Neurosurg Clin N Am 13 (2002) 183–199

NEUROSURGERY CLINICS OF NORTH AMERICA

Radiology of nonaccidental trauma

Edward Rustamzadeh, MD[a], Charles L. Truwit, MD[b], Cornelius H. Lam, MD[a,*]

[a]*Department of Neurosurgery, University of Minnesota, Mayo Building, Code 96, 420 Delaware Street, Minneapolis, MN 55455–0374, USA*
[b]*Department of Radiology, University of Minnesota, Mayo Building, Code 96, 420 Delaware Street, Minneapolis, MN 55455–0374, USA*

In 1946, Caffey [1] provided one of the first medical accounts of child abuse, with descriptions of subdural hematomas (SDH) and characteristic long bone fractures in six children. In 1962, Kempe et al [2] introduced the phrase *battered child syndrome*, and discussed the incidence, clinical manifestations, radiologic findings, and insights into the psychiatric profiles of the batterer and the abused victim. These seminal works stimulated other investigators to explore the depth of child abuse.

Awareness of child abuse among clinicians has risen from 10% in 1976 to over 90% by 1996. Child abuse transcends all racial, cultural, religious, socioeconomic, and gender boundaries, but the statistics on the prevalence and type of injuries are fraught with inherent problems. First, the incidence of abuse is underestimated because many cases are not brought to medical attention and the experience in dealing with abuse varies widely between urban and rural trauma centers. The second problem in documenting abuse is the inherent limitation of obtaining a history from a young child; therefore, only abuse with obvious physical manifestations is reported. Even with these limitations, head injury is the most common cause of death and disability in childhood and the third leading cause of death in children less than 1 year of age [3–5].

Approximately 10% of all inpatients with neurologic injury are abused children [6,7]. Several studies have shown that if one looks at only the infant group, 64% of all head injuries, excluding simple skull fractures, were caused by child abuse, and 95% of intracranial injuries were caused by child abuse [8–10]. The reported mortality with head trauma is 10% [11–13], which is five times the mortality from childhood leukemia, and 18 times that of childhood brain tumors [14].

The sequelae of child abuse are not limited to skin or bone injury but its effects are long ranging [4]. The behavioral, cognitive, and emotional effects of child abuse result in a long-lasting impact on the individual and society [15]. Duhaime and Sutton [4] estimated the annual national cost of hospital care for head-injured children to be greater than 1 billion dollars. There are numerous texts written about pediatric radiologic injuries. This article does not discuss all these aspects, but rather summarizes the types of injuries involved in child abuse for which a neurosurgeon might be consulted. Also discussed are common pitfalls in interpreting images of suspected abused children.

Scalp injuries

Scalp injury might be the first and only clue of an underlying head trauma. Other than lacerations, there are two types of scalp-associated hematomas. The most common scalp injury is a cephalohematoma (Fig. 1). It occurs under the periosteum and results from shear forces between the pericranium and the calvarium [16–18]. The two most common sites for this type of lesion are the parietal and the occipital convexities. Twenty-five percent of cephalohematomas are associated with skull fractures [19]. The temptation to aspirate this lesion should be resisted because of the

* Corresponding author.
E-mail address: lamxx023@tc.umn.edu (C.H. Lam).

PII: S1042-3680(01)00004-3

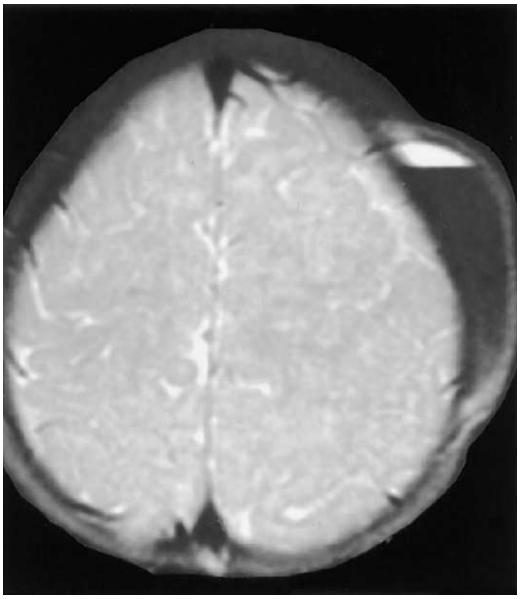

Fig. 1. Axial proton density image demonstrates cephalohematoma on the left, with fluid level typical of evolution involving blood products. The lesion seems to be contained within the suture confines.

possibility of infecting the hematoma. Over a period of several months these hematomas might result in remnant calcified subperiosteal lesions that can be resected at a later time for cosmetic reasons.

The other type of scalp injury is a subgaleal hematoma, also known as *caput succedaneum*. This results from the tearing of emissary veins that run from the diploe of the calvarium to the subgaleal tissue. Most subgaleal hematomas are results of birth injuries caused by either forceps injury or vaginal canal narrowing; however, forceful hair pulling may also be a cause [20]. The hematoma lies typically beneath the hair braid. These hematomas are less likely to be associated with an underlying skull fracture; however, in an infant they may result in significant blood loss. Unlike cephalohematomas, which are confined by the periosteal-fibrous union of the calvarial sutures and do not cross suture lines, subgaleal hematomas can expand widely and lead to hypovolemic shock in infants. In most cases conservative treatment with follow-up over several weeks is sufficient. On skull radiographs and CT the lesions appear as opacified oval masses above the outer calvarial layer. The two types of lesions can be differentiated by noting whether or not a suture line is crossed.

Skull fractures

Skull fractures are very common in children. Even from birth they can sustain one, such as from obstetric trauma, where the infant's head is compressed against the sacral promontory resulting in a depressed prominence leading to a ping-pong fracture [21]. More relevant to this discussion is that fractures can be the only evidence of child abuse (Fig. 2). Most abuse-related skull fractures in infancy are simple linear fractures; however, in older children depressed skull fractures are more prominent. When a skull fracture is detected emphasis should also be placed on the site of the fracture and the type of fracture. For instance, a linear fracture in and of itself might not carry the same importance as one that crosses a dural sinus, an air-filled sinus, a vascular groove, or the vestibular apparatus.

Skull fractures are divided into two categories: simple or complex. Within these two categories there are various subtypes. A simple fracture is defined as one that is 2 mm or less in width; limited to one bone; not crossing a suture line; and extending in a straight, jagged, or curved fashion. A complex fracture can have a branching pattern or a radial spiked pattern termed a *stellate fracture*. A high-velocity small surface injury results in a focal depressed stellate fracture. A high-velocity, broad-surface injury typically results in a central stellate fracture with an extending V-shaped peripheral fracture [22]. If the fracture is circumferential and isolated it is termed a *comminuted fracture*. If there is an overlying scalp laceration it is called a *compound fracture*. Wide complex fractures are termed *diastatic*. Complicated fractures are often caused by mid- or high-impact forces.

Many authors have noted the lack of association between fractures and underlying intracranial injuries, and based on clinical presentation defer to a head CT or MRI [9,23,24]. Skull radiographs can be helpful in documenting abuse and may demonstrate inconsistencies in the alleged mechanism of injury. Correct diagnosis of a skull fracture requires familiarity with the radiologic and developmental anatomy of the skull. It is imperative that the examining physician or radiologist be familiar with the various sutures, synchondroses, and fissures that may be present throughout the developmental stages. A common pitfall is in diagnosing these variants as a fracture. Fractures are radiolucent, sharply etched lines that may or may not branch but finally taper to indistinction. For example, a depressed skull fracture can be detected

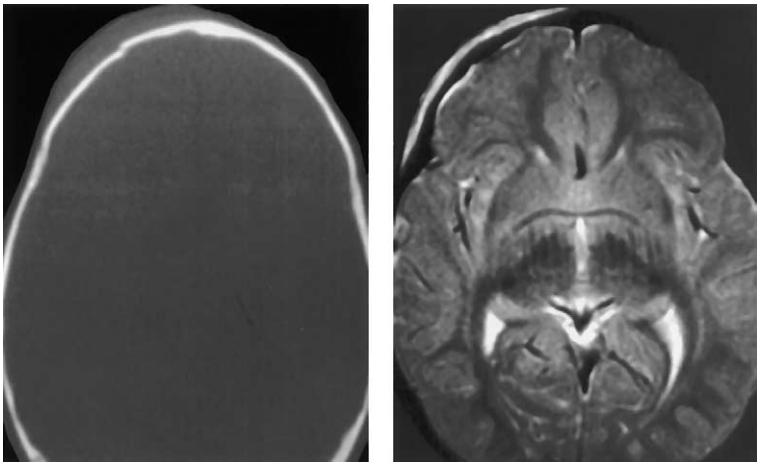

Fig. 2. Axial CT scan in a young child with apparently mild head bump demonstrates soft tissue swelling and subtly displaced skull fracture (*left*). Axial T2-weighted image demonstrates small amount of subdural hemorrhage not seen on the CT scan, and the soft tissue hyperintensity of the scalp (*right*).

by a sclerotic edge on one side of the fragment and a parallel disproportionate lucency on the other [22]. Sutures in turn can be differentiated by their serpiginous appearance, symmetry, sclerotic edges, and normal anatomic position.

