

# The ABCs of Pediatric Head Trauma

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## ABSTRACT

Traumatic injury is the leading cause of morbidity and mortality in children, and causes over half of all childhood deaths.<sup>1-4</sup> Mortality rates of 30 to 50% have been reported for children with traumatic brain injury (TBI). Although children have better survival rates than adults, the long-term sequelae are more devastating in children due to their age and extended life expectancy.<sup>5</sup> Costs involved in care of a child with head injury, extended over the lifetime of the patient, are tremendous. It is unfortunate that despite preventive measures, TBI remains the major morbidity and mortality factor for children.

**KEYWORDS:** Brain injury, diffuse axonal injury, intracranial pressure, pediatric, shaken baby, trauma

**Objectives:** Upon completion of this article, the reader should be able to: (1) summarize the key components of the initial trauma assessment in children with TBI; (2) know the indications for computed tomography and magnetic resonance imaging in pediatric head trauma; and (3) know the goals of treatment for these children.

## EPIDEMIOLOGY

There are over 150,000 new cases of pediatric head injury each year, resulting in over 7000 deaths and 29,000 new permanent disabilities annually.<sup>3,4,6-8</sup> While most victims suffer minor head trauma, long-term permanent neurological damage still occurs.<sup>9,10</sup> The most common mechanisms of injury are age specific. For example, children less than 4 years old most often suffer from injuries due to falls, motor vehicle collisions (MVCs), and child abuse. Child abuse, or nonaccidental trauma, sadly represents up to two thirds of severe traumatic brain injury (TBI) in some series.<sup>3,7,11</sup> In school-age children, ages 5 to 12 years, sports and MVC are the most common causes of TBI. Although it has been reported that approximately 10% of all head injuries are related to athletics, MVCs involving bicycles and pedestrians are the most common mechanism of injury in school-age children. For teenagers, MVCs represent the most com-

mon mechanisms of injury and account for 78% of all unintentional injuries.<sup>2</sup> However, violent crime and assault are now the second leading cause of death in teenagers.<sup>2</sup>

## PATHOPHYSIOLOGY OF BRAIN INJURY

Injury prevention efforts are directed toward preventing primary brain injury, the pathology that results directly from trauma. Devices such as seat belts, airbags, and helmets are helpful when used correctly to lessen or prevent the impact and the resultant primary brain injury. Secondary injury involves the physiological and biochemical events that ensue, and postinjury intervention is directed at reducing secondary brain damage. Research efforts are under way to develop better ways to optimize oxygen delivery, monitor intracranial response,

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and improve the outcome for children through proactive medical and surgical intervention. It is useful to differentiate "secondary injury" from "second insults."

Second insults are events following the primary injury that contribute to the severity of the secondary injury and worsen prognosis (e.g., hypoxia, hypotension). Compared with adults, children more often suffer diffuse brain injury as compared with focal trauma. The reason for this is likely due to differences in the biomechanical profile and tissue properties in the child; the child's brain has a much higher water content and incomplete myelination. It is possible that the incidence and response to the second insults might also contribute to the diffuse nature of their resultant injury.<sup>12,13</sup> Similar to the adult, biomechanical forces involved in pediatric TBI include contact and inertial forces. Contact forces result in direct injuries, for example, occur when the head strikes an object and is associated with linear force vectors. Inertial forces are exerted on the brain when there is an acceleration-deceleration or angular-rotational movement of the head. Either or both of these forces determine the injury observed. Autopsy studies have shown that children with head trauma often demonstrate pathology consistent with diffuse axonal injury and vascular congestion.<sup>14,15</sup> It is believed that this type of injury occurs because the child's head to torso ratio is much greater than that of the adult. Angular biomechanical forces exerted on the head, therefore, cause brain acceleration and deceleration to a much greater extent.

