

Traumatic brain and spinal cord injury

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Head injuries account for 75% of all pediatric hospitalizations for trauma [1] and contribute to nearly 80% of trauma deaths in children [2]. Severe childhood head injuries carry a 9% to 35% mortality [3]. Although the demographics, pathophysiology, and management of head injury in adults and children share many similarities, there are notable differences. Whereas adult head injuries are usually caused by motor vehicle accidents and assaults, childhood head injuries are often the result of domestic falls, motor vehicle accidents, and recreational injuries such as bicycle accidents and sports mishaps [4–8]. Child abuse (or nonaccidental trauma) remains a significant cause of head injuries in children under 2 years of age; 7% to 20% of child abuse victims have significant head injuries [9]. Finally, gang and drug-related assaults, although accounting for only a fraction of childhood head injuries, are increasing at an alarming rate. Brain injuries from firearms account for 12% of all pediatric deaths [10].

The pathophysiology and outcome of head trauma differs significantly between children and adults. Severe head injury occurs less frequently in children than adults [11]. Mortality is lower for children than for adults, even after stratification by severity of head injury [11–14]. The frequency with which children deteriorate rapidly after an initial lucid interval (the so-called “talk and die” syndrome) is also lower than in adults (3% versus 10%) [15]. Surgical mass lesions such as subdural and epidural hematomas occur less frequently in children than in adults and when present are associated with a lower mortality [11,16,17]. The biokinetics of the head and body of the young child and the deformability of the young skull and spine may influence both the frequency and severity of certain injuries as well as the outcome following childhood head trauma [14]. The plasticity of the developing brain may contribute to greater recovery from traumatic brain injury in children. Finally, a number of issues unique to pediatric head trauma deserve mention. Immediate posttraumatic seizures without serious head injury occur much more frequently in children than adults and are more frequent in younger children than in older children [18]. Posttraumatic

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Table 1
Glasgow coma score – possible scores 3–15

Score	Motor	Verbal	Eye opening
6	Obeys commands		
5	Localizes	Oriented	
4	Withdraws	Confused	Spontaneous
3	Flexor posturing	Inappropriate words	To voice
2	Extensor posturing	Incomprehensible sounds	To pain
1	None/Flaccid	None	None

migraine syndromes are largely confined to the pediatric age group. The pediatric postconcussive syndrome is characteristic and can cause confusion with an expanding mass lesion. Finally, the issue of child abuse remains an important part of the evaluation of pediatric head injury, particularly in the young age group.

Assessment of the head-injured child begins at the time of initial resuscitation. Information about the timing and mechanism of injury and the extent and effectiveness of resuscitative efforts and therapeutic interventions is sought from those present at the scene and from emergency personnel. Most importantly, the mechanism of injury, the extent of the neurologic signs and symptoms, and the evolution of the child's clinical status—whether improving, deteriorating, or remaining stable—all provide critical information that may aid in determining the extent of brain injury and establishing therapeutic priorities. The Glasgow Coma Scale (GCS) [19] and, for younger children, the Children's Coma Scale [20] are most often used to quantify the level of arousal (Tables 1, 2). GCS scores range from 3 to 15 points; scores of 13 to 15 are defined as mild, 9 to 12 as moderate, and 3 to 8 as severe head injuries. The clinical condition of the child often fluctuates and requires a change in the approach to treatment. In this article, management schemes are divided arbitrarily into those for children with mild head injuries and those for children with moderate and severe head injuries.

Evaluation and management of mild head injuries

It is estimated that as many as 90,000 children with mild head injuries (GCS scores of 13–15) are seen in hospital emergency rooms and pediatricians' offices each year [21]. Most of these children do well without any intervention, but

Table 2
Children's coma score – possible scores 3–15

Score	Motor	Verbal	Eye opening
6	Spontaneous		
5	Purposeful/localizes	Coos, babbles	
4	Withdraws	Irritable, cries	Spontaneous
3	Flexor posturing	Cries to pain	To voice
2	Extensor posturing	Moans to pain	To pain
1	None/Flaccid	None	None

significant health care resources are expended to identify the rare child who harbors a potentially life-threatening intracranial injury. The critical question is how best to identify those rare children who are at risk for complications without wasting precious health care resources. Deciding whether a child should undergo a CT scan or be admitted for hospital observation involves assessing the risk of an occult intracranial abnormality. The incidence of radiographic intracranial abnormalities in children with minor head injuries (GCS scores of 13–15) ranges from 3% to 53% [17,22–32]; even among those with a GCS score of 15, the risk of intracranial injury is 2.5% to 7% [24,29,30,33]. Choux and colleagues [17] have estimated that 57% of children and 85% of infants with epidural hematomas have no loss of consciousness at the moment of impact, and 7% have no alteration in consciousness at any time following the injury. It is therefore readily apparent that clinically significant, even life-threatening, intracranial injuries can exist in children with no significant initial clinical manifestations.

The incidence of delayed deterioration in patients with head injuries is estimated to be between 1% and 4% [34]. Although much has been written about delayed deterioration following minor head injury [15,31,34–53], a detailed review suggests that in virtually all of these reports (1) insufficient data were available to properly evaluate the case, (2) initial CT scans were either abnormal or were never performed, or (3) patients had moderate or severe brain injuries (GCS scores < 13) on the initial examination, or were taking anticoagulants at the time of the injury [35–38,41,44,46–49,54]. On the other hand, delayed deterioration in children with both a normal initial CT scan and neurologic examination is extraordinarily rare. In one series of 791 children with minor head injuries, only 2 (0.3%) deteriorated after a normal initial CT scan, one child from a delayed epidural hematoma and the other from diffuse brain swelling [29]. Both children had a GCS score of 13 on admission, and it is not clear whether they improved before their deterioration. Several recent retrospective reviews of minor head injuries in patients with GCS scores of 13 to 15 and normal CT scans also provide mounting evidence that late deterioration in the setting of a minor head injury and a normal initial CT scan is rare [21,24,28,29,41,42,54,55]. Five separate retrospective pediatric studies including a total of 1241 children suggest that these children could safely be discharged to home with an approximately 0.5% return rate and no significant complications [21,24,28,29,54].

Using a prospectively designed algorithm (Box 1) for 216 children older than 23 months of age meeting predetermined clinical criteria (Box 2) and no visible intracranial injury on initial CT scans undergoing a short period of observation and subsequent discharge from the emergency department for those meeting discharge criteria (Box 3), the author and colleagues found no untoward complications and calculated a statistical risk of less than 1% (using 95% confidence limits) of late deterioration (M.S. Dias et al, unpublished data). Other retrospective studies have demonstrated that children with uncomplicated skull fractures [56] and basilar skull fractures [57] can be similarly managed.

Although it is difficult to make absolute recommendations about the management of minor head injuries, this algorithm is a safe and cost-effective manage-

Box 1. Clinical parameters for emergency department management of children with minor head injuries

Age \geq 24 months

One or more of the following:

- More than a brief loss of consciousness (measured in seconds)
- Significant amnesia
- More than one episode of vomiting
- Lethargy/decline in mental status

Lowest recorded in-hospital GCS score of 13–15

No focal neurologic deficits referable to head injury

No posttraumatic seizure

No spinal fluid otorrhea or rhinorrhea

No shock or other major organ system injury that would preclude discharge

No anticoagulant or chronic anti-inflammatory drug use or bleeding diathesis

No suspicion of child abuse

No prior cranial neurosurgical procedure

ment scheme for children older than 23 months; children not meeting the criteria specified in [Boxes 1, 2, and 3](#) should be admitted for a period of in-hospital observation. For infants younger than 24 months there may be additional difficulties eliciting specific complaints and obtaining a detailed neurologic examination, and there is a greater possibility of unrecognized abusive head injury in this group. Retrospective studies, however, have suggested that infants younger than 24 months of age can be similarly managed with surveillance CT scans [25,26,58].