In the adult it is easy to differentiate the coronal, sagittal, and lamdoid sutures. Interpretation of an infant's skull radiograph, however, can be complicated. A classic example is the misdiagnosis of the metopic suture for a frontal bone fracture. Furthermore, in a Towne's view this suture could be mistaken for an occipital fracture. One can differentiate this by noting whether or not the fracture runs through the foramen magnum. A true occipital fracture typically ends at the posterior lip of the foramen magnum.

Radiographic markings in the occipital bone can present a challenge in differentiating fissures and sutures from true fractures. Embryologically, the occipital bone arises from six separate ossification centers that are separated by four cartilaginous segments [25]. Examples of this include a transverse occipital suture, which typically lies at the transverse sinus level and results from nonfusion of the intraparietal and supraparietal squama of the occipital bone [26]. A mendosal suture results from the mid-fusion of the transverse occipital suture with the resultant lateral portions appearing as paired fractures. This variance may persist into adulthood as either bilateral fissure or unilateral fissure. A superior median fissure is a remnant of incomplete union of the superior intraparietal segments often extending dorsally past the union of the sagittal and lambdoid sutures. A midline occipital fracture can be differentiated from a superior median fissure by assessing whether the defect extends the entire length of the occiput [27]; only a fracture could account for this.

Numerous small sutures known as the *lateral interparietal sutures* may be seen along the occipital margin of the lambdoid sutures. The existence of these intraparietal sutures should be kept in mind before diagnosing the so-called *eggshell fracture* of the calvarium.

Intraparietal sutures are symmetric, whereas an eggshell fracture is not [22]. Similar confusion can occur along the lateral skull radiograph. The greater wing of the sphenoid connects with the frontal, zygomatic, parietal, and temporal bones. These sutures vary in size and depending on the size can form an X or an H configuration [28]. The squamous suture of the temporal bone may also be misinterpreted as a diastatic fracture if the film is rotated so that the bilateral sutures are superimposed [27].

Vascular markings on skull radiographs may also pose a diagnostic problem. These can be differentiated from fractures on the basis of radiolucency, because fractures involve the inner and outer calvarial table. Furthermore, vascular grooves are more linear and have a near constant anatomic branching pattern. Venous lake tributaries can be confused with stellate fractures, but can be differentiated by additional skull views.

In infants, fractures can result in two late sequelae not observed in other age groups. The first

involves a fracture that crosses a suture line. This can result in a tear in the arachnoid layer that is fixed to the suture, resulting in a subepicranial cerebrospinal fluid (CSF) hygroma [29], which usually resolves over time. The second is a growing skull fracture known as a *leptomeningeal cyst*, which results from a combination of an enlarging skull and an underlying contused brain that swells and herniates through the dural tear further separating the fracture margins (Fig. 3). On tangential skull radiographs, a leptomeningeal cyst appears as a widened skull defect with scalloped or everted sclerotic margins. On CT it appears as a porencephaly, ventricular diverticula, or hypodensity of brain [25,30]. Clinically these infants have progressive neurologic deficits or seizures. Surgical intervention consists of separating the brain from the dural scar and repairing the dural defect with a graft and bone closure with a split-thickness bone graft [31]. Although skull fractures are not absolute indicators of child abuse, findings of multiple linear skull fractures that are bilateral and cross suture lines have been shown to be more characteristic of child abuse than accidental trauma [32].

Epidural hematomas

Although epidural hematomas (EDHs) are more common in older children, they still account for 2% of intracranial hematomas in infants [30]. Unlike a SDH, an EDH has a higher likelihood of being associated with a skull fracture. With

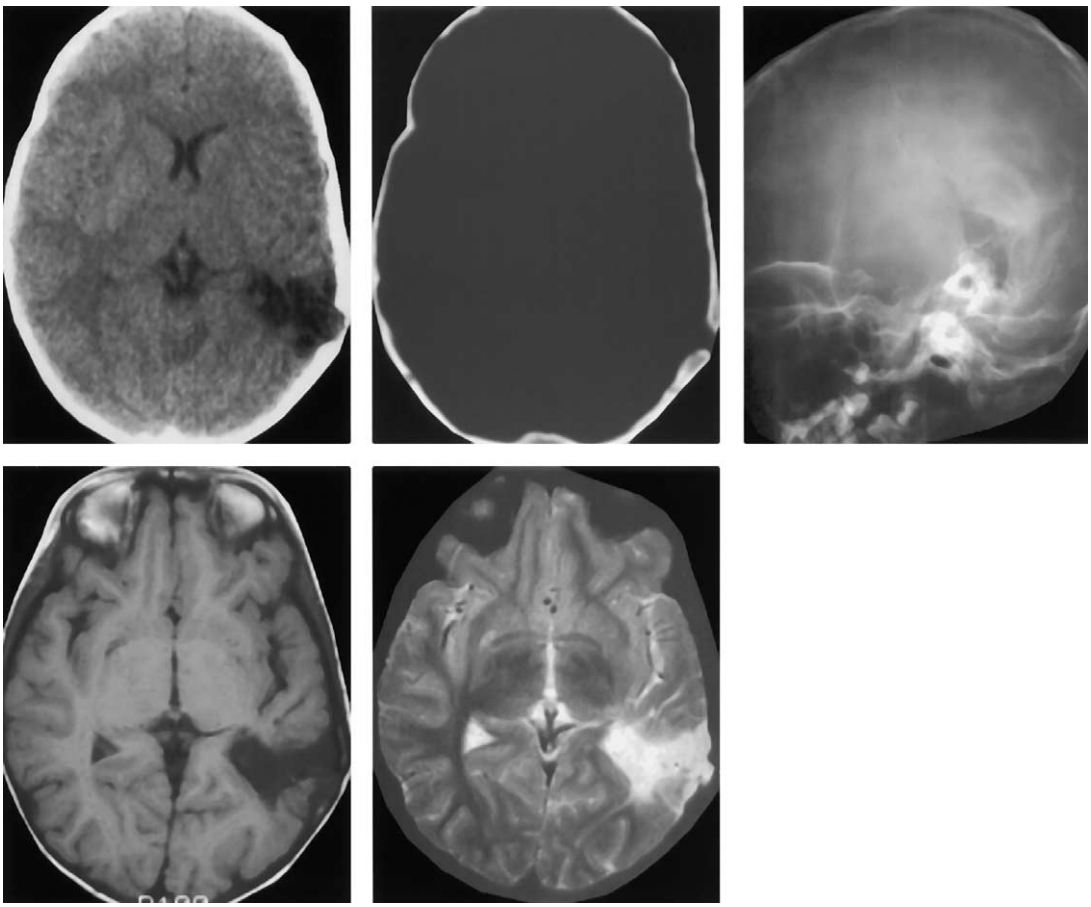

Fig. 3. Axial CT scan (*upper left*) demonstrates encephalomalagic area of the left posterior temporal region with obvious lytic defect of the bone best seen on the bone images (*upper middle*). Eburnated margins of the bone of the lytic lesion suggest that this is a chronic lesion. Lateral skull view demonstrates an oblique lesion, typical of the growing fracture (*upper right*). Axial T1- (*lower left*) and T2- (*lower right*) weighted images demonstrate the changes seen on the CT scan and the extensive gliosis in this case. Porencephalic involvement of the ventricular system is noted.

any fracture that crosses over the middle meningeal groove, clinical suspicion for an underlying EDH should increase. The source of the bleeding in 85% of cases is the posterior branch of the middle meningeal artery [23], with the remainder caused by either the middle meningeal vein or dural sinus tear.

Most EDHs are a result of obstetric trauma. The infant commonly presents with an edematous scalp, full fontanel, decreased level of consciousness, and neurologic deficit. If the EDH is associated with a diastatic fracture the possibility of hypovolemic shock can occur secondary to loss of a tamponade effect of the skull. In older children, the textbook presentation of brief loss of consciousness followed by a lucid interval then obtundation, contralateral hemiparesis, and ipsilateral pupillary dilatation occurs in less than 10% to 27% of cases [33].

In the pre-CT era, epidural bleeding could be diagnosed on angiographic basis by noting the medial displacement of the ipsilateral middle cerebral artery vasculature. On CT scans, EDHs have a characteristic biconvex, high-density appearance. This configuration is a result of the dural attachment to the inner calvarial table. Small EDHs, however, can be virtually impossible to differentiate from a SDH [34]. Furthermore, a lucent zone within the hematoma may represent an area of active bleeding [35].

