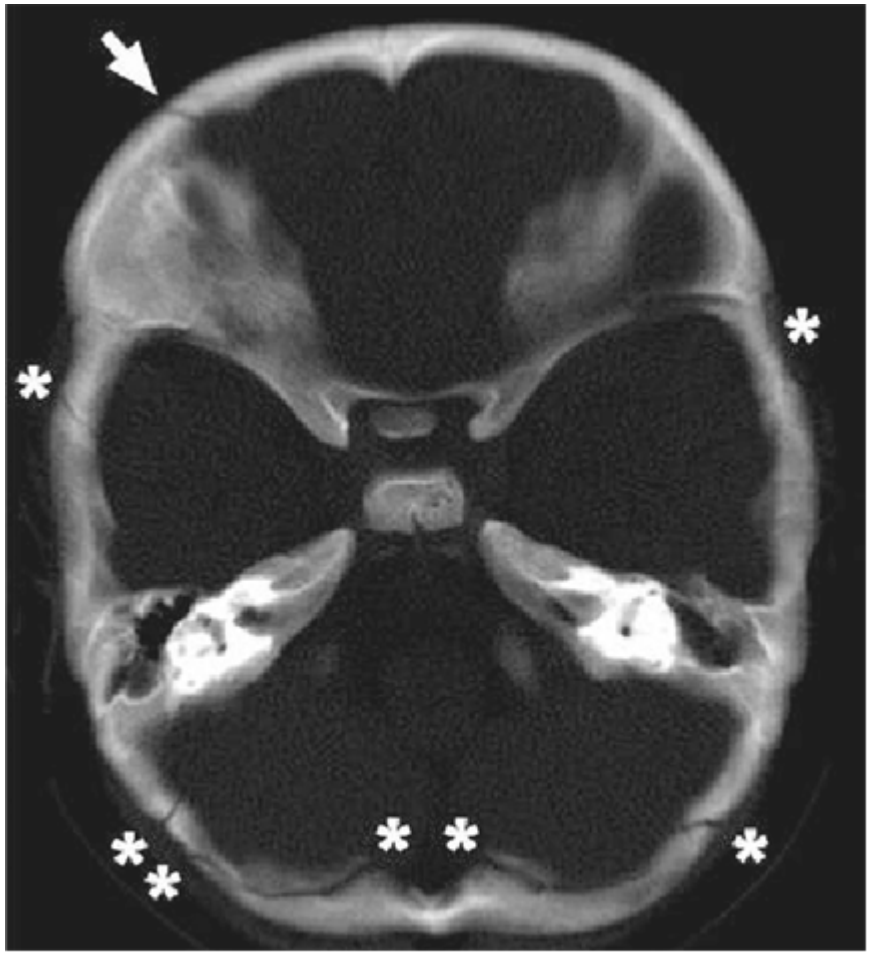
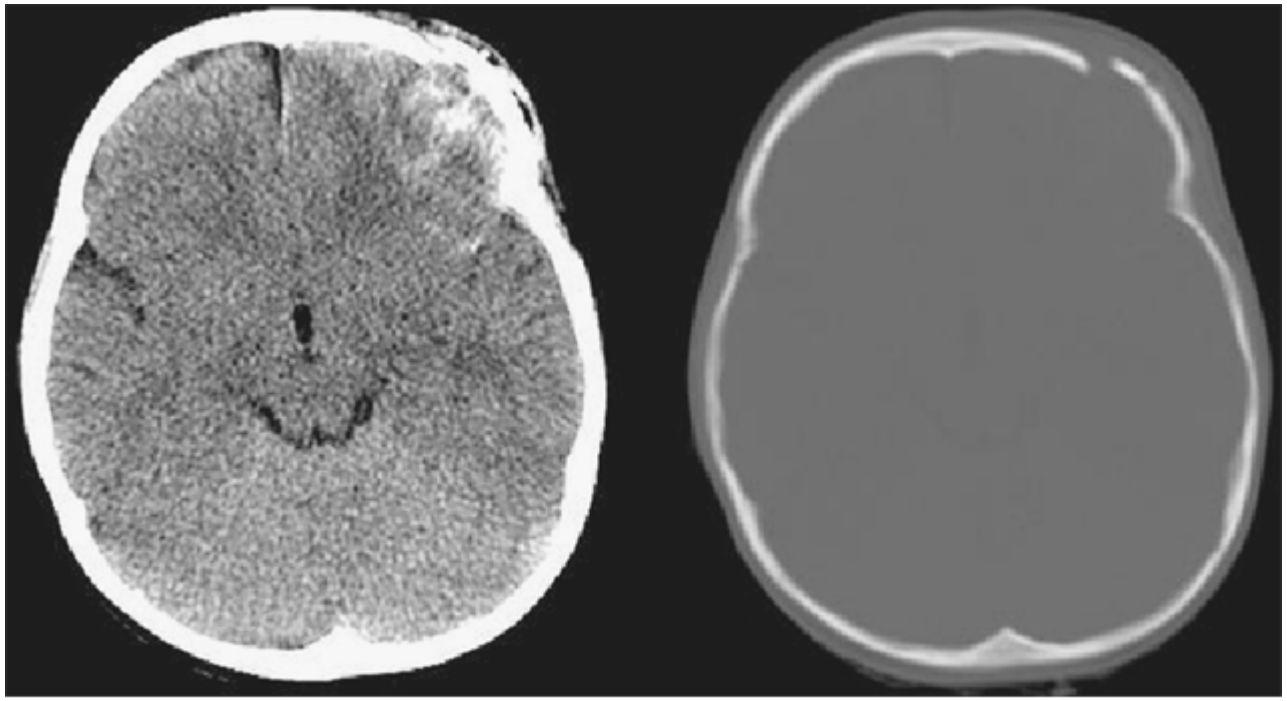


FIGURE 138-2.

Open skull fracture with underlying cerebral contusion. This injury was sustained from a fall of two stories. [Image used with permission of Joseph Piatt, Jr., MD, Division of Neurosurgery, A. I. duPont Hospital for Children, Wilmington, Delaware; Departments of Neurological Surgery and Pediatrics, Thomas Jefferson University, Philadelphia, Pennsylvania.]