Box 2. Radiographic parameters for emergency department management of children with minor head injuries

No intracranial abnormalities related to the head injury

Skull fractures allowed except

- Those that cross the middle meningeal artery (present on first three cuts of the middle fossa between the sphenoid wing and petrous ridge)
- Those that cross the dural venous sinuses (sagittal, transverse, sigmoid)
- Those that are depressed by more than the thickness of the adjacent skull

Box 3. Radiographic parameters for emergency department management of children with minor head injuries

No hemodynamic or respiratory changes
GCS score of 15 at time of discharge
No neurologic deterioration over preceding 2 hours
No focal deficits referable to head injury
Able to hold down liquids for at least the preceding 30 minutes
Reliable caretaker who is comfortable with discharge and understands discharge instructions

Medical management of moderate and severe head injuries

The initial treatment is directed toward maintaining ventilatory and hemodynamic stability and identifying the extent of the injuries [59]. Ideally, the initial assessment and resuscitative efforts proceed concurrently. As discussed later, endotracheal intubation is usually indicated for children with severe head injuries and coma. The cervical spine must be protected during intubation; a cervical collar and immobilization on a spine board are required, and cervical hyperextension must be avoided if possible. Orotracheal intubation is preferred, especially if there is strong suspicion of an anterior basilar skull fracture, to avoid further disrupting the anterior skull base or introducing bacteria or foreign bodies into the anterior cranial vault.

Hypotension is common following head injuries and usually suggests the presence of associated systemic injuries with blood loss or inadequate vascular volume. Rapid and aggressive treatment of hemodynamic instability is important to prevent secondary hypoxic-ischemic brain injury. Hypotension significantly increases the mortality from head injury [2,59–61]. Hypotension of neurogenic origin is rare but may occur in the presence of severe brainstem or cervical spinal cord injuries. Neurogenic hypertension, often with associated bradycardia and hypoventilation or apnea (Cushing's triad), is more common and suggests elevated intracranial pressure.

Hypothermia is more common in young children because of their small body mass and relatively large surface area and may be exacerbated by the presence of hypothalamic dysfunction or cervical spinal cord injury. The child should be kept in a thermally neutral environment and core temperature maintained with heat lamps or warming blankets [2].

During the initial resuscitation, the cervical spine should be adequately immobilized with a spine board and a cervical orthosis or sandbags. An urgent cross-table lateral cervical spine radiograph should visualize all seven cervical vertebrae and at least the top of the first thoracic vertebra. Anterior-posterior radiographs and, for children 8 years or older, an open-mouth odontoid view supplement the lateral radiograph and complete the series. Flexion and extension radiographs, CT, and MRI can complement the plain radiographs, depend-

ing upon the circumstances. Unstable cervical spine fractures should be immobilized immediately to preserve cervical spinal alignment and avoid further spinal cord injury.

As part of a thorough general examination, external evidence of head and spine injury should be sought. Scalp contusions or lacerations, depressions of the skull, or facial injuries raise suspicion for underlying brain injury. Periorbital or retroauricular bruising or swelling, hemotympanum, and clear rhinorrhea or otorrhea suggest a basilar skull fracture. In the infant, a bulging or tense anterior fontanelle suggests elevated intracranial pressure. Crepitations, swelling, or spinal deformity may suggest an associated spinal cord injury. Fundoscopic examination can give valuable information about the severity and mechanism of head injury and provide evidence of suspected abusive head trauma. Work-up or treatment should not be delayed by attempting to visualize the fundi in an obviously severely injured child, because papilledema usually takes 12 to 24 hours to develop after severe head injury [62], and fundoscopic evaluation rarely yields information that will affect acute management.

A rapid but detailed neurologic examination should be performed. A complete discussion of neurologic functions is beyond the scope of this article, which will touch on only the most critical points. The most important information is the level of consciousness and whether (and in what direction) it is changing. Transient impairment of consciousness (or concussion) is common following minor head injuries, whereas more prolonged alterations in level of consciousness follow severe head injuries. Both the duration of unconsciousness and the level of arousal at the time of evaluation may predict the severity of the head injury [59]. Intracranial pressure (ICP) monitoring is generally reserved for patients with a GCS score below 8. An even more important consideration is how the GCS score is changing with time. A worsening level of consciousness suggests rising ICP or an expanding intracranial mass lesion, whereas a lethargic but improving child may have a postconcussive syndrome. The GCS score should be documented at regular intervals.

In addition to the GCS score, focal neurologic deficits may suggest localized brain injury or a focal mass lesion; the pattern and temporal profile of neurologic deficits may localize the injury or suggest a cerebral herniation syndrome. For example, a unilaterally fixed dilated pupil with an associated contralateral hemiparesis or motor posturing suggests transtentorial herniation. Therefore, the secondary survey should include a brief and focused examination of certain neurologic functions.

Pupil size represents a balance between sympathetic and parasympathetic influences, and dysfunction of either system results in unopposed action of the other [63]. The size, shape, and the reactivity to light are important clinical parameters of pupillary function. An enlarged, nonreactive pupil (mydriasis) suggests an abnormality of the oculomotor nucleus in the midbrain, third nerve injury, or direct orbital trauma. Unilateral mydriasis in trauma most often suggests transtentorial (or uncal) herniation. Mydriasis (usually bilateral) may also accompany a seizure or postictal state. Finally, bilateral mydriasis can be caused

by pharmacologic agents such as atropine or sympathomimetic agents used during resuscitation.

A small pupil (miosis) suggests an injury to the sympathetic innervation of the pupil and may be caused by a common carotid artery injury or arterial dissection in the neck or skull base; associated often subtle ipsilateral ptosis and anhydrosis (Horner's triad) may be present as well [63]. The superficial temporal artery pulse may be diminished or absent on the affected side. These children should undergo magnetic resonance angiography (MRA), CT angiography, or a formal angiogram if necessary to exclude a vascular injury, because they are at risk for delayed stroke.

Abnormalities of ocular movements can reflect injury to the cranial nerves, brainstem, or cerebral hemispheres. For example, a destructive lesion of one frontal lobe may produce a conjugate tonic eye deviation toward the side of the injury or an incomplete gaze paresis toward the contralateral side, whereas an irritative focus such as a seizure may drive the eyes toward the contralateral side. Unilateral occipital lobe injury may result in a hemianopia and conjugate gaze preference toward the ipsilateral side [64]. Injury to brainstem gaze centers may produce a variety of extraocular movement abnormalities that can localize intracranial injury. For example, ipsilateral conjugate lateral gaze palsy suggests dysfunction of the parapontine reticular formation located within the pons [63,64].

The evaluation of brainstem responses can be valuable in localizing neurologic injuries. A unilateral facial palsy or hearing loss suggests facial or auditory nerve injury from a basilar skull fracture. The corneal reflex tests trigeminal function and provides an indication of pontine function. Vestibular function can be tested with the doll's eye maneuver (but only after cervical spine injury has been excluded) or cold-water caloric responses (only after a rupture of the tympanic membrane has been excluded). In a comatose patient, ice water instilled into one external ear canal induces an ipsilateral eye deviation, whereas in a stuporous or obtunded patient, nystagmus may develop with the rapid component directed contralateral to the side of instillation (known by the eponym COWS—cold opposite, warm same side—to describe the direction of the nystagmus) [63]. Both doll's eyes and cold caloric tests assess the function of the brainstem between the pontine vestibular nuclei and the oculomotor nuclei in the midbrain. Finally, the gag and cough reflexes test the integrity of the glossopharyngeal and vagus nerves and medullary swallowing centers.