An MRI can also be helpful in detecting epidural blood. On an MRI scan the dura can be detected as a thin, low-signal line between the hematoma and the brain further assisting in differentiating an EDH from a SDH. Furthermore the calvarial artifact that is seen on CT is not present on MRI, making detection of an EDH easier. The down side to using an MRI for this purpose is in the dating of the blood. Because MRI is a relatively recent technology, the appearance of blood on an MRI as it progresses through its stages from oxyhemoglobin to deoxyhemoglobin to methemoglobin is still controversial. The appearance of intracranial hemorrhage is based on empirical clinical observations, theoretical explanations, animal models, and in vitro studies of human blood clot composition and evolution [36]. In general though, blood on T1-weighted images progresses from a dark appearance in the hyperacute phase, to an isodense appearance in the acute phase, to a bright appearance in the subacute phase, to a heterogenous appearance in the chronic phase. Similarly, on T2-weighted images the blood appears bright in the hyperacute phase and progresses to a dark appearance in the acute phase until the late subacute phase, where it appears bright again, and finally becomes heterogenous in the chronic phase. In terms of surgical intervention, clinically significant EDHs should be evacuated emergently by a craniotomy.

Subdural hematomas

Acute subdural bleeding is more common in the neonate than in any other age group of children secondary to obstetric trauma [37]. In fact, SDHs have been reported to outnumber EDHs by as much as 10:1 [38]. SDHs are second only to congenital hydrocephalus as the primary cause for macrocephaly [39]. The posterior fossa is the most common location for SDHs in this age group. The hematoma results from a tear in the posterior fossa veins or more severely, a laceration of the tentorium with extension to the straight sinus, vein of Galen, or transverse sinus [40]. Another location common for SDHs is the convexity; the mechanism of injury involves tearing of the small bridging veins that empty into the sagittal sinus (Fig. 4). Nonevacuated hematomas become surrounded by endothelial cells with granular tissue invasion along the dural side, which in turn results in membrane encapsulation in 2 to 3 weeks. The hematoma then liquefies and takes on a serosanguinous appearance. Previously it was assumed that the breakdown in the red blood cells caused an increase in the osmolarity of the solution, causing an influx of fluid. There is evidence, however, that the increase in size of the lesion might be caused by neovascular permeability [41–43].

The typical appearance of a subdural bleed on CT is a crescentic lesion along the convexities. In infants bilateral SDHs are present 80% to 90% of the time [44]. Acute blood typically appears as a hyperintense lesion on CT, subacute blood appears as isointense to the brain, and chronic blood appears hypointense. Because of increased protein concentration in the SDH, the intensity is slightly more intense than CSF. Traditionally, the timing of SDHs has been classified as acute (less than 72 hours); subacute (1 to 4 weeks); or chronic (greater than 4 weeks) (Fig. 5). These parameters are based on the evolution of radiographic changes of SDH and intraparenchymal hemorrhages in adults and animals.

Dias et al [45] retrospectively reviewed 117 serial CT and MRI scans in 33 infants with shaken baby syndrome. Their data showed that the evolution of acute to chronic subdural fluid collection can occur

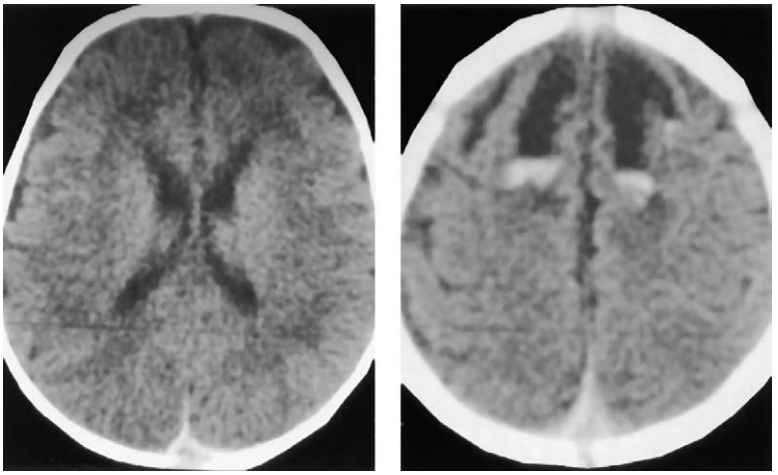

Fig. 4. Axial CT scans of young victim of nonaccidental trauma demonstrates subtle subdural hematoma posteriorly, and along the inner atmospheric fissure posteriorly (*left*). More cephalic images demonstrate the hemorrhagic contusions with fluid levels again consequent to the trauma (*right*).

as early as 20 hours and certainly by 3 to 12 days after the insult. Furthermore, they report that hypodensities can appear as early as 3.5 hours after the insult as compared with the traditional view of 6 to 48 hours.

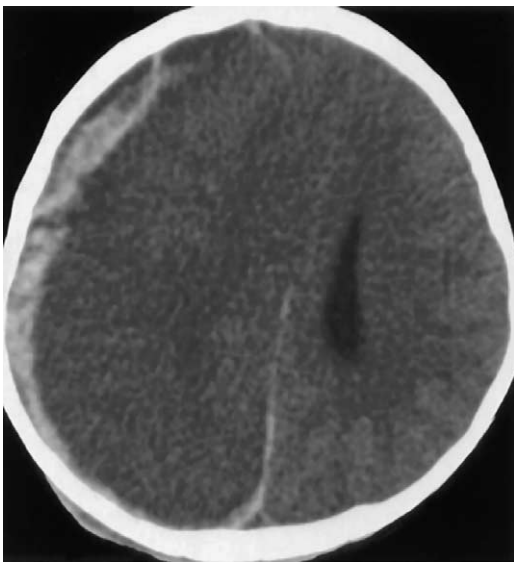

Fig. 5. Axial CT image of a 4-year-old girl demonstrating an acute subdural hematoma on the right with mass effect obliterating the right lateral ventricle and midline shift to the left. Note the less dense crescentic portion of the hematoma more superficially along the frontal regions with the higher density clotted subdural hematoma.

MRI can also be used to detect SDHs but the same controversy in dating the lesion as discussed previously exists. An important role of MRI lies in its ability to differentiate a chronic SDH from enlargement of the subarachnoid space caused by either benign extra-axial fluid collection of infancy or atrophy. Superficial veins draining the cortical convexity sulci penetrate the overlying arachnoid layer and then the inner dural layer to reach the superior sagittal sinus. These venous branches lie at a distance from the sagittal sinus. McCluney et al [46] coined the term *cortical vein sign* and proposed that enlargement of the subarachnoid space because of a fluid collection results in stretching or tethering of the cortical veins on MRI as compared with enlargement of the subdural space, which displaces the arachnoid membrane and the traversing cortical veins onto the cortical surface and results in a fluid collection void of vasculature structure. Furthermore, a chronic SDH results in visualization of the cortical veins only along the cortical margin emptying directly into the sagittal sinus.

Chen et al [47] used color Doppler ultrasound in a series of 18 consecutive infants with pericerebral fluid collection presentation to differentiate subarachnoid versus subdural space enlargement on the presence of cortical veins on Doppler ultrasound. All the patients with cortical vein detection on ultrasound had confirmation of enlarged subarachnoid space on MRI.

The presence or absence of cortical veins in infants with a pericerebral fluid collection is crucial in

differentiating a chronic SDH because of previous nonaccidental trauma versus benign enlargement of the subarachnoid space caused by extra-axial fluid collection of infancy (external hydrocephalus). In the latter case there is usually a family history of macrocephally, and more importantly the anterior interhemispheric fissure, the sylvian fissure, third ventricle, and the chiasmatic cisterns are larger than normal, whereas the posterior interhemispheric fissure and the occipital and posterior fossa CSF spaces are within normal limits [48]. Because of the tethered cortical veins in the infants with benign external hydrocephalus, a small percentage present with increased intracranial pressure caused by SDH. Because of the tenuous nature of these veins minor trauma, such as coughing or sneezing, can result in rupture of these veins. On MRI one typically sees a two-compartment fluid collection: a SDH overlying a dilated subarachnoid space [49].

Duhaime et al [50] coin the term the *disappearing subdural* in discussing a group of infants who presented with acute SDHs that disappeared within 24 to 48 hours. Distinguishing between the subarachnoid and the subdural compartment on an initial CT scan of an infant with an extra-axial fluid collection can be difficult at times. In this series of four patients all were diagnosed with an accidental inflicted SDH. Because the hemorrhage disappeared within 24 to 48 hours, however, the authors believe that hemorrhage might have been in the subarachnoid space with the CSF washing out the hemorrhage. The authors provide several characteristics that might help localize the fluid collection to the appropriate compartment. The first is that the density of the clot is less than that seen in a surgically treated SDH, and it has an inhomogeneous appearance to it as a result of CSF dilution. Second, blood may be seen in the sulci and fissures of the corresponding hemisphere. Third, the underlying brain does not seem to have mass effect.

Subarachnoid hemorrhage

Trauma is the most common cause of subarachnoid hemorrhage (SAH). Most traumatic SAHs are associated with other injuries including SDHs, intracerebral hematomas (ICHs), skull fractures, and cerebral contusions [51]. In general a CT scan more readily detects a SAH than does an MRI. This is caused by the rapid dilution and clearance of the SAH by CSF. Two specific sequences, intermediate echo and FLAIR, have been shown to be the most sensitive in detecting SAHs [52,53]. Most SAHs are supratentorial and an infratentorial SAH should raise the suspicion for atlantoaxial dissociation or other craniocervical injury [54].

