Pediatric Minor Closed Head Injury

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Trauma is the leading cause of death and disability in childhood, with approximately 3000 deaths, 50,000 hospitalizations, and 650,000 emergency department (ED) visits per year in the United States [1]. The term concussion has been used interchangeably with mild head injury (MHI) or mild traumatic brain injury (TBI) and has been defined as a trauma-induced alteration in mental status that may or may not involve the loss of consciousness (LOC). Controversy exists over the use of clinical predictors of intracranial injury in pediatric MHI; nevertheless, authors have devised decision trees to help guide the management of minor head injury. Many authors recommend imaging younger children because these children have a higher risk of significant brain injury after blunt head trauma. Neuropsychologic evaluation can detect more subtle changes associated with concussion, and neuroimaging studies may be needed in more severe concussions. Management and return-to-play guidelines have been developed for sports-related concussions of different grades.

Epidemiology

Pediatric MHI comprises a large portion of pediatric trauma cases seen in emergency departments. Among children aged 0 to 14, TBI results in over 400,000 ED visits each year. Injury rates are highest among children 0 to 4. One to two percent of all pediatric patients seen in the ED present with minor head trauma, but only 3% to 5% of those patients have intracranial injury (ICI), and less than 1% of these patients require any neurosurgical intervention [2]. Among recent studies, the incidence of ICI varies from 5% [3] to
25% [4] of neurologically normal children. Over 85% of the 1.5 million TBIs (all ages) occurring annually in the US are considered “mild” [5]. In a recent study of TBI epidemiology of all ages, the average incidence of mild TBI was 503.1 per 100,000 population, with a peak among American Indians/Alaska Natives (1026/100,000 population) and in children younger than 5 years old (1115.2/100,000 population). The mechanisms by which children sustain head injury vary by activity, age, helmet use, and geographic location.

Falls

Many studies of pediatric head injury cite falls as the most common mechanism of injury, ranging from 32% to 91% [6–9]. In a recent epidemiologic study [5] of mild TBI among all ages, the authors found that the mechanism of injury varied considerably by age, with falls frequent at the extremes of age and assaults and motor vehicle trauma in the middle-aged groups. In one retrospective study of children less than 2 years old, 89% of the patients sustained a fall as the mechanism of isolated skull fracture, in which 60% were free falls (from adults arms, tables, beds, bathtubs, and banisters) and 30% were stair falls. Direct falls from heights greater than 3 feet and stair falls are more likely to result in ICI than falls less than 3 feet. However, there is still a 2% to 7% risk of ICI in the infants who fall less than 3 feet [8,10,11]. Although falls may be the most commonly cited mechanism of injury in pediatric head trauma, motor vehicle accidents have been reported to have more significant intracranial pathologies [6].

Infant walkers have remained a threat to child safety when they are not used in safe locations or under close supervision. Thirty-four deaths were reported from infant walker accidents from 1973 to 1999 [12]. Stairs were implicated in 75% to 96% of these accidents. Because of insufficient safety regulations and a high risk of head injuries, the American Academy of Pediatricians (AAP) recommends a ban on the manufacture and sale of mobile infant walkers. The AAP offers stationary play centers as an alternative to mobile walkers [12].

Bicycle helmet legislation has become a strong strategy for head injury prevention among both children and adults. One thousand people die annually from bicycle crashes, and 65% of bicycle-related head injury deaths occur in children less than 15 years old [13]. The Cochrane Collaboration [14] systemic review reported that helmets reduce the risk of head injury up to 88% among cyclists. In a systematic review of five case-controlled trials in which there were 7253 head-injured cyclists (all ages), helmet use was associated with a risk reduction of 65% to 88% from head and brain injury [15].

Pathophysiology of mild head injury

Variations in the definitions of MHI can confuse the way in which patients are classified, evaluated, and treated. The term concussion has been used interchangeably with MHI or mild TBI. If MHI is viewed on an injury severity
spectrum, concussion usually refers to milder injuries. The definition used more commonly is by the American Academy of Neurology (AAN): “Concussion is a trauma-induced alteration in mental status that may or may not involve loss of consciousness. Confusion and amnesia are the hallmarks of concussion.” [16]. The Head Injury Interdisciplinary Special Interest Group of the American Congress of Rehabilitation Medicine (ACRM) has adopted criteria to help define MHI [17,18]. Further discussion of concussion and mild TBI continues in the sports-related head injury section below.

Head injuries can result from different types of forces. Impact, acceleration–deceleration, and rotational forces directly affecting the head may cause skull and scalp injuries, neural tissue damage, or cerebral vasculature injuries. Brain injury can be subdivided into primary and secondary injuries. Primary brain injury is the result of a direct force to the brain. The severity and location of the primary brain injury dictates the patient’s immediate level of consciousness and mental status. Primary brain injury can lead to impaired autoregulation of cerebral blood flow (CBF). Altered CBF regulation, bleeding contusions, and blood–brain-barrier breakdown contribute to brain swelling. Secondary brain injury subsequently results from the traumatic event and multiple interrelated pathophysiologic processes. The loss of consciousness after head injuries, the development of secondary brain damage, and the vulnerability of the brain after an initial insult can be explained by ion fluxes, acute metabolic changes, and CBF alterations [19]. Biochemical mediators involved in secondary brain injury involve excitatory amino acids, free radicals, and opiate peptides [19]. Massive increases in extracellular potassium concentrations can lead to the inhibition of action potentials and ultimately to the loss of consciousness. Sometimes, it may require several seconds or longer for the potassium levels to increase above the threshold level, which can explain the delay to loss of consciousness when an athlete walks off the field and collapses [20]. Delayed brain swelling is a major cause of elevated intracranial pressure, which can lead to brain herniation and death. From minutes to hours after the brain injury, a total-body hypermetabolic state begins, with a marked increase glucose use. However, after the initial phase, cerebral hypometabolism evolves, with decreased protein synthesis and reduced oxidative capacity. Cells are more susceptible to death after a second insult [20]. The final common pathway involves impaired glucose and oxygen delivery to neurons and ultimately neuronal cell death [19].

Children’s heads are disproportionately larger in comparison with their body size. Approximately 72% of the adult intracranial volume of 1200 to 1500 mL is achieved by the age of 2 years. The intracranial volume approaches 90% the adult volume by the age of 8 years, and by adolescence, 96% of the volume is achieved. The sphenoid and petrous bones provide a buttress support to pediatric skulls, thus conferring an “architectural advantage” [21]. In addition, the cranial sutures function as joints in infants, allowing for a small degree of movement in response to a mechanical stress [21].

