Pediatric Head Trauma: A Review and Update

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Practice Gaps

There is still a considerable amount of confusion when it comes to managing concussions. An excessive number of head computed tomographic scans are being obtained for concussions, resulting in unnecessary exposure to ionizing radiation. Clinicians should be aware of the most recent guidelines for the management of concussion, including the need for imaging, and should be able to differentiate mild from moderate and severe traumatic brain injury.

Objectives

After completing this article, readers should be able to:

1. Differentiate a mild from a moderate or severe traumatic brain injury (TBI).
2. Acutely manage a child with a TBI, including deciding when further imaging is necessary.
3. Manage a child with a postconcussion syndrome and identify when referral to a specialist is necessary.

Traumatic brain injury (TBI) is the leading cause of death or severe disability in children older than 1 year. (1)(2) In a report to Congress published by the Centers for Disease Control and Prevention (CDC) in 2018, (3) the CDC reported the public health burden of TBIs. They noted that 640,000 emergency department visits and 18,000 hospital stays were directly related to TBI. The etiology of TBI varies among age groups. In the 0- to 4-year-old age group, the most common cause of TBI is falls. On the other hand, in the 15- to 24-year-old age group the distribution of injuries caused by falls, assault, and motor vehicle events are nearly equal. Epidemiologic studies have found that rates of TBI seen in the emergency department have increased in all age groups since 2001, with children 0 to 24 years old having the highest rates of TBI of all age groups. Children 0 to 4 years old have almost twice the rate of TBI compared with the next highest age group (15–24 years old), making pediatric traumatic brain injury an especially salient topic for the modern-day pediatrician. (4) Moreover, 61% of children with moderate to severe TBI experienced a disability. Estimates conclude that at least 145,000 children aged 0 to 19 years are currently living with long-term symptoms due to a TBI (likely an underestimate with underreporting of mild TBI [mTBI]), with

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ABBREVIATIONS

AAP American Academy of Pediatrics
AHT abusive head trauma
CDC Centers for Disease Control and Prevention
Child SCATS Child Sport Concussion Assessment Tool
CISG Concussion in Sport Group
CT computed tomography
DAI diffuse axonal injury
FLAIR fluid-attenuated inversion recovery
GCS Glasgow Coma Scale
MRI magnetic resonance imaging
mTBI mild traumatic brain injury
TBI traumatic brain injury
symptoms extending far beyond their initial hospital visit into the following months and years. (3) Even children without overt neurologic deficits resulting from their TBI can demonstrate impairment in academic performance, attention and concentration, memory, and executive function, some of which only become apparent months or years after the initial injury. (4)(5)(6) The economic impact of TBI is substantial, with estimates ranging from $77.9 million per year in direct costs to more than $1 billion per year for TBI-associated hospitalizations. (5)

With all the associated morbidity and mortality, it is vital that pediatricians are educated in recognizing and treating TBIs and their sequelae. In this article, we aim to provide current evidence on the recognition, treatment, and rehabilitation of TBIs. We begin by discussing mTBI, typically manifesting as a concussion, and then discuss moderate and severe TBIs that are more often encountered in the emergency department or hospital setting.

**MILD TBI**

mTBI commonly manifests as concussion, and this is the focus of our discussion. However, it is also worth noting that even patients with more severe brain injury can exhibit signs and symptoms of concussion, and these should not be ignored. Concussion is a broad clinical diagnosis defined by the American Academy of Neurology as “a clinical syndrome of biomechanically induced alteration of brain function, typically affecting memory and orientation, which may involve loss of consciousness.” (6) Due to various mechanisms of action for concussion, and the multiple disciplines involved, including neurology, sports medicine, rehabilitation medicine, and military medicine, there are multiple diagnostic criteria and treatment recommendations in place, which can make it challenging for the primary care provider evaluating a patient with a concussion. (7) Yet, concussion is a common complaint in children, occurring in approximately 69.2 of 100,000 children younger than 15 years, (8) indicating that an evidence-based, comprehensive plan for concussion diagnosis and management is imperative. (7)

In this section, we aim to summarize current recommendations on concussion evaluation and management for the general pediatrician based on guidelines for concussion management developed by the American Academy of Neurology, (6) the CDC, (9)(10) and the Concussion in Sport Group (CISG), an international multidisciplinary group of clinicians and researchers focused on concussion diagnosis and management. (11) The CDC recently published new guidelines on concussion management, and these recommendations are reflected herein. (10) Although concussion can be caused by any mechanical force on the brain, we focus on sports-related concussion because this is the focus of most research and clinical guidelines and is the most common presentation of concussion for the general pediatrician. (7) Note that although sports-related concussions have been studied most extensively, it is probable that most of the recommendations made thereafter apply equally well to concussions related to other accidental and nonaccidental injuries. In fact, in their most recent recommendations, the CDC (10) does not differentiate between these types of concussions as it relates to diagnosis and management.

**Recognizing Concussion**

The first step to treating concussion is recognition. The clinical phenotype of concussion can vary between patients, and it can be a challenge for the evaluating clinician to consider all the possible manifestations of concussion. One useful acronym is COACH CV, which was developed by Craton et al (12) and is based on the CISG guidelines. This acronym includes the most common clinical phenotypes of concussion: Cognitive dysfunction, Oculomotor dysfunction, Affective disturbances, Cervical spine disorders, Headaches, and Cardiovascular and Vestibular anomalies (Table 1).

Symptoms of concussion in these domains are broad and include impairment of memory or attention, blurred vision or abnormal extraocular movements, fatigue, mood changes, poor sleep, headaches, vestibular dysfunction, or heart rate variability (see Table 1 for a more extensive list of potential concussion symptoms). Of note, a patient with a concussion may have 1 or more of these symptoms. There is no loss of consciousness required for a diagnosis of concussion.