However, significant brain injury can occur in the absence of skull fractures in 50% of cases.[8] Plain films of the skull should therefore not be obtained as a replacement for CT to evaluate for underlying brain injury.[9] Depressed skull fractures occur in response to the application of significant force and require neurosurgical consultation, as surgical elevation is often required. Compared to adults, skull fractures in children are more common but less frequently associated with underlying brain injury.[6]

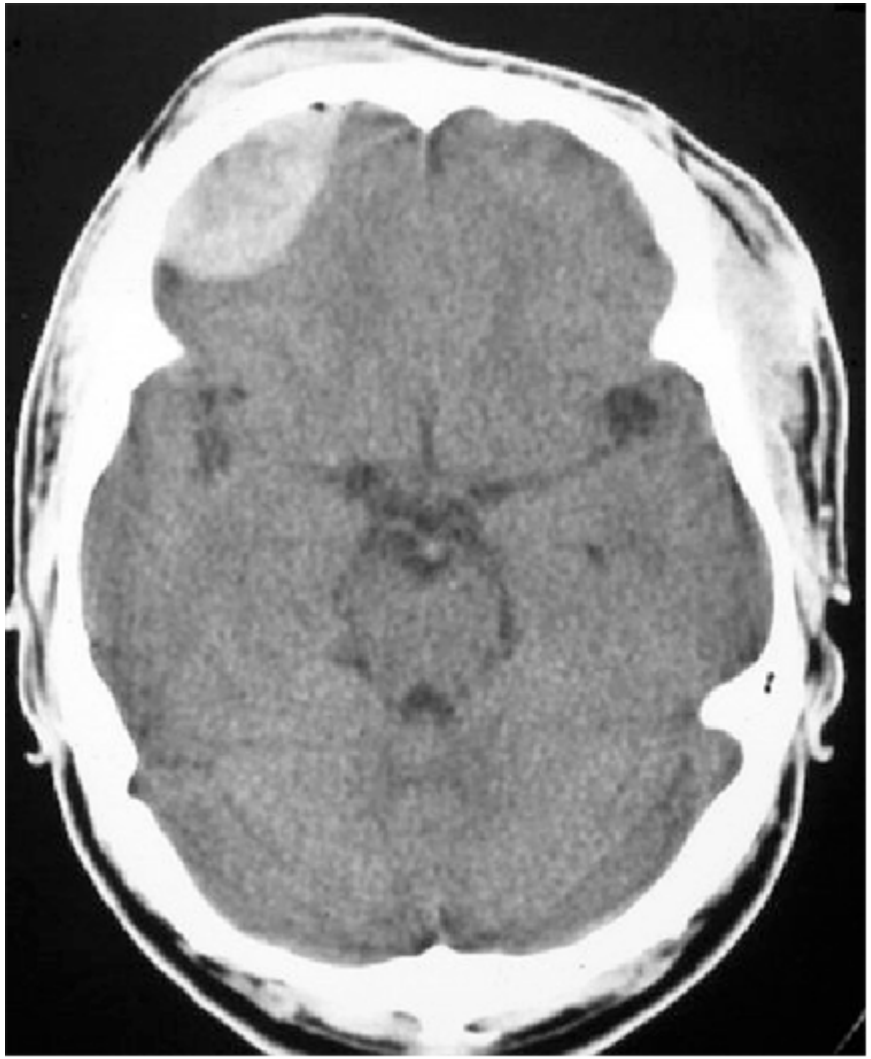
A growing fracture can occur when the leptomeninges are torn beneath the fracture, allowing for the formation of a cerebrospinal fluid leptomeningeal cyst that forces apart the fracture edges and leads to nonunion. **Growing skull fractures** typically present weeks to months following an injury resulting in skull fracture. This rare complication is unique to infants and requires neurosurgical repair.

INTRACRANIAL INJURIES

An **epidural hematoma** (Figure 138-3) is a collection of blood between the inner skull and the dura and can occur from rapid arterial bleeding from the middle meningeal artery or the dural or diploic vasculature.

FIGURE 138-3.

Epidural hematoma. Note the convex shape and focal location. [Image used with permission of Jack Fountain, Jr., MD, Emory University and Grady Memorial Hospital.]

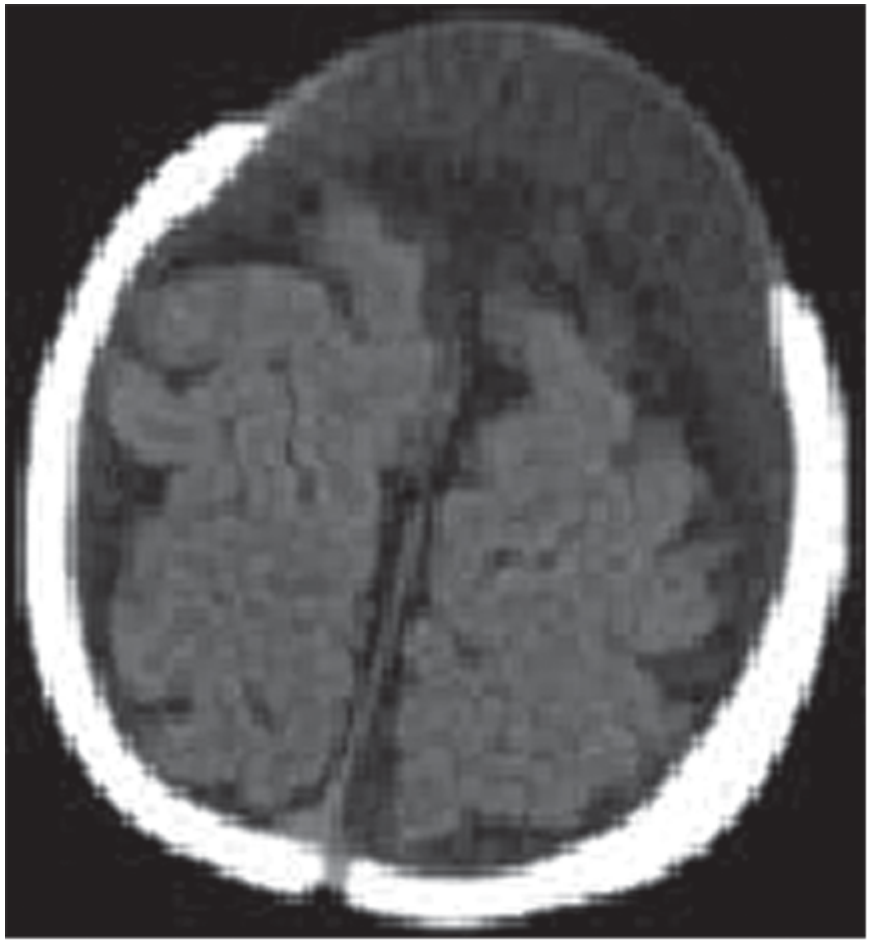


The classic presentation is a lucid interval after head trauma followed by rapid deterioration. The presence of a bi-convex hyperdense extra-axial lesion that does not cross the suture lines is indicative of an epidural hematoma on CT.[10] The long-term prognosis depends on the preoperative

