Acute Care Management of Closed Head Injury in Childhood

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Trauma is the most significant cause of death and disability among children in the United States. More than 22,000 children between the ages of 1 and 19 years die each year from trauma, and brain injury is the leading cause of death.¹ The mortality from head injury, however, is clearly overshadowed by the sheer magnitude of the morbidity in pediatric head injury. This morbidity may vary from minor reversible injury to lifelong disability resulting from a more serious injury. The incidence of head injury has been estimated between 90 to 250 per 100,000 population.² At our children's hospital emergency room alone, more than 1200 patients are seen for head injury each year. Even if only a small percentage of these injuries are serious, this represents a very large number of patients at the national level.

The mechanism of injury is often related to a child's developmental stage. In the infant, falls from tables or parents' arms are more frequent and usually not as severe as injuries in the older child involved in a high speed accident, while bicycling or driving a motor vehicle.² Assaults are more common in the older child and in the infant as a victim of abuse or neglect.

Mortality from head injury generally decreases during childhood until late adolescence, when motor vehicle crashes are more frequent and result in more severe injuries.³ Although children have a better chance of surviving brain injury than adults, there is no indication that children have any better recovery than adults from a given brain injury. In fact, they may be more susceptible to long-term cognitive deficits from minor injury, especially if younger than 4 years of age.

PATHOPHYSIOLOGY

Head injuries can be divided into primary and
secondary categories. Primary injuries are those inflicted immediately by the trauma and include skull fractures, contusions or lacerations of the brain, subarachnoid hemorrhage, and hematomas. Secondary injuries are a result of ischemia, hypoxia, hypotension, infection, hydrocephalus, or increased intracranial pressure (ICP). Many of these injuries are potentially preventable and are therefore the focus of most treatment plans.

Diffuse axonal injury results from shear stresses on the brain imparted by acceleration-deceleration forces, particularly when combined with angular or rotatory motion. This primary injury is more common in the child, whose head is proportionately larger in relation to the body and whose neck muscles are less developed. There is less restraint of motion, and greater forces are generated. On the microscopic level, there may be a spectrum of injury varying from only a functional disturbance of the neuron to complete disruption of the axons. The former is quickly reversible, while the latter results in permanent loss of function. Disruption of the myelin sheath around axons can result in severe loss of function that is only potentially reversible. The typical sites of injury include the internal capsule, corpus callosum, cerebellar peduncle, and brainstem. Obviously, even minimal shearing injury in these regions can result in severe neurological deficits.

Increased ICP results from the expansion of one or several components of the intracranial space: the cerebral blood volume (both venous and arterial), brain tissue volume, hematomas (intracerebral, subdural, and epidural), and cerebrospinal fluid (CSF) spaces. The ill effects of increased ICP result from direct compression of the brain structures, such as herniation under the falk cerebri or through the tentorium cerebelli, or indirectly through decreased cerebral blood flow with resulting focal or diffuse secondary ischemic injury. Increased ICP is more common in the more severely injured patient.

The CSF spaces can act as a buffer, decreasing in response to the expansion of the other components. If the spaces are trapped or obstructed, as may happen with subarachnoid or intraventricular hemorrhage, they can act as an additional mass and increase ICP. Children have less CSF buffering capacity than an open fontanelle or sutures, probably related to a smaller spinal CSF volume. This puts the child at risk for a more sudden decompensation from smaller changes in the ICP.

In the normal brain, the cerebral blood volume is closely regulated through a tight coupling of cerebral metabolism with the regional blood flow, a process known as autoregulation. Small changes in the partial pressure of carbon dioxide in the blood can result in rapid and very dramatic changes in cerebral blood volume. Areas of cerebral injury may lose autoregulation, with vasodilation unrelated to hypercarbia or metabolic demands, resulting in significant hypervolemia, increased volume, and ultimately increased ICP.

The volume of brain tissue, exclusive of CSF or blood volume, can increase due to interstitial edema, with a resultant rise in ICP. Even a focal area of edema may be particularly threatening if it impinges on vital structures, such as the brainstem, or obstructs the flow of CSF. Interestingly, diffuse axonal injury is less likely to result in an increase in ICP.