The parietal bone is the skull bone most commonly fractured (approximately 60%–70%), followed by the occipital, frontal, and temporal bones [22]. Fractures
are classified as linear, depressed, compound, or basilar. Linear fractures are the most common. In a depressed skull fracture (SF), the bone fragment is depressed below the inner skull table. “Ping-pong” fractures are a special variant of depressed SFs, in which the inner and outer skull table can be dented like a ping-pong ball. These fractures are seen more commonly in a newborn when the cranium is less well mineralized and more prone to distortion. Compound SFs are “open” fractures exposed through the full-thickness scalp laceration. Fractures that have an increased association with inflicted head injury can present as multiple, linear, and complex. These fractures may be comminuted or stellate [23].

The basilar SF is a fracture at the base of the skull involving the temporal or parietal bone. Clinical signs of a basilar SF include CBF rhinorrhea, CSF otorrhea, ecchymosis over the mastoid bone (Battle’s sign), periorbital ecchymosis (raccoon eyes), and hemotympanum or cranial nerve seven palsies. Fractures may extend into the petrous portion of the temporal bone and cause tears of the tympanic membrane, disruption of the auditory ossicles, and facial nerve injury [23]. Complications of basilar SFs can lead to permanent hearing loss if the fracture involves the cochlear–vestibular apparatus or facial nerve palsy. Undetected CSF fistulas can form as a result of a dural tear in association with a sinus fracture. Meningitis is a source of potential meningitis. The incidence of meningitis in basilar SF ranges from 0.7% to 5%, and in patients who do not have intracranial hemorrhage and Glasgow coma scores (GCS) of 13 to 15, the rate is 1% [22].

Clinical Indicators of intracranial injury

A physician’s goal in evaluating head trauma is to accurately identify and diagnose patients who are at risk for serious injury and complications. The management of primary brain injury demands early diagnosis and effective treatment to prevent secondary brain damage. In patients with depressed consciousness or focal neurologic signs, the ICI is easier to diagnose than in fully conscious patients, in whom an evolving ICI can be difficult to recognize [7]. Therefore, it is essential that the risk factors used to predict ICI are reliable. Many of the recommendations that physicians have followed are based on limited data and small study samples. More recent data are now available that involve prospective cohort studies with larger patient populations.

Physicians must determine which patient is at risk for ICI and who would benefit from head CT. Unfortunately, no predetermined set of clinical criteria exist that accurately and reliably predict the presence of ICI. The assessment of the GCS, LOC, mechanism of injury, and neurologic disability can help guide the physician in the evaluation and management. On examination, the physician needs to determine if there has been any change in mental status or other physical parameters from the time of the initial assessment, whether it was carried out by parents or paramedics.
Davis and colleagues [24] suggest that a normal mental status (GCS 15) after an isolated head injury does not warrant a head CT and that the child is not at risk for ICI. A similar suggestion is that only children with LOC, decreased GCS, or SFs should undergo head CT [9]. On the other hand, other studies have shown that historical and clinical factors are poor predictors of ICI. Even children with apparently minor head trauma and normal mental status can suffer ICI [4,25].

Pediatric patients can be difficult to assess clinically in terms of their mental status, behavior, and neurologic function. Therefore, much controversy exists over how to triage, evaluate, and manage these minor head injuries. Abnormal neurologic examination suggestive of ICI have signs and symptoms of headache, vomiting, drowsiness, irritability, amnesia, visual disturbance, focal neurologic signs, dizziness, altered consciousness, or signs of a fractured skull base [7]. An abnormal mental status in pediatric blunt head trauma should be considered if the patient has a GCS less than 15 or if the patient is confused, somnolent, repetitive, or slow to respond to verbal communication [1].

Many studies have attempted to establish clinical predictors of ICI, and although much progress has been gained, controversy still exists surrounding the management of patients with GCS 13 to 15 and brief LOC. A defined set of screening clinical criteria has not been validated or standardized, but many authors suggest clinical predictors of ICI [7,9,26]. The presence of a neurologic deficit is highly suggestive of an ICI [6,7,26,27]. Lloyd and colleagues [7] observed the presence of neurologic abnormalities had greater sensitivity and negative predictive value for ICI than did the presence of a SF. Similarly, Brown and colleagues [11] reviewed head-injured children less than or equal to 10 years old who underwent neurosurgical procedures. Altered mental status was the most common sign or symptom and was identified in 85% of the ICI patients.

Several studies of pediatric head injury have determined clinical predictors of ICI. Quayle and colleagues [27] prospectively studied a cohort of children who had nontrivial head injuries and identified five clinical predictors of ICI (altered mental status, focal neurologic deficit, signs of basilar SF, a SF, and LOC for \( \geq 5 \) minutes). There was also a trend toward an association between seizures and ICI, but it was not statistically significant. Nevertheless, over half of the children with ICI had a normal mental status and no focal abnormalities, two thirds of whom were infants. Taking this into consideration, the authors recommend scanning children who present with the study’s five identified clinical predictors as well as children with symptoms such as vomiting, amnesia, LOC, headache, and drowsiness. Although studies have determined independent predictors of ICI, children with ICI may present with subtle signs, especially infants younger than 1 year [27].

Clinical decision rules to predict ICI have been shown to be useful in pediatric populations. Palchak and colleagues [1] preformed an observational cohort investigation of over 2000 children younger than 18 years of age, 62% of whom underwent CT of the head, and found that 7.7% had TBI on CT scans, and 5% had a TBI requiring acute intervention. The study derived a decision rule that demonstrated high sensitivity for TBI and high negative predictive value for
identifying children without TBI. The authors combined the data of the two
decision trees for recognizing TBI on CT scans and TBI requiring acute intervention
(differing only by one clinical predictor). This combined decision tree identified
97 of 98 (99%) children who had TBI on CT, and 105 (100%) children who had
TBI requiring acute intervention. The authors conclude that important factors for
determining children at low risk for traumatic head injuries include the absence of
an abnormal mental status, clinical signs of skull fracture, a history of emesis,
scalp hematoma, and headache [1].

A normal mental status or the absence of neurologic deficit does not exclude
a serious brain injury. Studies have determined conflicting evidence on the
reliability of neurologic signs and symptoms for TBI. Some authors have found
that a detailed clinical examination is of no diagnostic value in detecting
ICI found on head CT [26,28,29]. This finding has led many practitioners to
recommend head CT in patients in whom LOC or amnesia and abnormal mental
status are observed as part of the evaluation, to avoid missing an ICI [29]. Several
studies have evaluated “most well” pediatric head trauma patients and have
determined that neither historical variables (history of seizure, LOC, amnesia, or
confusion) nor ongoing complaints (vomiting, headache, amnesia, sleepiness, or
irritability) are associated with ICI [4,6,8]. In the study by Dietrich and col-
leagues [6], LOC, amnesia for the event, a GCS of less than 15, and the presence
of neurologic deficit were significantly more common in children with ICI.
Nevertheless, no single characteristic consistently identified children with ICI
because 5% of the neurologically intact patients on presentation had CT scan
results that were positive for intracranial pathology.