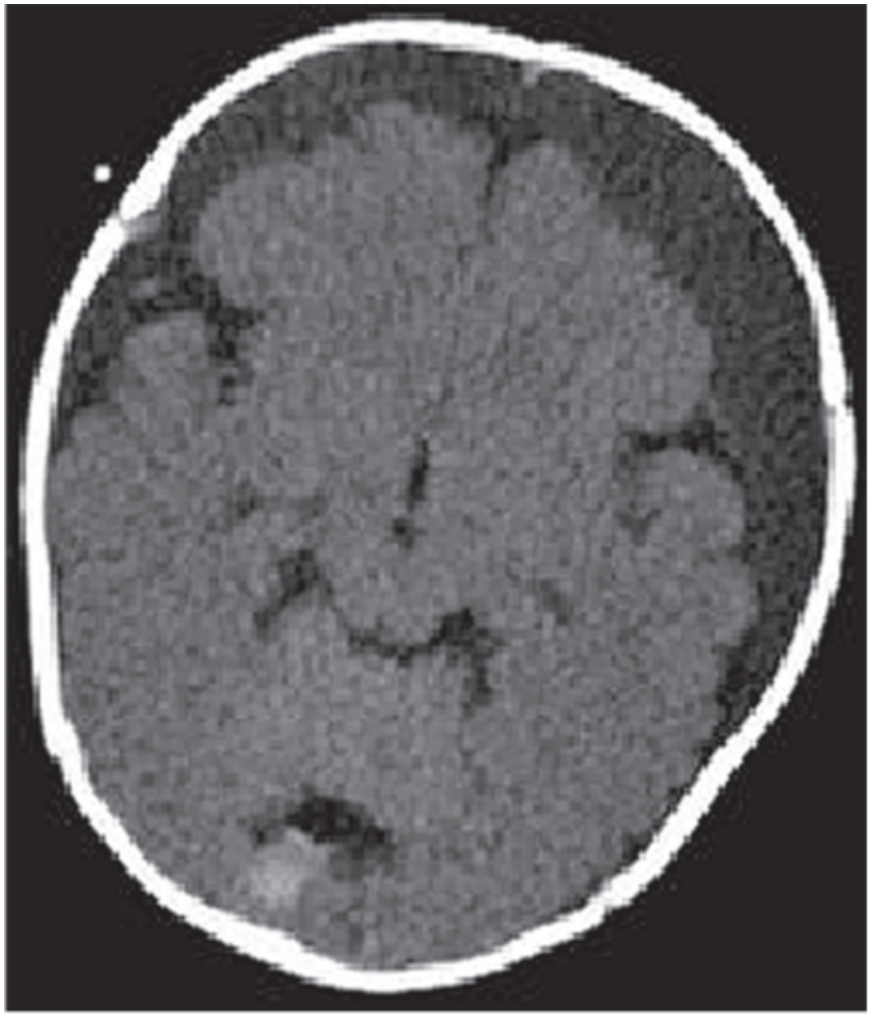
GCS and the extent of other underlying brain injury,[11] but is generally good if surgical evacuation can be undertaken in a timely manner.

Subdural hematomas (Figure 138-4), located between the arachnoid and the internal dural layer, are more common than epidural hematomas, particularly in infants and younger children. Subdural hematomas are caused by tearing of the subdural veins, are often extensive and bilateral (80% of cases), are frequently associated with underlying brain injury, and have a worse prognosis than epidural hematomas.

FIGURE 138-4.
A. Bifrontal chronic subdural hematoma extending through the anterior fontanelle in a 1-month old-child. B. Second image in the same child showing bifrontal chronic subdural hematoma as well as small, acute intraparenchymal hemorrhage in the posterior fossa.



