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Pediatric head injuries are common occurrences with the potential for serious morbidity or mortality. Fortunately, the incidence of traumatic brain injury (TBI) has been declining, mainly because of the development of effective prevention strategies (e.g., car seats and bicycle helmets). Although it is difficult to determine the exact incidence of head trauma in children due to variations in definitions and classifications, the majority of head injuries in children are minor and result in no long-term morbidity or mortality. However, early identification of a potentially serious injury and aggressive management of a child with a head injury facilitates the optimal possible outcome. The topic of pediatric TBI is extensive, and the majority of information is very familiar to the practicing emergency department (ED) physician. The author discusses two areas of controversy — patient selection for imaging and an update on management strategies for children with TBI. Selecting patients who require imaging following head trauma is easy if the child has an abnormal mental status or a Glasgow Coma Scale (GCS) score less than 15; he or she needs a head CT scan. The challenge is identifying high-risk patients with a GCS score of 15. The author reviews the available literature and presents currently available guidelines. Since TBI is the leading cause of death and disability, aggressive management of a child with a TBI is critical. The author reviews available therapies and their current application to pediatric patients.

—The Editor

Pediatric Controversies: Diagnosis and Management of Traumatic Brain Injuries

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Introduction

Trauma is the leading cause of childhood death,[1] and TBI is the leading cause of death and disability for children who sustain trauma.[2] Each year, more than 400,000 children younger than 14 years have emergent evaluations for head trauma.[3,4] Children younger than age 4 have considerable morbidity from head trauma. This age group has a prevalence of TBI that is more than twice the rate of the general population and nearly twice the rate for older children.[4] In addition, recent research indicates that even "minor" trauma may have the potential to result in life-long sequelae.[5,6] Thus, when evaluating children with head trauma, the practitioner must determine which patients are at risk, based on their history and physical exam, for significant injury requiring diagnostic imaging, careful monitoring, and aggressive intervention.

Evaluation of Children with Accidental Head Trauma

Injury patterns vary by the age of the child, with older children most likely sustaining an injury while participating in sports or when involved in motor vehicle collisions. However, children younger than 4 years most commonly sustain TBIs as a result of falls, motor vehicle collisions, or abuse. In the younger child, contact head injuries, such as linear skull fractures, hematomas, and cerebral contusions, can occur as the result of short, vertical falls.[7,8]

One study found that children who fell from a greater height were more likely to have injuries, but a number of patients had skull fractures or brain injury following falls from heights of less than three feet.[9] When there is a contact injury to the head, the point of impact causes the inner table of the skull to bend inward, which may injure blood vessels within the epidural or subdural space, as well as the parenchyma of the brain itself.[10] At the same time, there is also simultaneous outbending of the skull around the site of impact.[10] This puts the outer table of the skull under tension, and a fracture may result, either proximate to, or remote from, the site of impact. Children who sustain isolated skull fractures typically do not present with significant alterations in mental status, unless there is underlying brain injury with mass effect.[10]

Children Younger than 2 Years

Children younger than 2 years have been thought to be at high risk for significant brain injury after accidental head trauma.[11] Earlier studies often did not have enough data in the youngest age groups to recommend anything except a very cautious approach to evaluating head trauma in children younger than 2 years.[12,13] It has been estimated that the overall rate of brain injury after trauma in children younger than 2 years is approximately 5%, but infants younger than 2 months may have the highest prevalence of brain injury.[5]

Two studies in 1999 both evaluated infants younger than 1 year who presented to the ED with accidental head trauma.[5,9] The prevalence of brain injury was 12% in the 0-2 month age group, 6% in the 3-11 month age group, and 2% in infants older than 12 months.

Controversy exists in the literature regarding the ability of the physician to use clinical signs and symptoms to identify children at risk for brain injuries following blunt trauma. Obtaining an accurate history and a complete neurologic exam may be challenging, especially in younger children. Children younger than 2 years have been particularly identified as having subtle clinical presentations.[5] In addition, a computerized tomography (CT) scan of the head has disadvantages, including exposure to radiation, transport of the patient out of the ED, and a frequent requirement of sedation.[14-16]

Scalp Hematomas. Greenes and Schultzman sought objective markers of the presence of TBI and identified significant scalp hematomas as strongly associated with a skull fracture and brain injury in children younger than 2 years.[5] Another study also found the presence of a scalp hematoma to be the most important predictor variable for TBI identified on CT scan (e.g., intracranial hemorrhage, hematoma, or cerebral edema), in children 2 years and younger.[17] Finally, Greenes and Schutzman (2001) evaluated children younger than 2 years who sustained accidental head trauma, but had no neurological signs or symptoms.[18] The size and location of the scalp hematoma (e.g., parietal and temporal), and age younger than 3 months were each associated with skull fractures. This study also found that a skull fracture, large hematoma, and parietal location were associated with brain injury.[18] Children without a history of neurological symptoms and with a normal scalp exam were identified as a low-risk group.[9]