Glasgow coma scores

GCS scores of 13 to 15 have been considered reflective of MHI, yet patients
with ICI can present with these minor scores. In one study among children with
GCS 13, 14, or 15, the incidence of intracranial pathology was 33%, 11%, and
5%, respectively [1].

GCS scores can vary depending on the health care professional making the
assessment and whether the pediatric GCS system is used. Many times, the adult
GCS score is used to evaluate an infant when the pediatric GCS should be used.
Hahn and McLone [28] discuss the fact that verbal responses to the GCS system
cannot be applied to children under 3 years old. They recommend using the
Children’s Coma Scale (CCS), which modifies the best verbal response portion
of the GCS using the same scale of 3 to 15. Such a scale takes into consideration
the different responses that can be expected in a preverbal child (for a more de-
tailed discussion of the Children’s Coma Scale, see Hahn, 1993 [28]). Using this
scale, pediatric patient responses can be compared with adult responses [28]. In
a recent study of pediatric head trauma in children under 2 years of age, the
pediatric GCS performed significantly better than the standard GCS applied to
older children [30].
The application of the GCS has had mixed results. Wang and colleagues [31] have observed that the distribution of GCS scores did not differ substantially between children who accidentally fell from high- versus low-level heights. Confounding this observation is the finding that 60% of pediatric patients with intracranial hemorrhages were more likely to have improved scores than deteriorated scores in their GCS [25]. However, despite the inconsistency with minor head injury, more severe injuries do correlate with lower coma scores. In a study of head-injured children who required neurosurgical procedures, almost 70% had a GCS less than or equal to 8 [11]. Unfortunately, a normal coma score does not preclude ICI because as many as 28% of patients with ICI are neurologically intact [6]. Ultimately, significant injuries may still exist despite a normal neurologic examination, GCS 13 to 15, or no documented LOC.

**Loss of consciousness**

Because LOC is believed to be a reliable predictor of ICI, physicians frequently use a history of LOC as an indication to perform CT for the evaluation of head trauma [22,32,33]. Many studies have demonstrated that LOC is a significant indicator of TBI after blunt head injury [3,6,27]. Palchak and colleagues [1] found the risk of TBI to be higher in pediatric patients with a history of LOC or amnesia [34]. In one study of SFs in infants, lethargy (or altered level of consciousness) was the single sign and symptom that was the best positive predictor of ICI (78%) [9]. Conversely, several studies have observed that neither the absence nor the presence of LOC is a significant predictor of negative CT findings [10,24,35]. In the Gruskin and Schutzman study [10], a significant association was found between a depressed level of consciousness and SF or ICI. However, over 90% of the children with isolated SF and 75% of children with ICI (with and without SF) had normal levels of consciousness and nonfocal neurologic examination findings.

**Scalp abnormalities**

The incidence of SF in children with MHI ranges from 29% to almost 49% [28,35]. Many physicians use the presence of scalp abnormality to dictate ordering radiographic studies. Studies have identified abnormal scalp examinations and SF as important predictors of underlying ICI [1,8,10,24]. The physical finding of a scalp abnormality has been found to have high sensitivity (94%) and negative predictive value (97%) for SF and ICI, and the absence of scalp findings reduces the risk of SF and ICI [10]. In a retrospective study of 401 children with or without brief LOC, a deep scalp wound was the only clinical finding that correlated significantly with positive CT scan findings [35].
In one study by Greenes and Schutzman [36], scalp hematoma size and location and patient age were useful indicators of SF and underlying ICI in asymptomatic head-injured infants (0–24 months old). Patients with a large or moderate scalp hematoma were significantly more likely to have SFs than patients without scalp hematomas. Parietal and temporal hematomas were both highly associated with SF, and patients with occipital hematomas had a somewhat increased risk for fracture. Younger infants, especially less than 3 months old, had a higher propensity for SF even in the absence of scalp hematoma. A multivariate analysis determined that only a hematoma in the parietal area served as an independent clinical predictor of SF and of ICI. The authors used their data to formulate a scoring system to assess for the risk of SF based on hematoma size and location and patient age. Higher scores, therefore, and a higher risk of SF were assigned to parietal hematoma, large hematoma, and an age less than 3 months. Their decision rule had a sensitivity of 98% and specificity of 49% for detecting SF [36].

However, SFs do not have to be present for there to be an underlying head injury [7,25,27,37]. Quayle and colleagues [27] have found that the relative risk of ICI increased almost fourfold in the presence of a SF; however, the absence of a SF does not rule out ICI because almost 50% of children with ICI did not have fractures. Similarly, minor craniofacial soft tissue injury (hematoma or laceration) is a significant risk factor for ICI, but it has marginal predictive values [37].

**Clinical indicators and management in children less than 2 years old**

The evaluation of the pediatric patient also depends on the age of the patient. Historically, children less than 2 years old have been studied and clinically categorized differently than children older than 2 years. The AAP has published guidelines on the management of minor closed head injury (CHI) in children 2 to 20 years old, but children less than 2 years were not included [33]. Previous authors have excluded children less than 2 years old from their study protocols, which may dampen the portion of children identified with ICI. Children in their first 2 years of life also have been described as being at higher risk for significant brain injury after blunt head trauma. Therefore, infants traditionally have been evaluated differently than older children after head trauma. For example, Masters and colleagues [38] suggest that the age of less than 2 years itself be considered a moderate risk factor for ICI after head trauma. Some authors emphasize a lower threshold as an indication to order imaging studies of younger children because historical and clinical factors may not be available or present for assessment [6]. Many authors make broad recommendations for evaluating infants because even neurologically intact infants may have ICI [7,8,27].

Younger age can be a strong criterion for identifying the risk of ICI. High incidences of SF and ICI have been found in studies of head-injured children less than 2 years old [6,10,39], with a range from as low as 3.4% [10] to as high
to 30% [39]. A multidisciplinary expert panel [32] has defined the factors considered to be higher risk for delayed complications: younger age (especially infants 3–6 months old, who can be more difficult to assess and may lose a significant amount of blood into large scalp hematomas), large scalp swelling, fractures resulting from a high-energy mechanism, and threatening fracture locations (crossing a suture or dural venous sinus, vascular groove, or extending into the posterior fossa) [32].