A



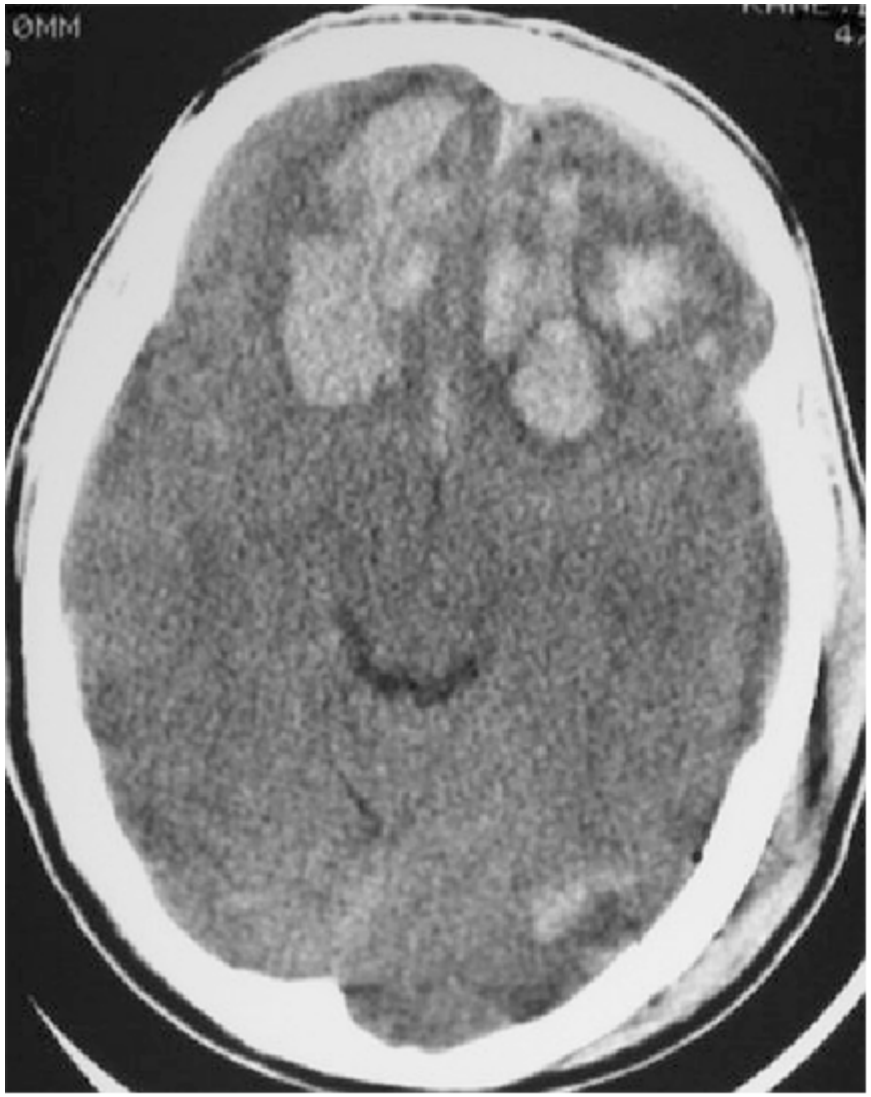
B

Radiographically, a subdural hematoma appears as a crescent-shaped fluid collection with the concavity facing the brain surface. Immediately after the injury, the subdural hematoma appears more dense (brighter white) than adjacent brain tissue. Due to the metabolism of blood products within the hematoma, its radiographic appearance changes over time. In the subacute phase (1 to 3 weeks after injury), the hematoma progressively assumes the same density of the brain tissue and can thus be difficult to recognize. Flattening of the sulci and the presence of a mass effect are indirect evidence of the presence of a subdural hematoma. Subsequently, in the chronic phase, the hematoma appears as a

hypodense fluid collection, with a density similar to cerebrospinal fluid.[10] In infants, loss of gray-white matter differentiation and diffuse hypodensity has been described with subdural hematoma.[7]

Cerebral contusions (**Figure 138-5**) are located in the cortex underlying the area of direct impact of a significant force (coup lesions) or on the opposite side (contrecoup lesions) where the brain has struck the cranial surface.

FIGURE 138-5.
CT scan demonstrating delayed intraparenchymal hemorrhages from a traumatic contusion. [Image used with permission of Jack Fountain, Jr., MD, Emory University and Grady Memorial Hospital.]



The severity ranges from minor CT findings in an asymptomatic patient to severe brain edema. On CT, the presence of ill-defined hyperdense areas within the cortex of the frontal and temporal lobe represent parenchymal contusions.

Traumatic subarachnoid hemorrhage is often associated with significant trauma and diffuse axonal injury (**Figures 138-6 and 138-7**). Infants more commonly present with diffuse injury and cerebral edema compared to adults because developmental differences render them susceptible to

rotational and deceleration forces. Clinically, the child with diffuse axonal injury presents with a profoundly depressed level of consciousness. Radiographic findings on CT may be minimal in the acute setting. MRI is more sensitive in the detection of diffuse axonal injury, which can be both hemorrhagic and nonhemorrhagic.[10]

FIGURE 138-6.
Diffuse axonal injury with intraventricular blood. [Image used with permission of Jack Fountain, Jr., MD, Emory University and Grady Memorial Hospital.]

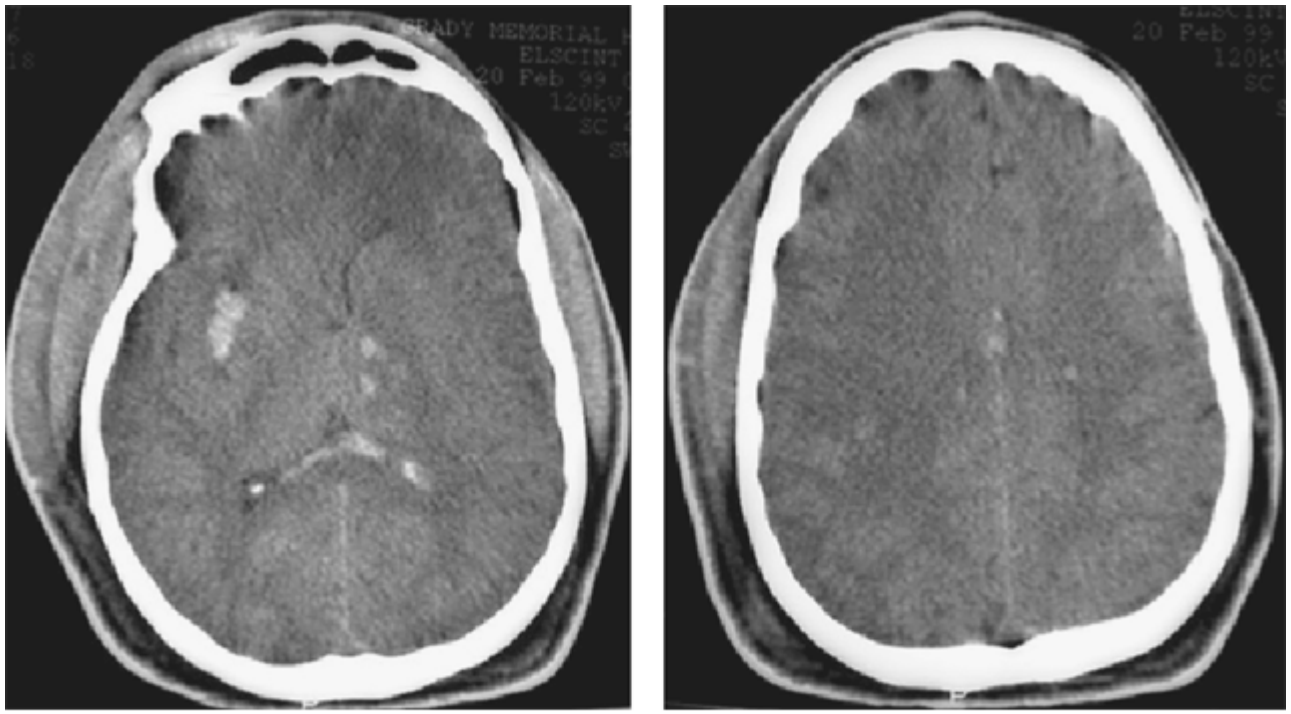


FIGURE 138-7.
Diffuse axonal injury with loss of the grey matter–white matter interface. [Image used with permission of Daniel Curry, MD, PhD, Texas Children's Hospital and Baylor College of Medicine.]