Secondary brain injury involves a complex cascade of physiological and biochemical events that cause increasing neural damage, both acutely and chronically following the primary injury. Cerebral autoregulation is often lost after severe TBI, which may contribute to the diffuse swelling and edema observed.<sup>16</sup> Cerebral blood flow (CBF) and oxygen content differ; and it is believed that there is increased susceptibility to hypoxia and hypotension in young children.<sup>12,13</sup> Studies demonstrate that soon after TBI, CBF reduction may cause cortical hypoperfusion and ischemia, which are associated with a poor prognosis.<sup>16</sup> After 24 hours, CBF increases with an apparent dissociation of CBF and oxidative cerebral metabolism (cerebral metabolic rate of O<sub>2</sub> [CMRO<sub>2</sub>]). Secondary damage is exacerbated by the release of excitatory neurotransmitters, including glutamate, aspartate, and glycine.<sup>17</sup> Supraphysiological glutamate concentrations have been found to be neurotoxic in both in vivo and in vitro studies.<sup>18,19</sup> Other factors that are involved in secondary injury include calcium and potassium influx and free radical formation.<sup>20</sup> Neural depolarization can result with these physiological changes, causing widespread acetylcholine release and traumatic capillary damage.<sup>14</sup> It has also been demonstrated that the immature brain is more susceptible to apoptosis or delayed cell death. Animal studies have shown the severity of neurodegeneration after trauma was highest in

the youngest animal brains; this age-dependent degeneration also corresponded to an increase in intracellular proteolytic activity.<sup>21</sup>

## DIAGNOSTIC MANAGEMENT

### Initial Assessment and Stabilization

The initial assessment and management by the paramedic team is paramount. Cerebral perfusion pressure (CPP) and oxygenation depend on adequate ventilation, cardiac function, and the maintenance of mean arterial pressure. Normotension or slight hypertension, normal oxygenation (> 93% oxygen saturation), and normocarbica should be maintained in children with TBI. Hyperventilation to reduce the arterial carbon dioxide content (pCO<sub>2</sub>) to less than 35 mmHg may be used in the acute setting of increased intracranial pressure (ICP), but should not be used chronically. When the pCO<sub>2</sub> is reduced below 30 to 35 mmHg, severe vasoconstriction may cause reduced CBF and resultant ischemia. In contrast, hypercarbia may cause deleterious vasodilation that can lead to increases in ICP. Because young trauma patients often become hypotensive despite aggressive intravenous fluid resuscitation, vasopressors should be administered for persistent hypotension. If there has been significant blood loss, expedient transfusion should be initiated. Hemoglobin and hematocrit should be kept above 10 mg/dL and 30%, respectively. In children, significant blood loss can occur in a short period of time due to the lower total blood volume.

### Neurological Exam

A brief initial neurological examination on admission can provide critical information about the extent of brain injury and overall prognosis. A more thorough evaluation of any head or spine injury can be obtained throughout the resuscitative phase. In addition, the eyes, orbits, and external auditory meatus should be examined. Other potential signs of injury include vomiting and/or seizures; however, these have been found to have poor sensitivity and specificity for detecting intracranial injury.<sup>22</sup> Michaud et al reported that the most significant predictors of survival were the severity of total traumatic injuries and pupillary responsiveness.<sup>23</sup> Although loss of consciousness alone is a poor indicator of patients with intracranial injury, the Glasgow coma score (GCS) has provided a standard guideline for the assessment of consciousness and function in head trauma patients.<sup>22</sup> Based on wakefulness and motor and verbal function, GCS is useful in the initial neurological assessment of adult trauma patients. In a study of 653 TBI patients younger than 15 years of age, Levi demonstrated that the most important prognostic indicators were presence of associated trauma, admission GCS scores, traumatic mass lesions with increased ICP, and the presence of diffuse axonal

injury.<sup>24</sup> With younger patients (< 3 years of age), the GCS must be age-modified, and neurological examination is often challenging. Some studies have failed to identify any clinical factors that were predictive of intracranial injury in pediatric head trauma patients.<sup>25</sup>

### Radiological Studies

Universal imaging of all children with minor head trauma would result in unnecessary cost, wasted time, and resources, but a computed tomographic (CT) scan of the head is indicated in any child with an altered level of consciousness, focal neurological deficit, or physical signs of head injury.<sup>22,26</sup> CT scans provide rapid and extensive information about extent of injury to the soft tissues, skull, and brain. Studies have demonstrated that, even in an intact child, CT scans have shown traumatic mass lesions requiring surgery, especially after high-risk injury.<sup>10,27-29</sup> If a patient needs to be taken to the operating room (OR) prior to a head CT to stabilize life-threatening, nonneurological injuries, and there is concomitant head trauma, then intraoperative placement of an ICP monitor may be necessary. Obvious depressed skull fractures or open injuries can be addressed without a CT scan. With the newer, rapid scanning CT, there are ways to quickly define cerebrovascular trauma. CT angiography (CTA) and venography (CTV) are becoming available at many centers. CTA can define regions of stenosis or flow void in the internal carotid artery caused by skull base fractures involving the carotid canal. If fractures are present or depressed above the sagittal sinus, a CTV may be able to show sinus compression or occlusion. Reconstructed, three-dimensional CT views may be useful by providing information on the nature and extent of brain injury. Xenon CT has been an invaluable resource in providing data about CBF and autoregulation following TBI in adults and in children. Adjustments in the patient's pCO<sub>2</sub> and blood pressure can be made to study the patient's autoregulatory function to optimize the physiological parameters for the patient. Positron emission tomography (PET) scanning has been used for adult TBI on a limited basis for research purposes, but has not been used in children.<sup>30</sup>