Some studies have found clinical signs and symptoms to be poor indicators of ICI in infants, and occult ICIs are more common among younger infants [6,33,40]. In the Greenes and Schutzman [40] study of occult ICI in infants less than 2 years old, children were considered symptomatic if they had LOC, a history of behavior change, seizures, vomiting, bulging fontanel, retinal hemorrhages, abnormal neurologic examination, depressed mental status, or irritability. Of the 101 infants studied, 19% had occult ICI. All occult ICIs occurred in infants younger than 1 year, and 95% of the occult ICIs also had SFs.

Although clinical signs and symptoms may be good indicators of either SF or ICI, they are insensitive predictors of head injury in children less than 2 years of age. The combination of the following variables was found in one study to be predictive of SF/ICI: age younger than 12 months, height of fall greater than 3 feet (0.9 m), and scalp abnormality [10]. However, among the subset of children who had fallen and undergone a nonfocal neurologic examination, this combination of variables would identify only 66% of children younger than 2 years with SF/ICI and 72% of children younger than 12 months. Sixty percent of children with SF/ICI had no history of LOC, emesis, seizure, or behavioral change. Furthermore, more than 90% of children with isolated SF and 75% of those with ICI (with or without SF) had nonfocal neurologic examination findings. Therefore, physicians should have a high suspicion for SF/ICI in any child 2 years or younger who has sustained head injury, especially in children younger than 12 months in whom complications are more common and clinical findings are less reliable [10].

Schutzman and colleagues [32] have devised a management strategy that categorizes children less than 2 years old into four subgroups based on the risk of ICI (Box 1). The first group consists of those at high risk for ICI, in whom CT is indicated. The second group consists of those at some risk for ICI, with potential indicators of brain injury in whom CT or observation is indicated. The third group consists of those without potential indicators of brain injury who are at some risk for SF or ICI, in whom CT, skull radiographs, observation is indicated. The fourth group consists of those at low risk for ICI, for whom imaging is not necessary. Generally, the authors emphasize that the younger the patient, the lower the threshold for imaging. The greater the severity and number of historical symptoms and physical signs, the stronger the consideration should be for obtaining an imaging study. Physicians most importantly need to individualize care based on the patient and the unique clinical situation [32].

In conclusion, many authors agree that the greater the forces involved (MVCs, falls from greater heights or onto harder surfaces) and the more pronounced the
Box 1. Management strategy for children less than 2 years old based on the risk of ICI

*High risk for ICI: CT scan is recommended*

1. Depressed mental status
2. Focal neurologic findings
3. Signs of depressed or basilar SF
4. Acute SF by clinical examination or by skull radiographs
5. Irritability (not easily consoled)
6. Bulging fontanel
7. Seizure
8. Vomiting $\geq 5$ times or $\geq 6$ per hour
9. $LOC \geq 1$ minute

*Intermediate risk with any potential indicators of brain injury: recommend CT or observation*

1. Vomiting, 3 to 4 episodes
2. Transient LOC $\leq 1$ minute
3. History of lethargy or irritability, now resolved
4. Behavior not at baseline, reported by caretaker
5. Nonacute SF ($\geq 24$ hours old)

*Intermediate risk with concerning or unknown mechanism or findings on clinical examination that may indicate underlying SF: recommend an imaging procedure (CT, skull radiography, or both) or observation*

1. Higher force mechanism (high speed motor vehicle crash or ejection, falls $\geq 3$ feet)
2. Falls onto hard surfaces
3. Scalp hematoma (especially if large, boggy, or located in temporoparietal area)
4. Unwitnessed trauma
5. Vague or absent history of trauma in the setting of signs or symptoms of head trauma (should raise the suspicion of child abuse or neglect)

*Low risk for ICI: recommend observation*

1. Low-energy mechanisms (fall $\leq 3$ feet)
2. No signs or symptoms more than 2 hours since injury
3. Older age is more reassuring (especially age $\geq 12$ months)
physical findings, the greater the risk of ICI. Younger children have a higher incidence of complications, asymptomatic ICI and are more difficult to assess.

**Imaging**

*Skull radiography*

Physicians continue to debate the use of skull radiographs to detect underlying head injury. Traditionally, skull radiographs were used to detect a fracture in a child with physical signs of a SF (eg, scalp hematoma). If a fracture was detected by radiography, the follow-up was to perform a head CT. Unfortunately, skull radiographs do not detect underlying brain injury. On the other hand, SFs have been found to be sensitive indicators of ICI in children less than 2 years old [8,27,32,41]. In fact, the presence of a SF is one of the strongest predictors of ICI in children less than 2 years old [32]. The benefits of skull radiographs are that they are readily available and do not require procedural sedation. The value of skull radiographs depends on the accuracy of interpretation because false-positives can stem from open sutures or vascular grooves that appear similar to fractures and vice-versa [41]. Critics of using skull radiography as a screening technique argue that misreading films can incorrectly classify a child as low risk because the fracture has not been identified [41]. Conversely, a child may unnecessarily undergo a head CT when the radiograph incorrectly identifies a fracture.

Studies of pediatric head injury have found that almost half of the ICI occur without a SF present on radiography [25,27]. Additional studies on the ability of plain radiographs to predict ICI have shown a sensitivity of 65% and a negative predictive value of 83%. With a high failure rate of detecting a SF on radiography, there is a large risk of missing a serious brain injury [7]. This high failure rate and poor prediction of underlying ICI have led many investigators to recommend not obtaining skull radiographs when CT is available. However, if CT is not available, skull radiographs can provide screening information, and authors do recommend radiographs in infants younger than 1 year with scalp hematomas or contusions because these infants are at greater risk of SF [27].

*CT imaging*

To avoid imaging every patient, physicians must decide which child is at risk for ICI and who would benefit from a head CT. The guidelines are clear for

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*a Represents expert consensus.

children with abnormal examination findings, altered mental status, or major mechanisms of injury. However, the guidelines are less clear for children who present with normal neurologic and physical examination results. Some authors have suggested specific clinical criteria that are indicative of ICI, and these children should have a CT scan [1,2,7,9,27], yet other authors have found clinical criteria to be inadequate as screening tests for ICI [4,6,8,35]. Articles have discussed the use of CT scan of children after MHI, which would conceivably identify all potentially life-threatening ICI. However, there is likely a subgroup of children in whom the potential for ICI is so low that the benefits of CT scans do not justify the costs [24].

The indications for CT scanning in minor pediatric head trauma remain controversial. Guidelines for imaging a child with a minor injury are ill defined and subjective to the practitioner and institution. One author points out that a liberal policy of CT scanning should be adopted in pediatric patients with significant mechanisms of injury despite normal findings on neurologic examination. Early CT scanning would capture the few patients who may require neurosurgical intervention [37]. In addition, CT scanning should be considered in any neurologically intact child who shows significant signs of brain injury (LOC, amnesia for the event, vomiting, seizures, headache, and focal neurologic deficits) [6].