In their recent recommendations for the diagnosis and management of concussion, the CDC recommends using a validated symptom rating scale in the evaluation of concussion. (10) The most commonly used tools include the Child Sport Concussion Assessment Tool (Child SCAT3) developed by the CISG, (13) the Acute Concussion Evaluation developed by the CDC, (9) the Postconcussion Symptom Scale, (14) and the Graded Symptom Checklist. (15)(16) These tools use a Likert scale completed by the patient and/or parent to assess symptom severity, with a higher score indicating more severe symptoms. Although some of the scales, specifically the Child SCAT3, were developed for sports-related concussion, the symptoms of concussion are generalizable to other concussion etiologies as well. Studies have further analyzed these scales, attempting to identify the underlying symptom groups contributing to higher scores in concussed patients (eg, neurocognitive, somatic, emotional), with mixed conclusions. (14)(15)(17) In practice, it is best to choose a scale and get baseline testing, followed by
testing at the time of injury and repeated testing throughout recovery to track changes in individual and total symptom scores. If symptoms in any 1 or more of the tested clinical domains are present, this suggests a diagnosis of concussion. (13) If further delineation of symptoms is required, additional testing, either in the office or via a referral to the appropriate provider, can be considered. Some toolkits, such as the Child SCAT5, include further testing that can be performed by the provider to screen for certain concussion phenotypes. These include the Balance Error Scoring System, which assesses postural stability, and the Sensory Organization Test, which assesses the patient’s equilibrium with altering visual and somatosensory inputs. However, these methods are not as sensitive at concussion diagnosis as the previously mentioned Likert rating scales. (6) Other targeted testing could include visual acuity for ocular motor dysfunction, orthostatic vital signs of cardiovascular dysfunction, or a detailed spinal neuromuscular examination to evaluate for cervical spine abnormalities (Table 1). (7)

Of note, computed tomography (CT) cannot be used to diagnose concussion and should generally be avoided to prevent unnecessary ionizing radiation exposure, although it may be used to rule out a more severe TBI, especially in patients with loss of consciousness, posttraumatic amnesia, persistent altered mental status, focal neurologic deficit, evidence of skull fracture, or signs of clinical deterioration. (6) The Pediatric Emergency Care Applied Research Network (PECARN) has established criteria that can be used in decision making for children presenting after a TBI. The clinical criteria for children 2 years and older include normal mental status, no loss of consciousness, no vomiting, nonsevere injury mechanism, no signs of basilar skull fracture, and no severe headache. If all of these criteria are met, they demonstrate a negative predictive value of 99.95% for clinically important TBI. Conversely, the presence of any 1 of these predictors has sensitivity of 96.8% in identifying clinically important TBI and indicates that further assessment with head CT is required. Criteria for children younger than 2 years are also included in the study. (18) Unfortunately, unnecessary head CT in children remains a common concern, and further education of community providers can help reduce this unneeded radiation exposure. (19)

Although clinical discretion is still required to make a concussion diagnosis, the previously mentioned tools can help identify symptoms and track recovery, aiding the clinician in decisions regarding return to play and when to pursue referral or further testing. With the typical natural history of concussion, an athlete’s symptoms should return to baseline in 2 weeks for adults and in 4 weeks for children. (20)

### TABLE 1. Clinical Phenotypes of Concussion (COACH CV)

<table>
<thead>
<tr>
<th>CLINICAL PHENOTYPE</th>
<th>SYMPTOMS</th>
<th>SPECIFIC TESTING</th>
</tr>
</thead>
<tbody>
<tr>
<td>C Cognitive function</td>
<td>Memory impairment, decreased attention and concentration, slowed processing speed</td>
<td>Neuropsychological testing (in person or computer-based, such as ImPACT testing)</td>
</tr>
<tr>
<td>O Oculomotor dysfunction</td>
<td>Convergence insufficiency, blurred vision, abnormal saccades and/or smooth pursuit, photophobia</td>
<td>Visual acuity testing, King-Devick test (assess saccadic eye movements)</td>
</tr>
<tr>
<td>A Affective disturbances</td>
<td>Fatigue, sadness, irritability, sleep disturbance, poor concentration, emotionality</td>
<td>Depression screen</td>
</tr>
<tr>
<td>C Cervical spine disorders</td>
<td>Neck pain, headaches, dizziness, balance difficulty</td>
<td>Neck range of motion, Palpation of bones and muscles of the neck</td>
</tr>
<tr>
<td>H Headaches</td>
<td>Migrainous, tension-type, or cervicogenic headaches</td>
<td>–</td>
</tr>
<tr>
<td>C Cardiovascular anomaly</td>
<td>Exercise intolerance, heart rate variability or elevation, postural orthostatic tachycardia syndrome, autonomic dysfunction</td>
<td>Orthostatic vital signs, Exercise stress test, Tilt table testing</td>
</tr>
<tr>
<td>V Vestibular dysfunction</td>
<td>Dizziness, vertigo, balance difficulties</td>
<td>Romberg test, Tandem gait, Vestibulo-ocular reflex, Balance Error Scoring System (see the Child Sport Concussion Assessment Tool)</td>
</tr>
</tbody>
</table>

This table includes the common clinical phenotypes of concussion that patients may endorse on a symptom scale. Listed are the corresponding symptoms of each phenotype, as well as further testing that can be considered to assess each symptom. See Craton et al. (12)
Rest and Return to Play

Once a concussion is diagnosed it is imperative that the patient is given the appropriate guidelines for rest and return to play/school. Previous recommendations for complete rest until symptom resolution are now outdated and likely stemmed from sports medicine literature in which there was a concern for second impact syndrome. Although this remains a concern, strict rest is not required for this entire period but rather guidelines now recommend complete rest for 24 to 48 hours, after which patients have a gradual return to full activity. In fact, a recent study comparing 2-day and 5-day strict rest periods in children with concussion demonstrated a slower resolution of symptoms in the group with a more prolonged rest. (21) After the initial rest period, children can follow a gradual return-to-activity protocol, which is outlined further in available toolkits, including the CDC Heads Up guidelines (9) and the Child SCAT5. The general strategy includes gradually increasing physical activity, beginning with nonaerobic daily activities and progressing through graduated steps until full return to sport (Table 2). The child should take at least 24 hours for each step of the plan, with return to the previous step for any worsening of symptoms. A similar progression can be used for return to school activities for children whose symptoms are exacerbated by mental activities, beginning with a few days of rest at home, followed by a gradual return to school full time. (13) Nonessential cognitive activities, such as playing video games, should be introduced as tolerated once a child is back to normal or near-normal physical routine.