The challenge lies not in identifying large SAHs, but in differentiating small SAHs from SDHs especially along the interhemispheric fissure. Three helpful clues in differentiating the two lesions are (1) SAH does not widen the interhemispheric fissure as does a SDH; (2) the falx does not extend to the rostrum of the corpus callosum, and any hyperintense extension is an indication for a SAH [55]; and (3) the hyperdensity of SAHs has an undulating margin, which conforms to the gyral surface of the brain [56].

Traumatic SAHs do not require surgical intervention; however, surgical intervention might be needed for other associated injuries and for possible late sequelae of hydrocephalus.

Intracranial hemorrhage

There are two basic types of forces involved in head trauma. The first is a translational force that involves an injury to the underlying brain directly at the site of the impact, with or without an overlying skull fracture. The mechanism involves a rapid acceleration and deceleration of the brain parenchyma. The second type of force is a rotational force that results in a shearing effect on the axons. This type of injury is discussed in depth in the next section. ICHs have a low incidence in neonates because of a lack of counter-coup injury [14]. Multiple sites of ICH in the neonate might be caused by an underlying coagulopathy. Cerebral injury can be documented in more than half of the child abuse cases [44]. Such injuries have the most significant and devastating sequelae of child abuse.

The most common sites of an ICH are the deep white matter of the frontal and the temporal lobes (Fig. 6). The hematoma results from either direct injury to a parenchymal blood vessel or as a result of extensive bleeding into a contusion site [35]. The typical CT finding is a hyperdense area with hypointense perilesional edema. Traumatic ICH can be differentiated from an underlying vascular malformation on the basis of the contour. The latter type has a more uniform margin [35]. Such factors as hemophilia, abnormal platelet function, defects in fibrin deposition, hepatic disease, excessive fibrinolytic activity, and anemia can result in an isodense appearance of the ICH [49,57–59]. In fact, the appearance of a fluid-fluid level within a

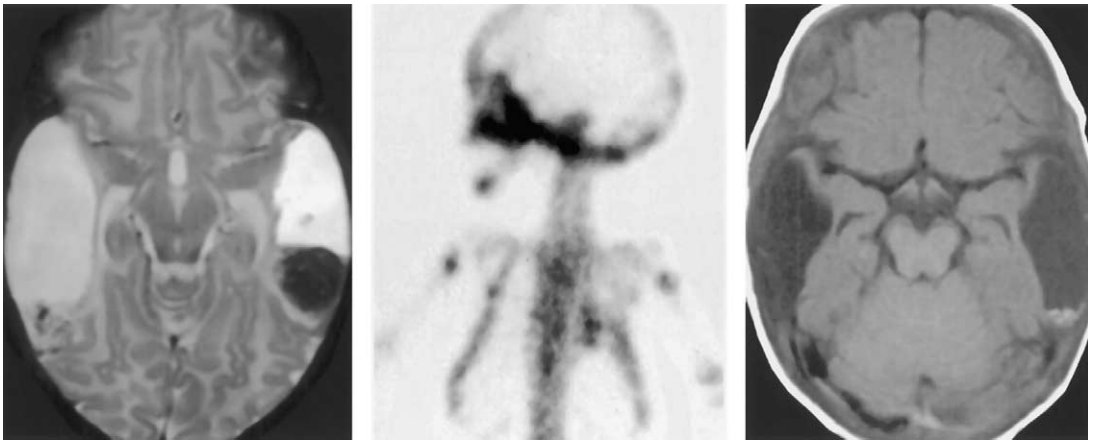

Fig. 6. Axial T2-weighted image of young victim of nonaccidental trauma demonstrating unusual bitemporal hematomas (*left*). Mass effect has resolved by the time of the MRI. Note the heterogeneity of the cystic hemorrhagic collections. Nuclear medicine study demonstrates increase uptake of posterior ribs, consistent with rib fractures (*middle*). Subsequent T1-weighted image from subsequent MRI demonstrates the malacic changes of temporal lobes seen earlier on the T2-weighted images (*right*).

clot represents an underlying coagulopathy in 50% of patients [28].

With the breakdown of the red blood cells the lesion takes on an isodense appearance that lasts for several weeks. During this time neovascularization occurs around the lesion. On contrasted CT, this appears as a ring-enhancing lesion [28]. A telltale sign of repeated head trauma is the so-called *target sign*. This appears as a hyperdense lesion within the subacute ring-enhancing lesion described previously.

The residue of ICH includes encephalomalacia (37%), slit-like lesions (25%), calcification (10%), and no identifiable abnormality in 27% of cases [60]. Associated edema either unilateral or bilateral is a frequent finding in an abused child with intracranial hemorrhage. The edema is a result of hyperemia and vasogenic edema. The edema becomes maximal at 3 to 6 days [61]. The mass effect on CT manifests as obliteration or compression of the ventricles, the sylvian fissure, and subarachnoid cisterns around the brainstem [56]. The mass effect does not resolve until resorption of the hematoma takes place.

Cerebral edema may also result from anoxic injury as a result of asphyxia, smothering, or strangulation. A useful key in differentiating the cause of the edema can be obtained by the findings on CT. In anoxic-induced edema, the gray matter of the cerebral cortex has a decreased density as compared with that of the thalamus, brainstem, and cerebellum (Fig. 7). This reversal of normal brain appearance is known as *Cohen's reversal sign* [62]. The composition, structure, sequential degradation, field strength of the magnet, and pulse sequence all affect the signal intensity of an ICH on a MRI scan. A CT scan is far more reliable in detecting ICH, although an MRI is far more sensitive to white matter changes including the extent of edema.

Surgical intervention for ICH in neonates and infants is technically difficult because of the soft and friable consistency of the yet incompletely myelinated white matter. Conservative treatment should be used to treat the underlying increased intracranial pressure.

Big black brain

Infants that survive the 26% acute mortality rate [63] associated with nonaccidental inflicted trauma may go on to develop severe parenchymal loss. Duhaime et al [64] followed 21 consecutive child-abuse infants with severe head trauma radiographically, and noted the development of unilateral or bilateral supratentorial hemispheric infarctions within 48 hours of admission. They termed these radiographic findings *big black brain* (Fig. 8). The radiographic findings consisted of loss of gray-white differentiation with uniform hemispheric hypodensity. These infarctions could

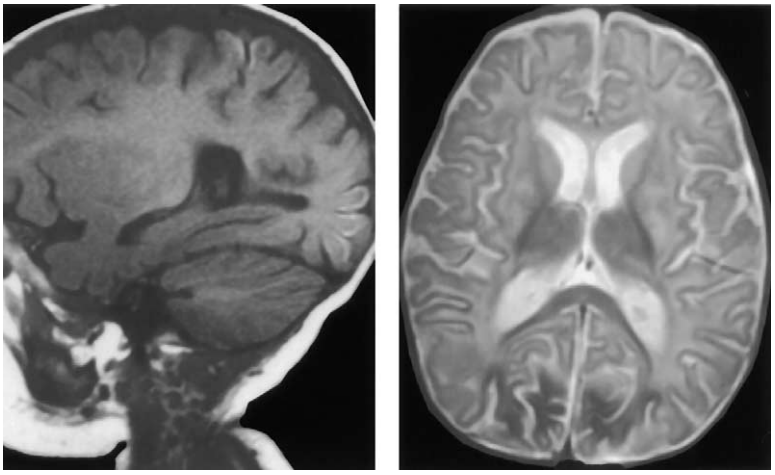

Fig. 7. Sagittal T1-weighted image of an asphyxiated child demonstrating hyperintense laminar necrosis of occipital cortex and portion of the posterior frontal lobe (*left*). Axial T2-weighted image demonstrates hyperintense basal ganglia, and the T2 shortening (hypointense) occipital region caused by the laminar necrosis (*right*).

not be explained solely on hypoxia because the infarcts were not symmetric, or on vascular occlusion because angiographically the vessels were patent. All the patients had extra-axial blood ranging from surgically significant SDH to thin fluid collections detectable only on MRI. They postulate that a combination of local pressure caused by the SDH or the release of vasoactive or excitotoxic factors may exacerbate the initial mechanical damage inflicted on the infant. Similar

