Cervical Spinal Cord Injury in Abused Children

Kenneth W. Feldman, MD, Anthony M. Avellino, MD,†*
Naomi F. Sugar, MD, and Richard G. Ellenbogen, MD†*

Abstract: Five infants and toddlers who sustained cervical spinal cord injury as the result of child abuse are described. Three cases are previously unreported. Diagnosis was complicated by coexistent brain injuries and their treatments, subtle and/or evolving paralysis, and central cord syndrome, in which arm function is diminished but leg function is preserved. Definitive spinal imaging by magnetic resonance imaging (MRI), computed tomography, and plain radiographs was delayed because of life support efforts. When completed, the MRI was most sensitive to cord injury. Evidence of associated bony spinal injury was often absent or unapparent until healing occurred; 4 children had spinal cord injury without (or with minimal) radiological abnormality. The 3 children presenting to our hospital with cord injury represent 1% of the estimated cases of inflicted head injury seen during a 23-year period.

Key Words: child abuse, craniocervical injury, cord injury, spinal, inflicted head injury, spinal cord injury without radiological abnormality

Clinically evident cervical spinal cord injuries are rare in abused children,[1–3] and recognition may be difficult because of subtle signs, coexistent traumatic brain injury, and "central cervical cord syndrome."[1,4–6] Furthermore, children may sustain spinal cord injury without bony spine injury (spinal cord injury without radiological abnormality [SCIWORA]), and spinal cord vascular events may result in delayed onset of cord injury symptoms.[1,6–10] During the first author's 23-year child abuse consultation practice at a major pediatric referral hospital, only 3 abused children treated there have been recognized clinically to have cervical cord injury. This author has also consulted on the legal cases involving 2 other children with cervical cord injury, one of whom subsequently obtained neurosurgical care at our institution. The 3 previously unreported cases are presented in detail, and the 2 previously reported cases are presented in tabular form to highlight the diagnostic difficulties.

This paper was deemed institutional review board exempt by the Children's Hospital and Regional Medical Center Human Subjects Review Board.

*General Pediatric Division, Department of Pediatrics; and †Pediatric Neurosurgery Division, Department of Surgery, Children's Hospital and Regional Medical Center, and University of Washington School of Medicine, Seattle, WA.

The authors have no conflicts of interest to disclose.

Address correspondence and reprint requests to Kenneth W. Feldman, MD, 2101 East Yesler Way, Seattle, WA 98122. E-mail: kfeldman@u.washington.edu.

ISSN: 0749-5161/08/2404-0222

CASE

Case 1

A 2-month-old infant had 3 days of fussiness, decreased feeding and activity, congested and grunting respiration, and an altered high-pitched cry. Symptoms were reported to have begun after she fell from her dad's chest to the floor while both had been resting in bed. Her mother reported an elevated temperature on 1 measurement at home. Physical examination was considered normal except for the presenting symptoms and decreased tone. Spinal tap, complete blood count, and iron and coagulation studies were normal except for anemia with a hematocrit of 0.26. Urinalysis contained ketones and blood. She was thought to be septic and received 48 hours of intravenous antibiotics, but bacterial cultures and viral studies were normal.

She had been the product of her mother's first pregnancy, which was term and uncomplicated. She was breast-fed, growing appropriately thereafter.

The day after admission, a chest film was obtained for cough. A healing fracture of the left seventh rib was noted, leading to a full skeletal survey 2 days later. This revealed split cranial sutures, a linear vertical right midparietal fracture, and a high left parietal fracture; the right fracture was accompanied by scalp swelling. Numerous additional acute and healing fractures were identified, including the right second and left second to seventh rib necks, the right distal radial and ulnar diaphyses, left distal tibial and bilateral distal femoral metaphyses, bilateral fractures of the distal acromions and proximal humeri, and buckle fractures of the distal left third to fifth metatarsals. Dilated ophthalmology examination revealed no retinal hemorrhages.

On the fourth day after admission, it was observed that she moved her legs normally, but her arms moved little and were hypotonic, right more so than left. Computed tomography (CT) of her head that day showed the skull fractures, prominent frontal extra-axial spaces, and an acute left frontal subdural hemorrhage. The following day, magnetic resonance imaging (MRI) revealed a right posterior medulla hemorrhage extending to the peduncle, patchy increased spinal cord T2 signal and gradient sequences at the C3-4 level, and a posterior bulge of the body of C4 into the spinal canal (Fig. 1).