In children, the infrequency of subdural hematoma and the greater frequency of epidural hematoma may relate to the greater flexibility of the immature skull and the lack of dural adhesions. With open cranial sutures and thin bone, there may be greater transmission of force to the brain, resulting in greater shear injury, dural lacerations, and parenchymal injury with subsequent edema.

CLINICAL EVALUATION

The pertinent elements of the head-injured child's clinical history include the mechanism of injury and determination of any changes in the patient's clinical examination over time. The patient who has sustained a high-speed, high-force mechanism of injury, such as a motor vehicle crash or a blow to the head from a baseball bat, raises a greater level of concern for a significant injury even if the patient otherwise appears well. A subtle but distinct decline in a child's level of function, documented prior to arrival in the emergency unit or office, might be the only clue to a significant injury.

In examining children with head injury, emphasis should be placed on the trend of serial examinations and the presence of any focal findings or irritability and lethargy. The classic signs of increased ICP are frequently absent or incomplete in children, even with a significantly elevated ICP. Papilledema requires days to develop, and the Cushing response to increased ICP (bradycardia with systemic hypertension) is not as predictable in children and may be a preterminal event. Systemic hypotension or hypoxia also can cloud the examination, causing an artificial decrease in the neurologic state that quickly reverses with correction of the underlying systemic abnormality.

The Glasgow Coma Scale (GCS) has become the
TABLE

Glasgow Coma Scale* Modified for Pediatric Patients

<table>
<thead>
<tr>
<th>Eye-Opening Response</th>
<th>&gt; 1 Year</th>
<th>&lt; 1 Year</th>
</tr>
</thead>
<tbody>
<tr>
<td>Score</td>
<td>Spontaneous</td>
<td>Spontaneous</td>
</tr>
<tr>
<td></td>
<td>To verbal command</td>
<td>To shout</td>
</tr>
<tr>
<td>4</td>
<td>To pain</td>
<td>To pain</td>
</tr>
<tr>
<td>3</td>
<td>None</td>
<td>None</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Motor Response</th>
<th>&gt; 1 Year</th>
<th>&lt; 1 Year</th>
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<tbody>
<tr>
<td>Score</td>
<td>Spontaneous</td>
<td>Spontaneous</td>
</tr>
<tr>
<td>6</td>
<td>Localizes pain</td>
<td>Localizes pain</td>
</tr>
<tr>
<td>5</td>
<td>Withdraws to pain</td>
<td>Withdraws to pain</td>
</tr>
<tr>
<td>4</td>
<td>Abnormal flexion to pain</td>
<td>Abnormal flexion to pain</td>
</tr>
<tr>
<td></td>
<td>(decorticate rigidity)</td>
<td>(decorticate rigidity)</td>
</tr>
<tr>
<td>3</td>
<td>Abnormal extension to pain</td>
<td>Abnormal extension to pain</td>
</tr>
<tr>
<td></td>
<td>(decerebrate rigidity)</td>
<td>(decerebrate rigidity)</td>
</tr>
<tr>
<td>2</td>
<td>None</td>
<td>None</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Verbal Response</th>
<th>&gt; 5 Years</th>
<th>2 to 5 Years</th>
<th>0 to 23 Months</th>
</tr>
</thead>
<tbody>
<tr>
<td>Score</td>
<td>Oriented and converses</td>
<td>Appropriate words and phrases</td>
<td>Babbles, coos appropriately</td>
</tr>
<tr>
<td></td>
<td>Confused conversation</td>
<td>Inappropriate words</td>
<td>Cries, but is consoleable</td>
</tr>
<tr>
<td></td>
<td>Inappropriate words</td>
<td>Persistent crying or screaming to pain</td>
<td>Persistent crying or screaming to pain</td>
</tr>
<tr>
<td>5</td>
<td>Grunts or moans to pain</td>
<td>Grunts or moans to pain</td>
<td>Grunts or moans to pain</td>
</tr>
<tr>
<td>4</td>
<td>None</td>
<td>None</td>
<td>None</td>
</tr>
</tbody>
</table>