With the increasing availability of CT, physicians face the conflict of selecting the patients who would benefit from obtaining the scan, yet they are confronted with the consequences of failing to diagnose ICI if scanning is not done. The use of CT scan as a method of pediatric MHI evaluation must be weighed against health care costs, time-consuming measures for staff, the need for procedural sedation, overall ED visit times, lost parental work time and wages, travel expenses, and parental anxiety [6,42]. A 2000 Canadian study compared the usage rates of CT scans at nine pediatric hospitals [3]. The authors reported a significant difference in the rate of ordering CT scans (6%–26%) among the participating hospitals but no significant difference in the rate of abnormal CT scans (1%–9%). Reasons posited for the various usage rates are the differing criteria for imaging among clinicians, the availability of CT scanning at the various hospitals, and the differences in the patient populations. Nevertheless, the frequencies of positive findings on CT scans did not differ based on the number of scans ordered.

Radiation risk has been a concern for children who undergo CT scanning after head injury [11,43–45]. Unfortunately, higher radiation doses improve image quality, and children receive a higher radiation dose than older children or adults for the same CT settings [46]. Likewise, children have a longer postimaging lifetime in which to manifest radiation-related cancer, and the cancer risk is cumulative over time. The increase in cancer risk has been reported to be as low as 0.35% for overall lifetime risk [43]. Authorities have also suggested that instead of omitting CT scanning, adjusting CT scanners to appropriate setting for the small heads of children might be sufficient to balance the risks and benefits of CT imaging [43,46].
American Academy of Pediatrics mild head injury guidelines

The AAP has published a practice parameter for the management of minor CHI in children [33]. The Committee on Quality Improvement of the AAP worked in collaboration with the Commission on Clinical Policies and Research of the American Academy of Family Physicians and experts in neurology, emergency medicine and critical care, research methodologists, and practicing physicians. The practice parameter makes recommendations for managing previously neurologically healthy children ages 2 to 20 who suffer minor CHIs. On presentation, the child may have experienced a temporary LOC (≤1 minute), an impact seizure, vomiting, or other signs and symptoms. The parameter defines children with minor CHI as those who have normal mental status at the initial examination, no abnormal or focal findings on neurologic examination (including fundoscopic), and have no physical evidence of SF. The parameter is not intended for victims of multiple trauma, children who have unobserved LOC, or patients who have known or suspected cervical spine injury. Finally, the parameter is directed only to the management of children evaluated by a health care professional within 24 hours after the injury.

For children with CHI and no LOC, a thorough history and an appropriate physical and neurologic examination should be performed, followed by observation. Observation includes the setting of the clinic, office, ED or at home, under the care of a competent caregiver. The use of CT, skull radiography, or MRI is not recommended. If the patient’s findings on examination appear normal, no additional tests are needed, and the child may be discharged under the care of a responsible guardian. The Quality Standards Subcommittee of the AAN [16] believes that the low prevalence of ICI and the marginal benefits of early detection of ICI afforded by imaging studies were outweighed by the considerations of cost, resource allocation, inconvenience, and possible side effects of sedation. The parameter discusses the limited role of skull radiographs in evaluating children with MHI, no LOC, and no signs of SF. Some situations may arise in which the physician may order an imaging study (eg, scalp hematoma over the course of meningeal artery). The Subcommittee believes that cranial CT scanning is the imaging modality of choice based on its increased sensitivity and specificity and the low predictive value of skull radiographs. When CT is not available, skull radiography may assist the practitioner in determining the risk for ICI. For children with minor CHI and brief LOC (≤1 minute), the parameter recommends observation or cranial CT scan. However, skull radiography and MRI are not recommended.

Disposition

Multiple factors contribute to the decision on whether to admit a child after head trauma. Some of these factors involve concerns of associated injuries, neurologic observation, and the child’s home environment. Many children who
sustain isolated MHI have traditionally been hospitalized for observation, despite normal findings on neurologic examination and even on imaging studies. Most of these patients do well and do not have adverse events during their admission [28,35,47–49].

Several prospective studies have shown that normal CT scans and normal examinations may be helpful criteria in deciding to safely discharge patients from the ED. In three studies (a total of 261 patients), there was a late deterioration incidence rate of zero in children with normal CT results [4,8,22,27]. Similarly, in six studies (a total of 349 patients) there was a zero incidence rate for clinical deterioration for children with isolated SF and no ICI present [4,8,9,22,27,50,51]. Several other trials demonstrate that in otherwise stable patients with a normal mental status, cranial CT and neurologic examination can be performed and the patient can be safely discharged from the ED [24,42,47]. A thorough evaluation, including normal physical examination and neuroimaging, would be more cost-effective than 1 to 2 days of hospital observation [42]. A large study that provided a comprehensive statewide follow-up of 400 children who had normal CT scan results after head injury observed that the children remained stable after discharge [42]. Only four children had delayed symptoms requiring hospitalization, and only one required operative neurosurgical management (this patient had a known risk factor for a bleeding diathesis). Many authors have demonstrated that patients with a normal CT scan and neurologic assessment after MHI are at very low risk for subsequent intracranial bleeding or neurologic sequelae and may be discharged from the ED if a competent observer is available [42,47,49].

Outcome and seizures

The short- and long-term complications of MHI in children are poorly understood. Studies have investigated both short-term and long-term outcome from MHI, specifically studying symptoms and behavioral and cognitive effects. The term postconcussive syndrome refers to the constellation of acute symptoms after MHI. These symptoms can be somatic (headache, dizziness, blurriness), emotional (irritability, anxiety), and cognitive (concentration and memory) [18]. The complete diagnostic criteria for postconcussive syndrome can be found in the “International Statistical Classification of Diseases and Related Health Problems,” 10th edition [52].

Short-term outcome

Hahn and McLone [28] studied the risk factors in the outcome of children who had MHI. In their prospective study of admitted children, 84% had minor head injuries. Of the 780 children who lost consciousness for 0 to 15 minutes, none had a poor outcome (defined as suffering severe disabilities, vegetative state, or death). Almost 6% developed various forms of seizures after the trauma. The
authors did not find a statistical difference between CCS scores and the incidence of posttraumatic seizure. However, children with seizures had twice the risk of having a poor outcome ($P = .001$). Of the 94% who did not have seizures, 3% had a poor outcome, compared with 6.4% of the children who had seizures with a poor outcome. The authors found that children with punctate hemorrhages on CT scans did not have an additional risk of a poorer outcome than children did with normal CT scans ($P = .001$).