Although magnetic resonance imaging (MRI) does not add to the diagnosis of operative head injury, studies show a significant correlation between the extent of neural injury on MRI to the cognitive impairment in outcome studies (Fig. 1). Diffusion weighted imaging (DWI) reveals nonhemorrhagic infarction hours to days before conventional CT scanning and provides an indicator of severity that is more complete than any other imaging modality.<sup>31</sup> Apparent diffusion coefficient (ADC) maps have shown that cytotoxic edema plays a significant role in secondary injury. Functional MR identifies changes in CBF relative to neurological deficits, and MR spectroscopy (MRS) assesses cerebral metabolism

through the relative quantification of molecules such as N-acetylaspartate (NAA), creatine, phosphocreatine, choline, lactate, myoinositol, glutamine, glutamate, and adenosine triphosphate (ATP). NAA is a well-characterized marker of neuronal integrity and is reduced following TBI.<sup>32</sup> Elevation of choline reflects membrane breakdown and inflammation.

### Electrophysiological Monitoring

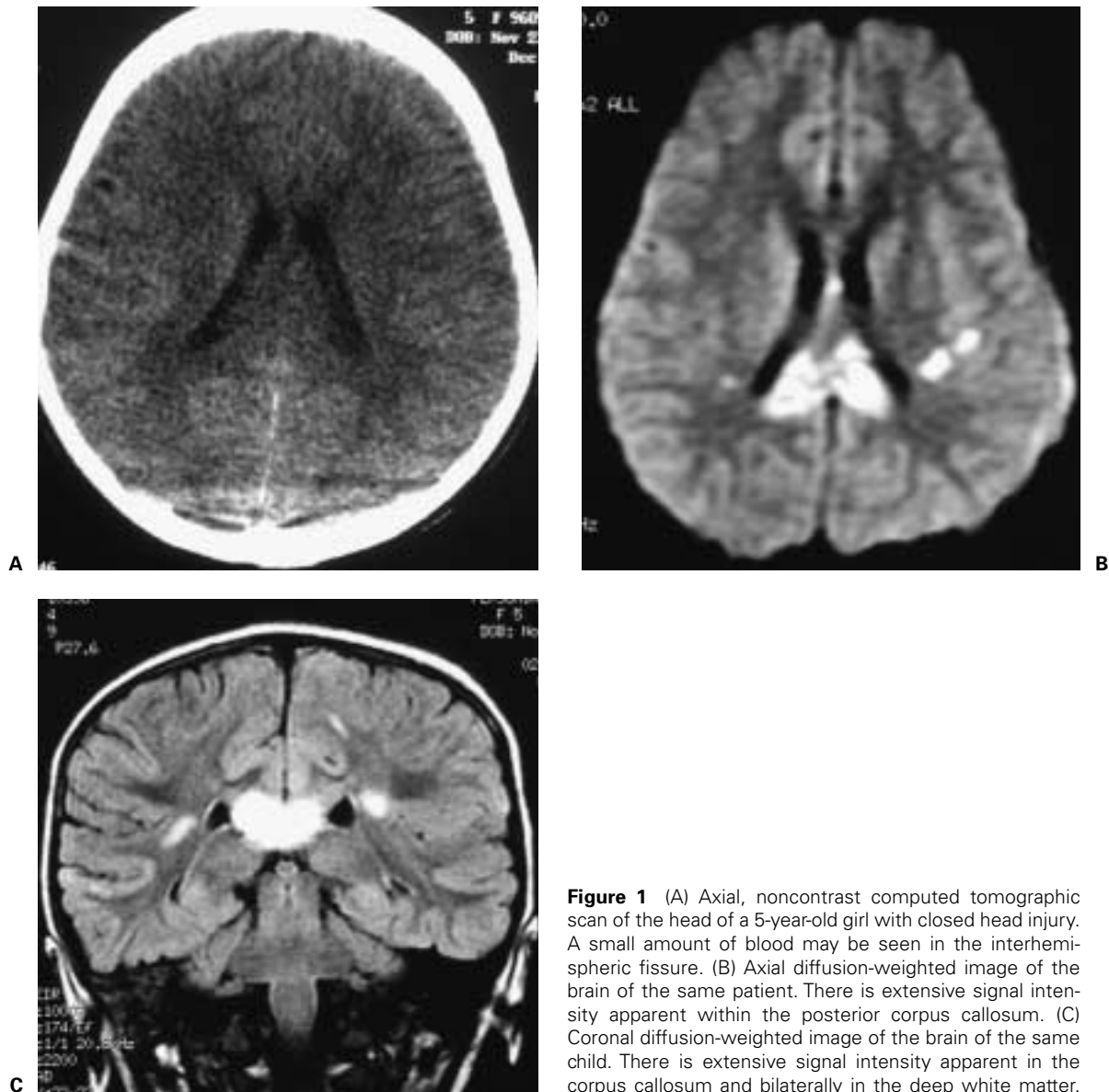
Neurophysiological monitoring is important in the management of TBI because it may provide an "early warning" of impending deterioration with regard to oxygenation, hypotension, neurological decline, and intracranial hypertension. Serial or continuous somatosensory evoked potentials (SSEP) and electroencephalograms (EEG) are more controversial, but advanced computer software is being developed to provide even earlier evidence of worsening status. SSEP's are relatively unaffected by cerebral suppressive medications; bilateral absence of cortical peaks of the SSEP is the strongest electrophysiological positive predictor of poor functional outcome in children.<sup>33</sup> There have also been studies to determine the prognostic significance of abnormal EEG patterns in children. In general, mild slowing is associated with a good outcome, whereas the best predictor of a bad outcome is lack of variability and reactivity.<sup>34,35</sup> Brainstem auditory and visual evoked potentials (BAER and VEP) have also been used to assess brainstem function after TBI, with normal BAER correlating well with survival.<sup>36</sup>