* Glasgow Coma Score = sum of best eye opening, motor, and portal responses. Range = 3 to 15. Usual definitions of severity of head injury: Severe — score of < 9; Moderate — score of 9 to 12; Mild — score of 13 to 15.
Adapted from Simon J.E. 6

IMAGING

The available imaging methods include plain radiographs, computed tomography (CT), and magnetic resonance imaging (MRI). Plain radiographs are quite inexpensive, but define only skull fractures. Only depressed or open fractures are clinically important in themselves and are usually well defined by clinical examination combined with CT. Harwood-Nash et al found skull fractures in 26.6% of their patients, but the finding was only significant when accompanied by another clinical finding. 7

The presence of a skull fracture, however, is associated with an increased risk of intracranial injury. 8,9 One study found the risk of subdural or epidural hematoma increased two to three times when a skull fracture was present. 10 However, the absence of skull fracture does not reliably exclude a significant intracranial injury. Harwood-Nash et al found 8% of children without a skull fracture had a brain injury, or a subdural or epidural hematoma. 8 Hahn and McLone reported epidural hematomas in 3% and subdural hematomas in 1.5% of head-injured children without skull fracture. 10

Computed tomography is now nearly ubiquitous at hospitals. The newest generation of scanners is fast, generating a complete head study in 5 to 10 minutes, with slice acquisition times of several seconds negating most patient motion. Computed tomography is able to identify intracranial hematomas easily and to define cerebral contusions and shifting of the brain structures. Increased ICP can be inferred when obliteration of normal CSF spaces is seen. When the images are adjusted to evaluate bone density, depressed and most clinically important linear skull

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fractures are identified. Computed tomography allows for the prompt identification and treatment of the injuries that are potentially reversible, and may be the single factor most responsible for the recent improved outcome of head-injured patients.

Magnetic resonance imaging has little utility in the acute management of head injury. Magnetic resonance imaging is much less available than CT. It is relatively insensitive to acute hemorrhage, and the scan times are long with the image quality easily degraded by even minimal motion. Magnetic resonance imaging does have a role in the subacute care of some head-injured patients, being extremely sensitive in detecting diffuse and deep shearing injuries of the brain and brainstem, allowing a more accurate assessment of the extent of injury and the prognosis for recovery.

There is great debate regarding the evaluation of "minor" head injury, typically those patients with a GCS score of 14 to 15. Radiographic evaluation using CT is clearly indicated for pediatric patients presenting with a GCS score ≤13, with immediate scanning for severely injured patients (GCS score ≤8). There is no acute diagnostic role for plain radiographic evaluation in these patients unless indicated for surgical planning.

Stein et al. detected significant abnormalities on CT in 17.6% of 658 adult patients with a GCS score of 13 to 15, including 13% of 454 patients with a GCS score of 15.11 Jeret et al. detected CT findings in 9.4% of patients who had a GCS score of 15 and who had a history of loss of consciousness or amnesia.12 They were unable to identify any other combination of clinical findings that would predict CT abnormalities. There also have been recommendations that the GCS score threshold for radiographic evaluation be lowered for young children due to their deformable skull and possible increased risk of significant injuries.13,14

We recommend CT scanning for any child with a head injury who has a neurological deficit or a GCS score ≤14. Other factors that raise the suspicion of injury and warrant CT scanning regardless of the GCS score include a high force mechanism of injury, a history of change in the level of consciousness, or a planned intervention or surgery where the clinical examination will be lost for more than a brief period of time. The GCS score threshold for scanning also is reduced for infants younger than 2 years of age (Figure 1).