Disordered breathing may localize brainstem dysfunction. Periodic (Cheyne-Stokes) breathing suggests bilateral hemispheric or diencephalic injury but may reflect injury as caudal as the upper pons [64]. Apneustic breathing (having a prolonged inspiratory plateau) suggests mid- to caudal pontine injury, whereas ataxic breathing (with irregular stuttering respirations) suggests injury to the medullary respiratory generator centers [64].

ICP monitoring and treatment is the cornerstone of acute brain injury management. ICP is generally measured with a fiberoptic sensor inserted into the brain parenchyma or subdural space or with a catheter inserted directly into the lateral ventricle (Fig. 1). A ventricular catheter has the advantage of being able

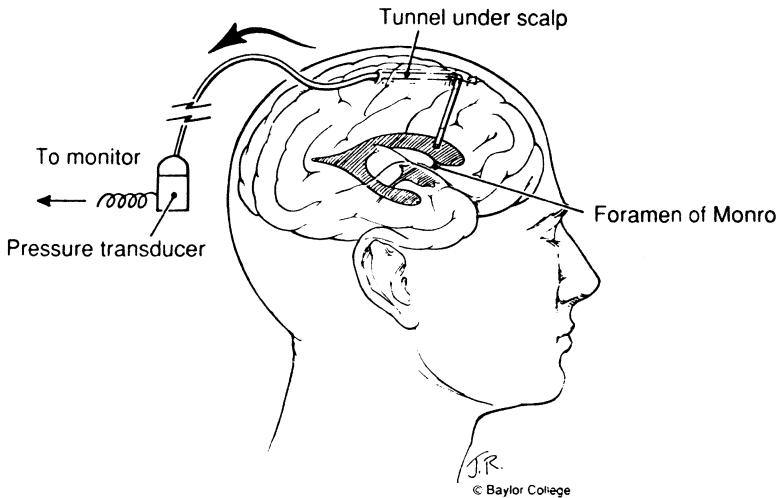


Fig. 1. Insertion of intracranial pressure (ICP) monitor into the lateral ventricle. (From Kanter MJ, Narayan RK. Intracranial pressure monitoring. *Neurosurg Clin N Am* 1991;2:257; with permission.)

both to monitor and to treat ICP elevations (by allowing cerebrospinal fluid [CSF] to be withdrawn) but is more difficult to insert and has a higher infection rate than parenchymal fiberoptic monitors. A complete discussion of ICP management is beyond the scope of this article. Normal ICP is usually less than 10 mm Hg for adults [65] but varies from 3 to 7 mm Hg in younger children and from 1.5 to 6 mm Hg in infants [66–68]. Most studies of adult head trauma have rather arbitrarily used a threshold of 20 mm Hg to initiate measures to control ICP following head trauma. Because ICP is normally lower in younger children and infants, one might reasonably treat an ICP of 15 mm Hg in a young child, or even an ICP of 10 mm Hg in an infant, although outcome studies for this approach have not yet been undertaken. Cerebral perfusion pressure (CPP) can also be calculated as the difference between the mean systemic arterial pressure and the mean ICP. In adults, a CPP of greater than 50 to 60 mm Hg is sought following trauma, although a CPP of 70 mm Hg or more may be necessary in some cases [69]. Both the ICP and mean blood pressure are demonstrably lower in children than adults; however, the CPP necessary to perfuse the infant and developing brain adequately is unknown [70]. One scheme for the management of brain injury seeks to optimize cerebral blood flow by reducing ICP or by maintaining or even artificially increasing mean arterial pressure (thereby raising CPP).

ICP management has undergone several important changes over the past decade; management guidelines for both adult and pediatric head injuries have been published [71,72]. Three particularly critical management changes have been made. First, a large number of well-designed studies have failed to demonstrate any benefit of steroids in head injury [73–78], and steroids currently have no place in the management of traumatic brain injury. Second, hypovolemia

is no longer induced to reduce cerebral edema. Instead, the present goal is to maintain euvolemia or even mild hypervolemia using isotonic or even hypertonic saline solutions at maintenance rates or greater. Third, prolonged hyperventilation is no longer routinely employed (although brief periods of hyperventilation are still indicated in the setting of acute herniation); severe hypocapnia, by causing vasoconstriction of cerebral vessels, can cause secondary cerebral ischemia and further exacerbate brain injury [79]. The goal of therapy is therefore to maintain eucapnia or mild hypocapnia (PaCO_2 of 30–35 torr or greater).

Management of intracranial hypertension therefore begins with intubation and adequate oxygenation, eucapnia or mild hypocapnia, adequate sedation and pain control with continuous infusions of short-acting agents such as fentanyl and midazolam, and occasionally paralytic agents. Transient rises in ICP caused by intubation, endotracheal suctioning, or other endotracheal manipulations may be minimized by prior intravenous or endobronchial lidocaine [80–82]. Occult seizures are a potential cause of unexplained elevations in ICP [83], and prophylactic anticonvulsants should be considered throughout the period of ICP monitoring. Fevers raise ICP several torr for each degree centigrade [83] and should therefore be treated. Recent investigative trials using hypothermia as an adjunct to lower ICP have given inconsistent but encouraging results; however, its use in children is still considered investigational [84]. Finally, adequate bladder drainage, nonrestrictive cervical collars, head elevation to 30°, and reducing environmental stimulation may provide additional nonspecific measures to reduce ICP.

Specific measures to reduce ICP are employed if nonspecific measures fail. If a ventricular monitor is in place, transient rises in ICP are first treated with CSF drainage [83]. If CSF drainage is not available or fails, an osmotic diuretic such as mannitol is employed. Mannitol may work by several mechanisms including promoting bulk flow of extracellular edema fluid into the vascular system. In addition, mannitol significantly reduces blood viscosity and improves rheology (and therefore cerebral blood flow) [85]. The resulting constriction of cerebral vessels through cerebral autoregulation decreases cerebral blood volume and reduces ICP [86]. A dose of 0.25 to 2 g/kg is used, although 0.25 g/kg may be as effective as larger doses in reducing ICP [87]. Recurrent doses are generally reserved for further documented elevations in ICP. Barbiturates may be used if the other measures fail [83] but entail significant risks including hypotension, anergy, pneumonia, sepsis, and hyponatremia [88]. Adequate volume expansion with close monitoring of central venous or capillary wedge pressures is necessary, and inotropic support with dopamine or dobutamine together with levaterenol or phenylephrine may be required. Finally, intriguing recent studies suggest that continuous lumbar drainage may, under certain circumstances, provide an additional adjunct to control ICP despite empiric concerns about inducing cerebral herniation [89]. This method should be employed only with extreme caution and in consultation with (or under the direct care of) a neurosurgeon, and only after specific pre-existing conditions are met including (1) open basilar CSF cisterns demonstrated on CT scan, (2) no significant intracranial mass or midline

shift, (3) a pre-existing and functional intraventricular drain in place, and (4) ICP that is refractory to conventional treatments including ventricular drainage and mannitol.