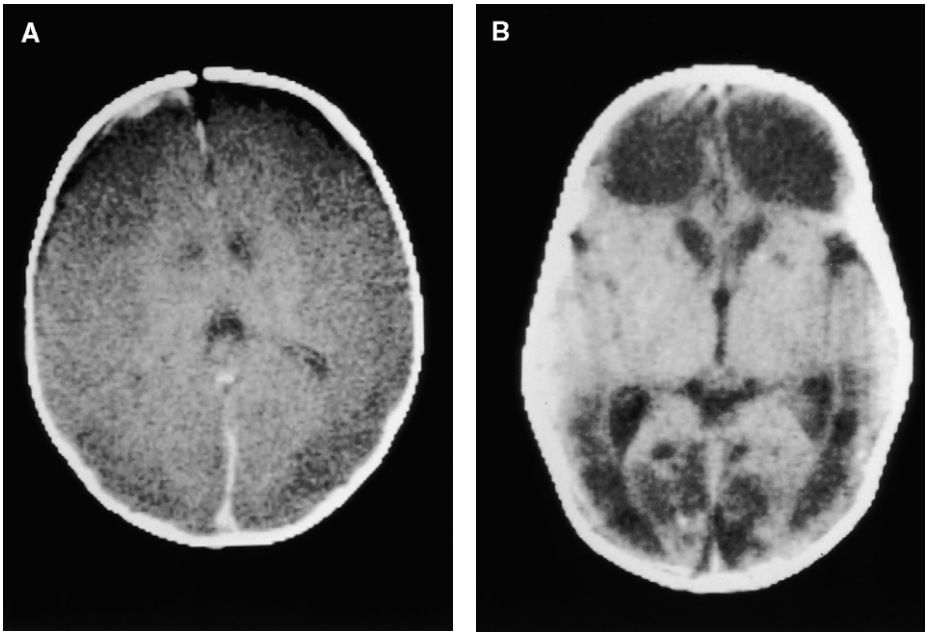

Fig. 8. (A) Axial CT image of an infant with inflicted shaking injury showing a small subdural hematoma and extensive loss of normal cortical architecture (also referred to as *big black brain*). (B) Same patient at follow-up showing diffuse encephalomalacia and cystic changes, which are common sequelae to early diffuse changes and are associated with uniformly poor outcome in survivors. (*From* the American Academy of Pediatrics; with permission.)

findings have been seen in experimental models of SDH [65].

Diffuse axonal injury

The types of injuries discussed so far have resulted from direct impact to the skull and its underlying content; however, many times the child can present with severe neurologic deficits without any evidence of direct head trauma. Transmission of rotational forces as a result of impact or nonimpact (whiplash) injuries can lead to sudden acceleration then deceleration of the brain causing a shearing and tearing of the brain parenchyma, known as *diffuse axonal injury* (DAI) (Fig. 9). The most common cause of DAI is shaken baby syndrome. The presentation often involves severe neurologic deficits, seizures, or sudden infant death [66–68]. The functional outcome of shaken baby syndrome is poor with up to 30% resulting in death, 30% to 50% left with severe cognitive or neurologic deficits, and only 30% having a full and normal recovery [31]. Differences in the mobility of various parts of the brain are thought to be the underlying cause of shearing effect seen on radiologic and pathologic findings [69].

The effects of rotational forces on the parenchyma were studied by Holbourn [70] by using a gelatinous model. The areas that were found to be most affected were the parasagittal, fronto-orbito-temporal surfaces, corpus callosum, and the dorsolateral aspect of the upper brainstem. Zimmerman et al [71] attributed this to the differential density of the white and gray matter. Several factors put infants and toddlers at a higher risk for DAI. First, the incomplete myelination in infants predisposes to shearing injury as compared with contusion in older children. Second, the relative large size of the head to the torso and the laxity of the ligaments and underdeveloped neck muscle strength results in poor resistance to any positive force. Finally, the thin and pliant skull bones allow more effective transmission of the force to the brain [72].

The initial CT scan can miss 50% to 80% of DAI cases [73,74]. Acute DAI on a CT scan is diagnosed based on the presence of small petechial hemorrhages in the gray-white matter junction and in the corpus callosum [75]. MRI is far more sensitive to changes in the white matter. The appearance of DAI on MRI depends on the presence or absence of hemorrhage and the duration of the lesion. In nonhemorrhagic shearing lesions T1-weighted images are often unremarkable. T2-weighted images often have a multifocal hyperintense foci at the gray-white matter junction and in the corpus callosum. The hyperintensity of the lesions diminishes over time. Hemorrhagic shearing lesions are seen as petechial hemorrhages on T1-weighted imaging and as diminished signal on T2-weighted imaging and gradient echo scans. In

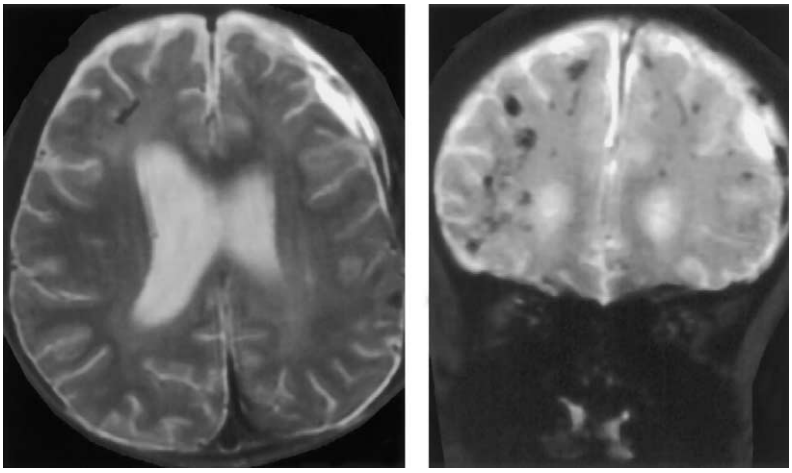

Fig. 9. Axial T2-weighted imaging demonstrates small foci of sheer hemorrhage in the right and left frontal lobe and left temporal parietal region (*left*). Coronal gradient echo image, however, is clearly superior for the detection of sheer injury compared with conventional imaging. Note the numerous sheer injuries through the frontal lobes not readily evident on the T2-weighted images (*right*).

some cases nonspecific atrophic changes might be the only abnormality on imaging [76].

Medical intervention is limited to close monitoring and management of any signs of increasing intracranial pressure.

Pediatric spine

Spinal fractures are uncommon in children, but if detected provide for strong evidence of child abuse. The prevalence of spinal trauma in child abuse ranges from 0% to 3% [77]. The sequelae of spine injury, however, can be devastating. Approximately 50% of children with spinal injury resulting in a major neurologic deficit have normal roentgenograms [78]. The anatomic distribution of spinal injuries relates to the age and the biomechanics of the spine. In the neonatal group cervicothoracic injury is common. In children less than age 8, craniocervical junction injury is unusually common because of the large fulcrum of the head and the hypermobile spine. In older children, a thoracolumbar injury is the second most common site of fracture or fracture-dislocation [79].

Just as with head imaging films, one must be aware of the normal variations in the presentation of the pediatric spine when documenting injury of the spine. There are several diagnostic pitfalls in evaluating pediatric spine radiographs. These are categorized as (1) hypermobility in which angular or translational motion appears excessive; (2) variations in normal lordosis, which may mimic ligamentous injury; and (3) deciphering between synchondrosis and fracture [80].

Cervical

Cervical injuries occur as a result of four types of forces: (1) flexion, (2) extension, (3) axial, and (4) rotational. A frequent cause of misdiagnosis of cervical spine instability is physiologic hypermobility. This in turn is a result of three factors. First, facet joints in infants are shallow and their surfaces are oriented horizontally allowing for an increase degree of motion on flexion and extension as compared with an adult's spine [81–84]. Second, the vertebral bodies have an anterior wedged shape permitting pseudosubluxation of one vertebral body on another [82]. Third, the uncinate processes are not fully developed allowing for both rotational and lateral movement [85].

Variations in cervical lordosis result from a combination of ligamentous laxity and underdeveloped cervical musculature. Because of the greater content of cartilage present in children until the age of 8, lucent areas in the cervical spine can be mistaken for fractures [81,86]. Before discussing specific injuries, it is important to list some local variations in the alignment of the cervical spine. First, the altantodens interval that spans from the posterior border of the anterior arch of the atlas to the anterior margin of the dens can vary up to 5 mm in children. Second, the anterior arch of the atlas can perch the tip of the dens in extension giving the false impression of an odontoid hypoplasia. Third, up to 45% of children can have anterior displacement of C2 on C3 exceeding 3 mm. A simple means to differentiate pathologic subluxation from that caused by physiologic is the posterior cervical line rule [87]. The line is drawn from the anterior aspect of the cortex of the C1 spinous process to the same point on the C3 spinous process. The relationship of the C2 spinous process is compared with this line. A deviation of 2 mm or more is indicative of a pathologic subluxation. Last, up to 14% of children can have anterior displacement of C3 on C4 that exceeds 3 mm [80]. Such normal variations in pediatric spine usually disappear by the age of 8 when the spine conforms to the usual adult appearance. The facet joints deepen, the vertebral bodies become more rectangular, the connective tissue becomes more rigid, and the uncinate process enlarges [80,82,84,88].