There is some evidence to suggest that the period for full physiologic and metabolic recovery from concussion may extend beyond that for clinical symptom recovery and that a repeat concussion during this period could further prolong recovery. The NCAA Concussion Study on collegiate football players found that 11 of the 12 players with a repeat concussion in one season experienced their second concussion within 10 days of their initial concussion, indicating that athletes are especially prone to recurrent concussion during this period.

### TABLE 2. Return to Play Progression

<table>
<thead>
<tr>
<th>EXERCISE STEP</th>
<th>EXAMPLE ACTIVITIES</th>
<th>ACTIVITY TIME</th>
<th>GOAL OF EACH STEP</th>
</tr>
</thead>
<tbody>
<tr>
<td>No activity</td>
<td>Complete physical and cognitive rest for 24–48 h</td>
<td></td>
<td>Reintegrate into work and school activities</td>
</tr>
<tr>
<td>Nonaerobic activity</td>
<td>Normal daily activities that do not provoke symptoms</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Light aerobic activity</td>
<td>Exercise bike, walking, light jogging at a slow pace (no weight lifting, jumping, or running)</td>
<td>5–10 min</td>
<td>Light activities leading to a mild increase in heart rate</td>
</tr>
<tr>
<td>Moderate activity</td>
<td>Jogging, brief running, moderate-intensity stationary biking, light resistance activities</td>
<td>Reduced from normal routine</td>
<td>Limited body and head movement</td>
</tr>
<tr>
<td>Heavy, noncontact activity</td>
<td>Running, noncontact drills, weight lifting, stationary biking</td>
<td>At or near normal routine</td>
<td>Intense activity without contact. Cognitive activity during exercise can be added</td>
</tr>
<tr>
<td>Full contact</td>
<td>Normal full-contact physical activities</td>
<td>Normal routine</td>
<td>Return to full-contact activities</td>
</tr>
<tr>
<td>Competitive activities</td>
<td>Return to full competitive activities</td>
<td>Normal routine</td>
<td>No further restrictions in activity</td>
</tr>
</tbody>
</table>

The table is adapted from the Centers for Disease Control and Prevention Heads Up guidelines and the Child Sport Concussion Assessment Tool for returning to play. Each step should take a minimum of 24 hours. During the above progression, the child, family, and health-care provider should pay special attention to any new or worsening symptoms. If any symptoms worsen while exercising, the child should return to the previous step.
Although pharmacologic treatment has not been shown to facilitate recovery from concussion, it may be considered in patients who have longer recovery periods or whose quality of life is significantly affected by their symptoms. In these cases, treatment should focus on symptom management, including medications such as melatonin for sleep disturbance, nonopioid analgesics for acute headaches and amitriptyline or topiramate for headache prevention, and selective serotonin reuptake inhibitors or amantadine for emotional or cognitive effects, respectively. Of note, in patients using pharmacotherapy, this could mask the symptoms of concussion, and these medications should be weaned or careful consideration should be given before returning to full play.

Referral
Occasionally, the situation will still arise in which children do not have a complete recovery with the previously described strategies. Children with symptoms persisting beyond 4 weeks and adults with symptoms persisting beyond 2 weeks should be referred to a health-care provider specializing in concussion. Studies have shown that higher symptom scores on immediate postconcussive testing can indicate more severe or prolonged postcognitive effects with a longer time for return to play. Certain preexisting conditions may delay concussion recovery, including history of postural orthostatic tachycardia syndrome, motion sickness, strabismus or ocular abnormalities, attention-deficit/hyperactivity disorder and learning disabilities, and mood disorders. These children may require additional school accommodations to facilitate their return. Other factors such as history of previous concussions, more severe presenting or postconcussive symptoms, memory problems, fatigue/fogginess, and disorientation may also contribute to a more prolonged recovery. Some intrinsic factors, such as low socioeconomic status, Hispanic race, and high school age (especially in girls), also place children at risk for more prolonged symptoms compared with other patient populations.

Residual Effects
Unfortunately, approximately 10% to 15% of concussion patients have persistent symptoms beyond the first few weeks. As recently as 2014, the Diagnostic and Statistical Manual of Mental Disorders criteria included a diagnosis of postconcussion syndrome, although this has now been renamed “major or mild neurocognitive disorder due to TBI.” It is up to the clinician to consider the severity and functional disability of the patient when assigning a diagnosis. Postconcussive symptoms can be widely variable and depend on preexisting comorbidities, including neurocognitive disorders, vestibular dysfunction, affective symptoms, and medication/substance use.

Additional evaluation and therapy should be considered for children with persistent postconcussive symptoms. Although most concussion patients will have normal cognitive function by 3 months after injury, some children could have cognitive deficits persisting up to 1 year, especially in the presence of a severe original injury, a history of previous concussions, and psychological risk factors. These children should be referred for formal neuropsychological testing. Magnetic resonance imaging (MRI) can be more sensitive at detecting certain types of brain injury, such as diffuse axonal injury (DAI) or petechial hemorrhages, which are not detected in 25% to 30% of CT scans (see the "Management" subsection later herein). Patients with vestibular or oculomotor dysfunction should be referred to the appropriate therapy for rehabilitation. Finally, the possibility of depression after concussion should not be ignored because it may often masquerade as cognitive or neurosensory dysfunction.