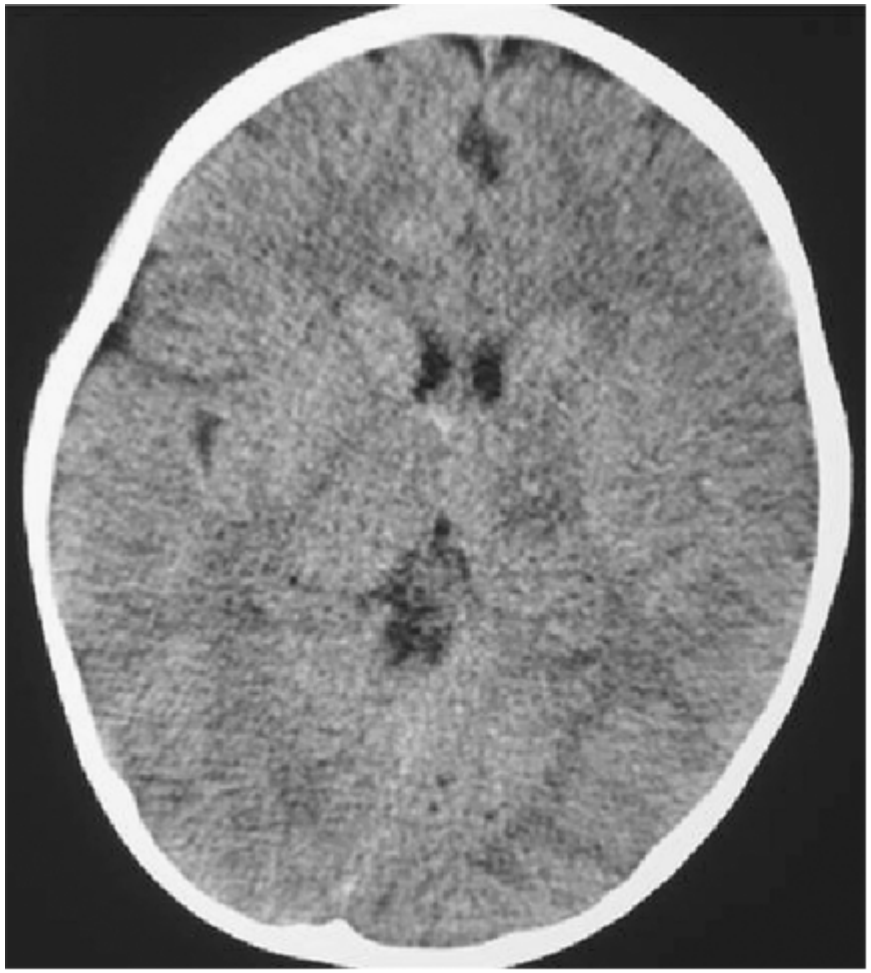
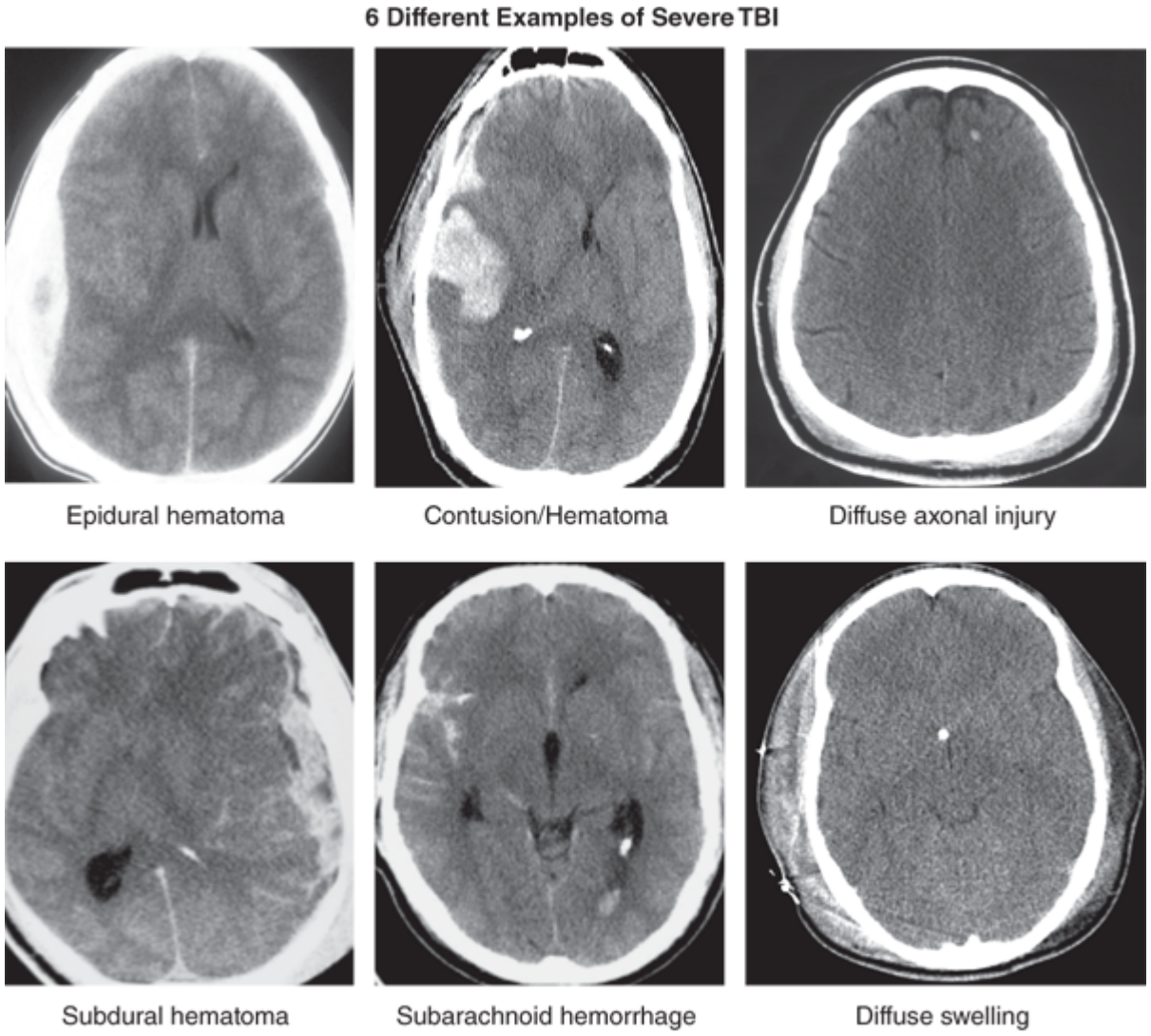


Figure 138-8 provides noncontrast head CT images of a variety of cerebral injuries.