Overview of specific types of pediatric head injuries

Subgaleal hematomas are collections of blood in the loose areolar tissue beneath the galea aponeurotica and above the periosteum. In contrast, cephalohematomas lie between the periosteum and the skull. Cephalohematomas are usually restricted from crossing suture lines by dense connections between the periosteum and the suture, whereas subgaleal hematomas are not so bound and may be more diffuse. Underlying skull fractures are more commonly associated with cephalohematomas [90]. Neither subgaleal nor cephalohematomas require acute surgical treatment. Needle aspiration is neither necessary nor desirable because of the risk of introducing bacteria. Large subgaleal hematomas in infants may produce anemia and even hypotension from blood loss [14,91] and require serial assessments of blood pressure and hematocrit. Rarely, cephalohematomas may later calcify and produce a disfiguring skull mass that requires surgical correction.

Skull fractures are frequent in pediatric head trauma. The importance of skull fractures as predictors of underlying brain injury is a matter of debate. Some studies suggest an increased risk of intracranial injury in the presence of a linear skull fracture [34,92], whereas others suggest that associated intracranial injuries are rare, particularly in the presence of a normal neurologic examination [93–95]. Skull fractures may be open (associated with an overlying scalp injury) or closed and may be linear, comminuted (having multiple branches), diastatic (having bone edges that are split apart), depressed, or basilar. Simple linear fractures comprise three quarters of skull fractures in children [96]; the mid-parietal bone is most frequently involved [96]. Most diastatic fractures require no further treatment. Diastatic fractures in infants less than 3 years old, however, are sometimes associated with the later formation of a so-called “growing skull fracture” or leptomeningeal cyst [97–100]. In these cases, the displaced fracture fragments are associated with a tear in the underlying dura, and there is often a laceration of the underlying brain producing an area of encephalomalacia on the initial CT scan [101,102]. Continued pulsations of brain and cerebrospinal fluid enlarge the fracture over time and may be associated with seizures or focal neurologic deficits. An expanding scalp mass becomes apparent within weeks of the injury and is composed of CSF or extruded brain. Serial radiographs or CT scans demonstrate progressive widening of the fracture and surrounding bony sclerosis over weeks or months [103]. Surgical repair is necessary [97–99,101,102].

Depressed skull fractures are often the result of a focal blow (eg, from a baseball bat, golf club, or hammer). Those that are open represent an opportunity for intracranial infection and should be evaluated by a neurosurgeon. The indications for surgery for simple closed fractures are primarily cosmetic, because

neither the frequency of posttraumatic epilepsy nor the incidence of focal neurologic deficits is significantly affected by surgical repair [104]. Skull fragments that are depressed less than the thickness of the skull generally do not require elevation. More complex fractures, particularly those associated with an obvious underlying dural laceration and brain injury, should be elevated to prevent further injury. Open depressed skull fractures or those that involve the intracranial wall of the frontal sinus may be elevated together with débridement and repair of the scalp laceration or exenteration of the frontal sinus to minimize the risk of infection. Depressed skull fractures that overlie a dural venous sinus are particularly treacherous because of associated thrombosis or bleeding from the sinus; surgical repair, if necessary, should be approached with extreme caution [104,105].

Basilar skull fractures complicate 5% of pediatric head injuries [106]. The anterior skull base in the region of the cribriform plate and the petrous bone in the posterior fossa are the most common sites. Petrous fractures may be associated with sensorineural hearing loss and facial nerve palsy [107,108] or, more rarely, with other cranial neuropathies. Basilar skull fractures may be associated with bloody or CSF rhinorrhea, hemotympanum, or bloody or CSF otorrhea. Battle's sign (mastoid ecchymosis) or raccoon's eyes (periorbital ecchymoses) may be clues to the diagnosis. Focal neurologic deficits may include anosmia from traumatic olfactory nerve injury with cribriform fractures and hearing loss, tinnitus, vertigo, and facial paralysis from petrous bone fractures [104,109]. Hearing loss may be caused by (1) blood in the middle ear, (2) rupture of the tympanic membrane, (3) disruption of the ossicular chain, (4) shearing of the cochlear hair cell stereocilia, or (5) stretching, compression contusion, or disruption of the auditory nerve.

Thin-section CT scans through the skull base using both axial and coronal orientations and bone algorithms detect many basilar skull fractures, although some are not visible even with sophisticated imaging. Fluid within the mastoid air cells or paranasal sinuses or intracranial air may be present. Glucose levels in the draining fluid may be misleading, because glucose is also present in normal mucosal drainage [110]. Measurement of chloride in CSF usually is elevated in comparison with serum chloride values [96]. The presence of beta-2 transferrin in the fluid is more specific for CSF [111], but it generally takes several days to obtain results.

Patients are initially managed expectantly. The ear should not be packed in the presence of CSF otorrhea, because packing impedes CSF drainage and may increase the risk of infection. The role of prophylactic antibiotics is controversial, and their use may promote the growth of resistant organisms [112]. Fortunately, CSF rhinorrhea stops spontaneously in nearly all cases, and CSF otorrhea stops in 85% of cases [113]. Secondary meningitis occurs in 4% of patients with CSF otorrhea and in 17% of patients with CSF rhinorrhea [114]. Common organisms include nasal and sinus flora such as *Streptococcus pneumoniae* (which accounts for 80% of cases) and *Haemophilus influenzae*. Broad-spectrum antibiotics such as vancomycin and a third-generation cephalosporin are used initially and are subsequently tailored to the results of CSF cultures and sensitivities. Recalcitrant

or persistent leaks may be successfully treated with CSF diversion through serial lumbar punctures, continuous lumbar drainage, or (more rarely) external ventricular drainage. Direct surgical repair is generally reserved for CSF leaks that have persisted beyond 1 to 2 weeks despite CSF diversion and those that have caused repeated episodes of meningitis.

Epidural hematomas complicate 2% to 3% of all head injury admissions in children [16,17,115–119] and are more frequent with advancing age; the peak incidence is in the second decade of life [118,120–123]. Bleeding may occur from a lacerated meningeal vessel, a dural venous sinus, or slow venous oozing from a diploic vein within a skull fracture. Associated skull fractures are seen in 60% to 75% of older children [124] but are less frequent in infancy because of the deformability of the young skull [116]. Skull fractures that cross the middle meningeal vessels in the temporal fossa or the dural venous sinuses (such as the superior sagittal sinus, transverse sinuses, or the torcular) are more frequently associated with epidural hematoma [125,126]. Childhood epidural hematomas are most often the result of a fall [16] and commonly occur in the setting of a relatively minor head injury. Changes in the level of consciousness are absent even at the moment of impact in up to 50% of children with epidural hematomas in such cases the hematoma is discovered on a screening CT scan performed because of the mechanism of injury or an associated subgaleal or cephalohematoma [17]. The classic lucid interval is present in only 33% of patients [127]. Persistent or progressive headaches, increasing confusion, lethargy or agitation, or focal neurologic deficits should suggest the presence of an epidural hematoma. Acute epidural hematomas appear as hyperdense, biconvex, or lentiform masses located immediately beneath the skull on CT scans (Fig. 2).