The infant was discharged to foster care with cervical immobilization for her central cord syndrome after a 20-day hospitalization. Three months after injury, she was moving all of her limbs normally. She still had an atrophic narrowed spinal cord with persistent central cord increased gradient and T2 signals on MRI. Her cranial MRI lacked evidence of residual brain injury or extra-axial fluid collections.[a]

[a]*Comment on Case 1:* Abused infants may present with vague symptoms that may be attributed to other causes such as infection. As a result, the correct diagnosis is often delayed.[11] In this infant, the intracranial injury and multiple fractures led to a clear diagnosis of abusive injury. The preserved function of her lower extremities in the face of decreased movement of her arms contributed to the delay in diagnosis of cervical cord injury. Magnetic resonance imaging was critical in documenting her medulla and spinal cord hemorrhages.

FIGURE 1. Sagittal T2 MRI 8 days after the injury of a 2-month-old child with central cord syndrome. Hemorrhage (confirmed by a gradient echo sequence) extends from the lower medulla (thick line) down the central cord to C7 (posterior arrows). The body of C4 protrudes posteriorly into the spinal canal (anterior arrow).

Case 2

In the early 1980s, a 4-month-old female infant acutely developed fever, respiratory distress, lethargy, and semiflaccidity while in her father's care. She required intubation at an outside emergency department (ED) and remained obtunded thereafter. She experienced a full respiratory arrest during transport. Her initial chest film revealed several healing rib fractures, whereas one taken a month before for pneumonitis had not.

The child had been born at 35 weeks of gestation and had transient tachypnea of the newborn and hyperbilirubinemia but thrived thereafter. Her father did not reside with the family but regularly visited and provided child care. After those visits, the mother observed bruises and, on 1 occasion, a facial burn on the child.

On initial examination, the infant was intubated and was on a ventilator. Multiple bruises were noted. She had variable lethargy with a left gaze preference. She exhibited occasional spontaneous movement of all but her right arm and had purposeful withdrawal of all extremities. Her toes were up going, and reflexes were 1 to 2^{+}. Neurological and neurosurgical examinations confirmed her purposeful extremity motion. Retinal hemorrhages were absent.

Head CT demonstrated a small right parietal subdural hemorrhage and contusion beneath a parietooccipital skull fracture. At least 19 healing and acute posterior and lateral rib fractures were observed. Both distal femurs and proximal tibias had acute classic metaphyseal fractures, and both distal tibias had healing metaphyseal fractures.

Over the next few days, her sensorium improved with a waxing and waning course. She had variable presence of spontaneous and stimulated limb movement, some of which appeared to be a triple flexion response. Urinary retention was noted on day 2. Spinal functional integrity was questioned that day but was felt normal the next day. Five days into the hospital course, as her sensorium was improving, her examination became more concerning for cord injury. Spine radiographs and CT scan remained normal except for a widened C5-6 disk space. The cervical subarachnoid space seemed preserved.

By 2 weeks after injury, linear densities projected from the inferior margin of C5 and superior margin of C6, suggesting healing anterior longitudinal ligament injury. Myelography 1 month after presentation demonstrated a diffuse thoracic extradural mass. At the cervicothoracic junction, contrast material leaked from the subarachnoid to the extradural space, flowing both upward and downward. However, contrast failed to enter the subarachnoid space above this point. By 6 weeks after injury, her cord contour was narrowed and irregular in the lower neck, and no cord was seen on myelography at C7, suggesting cord transaction.

She remained permanently quadriplegic. Her father pled guilty to her assault.[b]

Case 3

A nearly 6-month-old female infant developed decreased arousal and respiratory effort while in her father's care. Emergency aid was called, and she was taken to an outside ED, where pneumonia was suspected. Her chemistries were normal, hematocrit was 32%, and white blood cell count was 11,700/μL. Her chest film suggested a right upper lobe infiltrate. On transfer to our ED, she was noted to have hypotonia, right eye deviation, lethargy, and a tense and full fontanelle. No skin trauma was seen. A spinal tap yield cloudy yellow-tinged fluid with 3610 red blood cells and 50 white blood cells per microliter and slight xanthochromia. Cranial CT scan showed a vertical right parietal fracture with accompanying soft tissue swelling but no intracranial lesions. The family had no explanation for her injury.

She had been a normal term infant who grew well on formula thereafter. She had a history of bronchiolitis. She had been seen at an outside ED at age of 4 months with left face bruising and swelling, attributed to a fall off a bed while in her mother's care. A CT scan at that visit confirmed the soft tissue swelling, but no skull fracture or intracranial injury was present.