FIGURE 138-8.

Six different examples of severe traumatic brain injury (TBI). [Image used with permission of Alisa Green, MD, University of California, San Francisco.]



Source: J.E. Tintinalli, J.S. Stapczynski, O.J. Ma, D.M. Yealy, G.D. Meckler, D.M. Cline: Tintinalli's Emergency Medicine: A Comprehensive Study Guide, 8th Edition
www.accessmedicine.com

CONCUSSION

The pathophysiology of concussion is complex. Although cerebral blood flow is increased in some children immediately following a concussion, the predominant pattern is transiently decreased blood flow. This is followed by a period of hyperemia from days 1 to 3, followed by return of the low-flow state.[12] In more than one third of concussed children, this phenomenon continues for a month or more.[13] Concussions do not cause

gross structural changes, making them difficult to diagnose by conventional radiography. However, animal models indicate that the acceleration-deceleration forces of a concussion initiate a neurochemical cascade that results in neuronal membrane disruption and axonal stretching. This in turn leads to significant ion flux, initially coupled with a transient increase in the cerebral glucose metabolism, followed by hypometabolism lasting days to weeks. Cytokine-mediated inflammation, stretch-mediated axonal disconnection, and neurotransmitter-mediated oxidative dysfunction also contribute to concussion-related impairments.[12]

CLINICAL FEATURES

HISTORY

Obtain the history from adult caregivers, witnesses, and EMS personnel and focus on the mechanism and time of the accident, and whether the child has experienced loss of consciousness, seizure, changes in behavior, or vomiting. Loss of consciousness itself is difficult to correlate with pathophysiology: the absence of loss of consciousness alone is not necessarily reassuring, particularly in children under 2 years of age,[14] nor does loss of consciousness alone predict a significant intracranial injury. Some authors report that falls from <1.5 m (5 ft), result in death only in extremely rare cases (<0.48 deaths per 1 million young children per year); however, head injury requiring neurosurgical intervention has been reported with this mechanism.[2,14,15]

A history that is incompatible with the child's age or situation (e.g., a report that a 1-month-old rolled off of the changing table or that a 6-month-old crawled out of the crib) raises a suspicion of nonaccidental injury.

Although the mechanism of injury contributes to an understanding of forces involved, symptoms such as headache, vomiting, and changes in behavior (e.g., irritability, lethargy) and their progression or resolution guide most decision rules. If possible, ask the child directly. For preverbal children, caregivers who know the child's normal behavior can identify behaviors suggesting headache or pain. Particularly in the young child, symptoms of neurologic injury may be subtle. Lethargy, irritability, seizures, and alterations in muscle tone or level of consciousness, as well as vomiting, poor feeding, breathing abnormalities and apnea, raise the suspicion of significant head injury.[7] Worsening of symptoms suggests intracranial injury. The persistence of symptoms such as headache, confusion, and amnesia suggests either an intracranial injury or a concussion.

PHYSICAL EXAMINATION

Focus the initial assessment on the immediate evaluation of airway, breathing, circulation, level of consciousness, and GCS (Table 138-1). Protect the cervical spine with a *properly sized* cervical collar. Most children with minor head injury have stable vital signs and are alert, active, and

appropriately interactive, and often the cervical spine can be clinically cleared after a thorough evaluation (see discussion on cervical spine injury later in this chapter).

If the child is sleeping on ED arrival, gently arouse the child to assess the level of consciousness or irritability. If the child is upset, allow time for the child to become comfortable with the environment before continuing with the examination. A knowledge of appropriate developmental milestones (e.g., stranger anxiety) and consideration of the child's normal nap time are important factors in assessing the mental status of the head-injured child.

Perform a thorough head-to-toe assessment for trauma, including an age-appropriate complete neurologic examination, musculoskeletal examination, and funduscopic examination. Note any evidence of trauma to the skull and examine the fontanelle in the calm upright child for signs of increased intracranial pressure. **A scalp hematoma in a child <2 years old is associated with an increased risk of skull fracture and intracranial hemorrhage.**[2,10] Hemotympanum or cerebrospinal fluid otorrhea and rhinorrhea may signify basilar skull fracture. Periorbital ecchymoses ("raccoon eyes") and bruising behind the ears ("Battle's sign"), although rare, also suggest basilar skull fracture. Evaluate the cervical spine for tenderness to palpation and evidence of injury. When nonaccidental trauma is suspected in an infant, consult with an ophthalmologist to identify retinal hemorrhages. Anisocoria, hypertension, and bradycardia are clinical signs of impending herniation and are preterminal events.

DIAGNOSIS

Several factors have led to a disparity in practice patterns and more frequent use of CT scan (12.8% of pediatric head trauma victims in 1995 to 22.4% in 2003) in the evaluation of minor head trauma in children.[16] Significant intracranial injury after a minor head injury is a relatively rare but potentially preventable cause of death and disability in the young. No single clinical finding is pathognomonic for intracranial hemorrhage; therefore, clinical practice has come to rely on CT evaluation. However, ionizing radiation is a long-term carcinogenic risk and should be used only when clinically indicated.[17] Thus, several clinical decisions rules have been developed to guide imaging. Three large studies demonstrate high sensitivity and specificity and include **CHALICE**[18] (Children's Head Injury Algorithm for the Prediction of Important Clinical Events), **CATCH**[19] (Canadian Assessment of Tomography for Childhood Head Injury), and **PECARN** (Pediatric Emergency Care Applied Research Network). PECARN is considered the "best for children and infants, with the largest cohort, highest sensitivity and acceptable specificity of clinically significant ICI (intracranial injury)."[20]

In 2009, PECARN derived and validated a novel prediction rule to identify children with minor head injury who are at very low risk of clinically significant intracranial injury. To address concerns of applicability to children of all ages, two prediction rules (**Tables 138-2 and 138-3**)[4] were developed and validated: one for children <2 years old and one for children >2 years old. Among enrolled children <2 years of age, there were 8502 patients in the derivation group and 2216 patients in the validation group; among enrolled children >2 years old, there were 25,283 patients in the derivation group and 6411 patients in the validation group.