Epidural hematomas that are large or cause significant mass effect, those in the temporal or posterior fossae, and those that cause focal neurologic deficits or alteration in level of consciousness should be removed urgently by craniotomy. Certain epidural hematomas can be treated nonoperatively [128–136]. To be considered for nonoperative management, the hematoma should produce minimal or no symptoms, be located outside of the temporal or posterior fossae, be less than 40 cm³ in volume, should not be associated with intradural lesions, and should be discovered at least 6 or more hours after the injury. Asymptomatic hematomas that are discovered in a delayed fashion are particularly amenable to nonoperative management. Nonoperative management requires an intensive care setting with a neurosurgeon immediately available. The overall outcome for childhood epidural hematomas is generally good, with mortality of 0% to 17% [16,17,115,117,118,137]. The clinical presentation is the single greatest predictor of outcome; mortality is nearly zero for those who are awake and alert but is much higher for those presenting in coma.

Acute subdural hematomas are common in infants and decrease with advancing age into adolescence [5,138]. Subdural hemorrhage is usually the result of a high-velocity impact injury such as an automobile accident, assault, child abuse, or fall from considerable height [139,140]. Associated and usually extensive primary brain injuries, including contusions or diffuse axonal injuries (DAI), are



Fig. 2. CT scan showing an acute epidural hematoma (arrows).

often present [141,142]. Most subdural hemorrhages arise from tears in one or more cortical bridging veins as they traverse the subdural space to the dural venous sinuses. Hemorrhage overlying the cerebral convexities are more common in older children and adults, whereas hemorrhages within the posterior falx cerebri and overlying the tentorium cerebelli are more common in infants and toddlers [116]. The presence of these parafalcine and tentorial subdural hemorrhages in infants without a history of significant trauma are particularly suspicious for child abuse.

The clinical presentation of subdural hematoma depends on the size and location of the hemorrhage and the presence of associated brain injuries [143]. As many as 50% of patients are rendered immediately unconscious because of the associated injuries [11,144]. Focal neurologic deficits are common and are often attributed to the associated injuries; hemiparesis is seen in up to 50% of patients [118,144–146], and pupillary abnormalities are seen in 28% to 78% [144,146,147]. Seizures occur in 6% to 22% [145–147]. Acute subdural hematomas are high-density collections on CT scans, overlying and conforming to the convex surface of the brain (Fig. 3) in contrast to the lentiform shape of epidural hematomas. Large subdural hematomas that are producing mass effect commensurate with their size and are associated with significant change in consciousness or focal neurologic deficits would probably need to be removed. In contrast, small



Fig. 3. CT scan showing a convexity subdural hematoma.

hematomas with significant associated brain injuries and mass effect out of proportion to the size of the hematoma might be better managed nonoperatively. The outcome for subdural hematoma is worse than for epidural hematoma [11] with mortality ranging from 42% to 90% [116]; subdural hematoma associated with child abuse carries a 20% mortality and a 50% neurologic morbidity [148–150]. The neurologic status at presentation is the most important predictor of outcome [116]. Timing of surgery also significantly influences outcome, with lower mortality for patients operated on within 4 hours of the injury [142]. Associated injuries such as high ICP and DAI are associated with higher mortality and worse outcome [142,144,151,152].

Intraparenchymal hemorrhages can be focal contusions, DAIs, or intracerebral hematomas (Fig. 4). Small petechial hemorrhagic contusions are usually superficial in the cortex and subjacent white matter of the anterior temporal and inferior frontal lobes and are caused by the gliding of these vulnerable areas against the lateral sphenoid bone and the floor of the anterior fossa, respectively [124], or by a focal blow. Hemorrhages in the deep white matter, basal ganglia, or thalamus are more often caused by sudden and often violent angular acceleration and appear as punctate hyperdensities on CT as DAI [124]. Finally, deep brainstem hemorrhages are caused either by angular accelerations or by stretching

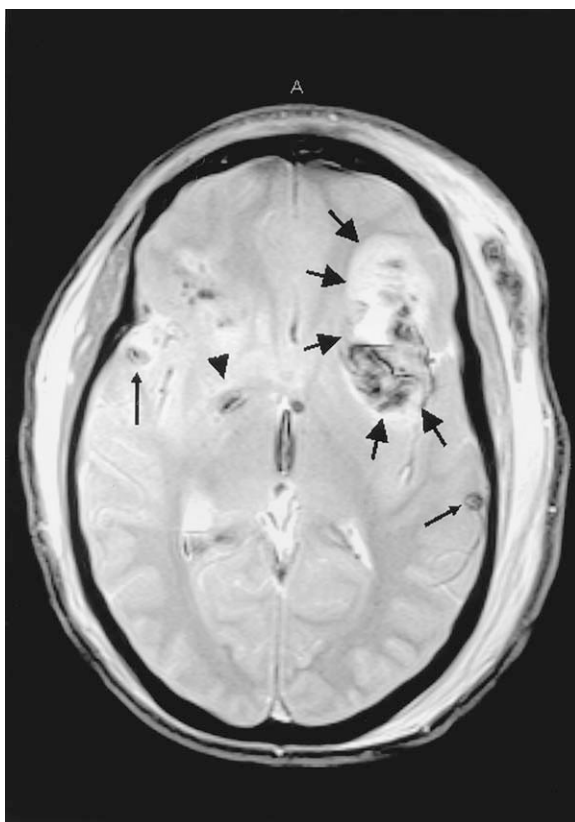


Fig. 4. MRI showing multiple intraparenchymal brain hemorrhages including cortical contusions (*smaller arrows*), deeper white matter shear injuries (*arrowheads*), and an intracerebral hematoma (*larger arrows*) that resulted from the coalescence of adjacent smaller contusions.

and tearing of perforating vessels during transtentorial herniation. Large intracerebral hematomas are rarely encountered in pediatric head trauma [115,153] and usually are caused by coalescence of smaller hemorrhagic contusions into a larger intracerebral hematoma [154,155]; one must be vigilant, especially in patients with contusions who deteriorate clinically or who have an unexplained rise in ICP. Routine follow-up scans within 12 to 24 hours of admission are advisable in all patients with significant contusions.

Distinctive sequelae of childhood head injury

Postconcussive syndromes of childhood

Delayed lethargy, irritability, and behavioral changes frequently follow minor head injury in children and may raise concerns about developing intracranial

hypertension or intracranial mass lesions. The history is usually stereotypical [15]. Following a relatively minor injury, with or without a transient loss of consciousness, the child recovers temporarily but then within 10 to 30 minutes becomes progressively more irritable, disoriented, and listless; lethargy or even stupor may develop. Pallor and diaphoresis are common; vomiting occurs in 95% [15]. Symptoms may persist or progress over the next 1 to 2 hours and may mimic an expanding intracranial mass lesion. Gradually and spontaneously, complete recovery occurs over the next 2 to 12 hours. The mechanism is unknown; spreading waves of depression, epilepsy, transient alterations in blood flow, and migraines have all been implicated [14,15,156,157]. Close observation is warranted, and if a CT scan excludes edema or a mass lesion, no further treatment is generally required. Rarely, intubation and institution of measures to control ICP are required if the GCS score falls below 8.

Postconcussive vomiting is particularly common, following 50% of childhood head injuries [158]. The head injury is rarely severe—children with minor head injuries vomit frequently and repeatedly, whereas severely injured children rarely vomit [14]—and there usually is no visible intracranial abnormality on CT scan. Management is expectant, and full recovery is universal; intravenous fluids may be necessary to prevent dehydration if the episode is prolonged.