Abnormal Mental Status. Other series have examined the ability of an abnormal mental status to predict an abnormality on CT. Palchak et al found that of 194 children age 2 years and younger, all 15 children with a TBI on CT were predicted by the presence of a scalp hematoma and an abnormal mental status (sensitivity 100%; 95% CI 81.9—100%).[17] Of the 60 chil-

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dren in this series age 2 years and younger who underwent CT and had a normal mental status examination and no scalp hematoma, none had a TBI identified on CT scan (negative predictive value 100%; 95% CI 95.1—100%). Lethargy, irritability, full or bulging fontanel, and vital signs suggestive of increased intracranial pressure (ICP) also have been associated with brain injury, while vomiting and loss of consciousness, at least in this age group, were not.[5]

Skull Fractures. Palchak et al found that of the 194 children age 2 years and younger who underwent CT scan, 15 (7.7%) had skull fractures on CT, and 46.7% had an associated TBI identified on CT.[17] Another study reported on 102 infants younger than 13 months with skull fractures. Fifteen of the 102 patients were found to have a brain injury. The authors found that patients with lethargy prior to presentation or in the ED and patients with parietal fractures were more likely to have sustained a brain injury.[19]

Guidelines. A multidisciplinary panel of nine experts in pediatric head trauma was convened.[20] All evidence gathered from a Medline search was reviewed, and using a modified Delphi technique, a set of guidelines for the evaluation of children younger than 2 years with minor head trauma was developed. Among the guiding principles the panel recommended were the following: One should have a lower threshold for diagnostic imaging in young children, with children younger than 12 months being at higher risk and children younger 3 months being at the highest risk for intracranial injury after head trauma; the greater the number and severity of signs and symptoms, the stronger the consideration should be for obtaining a CT. The greater the forces involved, the more pronounced the physical findings (e.g., scalp swelling), and the younger the age, the greater the risk for intracranial injury.

Specifically, the panel stratified the patients into risk categories based upon clinical features (e.g., history and physical examination), mechanism of injury, and absence/presence of a skull fracture.

High-risk patients had any of the following characteristics: depressed mental status, focal neurologic findings, signs of depressed or basilar skull fracture, seizure, irritability, acute skull fracture, bulging fontanel, vomiting greater than five episodes or for more than six hours, and loss of consciousness greater than one minute. All high-risk patients required a cranial CT scan.

Intermediate-risk patients had any of the following characteristics: vomiting three to four times; loss of consciousness less than one minute; history of lethargy or irritability, now resolved; caretakers concerned about current behavior; higher force mechanism; hematoma (especially large or nonfrontal in location); unwitnessed trauma; fall onto a hard surface; vague or no history of trauma with evidence of trauma; and nonacute skull fracture older than 24-48 hours. Patients in this category could be managed in one of two ways: a period of observation (4-6 hours recommended) and reevaluation, or a head CT scan.

Low-risk patients were defined as having low-energy mechanism (e.g., fall less than 3 feet), no signs or symptoms, and more than two hours since the injury; also, the panel found that as the patient's age increases, the risk decreases. These patients may be observed in the ED or at home with reliable caretakers.[20]

Apart from these findings and the panel recommendations, evidence exists suggesting that the youngest age group is more likely to have brain injury with no neurological findings.[6,21,22]

Children Older than 2 Years

For older children, it is easier to obtain historical information and an accurate physical examination. Many series have sought to determine historical factors and clinical features that are predictive of an intracranial injury. A recent prospective study found that in 2043 children younger than18 years with head trauma, an abnormal mental status, clinical signs of skull fracture or scalp hematoma (in patients younger than 2 years), history of headache and vomiting were predictive of intracranial injury.[17] The most important variable in this series was clinical findings of a skull fracture.

These five clinical findings identified 97 (99%; 95% CI 94.4—100%) of the 98 children with TBI on CT scan and all 105 children with TBIs that required acute intervention. Of the 304 (24%) children with CT scans who didn't have any of the five predictors, only one had a TBI on CT scan (0.3%; 95% CI 0—1.8%). Of the 825 patients who had none of the five predictors, no one had a TBI requiring acute intervention (negative predictive value 100%; 95% CI 99.6%—100%). Use of this rule would have decreased CT scan utilization by approximately 25%.[17] Similarly, another study found that children older than 2 years with closed head trauma who were neurologically normal and had no clinical signs of skull fracture could be managed safely without cranial CT.[23]