In reviewing cervical spine films, the goal should not be primarily in finding a fracture, because in many instances spinal instability might not have a concomitant discernable fracture. A systematic approach involving attention to the curvature of the spine, followed by assessment of the thickness of the prevertebral soft tissue can indicate possible spinal instability secondary to ligamentous injury. A rule of thumb in assessing acceptable prevertebral soft tissue thickness is to allow thickness of 7 mm or less above the glottis and 14 mm or less below the glottis [89]. Lack of prevertebral soft tissue thickness does not rule out spinal injury because it not uncommonly occurs with minimal anterior compression fractures and undisplaced dens fractures [90]. A pitfall in diagnosing increased prevertebral soft tissue thickness incorrectly can result if attention is not paid to the lack of distention of the airway or lack of extension of the cervical spine during a radiograph [90].

The predental space should be measured next. In children widths of 3 to 4 mm can be normal, but anything 5 mm or greater is considered abnormal [33]. Of note the altantodens interval can widen 1 to 2 mm between flexion and extension

views of the cervical spine, and only a lateral neutral film should be used for purposes of calculation [80].

Anterior vertebral body injury is the commonest form of pediatric spine injury. This can take the form of a compression fracture with a teardrop fracture, or subluxation of one vertebral body on another. Ligamentous injury is often present. In children a teardrop fracture consists of an avulsed epiphyseal ring of the vertebral body and indicates compromise of the anterior longitudinal ligament. These growth rings occur at either end of a vertebral body, and can sometimes be mistaken for a fracture. To avoid this pitfall, associated findings of spinal injury should be sought.

Radiographs can assist in discerning the type of injury, whether flexion or extension in nature, and this can be compared with the history of the injury. A flexion injury results in a compressive force anteriorly and a distracting force posteriorly. The radiographic findings consistent with this are wedging of the vertebral body anteriorly with avulsion of the inferior, anterior epiphyseal ring, narrowing of the disk space, widening of the apophyseal joint with variable degree of anterior subluxation, and splaying of the interspinous processes. In an extension injury the reverse is true. The vertebral body is seldom affected and when it is the superior, anterior epiphyseal ring is avulsed. The disk space is widened, and the posterior arch elements are frequently fractured [90]. An important pitfall to be aware of in evaluating flexion type injuries is that the spine can appear normal in a neutral or an extension view, and only in the flexion view is the injury realized [91].

Odontoid fractures occur as a result of either flexion or extension injury. Often there is paucity of clinical findings except for neck pain and resistance movement of the neck. In children less than 8 years of age the fracture usually occurs through the dens-body synchondrosis [87,92]. In flexion-type injuries, lateral and open-mouth views demonstrate increased radiolucency and widening of the synchondrosis, increase in the altantodens interval, and anterior subluxation of the dens [92]. In extension-type of injury the dens is displaced posteriorly, and frequently there is associated fractures of C1 arch or C2 pedicles. In older children, odontoid fractures usually are of the type 2 variety. A nondiagnosed odontoid fracture can manifest in two ways. The first can be progressive myelopathy, and the second an os odontoideum [93]. The latter consists of an ossicle that is separated from the C2 body as a result of traumatic vascular injury to the synchondrosis.

Another type of high cervical injury with minimal clinical findings is the Jefferson fracture, which involves the anterior or posterior rings of the C1 vertebrae. An axial loading force is the cause of this burst fracture. Diagnosis can be established with an open-mouth view. According to the "Rule of Spence," if the sum total overhang of the two lateral masses of C1 on C2 is 7 mm or greater, then there is a high likelihood that the transverse ligament is disrupted [94]. This type of fracture is uncommon in children less than 9 years of age because of the cartilaginous nature of the ring.

A hangman's fracture can result from either hyperextension or hyperflexion, although hyperextension with axial loading is more commonly the cause of the injury. A hangman's fracture caused by hanging differs in that the submentally placed noose causes a hyperextension with distraction [95]. On radiographs a bilateral fracture of the C2 pedicles with associated spondylosthesis is seen. Although most fractures result from motor vehicle accidents or diving, in infants such a fracture can result from violent shaking [96]. The three subclassification are (1) type 1: 3 mm or less subluxation of C2 on C3; (2) type 2: 4 mm or more subluxation of C2 on C3 or greater than 11 degrees of angulation; and (3) type 3: disruption of the C2-3 facet with fracture through the isthmus and resultant locked facet. Lateral C-spine radiographs show the fracture in 95% of the cases. A CT with thin bone slices picks up the rest. Ninety percent of the fractures heal with immobilization; however, before reducing a type 2 or 3 fracture, an MRI should be obtained to assess for any ruptured cervical disk that can result in further spinal cord injury during reduction. In an extremely small subpopulation of children a bony dysplasia, such as pyknodysostosis or Crouzon's disease, can mimic a hangman fracture [97,98]. These occur as a result of nonfusion of the dens-arch synchondrosis of C2. They appear on oblique cervical views and disappear on lateral views.

Lateral flexion injuries of the cervical spine can range from ipsilateral vertebral body compression to contralateral brachial plexus avulsion. The clinical presentation can vary from ipsilateral neck pain with no neurologic deficit, to contralateral Duchenne-Erb paralysis with injury to C4-6, to contralateral Klumpke's paralysis or an ipsilateral Horner's syndrome with a C7-T1 injury [90]. On anteroposterior views of the cervical spine this type of injury can manifest as an ipsilateral vertebral body wedging, a contralateral transverse process or an uncinate process fracture, or a widening of

the joints of Lushka [99]. The joints of Lushka are a series of joint-like structures that occur along the superior, lateral edges of the vertebral bodies of C3-T1. They have a spurlike structure that is covered with a synovial capsule. In an anteroposterior view of a lateral flexion stance, contralateral widening of all these joints can occur; however, if only one or two joints appear widened then significant underlying ligamentous tear with instability of the cervical spine should be suspected [90].

Thoracolumbar

Vertebral body compressions are the most common type of injury in this location. The mechanism is the same as in cervical injuries. The clinical manifestation ranges from midline lower back pain to abdominal pain. In infants fractures involve the epiphyseal end plates. A teardrop fracture in the lumbar region is known as a *limbus fracture*. In severe hyperflexion injury, the nucleus pulposus can herniate into the adjacent vertebral body. The herniation invariably occurs along the superior, anterior region of the vertebral body. The replacement of the vertebral body bone marrow with disk material results in a notched appearance of the vertebral body on radiograph because of loss of bone density [77]. Although diskitis can also result in this appearance, several features differentiate this etiology from trauma: notching caused by trauma involves a larger portion of the vertebral body, trauma results in mild disk narrowing, and notching caused by infection has an accompanying increase in the erythrocyte sedimentation rate and white count [100].

Dislocation of the posterior elements can result in a splaying of the spinous processes with avulsion of the tips. Because the spinous process is completely cartilaginous in infants, a radiograph of a chronically abused child shows progressive calcification and ossification of these fragments resembling a clay shoveler's fracture seen in a radiograph of an adult's spine. With increasing severity in the hyperflexion of the lower spine there is dislocation of the facets with either a perched appearance or a locked facet appearance. With fractionation of the neural arch, a so-called *wandering vertebral body* is seen on radiograph delineating a subluxation of the vertebral body as a result of separation from the pedicle [101]. An axial loading force can result in a burst fracture of the vertebral body. A combination of CT and MRI assists in detecting the structural compromise and the resultant degree of spinal cord injury.

Spinal cord injury without radiographic abnormality

A phenomena that occurs in children with spinal trauma is spinal cord injury without radiographic abnormality (SCIWORA). It is estimated to occur in up to 30% of children with spinal trauma [102]. The basis for this is caused by the elasticity of the spine in children. As discussed previously, the spine of a child is comparably more elastic than that of the adult's. Only after the age of 8 does the pediatric spine resemble the adult spine in biomechanical characteristics. In an attempt to define this quality, Leventhal [103] placed the spine of neonatal cadavers under tension and found that the spine could be stretched up to 2 inches without disruption, whereas the spinal cord could withstand only 0.25 inches in distraction. The region of maximal mobility in the cervical spine of a child resides at the C2-3 level in a child as compared with the C5-6 level in older children and adults. This is caused largely in part by the disproportionate head circumference and weak supportive structures in a young child [102]. Furthermore, in young children the surface of the lateral C1 arches and the occipital condyles are flat as compared with that of the adult's, which forms a groove for the vertebral artery's entrance into the skull. During extreme flexion and extension of the cervical spine, as occurs in infant shaking, the vertebral arteries can become compressed and result in a cervical spine infarct [104]. Clinical experience validates this theory because SCIWORA occurs predominantly in young children and affects primarily the upper cervical spine [105]. Furthermore, the younger a child is when sustaining a SCIWORA, the greater he or she is at risk for developing a complete neurologic deficit [105]. SCIWORA should only be diagnosed after complete radiologic work-up has excluded any structural defect that can explain the neurologic deficit.

Summary

The long-term outlook for infants subjected to nonaccidental trauma is bleak. In a retrospective study with a mean 9-year follow-up, Duhaime et al [63] noted the relationship between acute factors on presentation and the long-term outcome.