Trauma-triggered (or posttraumatic) migraine is a fascinating sequela of some pediatric head injuries [159]. The clinical history is characteristic. The child usually has suffered a minor head injury, often without a loss of consciousness, and initially behaves appropriately. Thereafter, the child becomes progressively more combative and agitated, often with violent or aggressive behavior, verbal abuse, and cursing (much to the chagrin of the parents). This behavior gradually abates, and the child sleeps, awakening the following morning with little or no recollection of the event. Although the child may complain of headache, this often is not a major feature of the syndrome. Many of these children have either a personal or a family history of migraine; sometimes a previous minor head injury has produced a similar response. Intracranial injury is rarely demonstrated on CT scans.

Transient cortical blindness is another rare but bizarre feature of childhood head trauma and may represent another manifestation of trauma-triggered migraine. An initially minor head injury is followed by transient, usually complete, blindness with spared pupillary responses. Malingering or hysterical behavior may be suspected. Again, CT scans are usually normal and complete recovery, usually within 24 hours, is the rule. Other possible mechanisms include vascular traction or transient ischemia from vasospasm unrelated to migraine [160–162].

Posttraumatic seizures

Posttraumatic seizures complicate 10% of pediatric head injuries [163]. Most follow relatively minor head injury and occur either on impact or within 24 hours of the injury (defined as immediate posttraumatic seizures) [164]. Sixty percent to 90% of immediate posttraumatic seizures occur within 24 hours, and approxi-

mately 50% occur within 1 to 3 hours [15,165–167]. Most are short-lived, generalized seizures that are not associated with any radiologically visible intracranial injury [18,92,163,168]. Immediate seizures do not predict later epilepsy; most do not require any treatment, and the neurologic outcome in these patients is quite good [18,168]. A recent study of 71 children with immediate posttraumatic seizures and no visible intracranial injury on CT scans found that 60 (85%) had a single or brief uncomplicated posttraumatic seizures; all made an uneventful recovery, and none of these children had a recurrent seizure once they had been admitted to the hospital nor any complications following discharge. Eleven children (15%) had prolonged seizures, status epilepticus, or apnea and required ICU admission; however, in all 11 the complex nature of their seizures was apparent before or immediately after their arrival in the emergency department. These results prompted the author and colleagues to consider emergency department observation for this group of children if initial CT scans are normal, although the number of patients in this study was too small to draw firm conclusions because the statistical risk of a bad outcome was as high as 9% using 95% confidence limits [18].

In contrast, early seizures (occurring 1–7 days after injury) and late seizures (occurring > 1 week after injury) are more frequently associated with visible intracranial injury including brain swelling, subdural hematomas, depressed skull fractures, and penetrating intracranial injuries, and with lower GCS scores. The risk of long-term epilepsy is higher in these groups [164,169], and prolonged anticonvulsant treatment (6–12 months) is generally recommended.

Seizure prophylaxis following severe traumatic brain injury has undergone considerable study. Previous uncontrolled studies have produced conflicting results. A randomized, prospective study in adults with traumatic brain injury demonstrated a beneficial effect of phenytoin only during the first week after closed head injury [170], and the group receiving longer-term phenytoin prophylaxis had more significant cognitive deficits during follow-up [171]. Although children have not yet been similarly examined, the results of this study suggest that prophylactic anticonvulsants be used only during the first week after head injury. The author currently uses prophylactic anticonvulsants (usually phenytoin) in children with significant head injuries and intracranial abnormalities for 7 days (or until the period of intracranial hypertension and ICP monitoring has passed) and then discontinues its use.

Child abuse

Child abuse is unfortunately a common cause of head injury in the infant and young child less than 2 years of age [168,172,173]. The syndrome of intracranial injury with DAIs, subdural or subarachnoid hemorrhages, and seizures, together with retinal hemorrhages and certain types of long-bone fractures, was described as the shaken infant syndrome by Caffey [174] in 1972. This constellation of findings is almost never encountered following accidental trauma [175]. Although shaking has been implicated as the cause of the injuries, Duhaime and

colleagues [176] report a high incidence of skull fractures in infants with fatal injuries and have coined the term shaken impact syndrome to emphasize the role of impact in many cases. The neuropathologic findings variably include skull fractures, cerebral contusions, subdural hemorrhage from tearing of bridging veins, DAIs involving the central white matter and corpus callosum, extensive cytotoxic and vasogenic edema, and ischemic-hypoxic changes in neurons and glia [176–178] and reflect a combination of linear or rotational acceleration injuries as well as ischemic/hypoxic injury. Finally, injuries involving the lower brainstem, cervicomedullary junction, and upper cervical spinal cord in many infants may interfere with respiration and exacerbate ischemic-anoxic injury [177,179,180].

In most cases, the child is left alone with a single individual, most often a parent (75% of perpetrators) or more specifically a father, stepfather, or mother's boyfriend (60% of perpetrators). Babysitters account for an additional 7% of perpetrators [181]. The stories offered by perpetrators are often remarkably similar from case to case. Some describe a fall from a low height such as a sofa, bed, table, or chair; this history is inconsistent with the observed injuries in that falls from such heights are rarely associated with significant intracranial injuries [182,183], and the pattern of injuries is different. Many deny any history of trauma [175], instead describing spontaneous seizure activity, lethargy, or apnea with no apparent cause. Often the perpetrator admits to gentle patting or shaking in an attempt to awaken or revive the infant and suggests that the injuries result from these resuscitative efforts.

On examination, the infant is often irritable, lethargic, or even comatose; seizures are common and may be frequent and difficult to treat. The anterior fontanelle is full and bulging; sometimes the head is enlarged, and the sutures are split [148], suggesting a chronic or repetitive injury. Focal neurologic deficits are less common. Retinal hemorrhages are present in up to 90% of abused children [168,176,184] and provide important corroborating evidence of abuse. Although retinal hemorrhages may be seen in up to 45% of newborn infants following birth [185], these usually are small, and only 3% persist beyond the fifth day of life [186]; essentially all are gone by 4 weeks following birth [185]. In contrast, retinal hemorrhages caused by child abuse are often massive, multilayered, and extend to the periphery of the retina (the ora serrata); retinal detachment, intraretinal hemorrhage with retinoschisis, and vitreous hemorrhage are present [184,185,187]. For legal purposes, a careful ophthalmoscopic examination by an experienced ophthalmologist should be performed in all cases of suspected abuse.

Radiologic studies reveal a variety of intracranial abnormalities, some of which are strongly suggestive of abuse. Although skull fractures are seen in many abused infants, they need not be present to make the diagnosis. Subarachnoid or subdural hemorrhage is common particularly in the occipito-parietal region along the falx and tentorium (Fig. 5). Chronic or mixed-density subdural collections may suggest a longer history of repeated abuse [188], although a more recent study of vehicular crashes in infants has suggested that a single episode of trauma can produce subdural hemorrhages of varying densities on CT scans [189].



Fig. 5. CT scan showing a subdural hemorrhage along the posterior and anterior falx and tentorium (*black arrowheads*), a small convexity subdural hematoma (*white arrowheads*), and bilateral occipitotemporal hypodensities in a child with abusive head trauma.

Patchy areas of hypodensity on CT scans, often involving the occipital lobes, suggest acute ischemic-hypoxic injury. Small petechial hemorrhages in the deep white matter, basal ganglia, and corpus callosum are sometimes present. MRI scans usually reveal more extensive injuries than do CT scans. Corpus callosum shear injuries are particularly well seen on sagittal MRI scans. Acute scans may be relatively unrevealing, but delayed scans (weeks or months following the abuse) often show severe atrophy of the cortex and underlying cerebral white matter with chronic subdural fluid collections.