In 1999, the American Academy of Pediatrics published guidelines for the management of closed head trauma in previously healthy children 2-20 years of age.[24] This consensus statement used the historical features of loss of consciousness and the presence of symptoms as an indication for obtaining a CT scan of the head. For those children without a loss of consciousness, a thorough history and physical examination should be performed, and a competent caregiver should observe the patient for any deterioration in mental status. For those who have a history of a brief loss of consciousness, along with amnesia, headache or vomiting at the time of evaluation, the prevalence of intracranial injury may be as high as 7%.[25-27] Though many of these brain injuries may have little clinical consequence, a minority of these children may require neurosurgical intervention.[26-28] Therefore, in this group of symptomatic children with a brief loss of consciousness, CT scanning of the head may be useful. However, with a brief loss of consciousness alone in an otherwise asymptomatic patient, observation of the patient for neurological deterioration may be an acceptable alternative to obtaining a CT scan of the head.[24]

While CT scanning is usually a safe procedure, some children may require sedation to obtain the study. Therefore, one must consider the risks of sedation against the benefits of obtaining a CT scan in this group of asymptomatic patients.

Once a TBI has been detected, the type of facility where the child will be evaluated and treated is important to the recovery. Several studies have examined the impact of pediatric trauma centers on the initial management of pediatric trauma. One study evaluated the morbidity and mortality rates among pediatric trauma victims in Pennsylvania and found that morbidity and mortality from TBI was reduced significantly in patients who were treated at pediatric trauma centers.[29] More neurosurgical procedures were performed in pediatric trauma centers, and there was concomitant lower mortality from TBI.[29] Another study found that the mortality rate was significantly higher for children with TBIs who were first transported to non-pediatric hospitals and subsequently transferred to pediatric trauma centers.[30] Thus, it is important that children with brain injuries be transferred to the nearest pediatric trauma facility as soon as it is feasible.

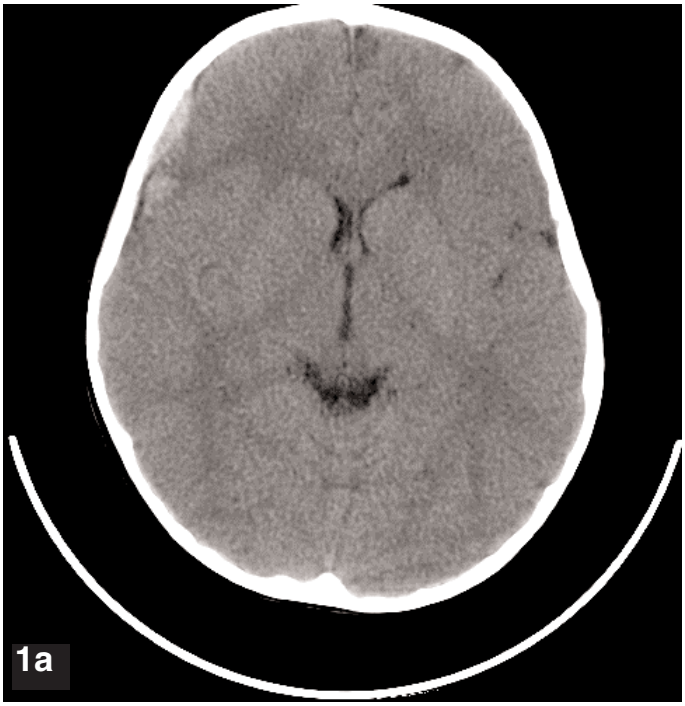
The guidelines for the acute management of severe TBI in infants, children, and adolescents made transfer to a pediatric trauma center a guideline based upon class II data (prospective and retrospective observation, cohort, and case control) and strong class III data (retrospective), and, as an option, an adult trauma center with qualifications for pediatric treatment.

Management of Intracranial Injuries

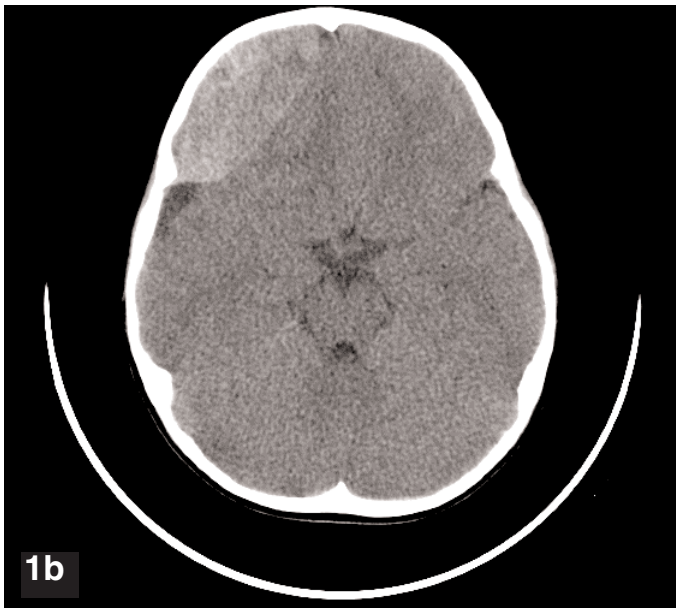
Group 1: Asymptomatic Intracranial Injuries. The optimal management and outcome of children who have intracranial injury as detected by CT scan, but who are otherwise asymptomatic, is controversial. Typically, such children are admitted to the hospital for close neurological assessment and monitoring. Many pediatric neurosurgeons have adopted an approach of expectant management for small intracranial and extradural hematomas, taking into consideration the size of the hematoma, its propensity to increase in size, shift of midline intracranial structures and surrounding cerebral edema.[24] (*See Figure 1.*) In some cases, children with subdural hemorrhage from minor trauma may do quite well with expectant management. Four patients were reported with unilateral subdural hemorrhage, of which three occurred from minor trauma and one from a fall out of a window. In all four cases, the subdural hemorrhage resolved spontaneously within 48 hours of injury.[31]