### Neuroinvasive Monitoring

Based on the Adult Guidelines, continuous ICP monitoring is indicated for adult patients with GCS  $\leq$  8.<sup>37</sup> Although pediatric guidelines for ICP monitoring are still being developed, present evidence supports the monitoring of ICP in children with severe TBI (GCS  $\leq$  8) (P.D. Adelson, personal communication, 2002). Intracranial hypertension and lower CPP contribute to the pathophysiological mechanisms involved in secondary brain injury. CPP needs to be maintained at normal levels to maintain adequate oxygen delivery. Intraventricular CSF samples obtained from pediatric TBI patients have shown elevated glycine and glutamate concentrations; these elevations were higher in children with more severe TBI and poor outcomes.<sup>17</sup>

### Treatment

The goals of treatment of TBI are directed at minimizing further neuronal damage from secondary injury. Interventions are generally directed toward lowering ICP and maintaining CPP and oxygen delivery to the brain. Elevated ICP, hypoxia, and hypotension should be treated



**Figure 1** (A) Axial, noncontrast computed tomographic scan of the head of a 5-year-old girl with closed head injury. A small amount of blood may be seen in the interhemispheric fissure. (B) Axial diffusion-weighted image of the brain of the same patient. There is extensive signal intensity apparent within the posterior corpus callosum. (C) Coronal diffusion-weighted image of the brain of the same child. There is extensive signal intensity apparent in the corpus callosum and bilaterally in the deep white matter.

aggressively to ensure an optimal milieu for cerebral recovery.

### Operative Intervention

Intracranial mass lesions, although less frequent in children as compared with adults, need to be treated expeditiously. Elevation of depressed skull fractures, removal of epi- and subdural hematomas, and resection of contused or infarcted brain should be done to lower ICP and maintain both focal and diffuse cerebral perfusion. Other operative interventions in the head trauma patient may involve repair of vascular injury, stenting of arterial dissection, decompressive craniectomy or lobectomy, and cranial reconstruction.