Studies have assessed physical, behavioral, and cognitive outcomes across the severity continuum of head injuries. Pavlovitch and colleagues [53] determined that speech and feeding difficulties were associated with an increasing severity of head injury, whereas walking was not. Headaches are among the most common postconcussive symptoms reported, but most headaches are resolved several months after injury [27,54–56]. Similarly, temper outbursts, dizziness, mood swings, anxiety, and aggressive behavior have been reported more significantly across the injury severity continuum. In one study, the parents of 28% of the children believed that the personality of their child had changed since the head injury, with significantly more reporting the higher the severity of head injury [54]. Another study of children with MHI reported more symptoms than controls at 1 week but did not demonstrate impairment on neuropsychologic measures [56]. Initial symptoms had resolved for most children by 3 months after the injury, but there was a group of children (17%) whose parents continued to report symptoms and behavioral problems. These children who had ongoing problems were more likely to have had a previous head injury, neurologic or psychiatric problems, a history of learning or behavioral difficulties, or family stressors. The authors emphasize that the “at risk” children who have the potential for poorer outcomes should be identified in the emergency department and monitored through follow-up. A similar study by the same authors, found that a careful assessment and providing an information booklet and coping strategies resulted in significantly reduced anxiety as well as reports of symptoms and behavioral changes 3 months after injury [55].

Children with MHI do well in their recovery, but studies have found that not all mildly injured children recover completely. In the study by Hawley and colleagues [54], half of the study group made a good recovery, but only 18.4% made a full recovery without discernible sequelae. The most frequent reason for placing a child in the good but not full recovery category was the presence of headaches. The authors state that a surprising number of children with MHI had moderate disability (43%) at follow-up. The authors were unable to identify a threshold of injury severity below which the risk of late morbidity could be discounted.

**Long-term outcome**

The long-term outcome after MHI continues to be a controversy. Satz and colleagues [57] performed a review of 40 outcome studies between 1970 and
1995, specifically examining three outcomes: neuropsychologic, academic, and psychosocial. Of the 40 studies, the results of the comprehensive review revealed 13 adverse, 18 null, and 9 indeterminate findings related to the three outcomes of interest. They did not find adverse effects on academic or psychosocial outcomes across the spectrum of MHI or on neuropsychologic outcomes at the more extreme end of the MHI distribution. When the studies were classified based on methodological merit, the stronger studies were generally associated with null outcomes. The authors concluded that MHI in children may result in mild but transitory alteration in cognitive functioning (attention and memory) but no reliable changes in academic or psychologic functioning. However, because severity within the MHI spectrum increased, more variability in findings was reported, and there may be a degree of mild injury that reaches a threshold of concern.

Authors have studied extensively the effects of MHI on measures of behavior, cognition, and physical and family functioning. Children with MHI recover fairly well, and those who experience more severe head injury are more likely to have long-term negative outcomes. Anderson and colleagues [58] examined the effect of MHI in young children (3–7 years old) using standardized measures of adaptive, behavioral, and cognitive functioning at the time of initial hospitalization and at 6 and 30 months after injury. No significant differences were noted between the study group and the matched controls on most cognitive measures (intellectual, speed of processing, attention, memory, receptive vocabulary, and auditory comprehension), both at the acute stage and at 30 months after injury. The MHI group performed significantly worse than the control group did on story recall and verbal fluency, suggesting that MHI may produce impairment in certain high-level language skills. The MHI group improved over time, and the authors comment that these impairments were transient interruptions of brain functions and delay in skill acquisition rather than permanent deficits. The authors conclude that children with MHI have generally good outcomes during the preschool years [59]. In another study by Anderson and colleagues [59], it was observed that a dose-response relationship exists for injury severity and physical and cognitive outcomes. Behavioral functioning was not related to injury severity, although results approached significance. For physical and cognitive recovery (acutely after injury and at 30 months), injury severity was a consistent predictor along with socioeconomic status. After injury, the child and family function were less associated with injury factors and more dependent on preinjury psychosocial functions. The authors conclude that preinjury factors play a key role in postinjury behavior and family function, which suggests that premorbid vulnerabilities increase the risk of poor outcome after TBI.

Another study examined long-term psychosocial outcomes after MHI in early childhood [60]. This study showed that children who experienced a MHI of sufficient severity to warrant temporary hospitalization between ages of 0 to 10 years old were likely to show adverse psychosocial outcomes in terms of hyperactivity and inattention and conduct-disordered behavior at 10 to 13 years of age, especially if the injury occurred before the age of 5. In contrast, children in the outpatient MHI group were comparable to the noninjured reference group
on psychosocial measures. The authors concluded that long-term psychosocial problems may be more likely at the upper end of the MHI severity spectrum. When children with head injury return to school, they may be expected to assimilate immediately with the class. The child’s teacher may not understand the head injury or even know the child suffered a MHI, which could affect how the child is treated, observed, or graded. In the study by Hawley and colleagues [54], teachers knew of the child’s head injury in only 39.8% of the children, and there was a significant linear trend across injury severity groups. Special educational needs were provided for only 65% of the children identified with such needs, which did show a significant linear trend across injury severity. At follow-up, 18.7% of the children were currently having difficulties with schoolwork, and there was a significant linear trend across severity groups. Interestingly, 18% of the children had been disciplined by the school for problem behavior after sustaining their head injury [54].

**Posttraumatic seizure**

Posttraumatic seizures (PTS) after MHI can be relatively common in children. Recent studies cite a 3% to 6% incidence of PTS [28,61]. Rarely do these children need treatment, but children with PTS traditionally have been admitted to the hospital for observation. PTS can be defined as immediate (occurring within 24 hours of the head injury), early (occurring between 24 hours and 7 days after the injury), and late (occurring beyond 7 days after the injury) [62]. Impact seizures have also been described as developing at the time of the accident [28].

PTS after blunt head trauma may be associated with the presence of TBI. Although some studies indicate that mildly injured children without PTS have normal findings on neurologic examinations and head CT scans do not need routine hospitalization, some authors have questioned the traditional admission of children after PTS [61,62]. Studies have found that hospitalized patients after both MHI and PTS do well without any further seizure activity and normal neurologic exams. These authors emphasize that children who have isolated head injury and simple PTS who recover fully in the ED, whose CT scans do not show any ICI and no history of neurologic disease, are at low risk for recurrent seizures or neurologic complications. The authors suggest that these low-risk patients can potentially be sent home with a reliable caregiver [61,62].