Critical to the management of children with an acute TBI is the initial assessment of the child's neurologic status and ongoing monitoring. Standardized assessment scores are the most accurate for detecting subtle changes in a patient. The GCS is useful for repeated neurological assessments in children with TBI. (See *Table 1.*) In one study, the most important prognostic indicators for pediatric TBI were demonstrated: the presence of associated trauma, admission GCS scores, traumatic mass lesions with ICP, and the presence of diffuse axonal injury.[32] There are modifications to the GCS to accommodate children who are preverbal or who are unable to verbally communicate due to sedation or endotracheal intubation. Such modifications include the Children's Coma Scale and the Infant Face Scale.[33,34] (*See Table 2.*)

Figure 1a and 1b. Rapidly Expanding Epidural Hematoma



1a. A head CT of a child performed two hours after a fall. The child had progressive emesis and lethargy. **1b.** Same patient's head CT five hours after the head trauma done secondary to increasing lethargy. Note the rapidly expanding epidural hematoma.



Group 2: Symptomatic Intracranial Injuries. The primary injury is the injury that occurs to the brain as a direct result of the trauma. Once an intracranial injury has occurred, management is directed at preventing secondary insults, which can exacerbate the primary brain injury and make the patient susceptible to progressive brain injury. The major, avoidable secondary insults include hypoxia and hypotension, which may

Table 1. Glasgow Coma Scale

EYE OPENING	
Spontaneous	4
Verbal stimulation	3
Painful stimulation	2
None	1
MOTOR	
Obeys commands	6
Localizes	5
Withdraws	4
Flexion	3
Extension	2
None	1
VERBAL	
Oriented	5
Confused	4
Inappropriate	3
Incoherent	2
None	1

Table 2. Glasgow Coma Scale — Modifications for Children

CHILDREN'S COMA SCALE (HAHN ET AL 1988) BEST SCORE = 15	
• Modification to best verbal response	
Smiles, orients to sound, follows objects, interacts	5
Consolable	4
Inconsistently consolable	3
Inconsolable	2
No response	1
INFANT FACE SCALE (DURHAM ET AL 2000) BEST SCORE = 15	
• Modification to best motor response	
Spontaneous normal movements	6
Hypoactive movements	5
Nonspecific movement to deep pain	5
Abnormal, rhythmic, spontaneous movements	3
Extension, either spontaneous or to pain	2
Flaccid	1
• Modification to best verbal response	
Cries spontaneously to handling or pain, alternating with quiet wakefulness	5
Cries spontaneously to handling or minor pain, alternating with sleep	4
Cries to deep pain only	3
Grimaces only to pain	2
No facial expression to pain	1

occur in the patient with multiple trauma; and intracranial hypertension, which may occur after the primary brain injury. Secondary brain injury causes a loss of cerebrovascular autoregulation and may result in cerebral edema, thereby reducing cerebral blood flow. Secondary brain injury also may be due to release of excitatory neurotransmitters, which can alter intracellular ion concentrations; and to the formation of inflammatory mediators, which can disrupt the blood-brain barrier and exacerbate neuronal damage.[35-37] Therefore, the goals of treatment of children with significant brain injury are to lower ICP and maximize cerebral perfusion pressure and oxygen delivery to the brain.

Monitoring of the ICP is appropriate in patients who have GCS score of 8 or less; have an abnormal initial CT scan of the head that demonstrates hematomas, contusions, or cerebral edema; or in whom serial neurological examinations are not possible due to other injuries, sedation, or neuromuscular blockade. There have been several studies in children that demonstrate an association between intracranial hypertension and poor neurological status at hospital discharge.[38,39]