### Head Position and Intracranial Pressure

Optimally, the head should be kept in a neutral position, and jugular venous obstruction should be avoided by endotracheal (ET) ties or tape to prevent elevation of ICP. Elevation of the head of the bed (HOB) to thirty degrees usually decreases ICP with maintained MAP, and is a level that has no detrimental effect on CPP or CBF. Elevating the HOB above 30 degrees has been shown to reduce CPP in some adult patients.<sup>38</sup>

### Cerebrospinal Fluid Diversion

CSF diversion can be used as a treatment methodology for elevated ICP when an external ventricular drain (EVD) has been placed. Drains can be kept clamped

and continuously transduced, draining intermittently for ICP elevations or kept open at or above the level of the tragus for continuous drainage.

### Sedation and Pain Management

Studies demonstrating the utility of short-acting sedatives have been done in adult TBI, though sedation of the pediatric TBI patient is controversial. Midazolam (Versed) or narcotics are often used in the acute setting, but long-acting sedatives are not desirable because they cloud frequent neurological exams. As a result, lorazepam (Ativan) and diazepam (Valium) should be avoided. Pain control is managed by fast-acting morphine or fentanyl. Ketamine is avoided because it elevates ICP.

### Osmolar Agents and Diuretics

Osmotic agents such as mannitol and diuretics have been employed for treatment of elevated ICP in TBI. Mannitol is believed to exert its effects in two ways: decreasing blood viscosity and reducing the extravascular volume. Through normal autoregulation, decreased blood viscosity increases CPP while reducing extravascular volume and increasing intravascular volume and CBF. Older studies challenged the utility of mannitol in head trauma; however, even though there are no prospectively randomized studies that show benefit for use in pediatric head trauma, there is clinical evidence that demonstrates its positive effect.<sup>39</sup> The half-life of mannitol is 3 to 4 hours, and the dosing interval can range from every 2 to every 4 hours. Doses range from 0.25 gm/kg of total body weight to 1 gm/kg IV. Normonatremia should be maintained with normotension and normal to slightly elevated serum osmolarity ( $\leq 320$  mOsm). To avoid hypotension, hypovolemia, or hypernatremia, central venous pressure (CVP) should be monitored; hypotension should be treated with saline or albumin boluses. Diuretics, such as furosemide (Lasix), have been used in conjunction with mannitol in TBI. Furosemide reduces intra- and extravascular volume and induces the secretion of sodium (Na<sup>+</sup>). Patients should be carefully monitored if diuretics are used in TBI because of possible hypovolemia and hypotension. Recently, there has been renewed interest in hypertonic, 3% sodium chloride (3%NaCl).<sup>13,40</sup> Rat head injury models and clinical studies in children demonstrate that both mannitol and hypertonic saline reduce ICP, but the effect of hypertonic saline may be greater and longer lasting.

### Mechanical Ventilation

For severe head trauma patients, mechanical ventilation is necessary to (1) maintain adequate oxygen (O<sub>2</sub>) saturation (> 90%) and (2) avoid hypercarbia (pCO<sub>2</sub> should

be 35–38 mmHg). Hyperventilation should be avoided because hypocarbia induces vasoconstriction and may lead to relative ischemia.<sup>41</sup>

### Antiepileptic Medications

Antiepileptics are commonly started during the initial hours after severe head injury, especially if there is parenchymal injury apparent on the initial CT scan. Seizures can increase ICP by increasing metabolic demand and Valsalva effect, releasing neurotoxic excitatory amino acids, and decreasing or increasing systemic blood pressure.<sup>42,43</sup> Adult studies show that the benefit of giving 1 to 2 weeks of phenytoin outweighs the minimal risks. Infants are especially at risk for posttraumatic seizures, occurring in upwards of 80% of children under 1 year old, even after minor head trauma. In infants, phenobarbital may be given IV or orally for seizure prophylaxis; carbamazepime (Tegretol) has also been used on occasion.

### Barbiturates

Barbiturates exert neuroprotective effects by reducing cerebral metabolism, lowering oxygen extraction and demand. In addition, ischemia is better tolerated, free radical injury is prevented, and vascular tone is improved. Early initiation of pentobarbital and “barbiturate coma” may prevent malignant cerebral edema and secondary injury. Because data suggest that complete burst suppression on electroencephalography (EEG) may not be necessary for the protective effects of pentobarbital, lower doses (5 mg/kg IV every 4–6 hours) may be given to avoid myocardial depression and resultant systemic hypotension. Starting at a lower dose also allows the increased titration of pentobarbital to treat unresponsive elevated ICP. We recommend that during this period of pentobarbital treatment, enteric feeding be avoided due to the higher incidence of gastroparesis and ileus seen with barbiturates. Care of the comatose patient must involve monitoring of liver function and prophylaxis of deep venous thrombosis and pressure-induced decubiti.