Risk Reduction
General risk reduction for concussion centers around preventive safety measures to decrease risk of head injury. This includes the use of appropriate car seats, booster seats, or seat belts in automobiles; helmets while on bicycles or scooters; stair gates; and soft surfaces in play areas. However, the use of equipment in sports to protect against concussions is a controversial topic. The use of mouth guards does not seem to provide protection from concussion. Helmets and headgear have been shown to reduce the risk of concussion in skiing and snowboarding, but the effect in full-contact sports such as hockey and football has not been as conclusive. This, in part, has led to the new rules for helmet contact implemented recently by the National Football League to reduce unnecessary risk of head injury in its players. Research on risk reduction for sports-related concussion has also focused on analyzing age, level of competition, sex, and type of sport to determine whether any individual factor can affect concussion symptoms and risk. A recent study found that females with concussion are more likely to report a higher level of symptoms and to experience postconcussive headaches, whereas males are more likely to experience loss of consciousness, confusion, and amnesia with a
concussion. (33) Although rates of concussion in males are greater due to larger total numbers of athletes, concussion risk seems to be greater in female athletes playing soccer or basketball. In addition, for all athletes there seems to be a higher risk of concussion with American football and rugby compared with other sports, with baseball, softball, volleyball, and gymnastics having the lowest risk. (6)

There is also evidence that children experiencing a first concussion before age 10 years have approximately twice the risk of sustaining a subsequent concussion before age 18 years compared with patients experiencing their first concussion in adolescence. It is not known whether this is due to the early concussion itself, the duration of participation in contact sports, or intrinsic factors affecting an individual’s concussion risk (eg, risk-taking behavior). (34)

Ultimately, health-care providers caring for amateur athletes with a history of an early concussion, recurrent concussions, or persistent concussive symptoms may need to discuss the previously mentioned evidence as well as implement formal neurologic and neuropsychiatric assessments to aid in the discussion of concussion risk management or possible retirement from play. (6)(35)

Second Impact Syndrome
One of the greatest concerns of parents and clinicians surrounding concussion is the threat of second impact syndrome. Second impact syndrome is described as a clinical syndrome of catastrophic cerebral edema that results when a second concussion occurs before resolution of symptoms from the initial concussion. (35) The second impact may be much less severe and not even a consequence of a direct impact to the head. (35) It is thought to be due to a failure of cerebral autoregulation coupled with a stress-induced catecholamine surge, leading to cerebral edema and consequent herniation, resulting in severe disability or death. (36) Although it seems to be extremely rare, a recent review of 17 cases reported in the literature from 1946 through 2015 noted an age range of 13 to 23 years, indicating that this is a syndrome that is particularly impactful in the pediatric population. (37) Although cases seem to be most common with repeated concussions within the first 2 weeks of the initial injury, children are considered to be at risk as long as they continue to be symptomatic from their initial concussion. This diagnosis has gained a lot of attention in the medical literature owing to its devastating consequences, yet there remains some controversy around its existence. It is known that diffuse cerebral swelling can occur after a single head injury, so the occurrence of a second impact to create the clinical syndrome may not be required. (38) Nonetheless, all concussion practitioners agree on the importance of complete resolution of symptoms before return to play because this will decrease the risk of prolonged postconcussive symptoms and the possibility of second impact syndrome. (31)

MODERATE AND SEVERE TBI
In this section we focus on moderate and severe TBI. Although there are several scales that have been used to differentiate mild TBI from moderate and severe TBI, the most commonly accepted classification relies on the Glasgow Coma Scale (GCS), with moderate defined as a GCS score of 9 to 13 and severe as a GCS score less than 8 (39) (Table 3). These more severe injuries are differentiated from mTBI by the fact that they have clear imaging findings; mTBIs typically do not have any MRI or CT findings. Although TBI remains one of the leading causes of mortality and morbidity in children in the United States, the management of this entity continues to be applied quite unevenly despite the existence of American Academy of Pediatrics (AAP) recommendations. (40) The epidemiology of TBI was reviewed in the Introduction.

Pathophysiology of Pediatric Head Trauma
It is important to recognize that there are major differences between the pediatric brain and the adult brain in the pathophysiology of head trauma. Although it is generally true that the pediatric brain tends to be more resilient to focal lesions (stroke, surgical excision) as a result of plasticity, the opposite seems to be true when it comes to TBI. There is strong evidence that the younger a child is when experiencing a severe TBI, the longer he or she takes to recover. (41) Furthermore, the morbidity from TBI seems to be significantly higher in children than in adults. (42) This may be related to several factors, including incomplete myelination, the higher water content of the pediatric brain, and a critical period during development when synaptic pruning depends on complex physiologic mechanisms.

Space-Occupying Traumatic Injuries
The most urgent clinical factor associated with TBI is the rapid expansion of space-occupying lesions, including bleeds and progressing edema. Interestingly, posttraumatic hydrocephalus is much less common in children than in adults and can often be managed conservatively, obviating the need for decompression or evacuation. (43)

Space-Occupying Lesions: Bleeds
Bleeds caused by TBI can occur in several locations, including potential or anatomical spaces formed by the meninges and within the substance of the brain itself.
Epidural Hematoma. Epidural hematomas tend to be the result of arterial bleeds associated with skull fractures. These bleeds result from the accumulation of blood in the potential space formed by the junction of the dura mater and the skull (Fig 1). The classic example is a middle meningeal artery tear in the context of a temporal or parietal bone fracture. In this context, epidural bleeds can be catastrophic. Given the arterial nature of these bleeds they can lead to a very rapid mass effect with resulting herniation of the cerebral contents. However, it is important to recognize that epidural bleeds in children are usually much more forgiving than those in adults. (44) It is not completely clear why this might be, although in the study just cited smaller clots tended to be more frequent in younger children. Acute epidural bleeds are characterized by a hyperdense lens-shaped lesion on CT (Fig 2).