ICP Monitoring. Recently published guidelines for the management of severe TBI in children recommend that a ventricular catheter connected to an external strain gauge is the most accurate and reliable manner in which to monitor ICP. [41] Such a device also allows for therapeutic diversion and analysis of cerebrospinal fluid.[40] These guidelines also recommend that the ventricular ICP be used as the reference standard in comparing the accuracy of ICP monitors placed in other cranial compartments.[41] Intracranial hypertension is defined as an ICP greater than 20 mmHg. The guidelines recommend that therapy be instituted when the ICP is consistently between 20-25 mmHg.[41] Other authors have suggested that the treatment of elevated ICP should be age dependent. In the young infant, treatment should begin when the ICP is greater than 15 mmHg; for children younger than 8 years, when the ICP is greater than 18 mmHg; and for older children and adolescents, when the ICP is greater than 20 mmHg.[35]

ICP Reduction.There are several methods to reduce ICP. Hyperventilation to reduce the pCO_2 below 35 mmHg may be useful in the setting of an acute rise in ICP or when signs of impending herniation are present. While hyperventilation may temporarily reduce intracranial hypertension, it also increases the volume of hypoperfused tissue in the injured brain; thus long periods of hypocarbia should be avoided.[41] The child's head should be maintained in a neutral position, and the head of the bed elevated to 30°. These maneuvers may decrease ICP without significantly changing cerebral perfusion pressure.[35] Jugular venous obstruction, which can elevate ICP, should be avoided by ensuring that cervical collars and endotracheal tube ties are not constrictive around the neck.[35]

Cerebral perfusion pressure (CPP) is defined as the difference between the mean arterial pressure and the ICP. The CPP is the gradient that promotes cerebral blood flow and substrate delivery to the brain. A CPP of 40-65 mmHg represents a spectrum to guide the efficacy of therapeutic interventions. Children with a CPP of 40-50 mmHg tend to have better survival after TBI.[42-45] Some authors have recommended that in young children, the CPP be maintained above 40-45 mmHg and above 50 mmHg in older children and adolescents.[35]

Therapeutic Interventions

Airway Management. *Hypoxia.* Patients should be well oxygenated throughout their ED course. Sedation and neuromuscular blockade may be useful to reduce the untoward effects of painful and noxious stimuli in patients with TBIs. Such stimuli include endotracheal intubation and mechanical ventilation, endotracheal suctioning, placement and maintenance of intravascular or intracranial catheters and monitoring devices, and transport for diagnostic procedures. Painful or stressful stimuli may increase the brain's oxygen consumption and increase sympathetic tone, leading to systemic hypertension and bleeding from operative sites.[46-48] There has been no systematic study of the efficacy of sedative and paralytic agents in children with TBI, and thus, there is no consensus as to what constitutes the ideal agents for sedation and neuromuscular blockade in this group of patients. There are case reports of the systematic, but limited, use of benzodiazepines, barbiturates, propofol, and non-depolarizing paralytic agents in children with TBI.[48] Prolonged use of propofol should be avoided in children because of reports of metabolic acidosis associated with its use. When using such agents, one must be aware of potential age-related differences in the response to pain and stress and in the level of sedation that patients may have.

Hypotension. Hypotension, which may occur in a pediatric multi-trauma patient, should be managed aggressively. Patients should be monitored carefully for the early signs of shock, including tachycardia, prolonged capillary refill, and loss of peripheral pulses. All volume deficits should be corrected and transfusions, when indicated, should not be delayed, to maintain hemoglobin and hematocrit at 10 mg/dL and 30%, respectively.[49]

Osmolar Agents. Osmolar agents, such as hypertonic saline and mannitol, have long been used in the treatment of children with TBI. Hypertonic saline works by increasing serum sodium concentration and serum osmolarity, creating an osmotic gradient by which water is pulled from the intracellular and interstitial compartments into the intravascular compartment. This increases intravascular volume and cerebral perfusion pressure, and reduces cerebral edema and ICP. One study reported results of a double-blind, crossover study comparing 3% saline and 0.9% saline boluses in 18 children with TBI.[50] During the initial trial boluses with hypertonic saline, the ICP decreased and there were reduced requirements for additional interventions. The guidelines for the acute management of severe traumatic brain injury in infants, children, and adolescents lists hypertonic saline as an option. The guidelines point out that hypertonic saline has evidentiary support, but mannitol has clinical acceptance and safety. Though mannitol works in a similar fashion, the blood brain barrier is able to exclude sodium chloride from the intracranial compartment, making it less likely to accumulate in the interstitial space.[51] Hypertonic saline also causes a reduction in vascular resistance by decreasing edema in the vascular endothelium of injured tissues.[52] Hypertonic saline also may normalize resting membrane potentials and cell volumes by restoring normal intracellular electrolyte balance in injured brain cells.[53] Rapid lowering of the serum sodium concentration should be avoided. Rebound cerebral edema can occur due to intracellular fluid shifts when the serum sodium concentration falls rapidly in the face of a residual hyperosmolar intracellular environment.[52]