### Steroids

There have been no conclusive studies to date that have demonstrated a significant advantage of the use of steroids in head trauma, in adults or in children. Steroid use in children though may lead to increased complications of infection and cortisol suppression.

### Antibiotics

The prophylactic use of antibiotics is not supported by the literature, although antibiotic therapy is often initiated at the time of monitor or EVD placement. We ad-

minister antibiotics while an EVD is in use, to cover *Staphylococcus aureus* and *S.epidermidis*. In adult populations, preliminary results investigating IV immunoglobulins as a prophylaxis against infection in trauma victims are encouraging, but no studies have been done in children.<sup>44</sup>

### Hypothermia

As early as the 1950s, hypothermia was used for the treatment of TBI in children.<sup>45-47</sup> Numerous adult Phase II and II TBI studies have shown a correlation between induced hypothermia and a good outcome, although the most recent adult trial did not prove efficacy. In the United States there is an ongoing Phase II prospective trial to study the safety of hypothermia in pediatric TBI and a Phase III trial that was recently initiated in Canada. Previous studies have suggested a potential benefit of hypothermia in younger patients with TBI.

### MORTALITY AND OUTCOMES

In the past 2 decades, there has been a significant decline in overall morbidity and mortality associated with pediatric TBI.<sup>3,48</sup> However, children under 4 years of age and older adolescents have higher morbidity and mortality rates as compared with their school-age counterparts.<sup>11,49-52</sup> This may be in part due to the different mechanisms of injury that occur at different ages. Luerssen et al reported that for all initial GCS scores, patients younger than 15 years of age had lower mortality rates than those over 15 years old, but the youngest children had the highest mortality. Mortality rates decreased with increasing age, until age 14, and then increased until adulthood.<sup>3</sup> In contrast, Levi found that outcomes worsened with increasing age during childhood, corresponding to increasing severity of injury.<sup>24</sup> Tilford et al studied the correlation between various interventions and outcomes for pediatric TBI in different pediatric intensive care units.<sup>53</sup> Admission severity, the use of paralytic agents, induced hypothermia, and ICP monitoring were evaluated. Despite significant variations in the modalities of treatment, there was no significant correlation with outcome. It is clear that multicenter, prospectively randomized, controlled studies are needed to determine the significance of these interventions.

Posttraumatic seizures may complicate or prolong the recovery period after TBI. During childhood, the developing brain is in the process of synaptogenesis and may be more vulnerable to traumatic insult. This may explain why infants have high morbidity rates and are prone to posttraumatic seizures. Chronic seizure disorder has been reported to be as high as 7.4% in long-term follow-up studies of children with severe head injury. In infants, the incidence of acute, posttraumatic

seizures may be as high as 80%.<sup>54</sup> In children with only mild or moderate TBI, long-term incidence of seizures is 0.2% and 1.6% respectively, not much higher than in the general population.

Children are also more likely to have good functional outcomes after TBI as compared with adults.<sup>55</sup> In a long-term follow-up of 50 pediatric trauma patients, most patients were employed or in school, and only 5% remained dependent and disabled.<sup>56</sup> Long-term outcome may be assessed by the Glasgow Outcome Scale (GOS) and/or the Functional Independence Measure (FIM), which analyzes 18 items covering self care, mobility, locomotion, communication, social skills, and other abilities.<sup>57,58</sup> Perhaps due to the diffuse nature of pediatric brain injury, even children with mild to moderate TBI may suffer significant, long-term cognitive defects that affect their abilities to function independently.<sup>59</sup> Intellect, concentration, memory, complex thought, judgment, speech, language, and motor deficits impact long-term outcome.<sup>60-64</sup> Recent studies have focused on the development of standardized tests to assess cognitive and linguistic skills of school-aged children after TBI.<sup>65</sup> The Pediatric Test of Traumatic Brain Injury (PTBI) is a tool created to measure attention, memory, language, reading, writing, and cognitive abilities in the pediatric head trauma population. Studies of performance in school and intellectual abilities have shown that children may suffer from posttraumatic attention deficit or postconcussion syndrome for months after head injury.<sup>18,59,66</sup> Preexisting emotional problems may be compounded or magnified after TBI, thus making it important to determine both the preinjury and postinjury behavioral characteristics of the child.<sup>67</sup> Collins et al reported that lower neuropsychological testing scores were found in college football players that had sustained previous concussion.<sup>68</sup> Football players with multiple concussions had even worse performance on cognitive testing. Master and colleagues reported that teenage soccer players had worse performance on planning and memory tests than their age matched counterparts, in noncontact sports.<sup>69</sup> Ewing-Cobbs demonstrated that after testing 35 children with mild to moderate posttraumatic head injury, subtle changes in performance may be long lasting, with language deficits persisting 2 years after injury in some subjects.<sup>59</sup> In contrast, other studies have suggested that children with only mild head injury performed similarly to noninjured control children when testing was done 1 year after the initial insult.<sup>70</sup>