Subdural Hematoma. Subdural hematomas result from the accumulation of blood in the potential space between the dura mater and the arachnoid layers (Fig 1). These bleeds are usually caused by the rupture of bridging veins that traverse this space. They are quite frequent in infants, especially in the context of abuse. (45) They are also frequently caused by other forms of rapid shearing injuries to the brain, such as motor vehicle accidents. Subdural bleeds typically layer in a crescentic shape that, when acute, appears hyperdense on the CT scan of the brain (Fig 2).

Subarachnoid Hemorrhage. Subarachnoid hemorrhages result from the accumulation of blood in the subarachnoid space. As opposed to the 2 previous types of bleeds, subarachnoid hemorrhages occur in an anatomical space normally filled with cerebrospinal fluid (Fig 1). These bleeds are extremely common in the context of TBI and frequently cause seizures because blood is an irritant to the cerebral cortex. CT scans of an acute subarachnoid hemorrhage show hyperdense layering along the convexities of the cerebral cortex extending into the sulci and often the basilar cisterns (Fig 2).

Space-Occupying Lesions: Contusion and DAI
Contusions occur as a result of mechanical compression of the brain tissue, such as often occurs after a very rapid acceleration, and tend to have the greatest impact to the orbital frontal region. These types of injuries frequently cause a corresponding injury in the diametrically opposite side of the brain, likely a result of a low-pressure area of injury. These types of injuries are frequently referred to as coup and contrecoup injuries (Fig 2). Interestingly, the contrecoup injury is often more severe than the coup injury. (46)

DAIs are deep white matter track injuries typically caused by rapid rotational acceleration of the brain content. These injuries result in axonal damage and often axonopathy (axon disconnection). Technically, DAI can be diagnosed postmortem only. However, a patient presenting with a closed head injury caused by a high-velocity impact and found to have a GCS score less than 9 is highly likely to have sustained DAI. (47) These lesions can have devastating morbidities, especially when they affect white matter tracts arising from the frontal lobes and connecting to the limbic system. The frontal lobes are the seat of important higher cognitive functions, including premotor planning, executive function, motivational states, and social behaviors. (48) It has been well

### TABLE 3. Pediatric Glasgow Coma Scale

<table>
<thead>
<tr>
<th>&lt;1 Y</th>
<th>&gt;1 Y</th>
<th>SCORE</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Eye opening</strong></td>
<td><strong>Motor response</strong></td>
<td><strong>Verbal response</strong></td>
</tr>
<tr>
<td>Spontaneously</td>
<td>Spontaneously</td>
<td>Spontaneously</td>
</tr>
<tr>
<td>To shout</td>
<td>To verbal command</td>
<td>Localizes pain</td>
</tr>
<tr>
<td>To pain</td>
<td>To pain</td>
<td>Flexion withdrawal</td>
</tr>
<tr>
<td>No response</td>
<td>No response</td>
<td>Decorticate</td>
</tr>
<tr>
<td>Spontaneous</td>
<td>Obey</td>
<td>Decerebrate</td>
</tr>
<tr>
<td>Localizes pain</td>
<td>Localizes pain</td>
<td>No response</td>
</tr>
<tr>
<td>Flexion withdrawal</td>
<td>Flexion withdrawal</td>
<td></td>
</tr>
<tr>
<td>Decorticate</td>
<td>Decorticate</td>
<td></td>
</tr>
<tr>
<td>Decerebrate</td>
<td>Decerebrate</td>
<td></td>
</tr>
<tr>
<td>No response</td>
<td>No response</td>
<td></td>
</tr>
<tr>
<td><strong>0–23 mo</strong></td>
<td><strong>2–5 y</strong></td>
<td><strong>&gt;5 y</strong></td>
</tr>
<tr>
<td>Smiles/coos</td>
<td>Appropriate words</td>
<td>Oriented</td>
</tr>
<tr>
<td>Cries, consolable</td>
<td>Inappropriate words</td>
<td>Disoriented, confused</td>
</tr>
<tr>
<td>Cries, inconsolable</td>
<td>Cries, inconsolable</td>
<td>Inappropriate words</td>
</tr>
<tr>
<td>Grunts, agitated</td>
<td>Grunts</td>
<td>Incomprehensible sounds</td>
</tr>
<tr>
<td>No response</td>
<td>No response</td>
<td>No response</td>
</tr>
</tbody>
</table>

established that subcortical injuries in these regions can result in significant neuropsychiatric impairments, including lack of motivation, depression, disinhibition, aggressivity, and cognitive impairments, especially in the area of motor planning. Intraparenchymal hemorrhages as a result of head trauma also occur frequently in the context of DAI, either as microbleeds or as much larger hemorrhagic lesions (Fig 2). 

Herniation Syndromes

Herniation syndromes occur as a result of a rapidly growing space-occupying lesion (over hours to days), such as a hemorrhage or developing edema. Herniation of the brain contents can occur around any of the major fixed intracranial structures, including the tentorium cerebelli (transient or uncal herniation), the falx cerebri (cingulate or subfalcine herniation), and the foramen magnum (tonsillar herniation). Each of these herniation syndromes has clinically distinct presentations (Fig 1 schematically illustrates each type of cerebral herniation; see also Table 4). It is important to emphasize that the herniation itself is a consequence, not the cause, of the clinical symptoms observed. The cause of the syndrome is in fact the downward pressure on the individual anatomical structures of the cingulate gyrus, midbrain, and medulla. (49)

Transtentorial Herniation

Transtentorial herniation is probably the most common herniation in the context of TBI. It occurs as a result of a mass effect in the supratentorial (above the tentorium cerebelli) region. Transtentorial herniation can be unilateral (uncal herniation) or bilateral (central herniation) (many authors refer to a bilateral tentorial herniation as transtentorial).