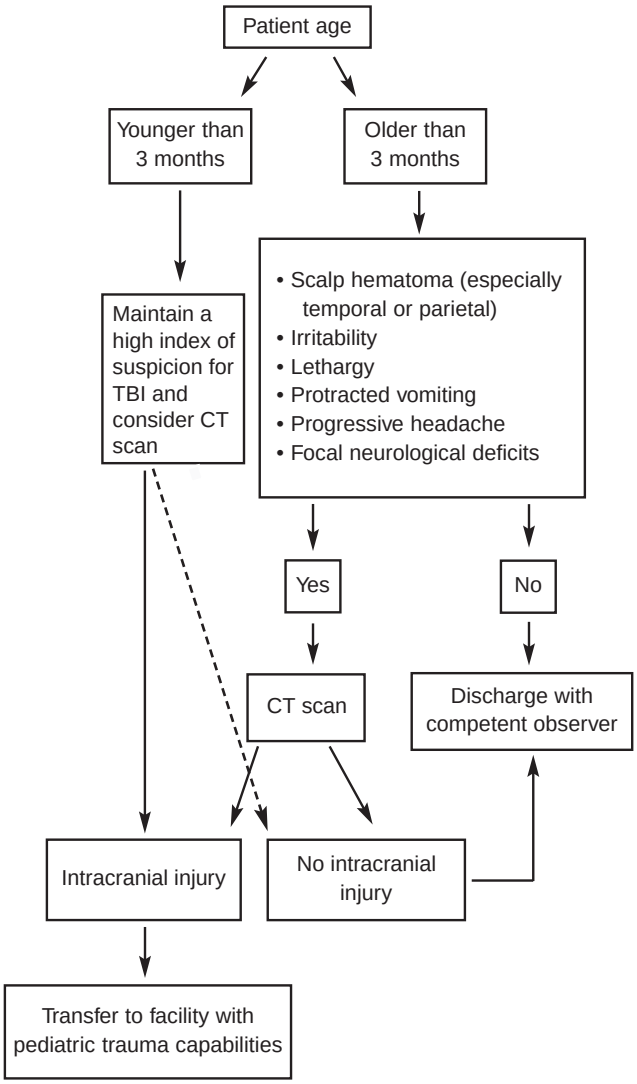
Mannitol works in a similar fashion by decreasing blood viscosity and, thereby the diameter of cerebral blood vessels. Cerebral blood flow is maintained by reflex vasoconstriction of the cerebral vasculature, but cerebral blood volume and ICP are reduced.[54] This mechanism relies on intact autoregulation of cerebral blood flow by the brain. Mannitol also reduces ICP by changing the osmotic gradient within the cerebral vasculature, causing water to move from injured tissues into cerebral blood vessels.[54] Mannitol should be administered as intermittent bolus doses. Prolonged administration of mannitol can result in its accumulation within injured tissues, reversing the osmotic gradient with the cerebral vasculature and worsening cerebral edema.[55]

Cerebral Metabolism Reduction. Reducing cerebral metabolism may be helpful in reducing ICP. Early initiation of barbiturate coma may reduce the risk of secondary brain injury. Barbiturates can lower ICP by reducing cerebral metabolism, altering cerebrovascular tone and reducing neuronal, free-radical injury.[35] Lower doses of pentobarbital initially may be given to prevent myocardial depression and systemic hypotension. It may not be necessary to use higher doses of pentobarbital to obtain burst suppression on the electroencephalogram (EEG), as lower doses still may have significant neuroprotective effects.[35]

Seizure Control. Seizures can cause a rise in ICP by increasing the brain's metabolic demands, releasing excitatory neurotransmitters, and raising systemic blood pressure. Antiepileptic drugs (e.g., phenytoin, fosphenytoin, or phenobarbital) may be helpful to prevent seizures within the first week after severe TBI, but their effectiveness in preventing late onset (i.e., longer than one week) seizures has not been demonstrated.[56] Some authors have recommended antiepileptic prophylaxis if there is significant parenchymal injury in children with severe TBI.[35] Children younger than 2 years of age are at high risk of post-traumatic seizures, with 44-70% of those with severe brain injuries having post-traumatic seizures.[35,57]

Hypothermia. The role of hypothermia in the treatment of children with TBI is unclear. While initial studies in adults demonstrated benefit in adults with TBI and intracranial hypertension, a recent randomized prospective study showed that hypothermia did not reduce morbidity and mortality in adults with severe TBI.[58-60] A similar degree of hypothermia has been shown to be efficacious in children with uncontrolled intracranial hypertension after TBI.[61] While intracranial hypertension was ameliorated after 48 hours of induced hypothermia when compared with the normothermic group, functional outcomes of survivors were similar between the two groups. A larger randomized trial is needed to definitively determine if induced hypothermia improves survival in children with TBI. Currently, the Guidelines for Acute Management of Severe Traumatic Brain Injury in Infants, Children and Adolescents recommend as an option, to avoid hyperthermia (i.e., temperature is higher than 38.5°C), and consider hypothermia (i.e., temperature is

Figure 2. Children Younger than 2 Years with a Head Injury



32-33°C) if refractory intracranial hypertension occurs.