**CHILD ABUSE**

The clinician involved in the evaluation and treatment of children with closed head injuries must be thoroughly versed in the symptoms and signs of child abuse. The abused child may present with only a head injury or an uncomplicated skull fracture. In one series of abused children, 22% had brain injury and 55% had skull fractures, while in another series two thirds had a skull fracture.15,16 The "whiplash shaken infant syndrome" recognizes the association of significant brain injury with retinal hemorrhages.17 The shaken child is subjected to acceleration/deceleration and rotational forces that can result in white matter shear lesions, interhemispheric subdural hematomas, and retinal hemorrhages.13,16,18 Retinal hemorrhages have not
been observed in children suffering mild to moderate accidental trauma and are extremely rare even with severe trauma.\textsuperscript{19,20} Retinal hemorrhages should always raise the suspicion of abuse, and in the child with less than severe trauma, retinal hemorrhages are diagnostic of a shaking injury.

**THERAPY**

The treatment of minor head injury that will identify all significant injuries with the minimum cost and resources has yet to be defined. We have adopted a conservative approach, evaluating many of these patients with CT scanning. Any child with a history of loss of consciousness or neurological deficit undergoes a CT scan, and those younger than 2 years of age also are admitted regardless of CT findings. Those patients with abnormal CT findings are admitted, and the others may be discharged home with supervision. All children with known or suspected skull fractures undergo CT scanning. Patients with fractures that cross dural venous sinuses are at risk for delayed epidural hematomas and are admitted for observation. Patients younger than 1 year of age with fractures are admitted; due to their size, even minimal emesis associated with a minor injury may lead to dehydration. Patients older than 1 year with normal examinations are admitted only for open or depressed fractures; other patients may be discharged home with supervision. Although physicians may be concerned that a patient with an undetected significant injury may be sent home and later deteriorate, the so-called talk and die patient, previously reported cases have all manifested some deficit or had a GCS score <15 at the time of initial evaluation, and surgically treatable lesions usually were not present.\textsuperscript{10,21,22}

The initial therapy of the severely head-injured child should establish an adequate airway and maintain ventilation and circulation. Endotracheal intubation should be undertaken in the unconscious patient, any patient who is unable to maintain an open airway, or in the agitated patient who will need additional diagnostic or therapeutic interventions. Ideally, intubation should use a pharmacologic technique with pre-oxygenation, a nondepolarizing muscle relaxant, and a rapid-acting anesthetic to minimize any increase in ICP associated with the intubation.

Fluid resuscitation uses isotonic intravenous fluids, avoiding hypotonic solutions that may contribute free water to brain edema. Fluid overload should be avoided, but "dehydration" is not the goal and can result in hypoperfusion. Although colloid may have theoretical advantages over crystalloid with respect to increased ICP, crystalloid is readily available and has no infectious risk associated with its use.

Surgically treatable lesions should be identified with prompt CT scanning (Figure 2). These lesions include epidural and subdural hematomas, skull fractures depressed more than one half the thickness of the skull or with in-driven fragments, and obstructive hydrocephalus. Children are more likely to have cerebral contusions rather than frank intracerebral hematomas, and evacuation of contused brain is reserved for a life-threatening mass or when other therapy fails to control the increased ICP. Burrholes in the emergency room are rarely, if ever, indicated in children. Most sudden decompensations in children are due to diffuse swelling, and epidural and subdural clots have a jelly-like consistency that resists easy drainage through a small hole.

Intracranial pressure monitoring is indicated for patients with a GCS score <9, those with a GCS score <13, and decreasing levels of consciousness or lesions on CT that have increased risk of swelling (ie, contusions). Patients who are at risk for swelling and will be under anesthesia or kept intubated and paralyzed for other injuries also will require monitoring. Fiberoptic catheters have been developed that can be placed quickly through a small hole in the skull, have minimal drift over many days, and can be placed into the subarachnoid space, cerebral tissue, or intraventricularly and maintain accurate ICP measurements. They have replaced the fluid-coupled subarachnoid bolts, screws, and catheters that were susceptible to occlusion and air bubbles, and did not allow drainage of CSF. Only a ventriculostomy can drain CSF to reduce the ICP, and it can be fluid coupled to a transducer or used with a fiberoptic catheter to provide ICP measurements.