TABLE 138-2

PECARN Low-Risk Criteria for Infants and Children with Minor Head Injury*

Age Group	Clinical Criteria
<2 years	Normal mental status No scalp hematoma except frontal Loss of consciousness <5 s Nonsevere mechanism† No palpable skull fracture Normal behavior per parents
>2 years	Normal mental status No loss of consciousness No vomiting Nonsevere mechanism† No signs of basilar skull fracture No severe headache

*Minor head injury was defined as a Glasgow Coma Scale score of 14 or 15 in this study.

†Severe mechanism: motor vehicle collision with ejection, rollover, or death of passenger; pedestrian or bicyclist without helmet struck by motorized vehicle; fall >2 m or 5 ft (age >2 y) or >1 m or 3 ft (age <2 y); head struck by high-impact object.

Source: Reproduced with permission from Kupperman N, Holmes JF, Dayan PS, et al: Identification of children at very low risk of clinically-important brain injuries after head trauma: a prospective cohort study. *Lancet*374: 1160, 2009. Copyright Elsevier.

TABLE 138-3

Pediatric Emergency Care Applied Research Network Low-Risk Criteria for Infants and Children with Minor Head Injury*

Age Group	Population	Sensitivity (95% CI)	Specificity (95% CI)	Negative Predictive Value (95% CI)
<2 years	Derivation (N = 8502)	98.6% (92.6–99.97)	53.7% (52.6–54.8)	99.9% (99.88–99.999)
	Validation (N = 2216)	100.0% (86.3–100.0)	53.7% (51.6–55.8)	100% (99.7–100.00)
>2 years	Derivation (N = 25,283)	96.7% (93.4–98.7)	58.5% (57.9–59.1)	99.95% (99.9–99.98)
	Validation (N = 6411)	96.8% (89.0–99.6)	59.8% (58.6–61.0)	99.95% (99.81–99.99)

*Minor head injury was defined as a Glasgow Coma Scale score of 14 or 15 in this study.

Source: Reproduced with permission from Kupperman N, Holmes JF, Dayan PS, et al: Identification of children at very low risk of clinically-important brain injuries after head trauma: a prospective cohort study. *Lancet*374: 1160, 2009. Copyright Elsevier.

The strengths of this study are the presence of both a derivation and a validation population, the strong statistical power provided by the large number of enrolled patients, and the inclusion of a separate study population of preverbal children age <2 years (25% of the study population). **The authors conclude that a CT scan is not indicated in patients meeting all low-risk criteria for age.** Of the clinical criteria suggesting intracranial injury in both age groups, the presence of altered mental status (or GCS of 14) or the presence of skull fracture most strongly correlated with intracranial injury (4.4% risk of clinically significant traumatic brain injury). Of note, the authors explicitly underscore that the findings in the study apply only to patients with a GCS of 14 or 15, because patients with a lower GCS have a higher risk of traumatic brain injury on CT.[4]

By comparison, the **CATCH** study identifies factors associated with high risk for significant intracranial injury requiring neurosurgical intervention among 3866 children <16 years of age presenting to the ED within 24 hours of minor head injury defined as GCS of 13 to 15 with witnessed loss of consciousness, vomiting, amnesia, and disorientation or irritability. High-risk children were those with GCS <15 2 hours after injury, suspected open or depressed skull fracture, worsening headache, or irritability. Medium-risk children were defined as those with signs of basilar skull fracture, a large, boggy scalp hematoma, or a dangerous mechanism of injury (fall >1 meter or five stairs, motor vehicle collision, or fall from a

bicycle without a helmet). Using these criteria, the CATCH rule had a sensitivity of 100% and specificity of 70% for identifying high-risk patients who require surgical intervention and 98% and 50% for identifying a CT abnormality among medium-risk patients. Compared to the PECARN rule, implementation of CATCH would lead to significantly lower rates of CT.[19]

The **CHALICE** study included patients with a range of injury severity and without a specific time of evaluation after injury. Conducted in the United Kingdom, 22,722 children <16 years of age were analyzed for factors associated with high risk of clinically significant intracranial injury (death, need for neurosurgical intervention, or significant CT abnormality). Identified risk factors included a witnessed loss of consciousness >5 minutes, amnesia >5 minutes, abnormal drowsiness, more than three episodes of emesis, seizure, suspicion of nonaccidental trauma, GCS <15 in children under 1 year of age or <14 in older children, penetrating or depressed skull injury or signs of basilar skull fracture, bulging fontanelle, scalp swelling, bruising or laceration >5 cm in children <1 year of age, abnormal neurologic exam, and significant mechanism of injury (high-speed motor vehicle collision, fall >3 m, or struck by high-velocity object). Using these high-risk factors, the CHALICE rule had a sensitivity of 98% and specificity of 87% and resulted in the lowest CT scan rate of the three studies.[18]

TREATMENT OF SERIOUS INTRACRANIAL INJURY

Treatment for children follows the same treatment principles as for adults (see chapter 254, "Trauma in Adults"). Treatment is outlined in **Table 138-4**.

TABLE 138-4

Treatment of Serious Head Injury

Cervical spine	Maintain spinal precautions	
Airway	Maintain airway, intubate for GCS < 8 or as needed for oxygenation and ventilation	
Oxygenation and ventilation	Oxygen saturation > 90; PCO_2 35–40	No prophylactic hyperventilation
Blood pressure	SBP >70 + (2 × age)	No permissive hypotension Consider sedation and neuromuscular blockade
GCS	GCS before paralytics if possible	Serial GCS to document changes
Stat neuroimaging (noncontrast head CT and cervical spine CT)	Look for signs of increased ICP and identify mass lesions	Transcranial Doppler may be useful in infants with open fontanelles and experienced pediatric radiologists
Glucose	Treat hypoglycemia and hyperglycemia	Maintain normal blood glucose
Increased ICP/impending herniation	Keep head of bed at 30 degrees	3% NS 5 mL/kg over 10 min *or* Mannitol 0.5 milligram/kg if normotensive (response is not dose-dependent)
Core temperature	Maintain temperature 36–38°C	Hypothermia in children not recommended; avoid hyperthermia
Seizure prophylaxis	Optional for children with witnessed seizures	Fosphenytoin or levetiracetam for first week following severe TBI

Neurosurgery/transfer	ICP monitoring and CSF diversion for ICP	
Anemia	Transfuse for Hgb <7 grams/dL	

Abbreviations: CSF = craniospinal fluid; GCS = Glasgow Coma Scale; Hgb = hemoglobin; ICP = intracranial pressure; NS = normal saline; PCO_2 = partial pressure of carbon dioxide; SBP = systolic blood pressure; TBI = traumatic brain injury.