Operative Intervention. Finally, operative intervention may be a necessary adjunct to medical therapy for severe TBI. Significantly depressed skull fractures should be elevated and intracranial and intraparenchymal mass lesions should be evacuated or debrided when ICP and CPP cannot be optimally managed by medical measures.[35] Some studies have demonstrated that decompressive craniectomy may be useful for pediatric patients with severe head injuries with uncontrolled intracranial hypertension.[62,63]

Predictors of Outcome

There has been a significant decline in the morbidity and mortality of pediatric TBI in the United States during the past two decades.[64] The overall mortality of children with TBI in the United States has been reported to be 6%, and those children with severe head injury requiring mechanical ventilation have a mortality of approximately 18%.[65,66]

There may be several reasons for such a decline in morbidity and mortality. One study analyzed consecutive admissions of children with TBIs to three different pediatric intensive care units. He found that while there was significant variation among centers with respect to the use of neuromuscular blockade, induced hypothermia and ICP monitoring, none of these modalities had an effect on mortality. Only the use of antiepileptic agents significantly reduced mortality in this study.[67] Another study found that in children with severe traumatic brain injuries, survival was associated significantly with the maintenance of supranormal systolic blood pressure (i.e., greater than 135 mmHg).[68] Mannitol was associated with a prolonged length of stay in the pediatric intensive care unit, but had no effect on survival. Similarly, Pigula found that children with severe head injuries and systemic hypotension had a much greater mortality rate.[69] Further study is needed to determine which interventions have an impact on morbidity and mortality in children with TBIs.

Several investigators have evaluated which factors may predict both survival and functional outcomes of children with TBI. In severe TBI, the GCS score and Pediatric Risk of Mortality Score (PRISM) may be predictive of survival.[66] In a retrospective study, children with GCS scores less than or equal to 5, but with lower PRISM scores, were more likely to survive and be discharged from the hospital. At hospital discharge, 40% of these patients were functioning independently; and at two years after the injury, nearly 66% were functioning independently. However, independent functioning in childhood may not persist into adulthood. In another study, 39 adults who had sustained TBI during the preschool years were evaluated.[70] While 59% of these patients attended a regular school after recovering from their TBI, only 29% eventually had full time employment as adults.[70] Most of these patients had sustained their TBI nearly 30 years ago, and it can be argued that recent advances in resuscitation of brain-injured children eventually may improve functional outcomes that persist into adulthood. Finally, serum levels of protein S-100 beta, a calcium-binding, dimeric protein found in astroglial and Schwann cells, when obtained and measured at the initial time of injury, may have predictive value in determining functional outcome in children and adults with mild to severe TBI.[71,72]

School-age children who survive TBI are at risk for having neuropsychological deficits and developing psychiatric syndromes. Children who survive severe TBI are at risk of having deficits in verbal reasoning, learning and recall, attention, executive functions, and constructional skills within 12 months of hospital discharge. Even when evaluated as long as four years after the injury, there may be little long-term recovery of such skills.[73] Children who recover from both mild and severe TBI are more likely than those who recover from orthopedic injuries to have psychiatric disturbances, such as organic personality disorder, attention deficit-hyperactivity disorder, major depression, and anxiety disorders.[74] Siblings and parents of children who survive severe TBI may also experience psychological distress during the patient's recovery and rehabilitation periods.[75,76]

Summary

TBI can cause considerable morbidity in young children. Children younger than 1 year, and particularly those younger than 3 months, are at higher risk of sustaining a TBI after head trauma than are older children. Scalp hematomas, especially those over the parietal region, altered mental status, and focal neurological signs, are the best clinical indicators of TBI in children.

Children with TBI are best managed at trauma centers, and transfer to such facilities should be expedited when TBIs are diagnosed in children. Once a primary brain injury, or trauma that results directly from impact, has occurred, the goals of management are directed at preventing secondary insults, which can exacerbate the primary brain injury and make the patient susceptible to secondary brain injury. Maximizing CPP and reducing ICP are the goals of management of children with TBIs. Sedation, neuromuscular blockade, hyperosmolar therapy, barbiturate therapy, and antiepileptic prophylaxis are management options in children with TBIs.

Finally, children and their families will require considerable support during the rehabilitation phase after a TBI. Psychological and psychiatric sequelae are common in children after a TBI, and significant family stress can occur during the patient's recovery and rehabilitation period.