The discussion of which child with PTS requires admission and which child can be discharged from the ED focuses on the question of what is the risk of sending home a child who suffered an uncomplicated PTS. Studies have found that the risk of PTS is more frequent among patients who have abnormal head CT scan results and altered mental status and that these patients are at higher risk for requiring neurosurgical procedures [61,63]. If all children with PTS have cranial CT scans, then a normal scan helps to eliminate that risk. Another concern is the risk of a prolonged seizure or status epilepticus. Furthermore, younger children have inconsistently been found to be more likely to suffer PTS. In the study
by Dias and colleagues [62], children with immediate PTS were significantly younger than a control group of children with uncomplicated MHI, 11% of the children had a prolonged seizure (≥ 5 min) and were treated with anticonvulsants and admitted to the ICU. Sixty-five percent of the children had the first seizure within 1 hour of impact, and 72% of the children studied had only a single seizure, yet no child suffered additional seizures or significant complications. The authors therefore concluded that as many as 85% of these children could have been discharged to home after normal imaging studies and a return to normal function after a single or brief seizure.

Seizure prophylaxis has been studied mostly in patients with moderate to severe head injuries. A 2004 randomized controlled trial studied phenytoin versus placebo in 102 patients 16 years old or less [64]. These children had a more serious head injury because the requirement for enrollment was GCS of 9 or 10, depending on age. The study did not find a significant difference in the rate of early PTS in the phenytoin versus placebo group.

**Sports injuries**

Recent research and news headlines on head injuries in athletes have raised concerns over the health of athletes who sustain concussions. Furthermore, these discussions have motivated clinicians and sports medicine specialists to develop practice guidelines and parameters for evaluating and managing the head-injured athlete. The various guidelines released have raised controversy because scientific foundation and delineation of concussion grades and return-to-play criteria are lacking [65]. In addition, there has been disagreement over the potential negative outcome of cumulative concussions. So far, there is no consensus on tests that assess baseline and posttraumatic neuropsychologic function. To begin the discussion of managing concussions in sports, physicians and researchers must have a common understanding and definition of what constitutes a concussion.

Many definitions of concussion have been proposed, but there is no universally accepted definition. Often, the term concussion has been used interchangeably with MHI [20]. The AAN defines concussion as “a trauma-induced alteration in mental status that may or may not involve loss of consciousness” and states that confusion and amnesia are hallmarks of concussion [16]. Many of the classifications of MHI and guidelines for management have been generated from studies of athletic injuries [20]. An agreement on a precise definition of concussion and the subtle delineations would help to solidify the general cohesiveness of how practitioners evaluate and manage athletes with concussions.

The three popular grading scales and guidelines are those of Cantu, the Sports Medicine Committee of the Colorado Medical Society, and the AAN (Table 1). In 1988, Cantu [66] developed guidelines for concussion by modifying the definition set earlier by the Congress of Neurological Surgeons and incorporated posttraumatic amnesia (PTA) into the definition. Cantu graded sports-related
concussions on the length of unconsciousness and PTA. In 1991, Kelly and colleagues worked with the Sports Medicine Committee of the Colorado Medical Society to develop another definition with associated guidelines [67,68]. Each level of concussion (mild, moderate, and severe) was described according to the length of unconsciousness and confusion or amnesia. The misunderstanding that concussion requires LOC still exists, and thus Kelly and colleagues reiterate that concussion should be defined as a traumatically induced alteration in mental status [67]. In 1997, the Quality Standards Subcommittee of the AAN developed a concussion severity rating scale in which both confusion and amnesia are important characteristics of concussion designations [16].

In 2001 and 2004, a panel of experts [69] met for an international symposium on concussion in sports and revised a consensus definition of sports concussion originally developed by the Committee on Head Injury Nomenclature of the Congress of Neurological Surgeons. The updated definition describes a sports concussion “as a complex pathophysiologic process affecting the brain, induced by traumatic biomechanical forces” [69]. Further delineations are made about concussion, stating that it is typically of rapid onset and a short-lived impairment of neurologic function that resolves spontaneously. In addition, concussion is typically associated with grossly normal structural neuroimaging studies. The group [69] proposed a new classification of concussion in sports as either simple or complex. In a simple concussion, an athlete suffers an injury that progressively resolves without complication over 7 to 10 days. Formal neuropsychologic screening does not play a role in these circumstances, although mental status screening should be a part of the assessment. The cornerstone of management is rest until all symptoms resolve. The group emphasized that all concussions mandate evaluation by a medical doctor; however, a simple concussion can be managed by a primary care physician or certified athletic trainer who has medical supervision. On the other hand, in a complex concussion, an athlete suffers persistent symptoms, specific sequelae (e.g., prolonged LOC over 1 min), or prolonged cognitive impairment after the injury. This classification may also include athletes who

<table>
<thead>
<tr>
<th>Cantu</th>
<th>American Academy of Neurology</th>
<th>Colorado Medical Society</th>
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</thead>
<tbody>
<tr>
<td>Grade 1 (mild)</td>
<td>Grade 1</td>
<td>Grade 1</td>
</tr>
<tr>
<td>No LOC</td>
<td>No LOC</td>
<td>No LOC</td>
</tr>
<tr>
<td>PTA ≤30 min</td>
<td>Transient confusion</td>
<td>Transient confusion</td>
</tr>
<tr>
<td>Symptoms resolve ≤15 min</td>
<td></td>
<td>No PTA</td>
</tr>
<tr>
<td>Grade 2 (moderate)</td>
<td>Grade 2</td>
<td>Grade 2</td>
</tr>
<tr>
<td>LOC ≤5 min, or</td>
<td>No LOC</td>
<td>No LOC</td>
</tr>
<tr>
<td>PTA &gt;30 min</td>
<td>Transient confusion</td>
<td>Confusion with PTA</td>
</tr>
<tr>
<td>Symptoms last &gt;15 min</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Grade 3 (severe)</td>
<td>Grade 3</td>
<td>Grade 3</td>
</tr>
<tr>
<td>LOC ≥5 min, or</td>
<td>Any LOC, brief or prolonged</td>
<td>LOC of any duration</td>
</tr>
<tr>
<td>PTA ≥24 h</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Data from Refs. [16,65,67].
suffer multiple concussions over time. Athletes with complex concussions should be managed by physicians with specific expertise in the management of concussive injury [69].

The management and return-to-play guidelines also differ among Cantu, the Colorado Medical Society, and the AAN (Table 2). The Colorado system distinguishes between grade 1 and 2 concussions according to the presence of symptoms, whereas the AAN makes this distinction according to the duration of symptoms [70]. Depending on which guideline is followed, an athlete may return to play at drastically different times, which may be too conservative or too hasty [65].