SPECIAL CONSIDERATIONS: CONCUSSION

Approximately 1.6 to 3.8 million athletes suffer a concussion annually in the United States,[12] with the majority affecting children.[21] Eight- to 13-year olds account for 40% of pediatric concussions.[22] Concussion is defined as a "complex pathophysiological process, involving the brain, induced by traumatic biomechanical forces." Common features include a direct blow or transmitted force, rapid-onset but short-lived neurologic impairment, characteristic symptoms that may or may not include loss of consciousness, and lack of structural abnormalities on standard imaging. Grading systems and the use of the terms *simple* and *complex* have been abandoned in lieu of a more symptom-focused approach.[23]

Sideline management involves attention to first aid and cervical spine protection in the appropriate clinical setting. Child and teen athletes suspected of having a concussion should not be returned to play the same day and should be observed for deterioration through the first few hours following the injury, because symptoms can progress over a 6- to 24-hour period. Standardized concussion assessment tools have been published and include the Sport Concussion Assessment Tool–Third Edition and the Child Sport Concussion Assessment Tool–Third Edition.[24] These tools are comprehensive, but impractical in the ED. In addition to assessment for serious head injury, focused tests of cognition and balance (designed for sideline use) are useful for the ED evaluation of the concussed patient, and two such exams, the Maddox questions and the Balance Error Scoring System, are detailed in **Table 138-5** and **Figure 138-9**.[25] Difficulty with definitive diagnosis on the sideline has led to the use of the phrase, "When in doubt, sit them out." Symptoms and signs of a concussion include somatic complaints (headache, dizziness, nausea or vomiting, blurred vision, balance problems, photophobia, phonophobia, fatigue), cognitive complaints (feeling of being in a fog, delayed reaction time, difficulty concentrating, confusion), emotional lability (irritability, depression, anxiety, apathy), loss of consciousness or amnesia, behavioral changes, and sleep disturbance (insomnia or sleepiness).[26]

TABLE 138-5

Maddox Questions for Sideline Evaluation of Concussion (any incorrect answer is suggestive of concussion)

Where are we at today?
Which half is it now?
Who scored last in the game?
What team did you play last week/last game?
Did your team win the last game?

FIGURE 138-9.

The athlete stands heel-to-toe with the dominant foot forward, eyes closed, and hands on hips for 20 seconds. More than five errors (lifting hands, opening eyes, stumbling, lifting forefoot or heel, or remaining out of the start position for >5 seconds) may suggest concussion. Note that shoes should not be worn during this test.

In the ED, neuroimaging for diagnosis of a concussion is not recommended, but CT should be performed on children in whom an intracranial injury is suspected. Advanced imaging with certain MRI sequences, magnetic resonance spectroscopy, and positron emission tomography may be abnormal and predictive of outcomes[27] but does not guide management and, thus, is currently used as a research tool. Computerized neurocognitive testing, such as ImPACT, is rarely available in the ED, but should be performed by the primary care physician, particularly if preseason testing of an athlete was done and a comparative baseline is available. In clinically equivocal cases, neurocognitive tests are >90% sensitive to diagnose concussion.[28] Scores may be useful in verifying readiness to return to play, and the 72-hour score is prognostic of recovery time.

Adults typically require 3 to 5 days to return to baseline neurocognitive testing scores, college students 5 to 7 days, and high school students 10 to 14 days. Premorbid conditions such as mental health disorders, migraines, or attention deficit/hyperactivity disorder predict a slower recovery.[29] Fourteen percent of children and adolescents will have persistence of symptoms beyond 3 months, or "postconcussive syndrome."[30] Concerns

regarding return to play prior to symptom resolution include second impact syndrome and recurrent concussions. **Second impact syndrome** is a rare entity characterized by loss of vascular autoregulation and cerebral edema in response to a second head injury prior to recovery from the first. It is exclusive to pediatrics, and mortality is high.[25] **Recurrent concussions** are more common in athletes who return quickly to play, and 80% of same-season concussions occur within 10 days of each other.[29]

Physical and cognitive rest are the cornerstones of concussion management. **Return to play should follow a six-step protocol of activity, provided that the child remains asymptomatic for at least 24 hours in between each step:**

1. No activity; complete rest
2. Light aerobic exercise (e.g., walking)
3. Sport-specific exercise, progressive addition of resistance training
4. Noncontact training drills
5. Full-contact training after medical clearance
6. Game play

Children who have recurrence of their symptoms upon progression should return to the previous level until they have been asymptomatic for 24 hours at that level.[28] Advise cognitive rest until the child is minimally symptomatic or asymptomatic. Cognitive rest includes rest from texting and video games and accommodations with schoolwork such as extra time for tests and assistance with note-taking.

Symptomatic treatment of concussive symptoms remains unproven, but described options include nonsteroidal anti-inflammatory medications, simple analgesics, and triptans for headache[31] and ondansetron for nausea and vomiting.[32] Prophylaxis with β-blockers or antiseizure medications can be considered in the outpatient setting.[31,33]

DISPOSITION AND FOLLOW-UP

Asymptomatic infants and children (e.g., not vomiting, normal neurologic and mental status examinations) who are at least 2 to 4 hours post-injury can safely be discharged to reliable caregivers without imaging. Provide careful instructions regarding signs and symptoms for which to **seek immediate care: lethargy, irritability, focal deficits, or intractable vomiting**.

Infants and children thought to be at *intermediate risk* can be observed for 3 to 6 hours, or a head CT scan can be obtained. If the CT is normal or they remain in stable condition on observation, they may safely be discharged to reliable caretakers with primary care follow-up.

Patients for whom a CT scan has been obtained and shows normal findings may safely be discharged to reliable caretakers. **The incidence of delayed deterioration requiring intervention after normal findings on CT is near zero.**[34,35] If a concussion is considered likely, parents should be informed of the specific return to activity guidelines, and encouraged to seek primary care or neurology follow-up.

Children with complex, depressed, or basilar skull fractures should be managed in conjunction with the pediatric neurosurgeon and typically require admission. Stable, asymptomatic infants and children with linear, nondisplaced skull fractures and no intracranial injury are safe for discharge with primary care or neurosurgical outpatient follow-up.[36] Parents should be warned that infants are at risk for "growing fractures" over the weeks to months following a skull fracture.

Patients with intracranial injury and those thought to be at risk of nonaccidental trauma may require transfer to a hospital with appropriate services if admission to an appropriately monitored setting is not available at the presenting institution.

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