In addition to an ophthalmologic examination, a skeletal survey or bone scan is required in suspected abusive head injury and may demonstrate occult long-bone or rib fractures of varying ages [173,190]. Skeletal surveys and bone scans may provide complementary information, with fractures being revealed on one but not the other [191]. A repeat skeletal survey 10 to 14 days later may also identify fractures that were not apparent on the first study. Finally, coagulation studies (prothrombin time, partial thromboplastin time, international normalized

ratio, fibrinogen, and fibrin split products) should be obtained to exclude a coagulopathy, because this defense can sometimes be used in court. The most common coagulation abnormality is disseminated intravascular coagulopathy secondary to released tissue thromboplastins from the severe brain injury and should not be confused with a primary coagulation disorder.

The medical management of abused infants involves the same intensive care monitoring as for all severe head injured children. Early seizures are particularly problematic and sometimes require multiple anticonvulsants. Significant subdural hematomas can be managed initially by intermittently tapping the collection with a needle through the anterior fontanelle [192,193] or with temporary external subdural drains. Continued enlargement of chronic subdural collections may require subdural-peritoneal shunts. Bilateral collections may require double-armed shunts, although many (even those that do not communicate radiographically) are successfully managed with a unilateral shunt [194,195]. Posttraumatic hydrocephalus in 4% of abused infants requires a ventricular shunt [196]. The outcome for infants with abusive head injuries is poor; mortality rates of 10% to 35% are reported [150,176,197], and half of the survivors have significant neurologic residua including seizures, focal neurologic abnormalities, developmental and motor delays or spasticity, and permanent visual loss or blindness caused by brain or retinal injuries.

One of the more challenging issues in abusive head injuries is determining the presence and certainty of an abusive injury, as well as the timing of the injuries and therefore the most likely perpetrator. This topic is beyond the scope of this article, and the interested reader is referred to other sources for reviews of additional information on various controversial aspects of abusive head trauma [185,198–201].

Pediatric spine trauma

A review of pediatric spine trauma is well beyond the scope of this article, and only a few critical principles are mentioned here. Spinal cord injuries in children are uncommon, accounting for only 0.3% to 10% of all spinal cord injuries [202]. Bony fractures and dislocations of the pediatric spine are even less common. Several unique features of the pediatric spine render it susceptible to distinct patterns of injury. The evaluation of the spine in children should be undertaken with knowledge of the developmental anatomy, the unique anatomic changes of the pediatric spine during development, and the various malformations that can masquerade as injury to the unwary.

The evaluation of the child with a suspected spinal cord injury begins with immediate spinal immobilization with sandbags, an external orthosis, and a spine board. Lateral cervical spine radiographs in the emergency department are completed as previously discussed and are supplemented by CT or MRI scans as required by the injury. The role of steroids in the management of spinal cord injuries is controversial, although randomized, prospective studies in adults (the

National Acute Spinal Cord Injury Study [NASCIS]-2 and NASCIS-3 trials) suggest that patients with motor weakness may benefit from high-dose methylprednisolone administered within 8 hours of injury (30 mg/kg load followed by a continuous infusion of 5.4 mg/kg/hour) [203,204]. The recommendations are to continue the infusion for 24 hours for patients beginning treatment within 3 to 8 hours after injury and for 48 hours for those beginning therapy greater than 8 hours [204]. These studies, however, have been criticized by many and are currently listed as options rather than as standards or guidelines in the evidence-based guidelines established for adult spinal cord injuries [205]. No such study has been undertaken nor have official recommendations been provided for children, although many centers employ the high-dose protocol for children with spinal cord injuries.

Although a thorough review of developmental spine anatomy is not possible in this article, a few points are relevant. The spine undergoes a number of anatomic changes throughout childhood and adolescence. Perhaps the most commonly misidentified radiographic peculiarity in children is pseudosubluxation (anterior translation of the second on the third vertebral bodies). Pseudosubluxation is a common normal finding on cervical spine radiographs in preadolescent children (Fig. 6) but occasionally is confused with a C2-3 dislocation (most commonly a hanged-man fracture). Pseudosubluxation is most readily identified by drawing a straight line between the posterior ring of C1 and the lamina of C3 on the lateral cervical radiograph; the lamina of the second cervical vertebra (the axis) should fall anterior to, on, or less than 5 mm posterior to this line. In contrast, a C2-3 dislocation will cause the lamina of C2 to fall more than 5 mm posterior to this line [206].

Lack of knowledge about the patterns of vertebral ossification, the locations of various ossification centers, and the timing of their fusion can lead to confusion. For example, failing to appreciate that the odontoid (dens) does not fuse with the body of the axis until 6 to 8 years of age can lead to the erroneous diagnosis of an odontoid fracture. Failing to appreciate that the vertebrae during childhood are normally wedge-shaped can lead to the erroneous diagnosis of vertebral compression fractures. Failing to identify properly the ring apophysis in the antero-superior or antero-inferior corners of the vertebrae on lateral radiographs can lead to the misdiagnosis of chip or teardrop fractures. These are but a few of the common challenges in interpreting pediatric spine radiographs.

The pattern of pediatric spinal injuries varies by age. Younger children up to age 8 years with relatively immature spines more frequently injure the atlas, axis, and upper cervical vertebrae whereas children older than 8 years with relatively mature spines more frequently develop adult fracture patterns. Perhaps most important is the recognition that children can sustain significant spinal cord injuries without visible bony fractures or dislocations. This spinal cord injury without radiographic abnormality (SCIWORA) accounts for 20% to 30% of all childhood spinal cord injuries [207]. Several features of the pediatric spine predispose children to SCIWORA: (1) increased ligamentous laxity in children; (2) a relatively large and heavy head compared with the neck; (3) poorly developed



Fig. 6. Lateral cervical spine radiograph of a 5-year-old child shows some of the typical features of the immature spine including C2-3 pseudosubluxation (*arrow*), wedge-shaped vertebral bodies, and the normal synchondrosis between the odontoid and body of C2, which have not yet fused.

cervical musculature; (4) anterior wedging of the vertebral bodies; (5) poorly developed uncinat processes; (6) a vulnerable growth zone at the vertebral endplate; and (7) shallow cervical facet joints. Clinical manifestations of SCIWORA can range from subjective sensory abnormalities, such as tingling dysesthesias or numbness, to frank weakness or paralysis, all without any visible radiographic abnormality. MRI may show edema or hemorrhage in the paraspinal ligaments or spinal cord; somatosensory evoked potentials may be abnormal as well. The management of SCIWORA is controversial, although a survey of pediatric neurosurgeons suggests that most will immobilize the cervical spine in an external orthosis (such as a Philadelphia or other rigid external collar) for several weeks and restrict children with SCIWORA from contact sports for an average of 4 to 7 months (Wong and Dias, unpublished data). Pang [207] has recommended up to 12 weeks of brace immobilization for children with SCIWORA. The outcome for children with mild injuries is good; for those with more severe injuries, incomplete recovery is the norm [207].