Unilateral (Uncal) Herniation. If there is a unilateral compression from a bleed or hemispheric edema, there is a risk of uncal herniation. In this case, the space-occupying lesion forces a displacement toward the opposite side to the lesion, resulting in compression of several critical structures below the tentorial incisura (ridge formed by the tentorium cerebelli). Critical structures compromised in this syndrome include the oculomotor nerve (CNIII) and the main motor pathways (pyramidal tracts). As a result, one will observe a unilateral mydriasis that will be ipsilateral to the herniation (compression of cranial nerve III) but contralateral to the lesion and a hemiparesis that will be contralateral to the herniation (compression of the pyramidal tracts before the decussation of the pyramids) but ipsilateral to the lesion. A classic pathologic finding that has been described in this context is the Kernohan notch, caused by compression of the cerebral peduncle against the tentorium opposite to the side of the space-occupying lesion.
lesion. It is important to recognize that anisocoria caused by transsentorial herniation is almost always accompanied by loss of consciousness as a result of a compression of the midbrain (including the periaqueductal gray and other structures essential for maintaining consciousness).

Bilateral (Central) Herniation. If there is diffuse cerebral edema or a large bleed as a result of bilateral and severe cerebral injury, transsentorial herniation may be bilateral, resulting from a downward compression force (aka central herniation). In this case the patient will first develop decorticate posturing (arms flexed and legs extended) as a result of midbrain compression, and as the herniation worsens, decerebrate posturing (arms and legs extended) with extension downward of the compression (a simple mnemonic is to remember decorticate posturing is caused by injury above the red nucleus and with the arms flexed and pointing toward the cortex, while decerebrate posturing is generally caused by injury below the red nucleus and the arms are extended and pointing away from the cortex). The pupils will initially be dilated and reactive. As the syndrome progresses they will become fixed and upward eye movement will be compromised (resulting in a “sunsetting appearance”).

Cingulate Herniation

Cingulate herniation (or subfalcine herniation) occurs as a result of the brain contents being displaced under the falx cerebri. This most often affects the frontal lobes as a result of a lateral rapidly growing mass lesion. The clinical signs of cingulate herniation are not as typical as those of the other herniation syndromes. Because the cingulate gyrus is compressed under the falx, the anterior cerebral artery may become compromised, with resultant ischemia of the medial motor...
cortex, leading to weakness of the contralateral lower extremity. However, compression of the foramen of Monro will eventually lead to increased intracranial pressure, and the patient will develop symptoms typically associated with uncal herniation.

Tonsillar Herniation

Tonsillar herniation is the least common type of herniation seen in the context of TBI (because the brain stem is much less frequently involved in TBIs). Tonsillar herniation results from mass effect in the brain stem. As a result of the mass effect, the cerebral contents will shift downward, forcing the cerebellar tonsils and the medulla through the foramen magnum. Initially the patient may complain of neck stiffness. On examination cranial neuropathies may be evident. As the syndrome progresses the children will often develop the Cushing triad: hypertension, bradycardia, and slow and irregular breathing from compression of the medulla oblongata.

Evaluation of the Child with Moderate to Severe TBI

Evaluation of the child presenting with TBI depends largely on the initial assessment. The management will be radically different depending on whether the child arrives with an mTBI (GCS score >13) or a moderate or severe TBI as evidenced by a GCS score less than 13 (for further details on the management of the child with an mTBI refer to the Recognizing Concussion subsection). The child with a decreased level of consciousness should be rapidly evaluated for symptoms of herniation while ensuring that airway, breathing, and circulation measures are addressed. The history in this case needs to be gathered rapidly but remains essential. A description of the trauma, the presence or absence of seizure activity, evidence of loss of consciousness at the time of impact, or change in the level of consciousness will all help guide the management of these children. In addition, any child presenting with a decreased level of consciousness or complaining of neck pain should immediately undergo cervical spine injury precautions with immobilization of the spine. If the GCS score is less than 9 or if the child’s mental status is fluctuating, an airway should be secured. The imaging modality of choice is CT because it is ideal to show both bone fractures and hemorrhage. However, DAI and contusion will not show as well on CT.

MRI versus CT

CT of the head is readily available in almost all emergency departments; it is a rapid study and is ideal to look for skull fractures or acute hemorrhages (acute hemorrhages will appear hyperdense on CT). In addition, if necessary, a CT of the C-spine can be added to evaluate for spinal fractures. The CT will also reveal areas of edema as evidenced by hypodense lesions, although these changes can take some time to become evident. It follows that a negative CT scan may require follow-up with MRI to fully characterize the lesion depending on the clinical presentation. However, CT is a poor study to evaluate for contusion or evidence of DAI. Furthermore, CT is associated with a significant amount of ionizing radiation. Note that the dose reduction techniques now commonly in use have resulted in significantly less dose exposure (refer to the Recognizing Concussion subsection for further discussion of imaging in mTBI).