Table 2
Return-to-play guidelines for concussion grading scales

<table>
<thead>
<tr>
<th>System</th>
<th>Grade 1</th>
<th>Grade 2</th>
<th>Grade 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cantu [66] 1988</td>
<td>Athlete may return to play that day in select situations if normal clinical examination at rest and exertion; if symptomatic, athlete may return to play in 7 days</td>
<td>Athlete may return to play in 2 wk if asymptomatic at rest and exertion for 7 d</td>
<td>Athlete may return to play in 1 mo if asymptomatic at rest and exertion for 7 d</td>
</tr>
<tr>
<td>Colorado Medical Society [68] 1991</td>
<td>Remove athlete from contest and examine immediately and every 5 min; permit return to contest if amnesia or symptoms do not appear for 20 min</td>
<td>Remove from contest and disallow return; examine athlete next day; permit return to practice after 1 wk without symptoms</td>
<td>Transport athlete to hospital and perform neurologic exam; admit to hospital if signs of pathology are detected, otherwise overnight observation; permit return to practice if asymptomatic for 2 wk</td>
</tr>
<tr>
<td>American Academy of Neurology [16] 1997</td>
<td>Examine athlete immediately for mental status changes; may return to contest if no symptoms or mental status changes at 15 min</td>
<td>Remove athlete from contest and disallow return; examine athlete on site for symptoms or mental status changes; athlete may return in 1 wk if asymptomatic</td>
<td>Remove athlete from contest and transport to hospital; perform neurologic exam and observe overnight; permit return to play if asymptomatic after 1 wk (if LOC was brief) or 2 wk (if LOC was prolonged)</td>
</tr>
</tbody>
</table>

Data from Refs. [16,65–68].

Second impact syndrome

The term “second impact syndrome” was first coined by Saunders and Harbaugh in 1984 [71]. Cantu has defined second impact syndrome as an injury in which “an athlete who has sustained an initial head injury, most often a
concussion, sustains a second head injury before symptoms associated with the first have fully cleared” [72]. The syndrome can propel a rapid cascade of cerebral vascular congestion, causing increased intracranial pressure and brainstem herniation and ultimately may lead to death [73]. McCrory and colleagues established criteria for definite, probable, and possible second impact syndrome and did not find any definite cases of second impact syndrome [74].

Many reports of complications and even death have been attributed to second impact syndrome; however, the diagnosis remains controversial. Maroon and colleagues [20] state that second impact syndrome “is an infrequent finding, predominately involves young athletes, and only rarely is fatal.” To prevent the second impact syndrome, popular guidelines have recommended that athletes not return to play until the postconcussive symptoms have resolved [16,67,68]. The delayed development of amnesia or postconcussive symptoms suggests that some pathologic processes occur gradually and could be missed if an athlete returns to play too early [67]. Kelly and colleagues [75] emphasize that prematurely returning to sports in which head injury is a risk could lead to a catastrophic outcome such as permanent disability or death.

Neuropsychologic testing of athletes

Most of the neuropsychologic testing of patients has been on adults or professional or amateur athletes of near adult age. Neuropsychologic testing for patients after mild TBI involves systematically testing the athlete at the beginning of the season for a baseline comparison, then 24 hours after the head injury, and then again at 5 days. Baseline assessments are essential to the neuropsychologic test because any deficits in the athlete’s performance need to be detected and attributed to the effects of the concussion or to previous unrelated factors [20].

Several studies have examined recovery from mild concussion in high school athletes. Researches have tried to identify diagnostic markers of concussion severity and how the markers relate to recovery. In a study of high school athletes who suffered mild concussion, the authors found significant declines in memory processes relative to the noninjured control group. Statistically significant differences between preseason and postinjury memory test results were still evident in the concussion group at 4 and 7 days after the injury. Self-reported neurologic symptoms, such as headache, dizziness, and nausea had resolved by day 4. The authors observed that on-field mental status changes such as retrograde amnesia and confusion were related to the presence of memory impairment at 36 hours and 4 and 7 days after injury as was also related to slower resolution of self-reported symptoms. The authors conclude that on-field mental status changes appear to have prognostic value and should be taken into account when making return-to-play decisions [76].

Chronic traumatic brain injury (CTBI) is the cumulative long-term neurologic consequence of repetitive and subconcussive blows to the head. Matser and colleagues [77] studied 33 amateur soccer players and 27 controls (amateur
swimmers and runners) to determine whether soccer players had evidence of CTBI. Compared with the control athletes, the amateur soccer players exhibited impaired performance on tests of planning and memory. The authors found that the number of concussions incurred in soccer was inversely related to the neuropsychologic performance on six of 16 tests. The authors conclude that the findings suggest participation in amateur soccer may be associated with CTBI, as evidenced by impairment on cognitive functioning [77].

Researches have tried to answer the question, how many concussions are too many? Collins and colleagues [78] assessed the relationship between concussion history and learning disability and the association of these variables with neuropsychologic performance. Of 393 college football players with a mean age of 20.4 years, 34% had experienced one previous concussion and 20% had experienced two or more concussions. The authors observed that both a history of multiple concussions and learning disability were associated with reduced cognitive performance. A history of concussion was significantly associated with deficits in domains of executive functioning and the speed of information processing. The authors found that a history of one concussion does not result in the long-term cognitive morbidity that is associated with two or more episodes of concussion. As commonly agreed, any neurologic symptom should signal that full recovery was not completed and that return to play was contraindicated. The authors suggest that neuropsychologic testing may detect subtle cognitive impairments and that mental status screening instruments are variable and may miss the subtle deficits associated with head injury [78]. Each concussion needs to be evaluated individually and correlated with appropriate neuropsychologic tests as well as neuroimaging studies [20].

**Summary**

Many studies have found conflicting evidence over the use of clinical indicators to predict ICI in pediatric MHI. Although altered mental status, LOC, and abnormal neurologic examination findings have all been found to be more prevalent among head-injured children, studies have observed inconsistent results over their specificity and predictive value. Children older than 2 years have been evaluated, managed, and studied differently than those younger than 2 years. Evidence strongly supports a lower threshold for performing a CT scan in younger children because they have a higher risk of significant brain injury after blunt head trauma.

Many authors state that children with MHI who have normal CT scan results and normal mental status and neurologic examination findings may be discharged from the ED with a reliable caregiver and detailed head injury instructions. Further research involving multicenter trials with prospective enrollment will help to validate the decision making and previous recommendations on neuroimaging, hospitalization, observation, and discharge. Currently, the Pediatric Emergency Care Applied Research Network is conducting a multicenter study to address these
questions. This study is currently in progress and has enrolled 16,000 children, with plans to enroll over 25,000 children. One of the goals is to create a neuroimaging decision rule on children with minor to moderate head trauma.

Most children with MHI make a full recovery; however, a subset of children with preinjury morbidity may develop neuropsychologic sequelae. The management of sports-related head injuries demands identifying potential neurosurgical emergencies and preventing catastrophic outcomes related to acute brain swelling and repetitive concussions.

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