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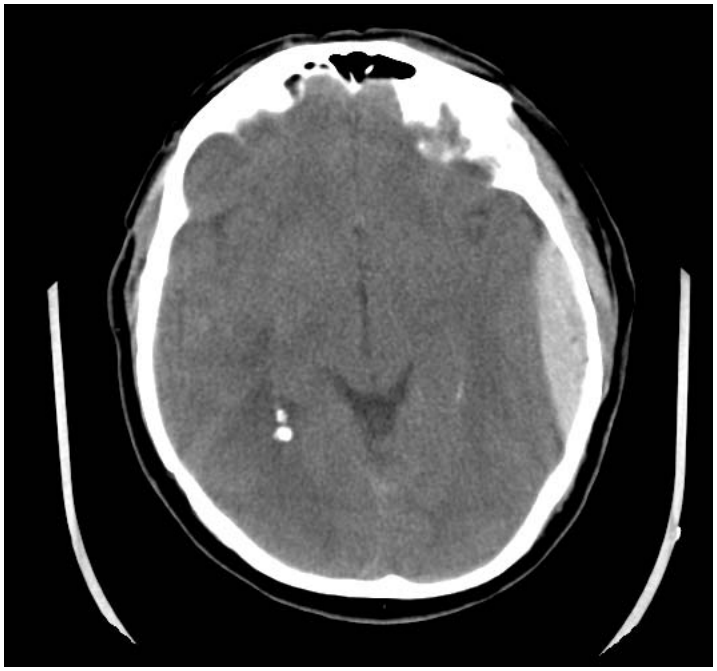
CE/CME Questions

1. Which of the following is true regarding a child younger than 2 years who sustains a head injury?
   A. The younger the child, the higher the risk for traumatic brain injury.
   B. The incidence of brain injury in a child younger than 2 years is about 5%.
   C. CT scans do have certain disadvantages, including exposure to ionizing radiation.
   D. All of the above

2. A 3-month-old male presents after his mother dropped him when she tripped. He fell approximately five feet. He is irritable, but consoles and has a large parietal hematoma. The most appropriate next test is:
   A. MRI.
   B. CT scan of the head.
   C. skull films.
   D. skeletal survey.

3. A 7-year-old male was involved in a fight at school four hours ago. He did not lose consciousness, remembers the entire event, and has had no vomiting. His neurologic examination is normal. On physical examination, he has a hematoma on his forehead. The next best test is:
   A. an MRI.
   B. a CT scan of the head.
   C. skull films.
   D. None of the above

4. Which of the following has/have been associated with an intracranial injury in a child younger than 2 years?
   A. Skull fracture
   B. Parietal scalp hematoma
   C. Large scalp hematoma
   D. All of the above



5. What is shown in the image above?
   A. Epidural hematoma
   B. Subdural hematoma
   C. Intraparenchymal hematoma
   D. None of the above

6. Which of the following is *not* considered to be high-risk criteria for TBI in a child younger than 2 years?
   A. Depressed mental status
   B. Signs of depressed or basilar skull fracture
   C. Two episodes of emesis
   D. Acute skull fracture

7. Which of the following children does *not* require a cranial CT following a fall?
   A. A 3-year-old with an occipital hematoma, no other symptoms, and a normal exam
   B. A 4-month-old who has a large scalp hematoma and is irritable
   C. A 1-year-old who has a GCS score of 13
   D. A 6-year-old with hemotypanum

8. Which of the following are critical in the initial stabilization of a child with a head injury?

CE/CME Instructions

Physicians and nurses participate in this continuing medical education/continuing education program by reading the article, using the provided references for further research, and studying the questions at the end of the article. Participants should select what they believe to be the correct answers, then refer to the list of correct answers to test their knowledge. To clarify confusion surrounding any questions answered incorrectly, please consult the source material. **After completing this activity, you must complete the evaluation form provided and return it in the reply envelope provided in order to receive a certificate of completion.** When your evaluation is received, a certificate will be mailed to you.

CE/CME Objectives

Upon completing this program, the participants will be able to:

a.) Recognize or increase index of suspicion for pediatric head injury;
b.) Identify how to correctly and quickly stabilize and manage pediatric head trauma;
c.) Employ appropriate diagnostic modalities for pediatric head trauma; and
d.) Recognize indications and potential risks with therapeutic options for children with head trauma.

A. Avoiding hypoxia
B. Avoiding hypotension
C. Maintaining an adequate cerebral perfusion pressure
D. All of the above

9. In which of the following scenarios is ICP monitoring *not* an appropriate consideration?
A. A child with a GCS score less than 8
B. A child with a GCS score of 12 five minutes after a seizure
C. A child who was intubated at the scene, is unresponsive and has cerebral edema on CT scan
D. A child who is intubated for a multi-system trauma and must be paralyzed and sedated

10. Which of the following may be used in the management of a child with a head injury and a GCS score of 8?
A. Early intubation
B. ICP monitoring
C. Correction of hypotension
D. All of the above

Answer Key:

1. D	**6. C**
2. B	**7. A**
3. D	**8. D**
4. D	**9. B**
5. A	**10. D**

In Future Issues:

Knee Injuries