MRI offers a better evaluation of hemorrhage. T1, T2, and T2 fluid-attenuated inversion recovery (FLAIR) series are

### TABLE 4. Summary of the Major Herniation Syndromes and Clinical Manifestations

<table>
<thead>
<tr>
<th>HERNIATION</th>
<th>CLINICAL PRESENTATION</th>
<th>ASSOCIATED CEREBRAL PATHOLOGY</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cingulate</td>
<td>Symptoms secondary to anterior cerebral artery compression and stroke include contralateral foot paresis and numbness, abulia, and urinary incontinence. As the syndrome progresses, uncal herniation may occur.</td>
<td>Cerebral lateral compressive mass such as epidural or subdural bleed.</td>
</tr>
<tr>
<td>Uncal</td>
<td>Ipsilateral pupillary dilation (cranial nerve III), contralateral hemiparesis (cerebral peduncles). Note that the mass compressing the brain will often be contralateral to the herniation. Always associated with altered consciousness.</td>
<td>Cerebral lateral compressive mass such as epidural or subdural bleeding compressing the cerebral peduncle against the tentorium toward the side opposite the mass (will result in a Kernohan notch).</td>
</tr>
<tr>
<td>Central</td>
<td>Initially bilateral fixed pupils, impaired upward eye movements (sunsetting appearance), decorticate followed by decerebrate posturing as the syndrome progresses.</td>
<td>Bilateral cerebral compressive mass such as diffuse TBI with associated edema and/or ischemia resulting in downward compression of the midbrain.</td>
</tr>
<tr>
<td>Tonsillar</td>
<td>Neck stiffness, cranial neuropathies, Cushing triad (hypertension, bradycardia, and irregular breathing).</td>
<td>Rare in the context of TBI, brainstem mass.</td>
</tr>
</tbody>
</table>

TBI = traumatic brain injury.
best to evaluate for extra-axial hemorrhages. T1 and T2 sequences are also useful in dating the age of the hemorrhage. More subtle parenchymal injuries, shearing injuries, and microhemorrhage are better seen using susceptibility-weighted imaging. T2 FLAIR images are considered the most sensitive MRI sequences to look for subarachnoid hemorrhage, although CT is very sensitive for acute subarachnoid hemorrhage as well. Edema and ischemia are best evaluated with T2, FLAIR, diffusion-weighted imaging, and apparent diffusion coefficient sequences. In addition, diffusion- and susceptibility-weighted imaging will often reveal evidence of shearing injury or DAI that may have been missed on T1 or T2 sequences. Furthermore, MRI does not expose the children to ionizing radiation. However, MRI is time intensive and is not available in all hospitals 24 hours a day.

Management
In 2012, the second edition of the guide for the acute medical management of severe TBI in infants, children, and adolescents was published. (50) These guidelines were endorsed by multiple organizations, including the AAP, the Child Neurology Society, and the American Association of Neurological Surgeons. Although a detailed discussion of the management of the child with severe TBI is beyond the scope of this review, it is worth mentioning a few basic principles from these guidelines.

First and foremost, children with TBI are at very high risk for elevated intracranial pressure. This is especially notable in children with evidence of diffuse cerebral swelling on CT, a finding noted to be 75% specific for the presence of intracranial hypertension. Children with an altered level of consciousness are also at markedly increased risk for elevated intracranial pressure. These children need urgent management in an ICU setting.

Finally, a word must be said about the use of prophylactic antiepileptic medications. The guidelines cited previously herein note that the risk of posttraumatic seizures in the pediatric patient is approximately 10%. Based on a single study the guideline states that prophylaxis with phenytoin may be considered. However, although phenytoin and fosphenytoin remain the most commonly used antiepileptic medications in the prevention of posttraumatic seizures, there is accumulating evidence that levetiracetam is an excellent alternative. (51) Levetiracetam is available in an intravenous formulation, does not have a significant protein-bound fraction (as opposed to phenytoin that is 96% protein bound), has essentially no liver metabolism, and has very few significant drug interactions.

Special Considerations: Abusive Head Trauma
Inflicted TBIs have been labeled by a variety of different names, including nonaccidental trauma and shaken baby syndrome. In 2009, the AAP recommended replacing all of these terms with the nomenclature abusive head trauma (AHT). (52) TBI inflicted on a child by an adult is unfortunately too frequent an occurrence. According to a recent CDC publication on AHT, it is the leading cause of head injury in infants. (53) Peak incidence occurs between the ages of 2 and 3 months, and AHT is rare after 2 years of age. The mortality and morbidity of AHT are considerable, with up to 20% of children succumbing to their injuries and two-thirds of the surviving infants having severe and permanent intellectual and physical impairments. The most common presentation of AHT is an infant or toddler with a decreased level of consciousness and/or seizures. Frequently in such situations there will be conflicting or vague histories from the parents or guardians. In addition, there may be a clear disconnect between the apparent severity of the injury and the described mechanism (“fell off a chair and hit his/her head on the floor” would be a very uncommon cause of TBI). One should also look for inappropriate affect in the adults accompanying the child. The evaluation of the child with suspected AHT should include an urgent CT scan of the head. Children with these types of injuries very frequently have a combination of subdural hemorrhages (54) and significant DIA (as evidenced by hypodense lesions on the CT scan). (55) In addition, skull fractures may be observed, but it is important to emphasize that the absence of skull fracture does not rule out AHT. Once there is suspicion of AHT and the child is clinically stable one should obtain a skeletal survey to look for evidence of new or old bone fractures elsewhere and a dilated funduscopic examination to look for evidence of retinal hemorrhages.