The medical treatment of increased ICP begins with simple maneuvers. Cerebral blood volume is
reduced by minimizing venous outflow obstructions, such as circumferential ties on the neck that compress the jugular veins. Ventilator end expiratory pressures are kept to the minimum required, and the head of the bed is elevated slightly. Hyperpyrexia increases cerebral metabolism, thus cerebral blood flow and ICP, and should be treated with cooling blankets and antipyretics. Anticonvulsant medication is used routinely, and any seizure activity is actively treated with additional medication. Often forgotten is the increased basal metabolic rate accompanying severe injuries, placing the patient into negative nitrogen balance. Vigorous caloric support should be instituted as soon as possible, using the enteral route when able and the parenteral route if necessary.

Cerebral vessel volume is reduced through hyperventilation and induced hypocarbia. The pCO₂ is initially reduced to 25 torr, but may be taken as low as 20 torr. Since this reduction in blood volume is based on intact autoregulation, and areas of injury may have impaired autoregulation, concern has been raised that severe hyperventilation may cause a steal phenomenon from the normal cerebral tissue, resulting in additional areas of injury. Hyperventilation, however, remains a good treatment for increased ICP in sedated patients.

Sedation, analgesia, and neuromuscular blockade must be used consistently in deeply comatose patients, who will respond to noxious stimuli with an increase in ICP. Short-acting narcotics blunt these responses, yet allow reversal for interval assessments of the neurologic condition. Muscular actions, such as coughing and gagging, will increase tracheobronchial and intra-abdominal pressure, decrease venous return, and increase ICP; neuromuscular blockade prevents this. The response to endotracheal suctioning also can be moderated by pretreatment with intravenous lidocaine (1 mg/kg) immediately before suctioning.

More aggressive treatment uses osmotic diuretics such as mannitol. Mannitol is given in bolus doses ranging from 0.25 to 1 g/kg of patient weight to reduce the ICP and increase the measured serum osmolality to approximately 300 mOsm/kg. Renal failure can result from prolonged maintenance of serum osmolality >315 mOsm/kg. Mannitol's mechanism of action was thought to be removal of water from the interstitial space, but more recent evidence suggests a complex interaction of changes in blood viscosity, cerebral blood flow, and vessel diameter.²³

Cerebrospinal fluid drainage can effectively reduce the ICP. With cerebral swelling, the compliance of the brain decreases, and very small intracranial volume changes result in significant pressure changes. Removal of as little as 1 mL of CSF can dramatically reduce the ICP in the severely injured brain. Cerebral spinal fluid drainage requires placement of a ventriculostomy with its associated risks of hemorrhage and infection, and extremely small ventricles in some children cannot be cannulated.

Even more aggressive therapy can be applied in an attempt to reduce the cerebral metabolic rate, thus reducing blood flow and ICP. Barbiturate-induced coma has no proven prophylactic role in head injury, but may be useful when the ICP remains refractory to other treatments. Extensive cardiac and hemodynamic monitoring is required for side effect hypotension and often requires pressor support. Hypothermia to core temperatures below 34°C also will reduce metabolism and ICP, but is complicated by a severe decline in cardiac output, cardiac arrhythmias, decreased renal function, and poor leukocyte function. Newer therapies under evaluation are directed at the biochemical events induced by hypoxia and ischemia and common to irreversible cell death. These include free-radical scavenging, inhibitors of excitatory amino acids, and calcium channel blockers.

The goal of treatment is to maintain the ICP in the normal or lowest range possible. As the injury progresses, one must often adjust the therapy and accept higher ICP values, escalating the intensity and invasiveness of therapy to maintain the cerebral perfusion pressure. Elevated ICP may require treatment for up to several weeks. Any change in the ability to control the ICP requires reevaluating the patient, assuring adequate ventilation, and repeating the CT scan to assess for the interval development of surgically correctable lesions.

CONCLUSION

Closed head injury encompasses a vast spectrum of pathology, from the minor falls with disorientation to the severe brain injury with coma and increased intracranial pressure. This overview of the pathophysiology of acute head injury, its radiographic evaluation, and the various treatments that are used should help the primary care pediatrician understand the difficulties encountered in treating these patients. With prompt evaluation and treatment, the outcome of these injuries can usually be improved.

REFERENCES