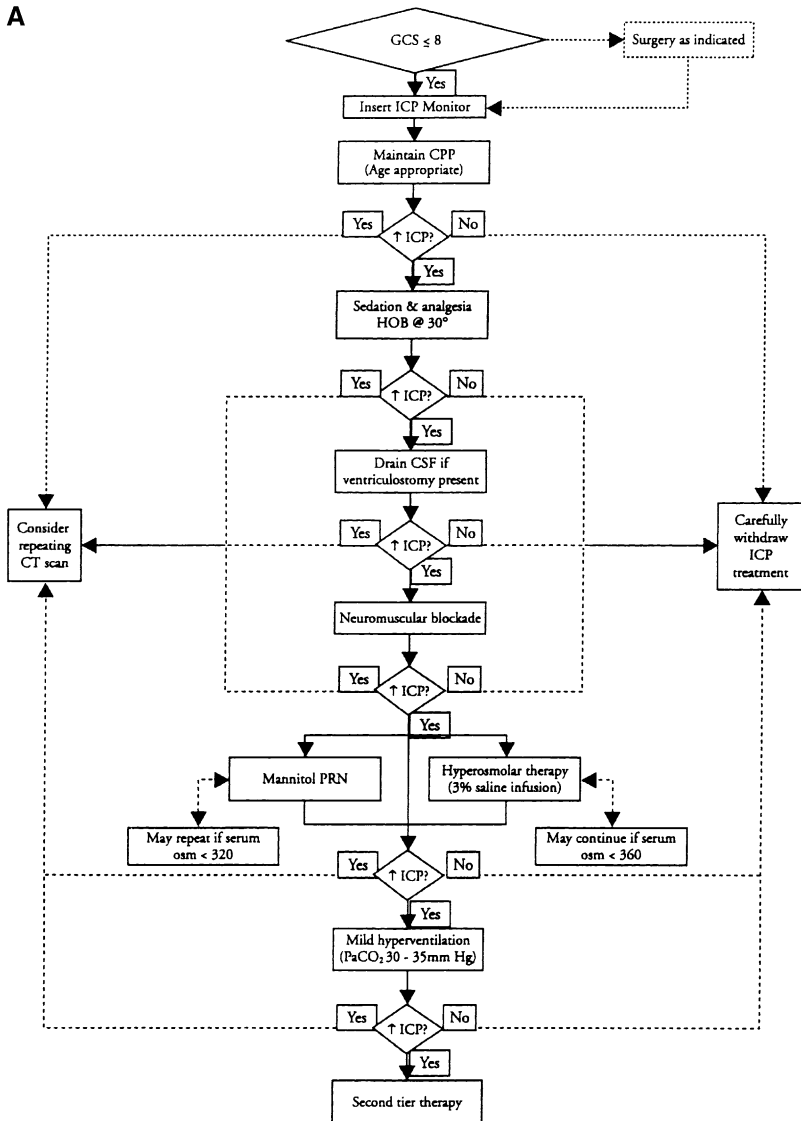


Fig. 7. Proposed management algorithm for the management of severe head injuries in children. (A) Primary therapies. (B) Second tier therapies. (From Adelson PD, Bratton SL, Carney NA, et al. Guidelines for the acute medical management of severe traumatic brain injury in infants, children, and adolescents. *Pediatr Crit Care Med* 2003;4(3 Suppl):S1–75; with permission.)

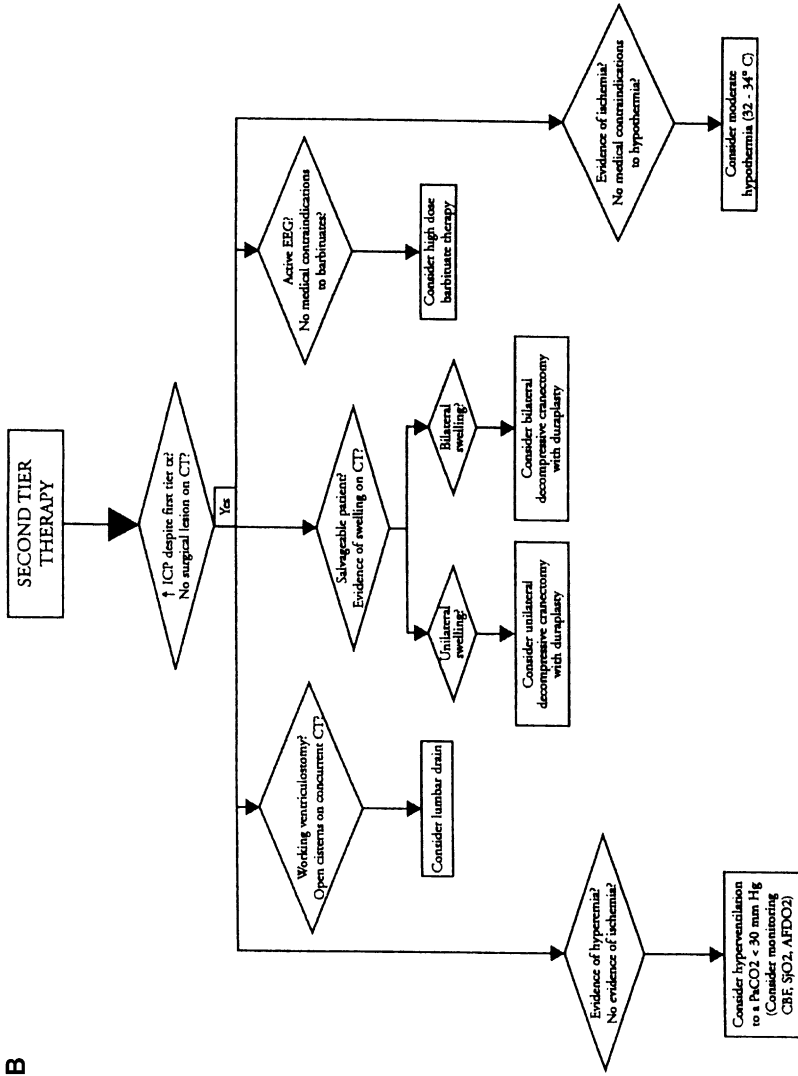


Fig. 7 (continued).

Last, the task of adequately clearing the child's spine is controversial. Some standards for clearing the cervical spine in adults have been established. For example, adults who are awake, alert, are not intoxicated, and have no other distracting injuries, neck pain or tenderness do not require any supplemental radiographic assessment to clear the cervical spine adequately [208]. These guidelines can reasonably be adopted for children as well, even among those who are nonverbal, if there is no evidence of any neck pain or tenderness. For children 9 years and older who have neck pain or other significant distracting injuries, a three-view (lateral, antero-posterior, and open-mouth odontoid) radiographic series is a standard of care. CT scans may be obtained through any suspicious or unevaluable areas. The available evidence suggests that for children younger than 9 years the odontoid view may safely be omitted if other views are normal. MRI within 48 hours of injury has been helpful in identifying ligamentous injuries in adults that were not readily apparent on other imaging studies [208]; the value of MRI has not yet been systematically studied in pediatric spine injuries.

The author and colleagues maintain cervical spine immobilization in children with significant head injuries or with significant neck pain or tenderness until an MRI can be performed to exclude ligamentous injury or until the child's pain subsequently resolves and flexion and extension radiographs reveal no apparent injury. Children with minor injuries and neck pain are discharged when medically stable and return for outpatient flexion and extension radiographs after the neck pain resolves. For obtunded or comatose patients who are not expected to regain consciousness adequately within a reasonable period of time, MRI or flexion and extension radiographs performed under fluoroscopy (and with the supervision of experienced personnel) are recommended to clear the cervical spine. No formal recommendations have yet been provided in children, however.

Summary

The management of pediatric head injuries has evolved over the past decade, and a number of significant advances have been made. Evidence-based guidelines and algorithms for the management of severe pediatric head injuries have recently been published (Fig. 7), and all pediatricians who care for children with severe head injuries should be familiar with these guidelines. It is hoped the guidelines will streamline the clinical management of these children and stimulate future research into the many areas that require further investigation.

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