Infants who were unresponsive on presentation remained vegetative or severely impaired at follow-up. Most of the infants who required intubations were severely impaired at follow-up. In those with acute seizures, 60% were severely impaired, and an equal number had moderately severe to good outcome. Less than 6 months of age at the time of insult resulted in severe disability in most infants. CT findings of diffuse hypodensity or loss of gray-white differentiation led to severe disability at follow-up. Focal areas of hypodensity or contusion had an equal probability of good outcome or severe disability.

Child abuse unfortunately is prevalent in our society, and as physicians our profession requires that we help our patients. When dealing with one of our most vulnerable group of patients, it is imperative when a child with injury is examined that physicians keep in their differential child abuse. This article has provided information in regards to radiographic studies that assist a physician in diagnosing child abuse. The importance of this not only is in providing appropriate care, but far more importantly in protecting a child or his or her siblings from future abuse.

References

[1] Caffey J. Multiple fractures in the long bones of infants suffering from subdural hematoma. Am J Roentgenol 1946;56:163–73.
[2] Kempe C, Silverman F, Steele B, et al. The battered child syndrome. JAMA 1962;181:105–12.
[3] Chen MH, Bullock R, Graham DI, et al. Ischemic neuronal damage after acute subdural hematoma in the rat: effects of pretreatment with a glutamate antagonist. J Neurosurg 1991;74:944–50.
[4] Duhaime AC, Sutton L. Head injury in the pediatric patient. In: Tindall G, Cooper P, Barrow D, editors. The practice of neurosurgery. Baltimore: Williams and Wilkins; 1996. p. 1553–7.
[5] Luerssen T. Head injuries in children. Neurosurg Clin North Am 1991;2:399–410.
[6] Craft AW, Shaw DA, Carlidge NEF. Head injuries in children. BMJ 1972;4:200–3.
[7] Mayer T, Walker ML, Johnson DG, Matlak ME. Causes of morbidity and mortality in severe pediatric trauma. JAMA 1981;245:719–21.
[8] Billmore ME, Meyers PA. Serious head injury in infants: accident or abuse? Pediatrics 1985;75:340–2.
[9] Helfer RE, Slovis TL, Black M. Injuries resulting when small children fall out of bed. Pediatrics 1977;60:533–5.
[10] Hobbs CJ. Skull fracture and the diagnosis of abuse. Archives of Disabled Child 1984;59:246–52.
[11] Annegers J. The epidemiology of head trauma in children. In: Shapiro K, editors. Pediatric head trauma. New York: Futura; 1983. p. 1–10.
[12] Goldstein F, Levin H. Epidemiology of pediatric closed head injury: incidence, clinical characteristics, and risk factors. J Learn Disabil 1987;20: 518–25.
[13] Kraus J, Fife D, Conroy C. Pediatric brain injuries: the nature, clinical course, and early outcomes in a defined United States population. Pediatrics 1987;79:501–7.
[14] Bleyer WA. The impact in the United States and the world of central nervous system cancer during childhood. In: Packer R, Bleyer W, Pochedly C, editors. Pediatric neuro-oncology. Chur, Switzerland: Harwood; 1992. p. 1–6.
[15] Bijur P, Haslum M, Golding J. Cognitive and behavioral sequelae of mild head injury in children. Pediatrics 1990;86:337–44.
[16] Churchill J, Stevenson L, Habhab G. Cephalohematoma and natal brain injury. Obstet Gynecol 1966;27:580–584.
[17] Hartley J, Burnett C. An enquiry into the causation and characteristics of cephalohematoma. Br J Radiol 1944;17:33–41.
[18] Ingram M, Hamilton W. Cephalohematoma in the newborn. Radiology 1950;55:503–7.
[19] Kendall N, Woloshin H. Cephalohematoma associated fracture of the skull. J Pediatr 1952;41: 125–32.
[20] Hamlin H. Subgaleal hematoma caused by hair-pull. JAMA 1968;205:314.
[21] Alexander E, Davis C. Intra-uterine fracture of the infant's skull. J Neurosurg 1969;30:446–54.
[22] Swischuk LE. The head. Emergency radiology of the acutely ill or injured child. 2nd edition. Baltimore: Williams and Wilkins; 1986. p. 491–555.
[23] Harwood-Nash DC, Hendrick EB, Hudson AR. The significance of skull fractures in children. Radiology 1971;101:151–5.
[24] Leonidas J, Ting W, Binkiewicz A, Vaz R, Scott RM, Pauker SG. Mild head trauma in children: when is a roentgenogram necessary? Pediatrics 1982;69:139–43.
[25] Kier EL. Fetal skull. In: Newton TH, Potts DG, editors. Radiology of the skull and brain, vol. 1. St. Louis: CV Mosby; 1971. p. 99–117.
[26] Koehler A, Zimmer EA. Borderlands of the normal and early pathologic in the skeletal roentgenology. 11th edition. New York: Grune and Stratton; 1968.
[27] Allen EW, Kier LE, Rothman LGS. Pitfalls in the evaluation of skull trauma. Radiol Clin North Am 1973;9:479–503.
[28] Pfleger MJ, Hardee EP, Hayman LA. Fluid/fluid levels in intracerebral hematoma: a sign of coagulopathy. Reported at the 30th Annual Scientific Meeting of the American Society of Neuro radiology. St. Louis, May 31–June 5, 1992.

[29] Epstein J, Epstein B, Small M. Subepicranial hygroma: a complication of head injuries in infants and children. J Pediatr 1961;59:562–6.
[30] Takagi T, Nagai R, Wakabayashi S, et al. Extradural hemorrhage in the newborn as a result of birth trauma. Childs Brain 1978;4:306–18.
[31] Weiner HL, Weinberg JS. Head injury in the pediatric age group. In: Cooper PR, Golfinos JG, editors. Head injury. 4th edition. New York: McGraw-Hill; 2000. p. 419–56.
[32] Meservy CJ, Towbin R, McLaurin RL, et al. Radiographic characteristics of skull fractures resulting from child abuse. AJR Am J Roentgenol 1987;149:173–5.
[33] Locke GR, Gardner JI, Van Epps EP. Atlas-dens interval (ADI) in children: a survey based on 200 normal cervical spines. AJR Am J Roentgenol 1966;97:135–40.
[34] Tsai FY, Zee CS, Apthorp JS, Dixon GH. Computed tomography in child abuse head trauma. CT: The Journal of Computed Tomography. 1980;4: 227–86.
[35] Zimmerman RA, Bilaniuk LT. Computed tomography in pediatric head trauma. J Neuroradiol 1981;8:257–71.
[36] Zyed A, Hayman LA, Bryan RN. MR imaging of intracerebral blood: diversity of the temporal pattern at 0.5 and 1.0 T. AJNR Am J Neuroradiol 1991;12:469–74.
[37] Levy H, O'Connor J, Ingall D. Bacteremia infected cephalohematoma and osteomyelitis of the skull in the newborn. Am J Dis Child 1967;114: 649–51.
[38] Matson DD. Subdural hematoma. In: Ingraham FD, Matson DD, editors. Neurosurgery of infancy and childhood. Springfield, IL: Charles C. Thomas; 1969. p. 328–47.
[39] Lagos JC. Differential diagnosis in pediatric neurosurgery. Boston: Little, Brown and Co; 1971.
[40] Drake J, Siddiqi S. Birth trauma. In: Youmans J, Becker D, Dunsker S, et al,editors. Neurological surgery. 4th edition. Philadelphia: WB Saunders; 1997. p. 1767–76.
[41] Mealey Jr J. Pediatric head injuries. Springfield, IL: Charles C Thomas; 1968. p. 166–201.
[42] Rabe EF, Flynn RD, Dodge PR. A study of subdural effusion in an infant. Neurology 1962; 12:79–92.
[43] Rabe EF, Young GF, Dodge PR. The distribution and fate of subdurally instilled human serum albumin in infants with subdural collection of fluid. Neurology 1964;14:1020–8.
[44] Mealey Jr J. Infantile subdural hematomas. Pediatr Clin North Am 1975;22:433–42.
[45] Dias MS, Backstrom J, Falk M, Li V. Serial radiography in the infant shaken impact syndrome. Pediatr Neurosurg 1998;29:77–85.
[46] McCluney KW, Yeakley JW, Fenstermacher MJ, Baird SH, Bonmati CM. Subdural hygroma versus atrophy on MR brain scans: "the cortical vein sign". AJNR Am J Neuroradiol 1992;13:1335–9.
[47] Chen CY, Chou TY, Zimmerman RA, Lee CC, Chen FH, Faro SH. Pericerebral fluid collection: differentiation of enlarged subarachnoid spaces from subdural collections with color Doppler US. Radiology 1996;201:389–92.
[48] Prassopolous P, Cavouras D, Golfinopoulos S, et al. The size of the intra- and extra-ventricular cerebrospinal fluid compartments in children with idiopathic benign widening of the frontal subarachnoid space. Neuroradiology 1995;37:418–21.
[49] Boaz JC, Edwards-Brown K. Hydrocephalus in children: neurosurgical and neuroimaging concerns. Neuroimaging Clin N Am 1999;9:73–85.
[50] Duhaime AC, Christian C, Armonda R, Hunter J, Jertle R. Disappearing subdural hematomas in children. Pediatr Neurosurg 1996;25:116–22.
[51] Kakarieka A. Review of traumatic subarachnoid hemorrhage. Neurol Res 1997;19:230–2.
[52] Ashikaga R. Clinical utility of MR FLAIR imaging for head injuries. Nippon Igaku Hoshasen Gakkai Zasshi 1996;56:1045–9.
[53] Ashikaga R, Araki Y, Ishida O. MRI of head injury using FLAIR. Neuroradiology 1997;39: 239–42.
[54] Przybylski GJ, Clyde BL, Fitz CR. Craniocervical junction subarachnoid hemorrhage associated with atlanto-occipital dislocation. Spine 1996;21: 1761–68.
[55] Zimmerman RD, Russell EJ, Yurberg E, Leeds NE. Falx and interhemispheric fissure on axial CT. 2. Recognition and differentiation of interhemispheric subarachnoid and subdural hemorrhage. AJNR Am J Neuroradiol 1982;3:635–42.
[56] Kleinman PK. Head trauma. Diagnostic imaging of child abuse. Baltimore: Williams and Wilkins; 1987. p. 159–200.
[57] Kirkpatrick JB, Hayman LA. Pathophysiology of intracranial hemorrhage. Neuroimaging Clin N Am 1992;2:11–23.
[58] Stein SC, Ross SE. Moderate head injury: a guide to initial management. J Neurosurg 1992;77:562–4.
[59] Walenga JM, Marmon JF. Coagulopathies associated with intracranial hemorrhage. Neuroimaging Clin N Am 1992;2:137–52.
[60] Kreel L, Kay R, Woo J, et al. The radiological (CT) and clinical sequelae of primary intracerebral hemorrhage. Br J Radiol 1991;64:1096–100.
[61] Hershey BL, Zimmerman RA. Pediatric brain computed tomography. Pediatr Clin North Am 1985;32:1477–508.
[62] Cohen RA, Kaufman RA, Myers PA, Towbin RB. Cranial computed tomography in the abused child with head injury. AJR Am J Roentgenol 1986; 146:97–102.
[63] Duhaime AC, Christian C, Moss E, Seidl T. Long-term outcome in infants with the shaking-impact syndrome. Pediatr Neurosurg 1996;24:292–8.