### CHILD ABUSE AND THE NEUROSURGEON

Sadly, child abuse is a frequent cause of injury in the pediatric population.<sup>7,8,71</sup> Child abuse was the cause of injury in 56% of TBI in infants less than 1 year of age and occurred less frequently in children over 3 years of age.<sup>7</sup>

Nonaccidental head trauma (NAHT) occurred in approximately 12% of all cases of confirmed child abuse.<sup>72</sup> In a review of 173 cases of NAHT in children under 2 years of age, 31.2% of these patients were initially "missed."<sup>71</sup> Missed diagnosis most often occurs in younger and less severely injured children, usually from intact families. Victims may suffer direct blows to the head, strangulation, suffocation, or shaking and often have poor outcomes in retrospective studies.<sup>73</sup> Physicians are required by law to report nonaccidental injuries and are often aided by hospitals that now have child advocacy teams. These teams provide services to help the victims and families of the abused child, to counsel, and to provide temporary or permanent shelter if necessary.

For the physician, a thorough examination often reveals subtle injuries that may be a result of abuse. Ophthalmological examination of the dilated pupil can demonstrate specific, characteristic findings. Besides examining the child for sluggish or nonresponsive pupillary reflexes, external signs of periorbital edema, ecchymoses, or lid lacerations should be investigated. A traumatic sixth nerve palsy or paresis may result in esotropia. Conjunctival lacerations, corneal abrasion, traumatic cataracts, hyphema, and elevated intraocular pressures may be found. All children with suspected NAHT should have a thorough dilated fundoscopic examination to rule out retinal hemorrhages, the hallmark ophthalmological finding in "shaken-baby syndrome."<sup>73,74</sup> Abusive head trauma may produce multilayer bleeds, retinal folding, and possible damage to the optic nerve or macula.

X-rays of the chest and long bones may be useful for identifying new or chronic fractures.<sup>73</sup> On neuroimaging studies, signs of preexisting brain injury may also be apparent.<sup>59</sup> Chronic subdural hematomas and subarachnoid bleeds were more often found in NAHT.<sup>73</sup> In 81% of positive cases of nonaccidental head trauma, DWI revealed more extensive brain injury than was demonstrated by conventional MRI. Findings include hemorrhages or injury within the corpus callosum, subcortical white matter, cerebellar peduncles, and brainstem.<sup>31</sup>

## CONCLUSION

Injury prevention education is the best treatment for TBI and remains the focus of such organizations as "Think First." Through education and advocacy, neurosurgeons can reduce this most common "disease." Educating children about the benefits of helmets and risks of driving while under the influence of drugs and alcohol can help deter these types of activities. Warning parents about the dangers associated with recreational vehicles, and infants in walkers with wheels, can also help prevent unnecessary accidents.<sup>73</sup> Once injury has occurred, the goals of management include normalizing

the ICP and optimizing CBF and perfusion. Although there have been significant advances in the understanding of the mechanisms of secondary injury, the long-term outcomes of young children with head injury remain poor.

## PEARLS OF MANAGEMENT OF PEDIATRIC HEAD TRAUMA

1. The goals of management of TBI should be directed toward maintaining normal oxygenation and cerebral perfusion in children, while preventing and treating conditions that would elevate ICP. One needs to take into consideration the different physiology of children (e.g., lower MAP in infants and toddlers).
2. Diffusion-weighted imaging provides a useful tool for the early diagnosis of brain injury in children with traumatic, diffuse axonal injury.
3. Prevention remains the best intervention of severe head injury in children.

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