Of note, there are extremely rare metabolic disorders that are associated with spontaneous subdural hemorrhages. It is important to keep these conditions in mind when considering the differential diagnosis of AHT. These conditions include coagulation disorders, galactosemia, and glutaric aciduria. (56) Glutaric aciduria, galactosemia, and coagulation disorders can all be ruled out with relatively simple laboratory studies. Another condition to consider is Menke disease because it has been associated with spontaneous subdural hemorrhages. (57) However, this disorder should be evident based on the child’s clinical appearance (severe developmental delay, epilepsy, and unusual and brittle appearance of the hair). One should also recognize that spontaneous subdural hemorrhages have been observed in children with benign extracerebral collections in infancy and minimal or no evidence of trauma. (58) Benign enlarged extracerebral spaces have been referred to by many names, including benign external hydrocephalus, benign communicating hydrocephalus, subdural hygroma, and others. It usually occurs in
CONCLUSION

TBI encompasses a broad spectrum of clinical presentations. These injuries represent a major challenge for the primary care physician because the prognosis varies from excellent, with complete resolution in a few days to a few weeks with mTBI, to catastrophic, with severe morbidity and mortality in the case of moderate to severe TBIs. However, a systematic approach to the child who experiences a TBI should help in the proper clinical management. Guidelines exist for the management of adult head injury (National Institute for Health and Care Excellence’s Head Injury: Assessment and Early Management [https://www.nice.org.uk/guidance/cg176]). These guidelines have been found to be highly reliable and easily adaptable to multiple environments, including low- and middle-income countries. (59) The CDC has now published guidelines for the diagnosis and management of mTBI in children. (10) The introduction and adoption of these guidelines will help resolve much of the guesswork that many primary care providers have to consider when evaluating children who have sustained some form of head trauma. (59)

Suggested Quality Improvement Project

With the increasing numbers of children presenting to emergency departments with TBIs, it is more important than ever that providers appropriately assess children to determine whether head CT is needed and to avoid unnecessary and harmful radiation exposure. Fortunately, there is a well-validated set of clinical criteria developed by the PECARN to assist in this decision-making process. (18) Previous efforts to implement these criteria in a community emergency department have resulted in a reduction in rates of head CT scan in children presenting with head trauma, (19) indicating that this is an excellent opportunity for further quality improvement projects.

Project: Reduction of head CT scan rates in children presenting with head injury.

Setting: Emergency department.

Prediction tool: PECARN prediction rule for clinically important TBIs.

Outcome Measure: Rate of pediatric head CTs in children presenting with TBI.

References for this article are at http://pedsinreview.aappublications.org/content/40/9/468.

Summary

- Traumatic brain injuries are the leading cause of death or severe disability in children older than 1 year, and the incidence is continuing to increase, making this topic especially relevant for the pediatrician.
- Based on a well-designed prospective cohort study, it is not recommended that all children presenting with head trauma obtain a head computed tomographic scan. (18) Rather, this decision should be based on the mechanism of injury and the signs and symptoms of the patient.
- Based on multiple cohort studies and expert opinion, concussion symptom checklists, such as the Child Sport Concussion Assessment Tool, the Postconcussion Symptom Scale, or the Graded Symptom Checklist, can be used to aid in concussion diagnosis and tracking symptom resolution in determining graduated return to play. (6)(11)(13)
- Children with moderate or severe traumatic brain injury are at high risk for elevated intracranial pressure; this is especially true of children presenting with altered consciousness. (50)
- Abusive head trauma is the leading cause of head injury in infants. The morbidity and mortality of abusive head trauma are considerable, with up to 20% of the infants succumbing to their injuries and two-thirds of the survivors having significant cognitive and/or physical impairments. (53)
1. A 6-year-old girl is seen in the office after falling from her bicycle and striking her unhelmeted head yesterday. She did not lose consciousness at the time of injury, but ever since she has complained of headache and her mother feels that she is “not acting herself.” An evaluation for the presumed diagnosis of mild traumatic brain injury, also known as concussion, is begun. Which one of the following is part of the COACH CV mnemonic for diagnosis of concussion?
   A. Cardiovascular anomalies.
   B. Head imaging normal.
   C. Loss of Consciousness.
   D. Orbital injury.
   E. Vomiting.

2. You are participating as a sideline medical staff for an elite youth soccer team. A 14-year-old girl collided with another player and her head struck the other player’s knee. She believes she lost consciousness for a few seconds, but she is verbally responsive when you evaluate her on the field. She complains of headache but is alert and oriented, with no neurologic deficits on your sideline physical examination. You pull her from the game, but she asks when she can return to her workouts. Which one of the following is the best guidance for the time she must be on complete rest, after which she can return to noncompetitive physical activity?
   A. Immediate return to activity.
   B. 2 days.
   C. 5 days.
   D. 7 days.
   E. 10 days.

3. A 7-year-old boy is brought to the office for follow-up after he was seen in the emergency department for a concussion 1 week earlier. He was discharged with a standardized symptom assessment checklist, which his mother has been performing daily. His mother is concerned that he complains of headaches and seems “out of it” and short-tempered. She wants to know if this is going to be a prolonged recovery. Which one of the following is a risk factor for postconcussive syndrome?
   A. Attention-deficit/hyperactivity disorder or learning disabilities.
   B. History of supraventricular tachycardia.
   C. Male sex.
   D. Nonsport mechanism of injury.
   E. Preadolescent age.

4. An 11-year-old boy is being seen for routine well-child care. His mother is concerned that he has had 2 concussions in early childhood and is reluctant about letting him participate in sports. Which one of the following sports would you advise her that he plays because of its lowest risk of concussion?
   A. Biking.
   B. Football.
   C. Skiing.
   D. Soccer.
   E. Volleyball.
5. A 14-year-old boy is being evaluated in the emergency department after a fall from a roof (approximately 20 feet). His vital signs include heart rate of 55 beats/min, respiratory rate of 29 breaths/min, and blood pressure of 140/90 mm Hg. He has a large laceration to the forehead and a bloody nose. He is minimally responsive, with occasional moans and no purposeful movement of his extremities. On secondary survey you note that the left pupil is larger than the right and poorly responsive. In addition, he has weakness of the left arm and leg. Which one of the following findings would be expected on head imaging given the clinical presentation?

A. Bilateral central herniation.
B. Cingulate herniation.
C. Diffuse axonal injury.
D. Tonsillar herniation.
E. Uncal herniation.
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