[64] Duhaime AC, Bilaniuk L, Zimmerman R. The "big black brain" radiographic changes after severe inflicted head injury in infancy. J Neurotrauma 1993;10(Suppl 1):559.
[65] Miller JD, Bullock R, Graham DI, Chen MH, Teasdale GM. Ischemic brain damage in a model of acute subdural hematoma. Neurosurgery 1990; 27:433–9.
[66] Bass M, Kravath R, Glass L. Death scene investigation in sudden infant death. N Engl J Med 1986;315:100–5.
[67] Cristoffel K, Zierserl E, Chiarmonte J. Should child abuse and neglect be considered when a child dies unexpectedly? Am J Dis Child 1985;139:876–80.
[68] Hobbs C. ABC of child abuse: head injuries. BMJ 1989;198:1169–70.
[69] Lindenberg R, Freytag E. Morphology of brain lesions from blunt trauma in early infancy. Arch Pathol 1969;87:298–305.
[70] Holbourn AHS. Mechanics of head injuries. Lancet 1943;2:438–50.
[71] Zimmerman RA, Bilaniuk LT, Genneralli T. Computed tomography of shearing injuries of the cerebral white matter. Radiology 1978;127:393–6.
[72] Kriel R, Krach L, Sheehan M. Pediatric closed head injury: comparison of children younger and older than 6 years of age. Pediatr Neurol 1989; 5:296–300.
[73] Gentry LR, Gordersky JC, Thompson B, Dunn VD. Prospective comparative study of intermediate-field MR and CT in the evaluation of closed head trauma. AJNR Am J Neuroradiol 1988;9: 91–100.
[74] Kelly AB, Zimmerman RD, Snow RB, et al. Head trauma: comparison of MR and CT- experience in 100 patients. AJNR Am J Neuroradiol 1988;9: 699–708.
[75] Besenki N, Jadro-Santel D, Grevic N. Patterns of lesions of corpus callosum in inner cerebral trauma visualized by computed tomography. Neuroradiology 1992;34:126–30.
[76] Gentry LR. Primary neuronal injuries. Neuroimaging Clin N Am 1991;1:411–32.
[77] Kleinman PK. Spinal trauma. In: Kleinman PK, editor. Diagnostic imaging of child abuse. Baltimore: William and Wilkins; 1987. p. 91–102.
[78] Burke DC. Traumatic spinal paralysis in children. Paraplegia 1974;11:268–76.
[79] Heffez DS, Ducker TB. Fractures and dislocations of the pediatric spine. In: Pang D, editor. Disorders of the pediatric spine. New York: Raven Press; 1995. p. 517–29.
[80] Cattell HS, Filtzer DL. Pseudosubluxation and other normal variation in the cervical spine in children. J Bone Joint Surg 1965;47:1295–309.
[81] Allen BL, Ferguson RL. Cervical spine trauma in children. In: Bradford D, Hensinger R, editors. The pediatric spine. New York: Thieme; 1985. p. 89–l04.
[82] Bailey DK. The normal cervical spine in infants and children. Radiology 1952;59:712–19.
[83] Townsend EH, Rowe ML. Mobility of the upper cervical spine in health and disease. Pediatrics 1952;10:567–73.
[84] Von Torklos D, Gehle W. The upper cervical spine. New York: Grune and Stratton; 1972. p. 10–94.
[85] Fielding JW, Hensinger RN. Fractures of the spine. In: Rockwood CA, Wilkins KE, King RE, editors. Fractures in children. Philadelphia: JB Lippincott; 1984. p. 683–730.
[86] Clark WM, Gehweiler JA, Laib R. Twelve significant signs of cervical spine trauma. Skeletal Radiol 1979;3:201–5.
[87] Griffiths SC. Fracture of odontoid process in children. J Pediatric Surg 1972;7:680–3.
[88] Fesmire F, Luten RC. The pediatric cervical spine: developmental anatomy and clinical aspects. J Emerg Med 1989;7:133–42.
[89] Penning L. Prevertebral hematoma in cervical spine injury: incidence and etiologic significance. AJR Am J Roentgenol 1981;136:553–61.
[90] Swischuk LE. The spine and spinal cord. In: Emergency radiology of the acutely ill or injured child. 2nd edition. Baltimore: Williams and Wilkins; 1986. p. 491–555.
[91] Scher AT. Anterior cervical subluxation: an unstable position. AJR Am J Roentgenol 1979;133: 275–80.
[92] Seimon LP. Fracture of the odontoid process in young children. J Bone Joint Surg Am 1977;59: 943–8.
[93] Fielding JW, Hensinger RN, Hawkins RJ. Os odontoideum. J Bone Joint Surg Am 1980;62: 376–83.
[94] Spence KF, Decker S, Sell KW. Bursting atlantal fracture associated with rupture of the transverse ligament. J Bone Joint Surg Am 1970;52:543–9.
[95] Wood-Jones F. The ideal lesion produced by judicial hanging. Lancet 1913;1:53.
[96] McGrory B, Fenichel GM. Hangman's fracture subsequent to shaking in an infant. Ann Neurol 1977;2:82.
[97] Currarino G. Primary spondylolysis of the axis vertebra (C2) in three children, including one with pyknodystosis. Pediatr Radiol 1989;19:535–8.
[98] Parisi M, Lieberson R, Shatsky S. Hangman's fracture or primary spondylolysis: a patient and a brief review. Pediatr Radiol 1991;21:367–8.
[99] Schaaf RE, Gehweiler JA, Miller MD, Powers B. Lateral hyperflexion injuries of the cervical spine. Skeletal Radiol 1978;3:73–8.
[100] Swischuk LE. The beaked, notched, or hooked vertebra: its significance in infants and young children. Radiology 1970;95:661–4.
[101] Faure C, Steadman C, Lelande G, Al Moudares N, Marsault C, Bennet J. The wandering vertebral body. Ann Radiol 1979;22:96–9.

[102] Pollack IF, Pang D. Spinal cord injury without radiographic abnormality (SCIWORA). In: Dang P. Disorders of the pediatric spine. New York: Raven Press; 1995. p. 509–16.
[103] Leventhal HR. Birth injuries of the spinal cord. J Pediatr 1960;56:447–53.
[104] Gilles FH, Bina M, Sotrel A. Infantile atlanto-occipital instability: the potential danger of extreme extension. Am J Dis Child 1979;133:30–7.
[105] Pang D, Pollack IF. Spinal cord injury without radiographic abnormality in children the SCIWORA syndrome. J Trauma 1989;29:654–64.