She was admitted to the intensive care unit where she was somnolent but arousable. Her eyes and head turned right. Her right-sided tone was reduced, and she was areflexic. She withdrew her legs but not arms from pain. She had prominent inspiratory retractions, expiratory prolongation, with diffuse mucoid and wheezy lung sounds. Her respiratory pattern was primarily abdominal. A small nodule of scar tissue was present on her upper lip frenulum, and the tip of her right tongue was bruised from recent injury. Her chest radiographs showed perihilar congestion, right upper lobe atelectasis, and healing fractures of the right lateral seventh to eight ribs. She was placed in a cervical collar, and a craniospinal MRI was scheduled. Dilated ophthalmology examination found no retinal hemorrhages.

On the second day of hospitalization, the MRI showed abnormal density (dark on T1 and bright on T2) of the upper posterior medulla that extended into the central spinal cervical cord down to C7 (Fig. 2). This was accompanied by cord swelling and

[b]*Comment on Case 2:* The infant's global impairment and waxing and waning motor and sensory examinations delayed definitive diagnosis of cord injury. The lack of MRI scanning during the early 1980s caused clinicians to rely on cruder and more invasive diagnostic methods, also delaying definitive evaluation.

FIGURE 2. Sagittal T2 MRI of a 6-month-old infant with central cord syndrome. High density of acute hemorrhage extends from the upper posterior medulla (line 2) to C7. The cord is swollen (line 1).

gradient echo evidence of blood products. No bony spinal injury was found on radiographs then or on subsequent flexion/extension views and CT. Skeletal survey on the fifth day of hospitalization showed a well-callused transverse fracture of the proximal third of the left tibia. Metaphyseal fractures were identified at the right tibial plateau and distal left tibia. Follow-up films 3 weeks after presentation also showed fractures of the necks of the seventh to eighth right and second to eighth left ribs.

Her father subsequently admitted shaking her before the presenting event as well as grasping and jerking her leg during a prior event of frustration.

She was discharged to assisted care 1 month after presentation but eventually returned to her mother's care. At discharge, she had some shoulder but no other arm movement and was developing contractures.

By 5 years old, she was doing well academically in kindergarten but had persistent bladder dysfunction, right greater than left weakness and spasticity, and an altered gait and required assistive devices for self-care. She was able to use both her arms.[c]

DISCUSSION

Anatomical and developmental differences cause different patterns of cervical spinal injury in infants and toddlers than occur in older children and adults. The infant's horizontally oriented facets, incomplete uncovertebral joints, lax ligaments, weak cervical muscles, and large head predispose to ligament and cord injury instead of bony injury.[12,13] Because the axis of maximum flexion is higher in the infant's cervical spine than in the adolescent's or adult's, infant injuries tend to involve higher levels of the cord.[9,13,14] The anterior vertebral artery, descending in the spinal canal, gives off segmental arteries that preferentially supply the central cord and the corticospinal tracts serving the arms.[4,6] Injury to these vessels or delayed vascular compression from swelling within the canal may result in delayed central cervical cord vascular compromise with onset of paralysis subsequent to injury and after the child's initial head injury presentation.[1,13] Fifty-two percent of Pang and Wilberger's[1] "spinal cord injury without radiological (bony) abnormality" (SCIWORA) children had delayed onset of paralysis. The loss of arm function may be overlooked in the child who still has normal leg function.[4,6] Two of our patients (cases 1 and 3) demonstrated this central cervical cord pattern, and an additional child (case 5) may have had delayed onset of paralysis after injury (Fig. 3 and Table 1).

In the past and again in the current series, the cervical MRI scan combined with a 3-dimensional formatted CT scan done to elucidate the bony anatomy/pathology are the most effective imaging modalities for cervical spine injury.[6,7,15] The MRI allows direct evaluation for cord damage and is a means of imaging the soft tissues surrounding and supporting the bony spine; this makes MRI the best modality for evaluation of SCIWORA. Gradient echo sequences highlight blood products in the cord, and fat-saturated T2 sequences are best to identify ligamentous injury.[16] However, using the MRI scanning sequences of the mid-1990s, scans in unselected children with inflicted head injury but no signs of cervical spine or cord trauma, there was a low yield of abnormality. On this basis, cervical MRI scans of infants with head injury but no spinal cord symptoms are not routinely recommended.[17] Although MRI usually does not image bony injuries as well as the CT, it provided the best image of the posterior bulge of the body of C4 in our case 1. The CT scan, combined with passive flexion extension plain radiographs, can help delineate essential bone anatomy and further elucidated stability issues.

Although cases of cervical spinal cord injury can result from child abuse, clinically apparent injuries have been rare.[1–3] Spinal cord injuries from abuse have composed 0% to 8.6% of all childhood spine injuries under 8 years.[1,8–10,13,14] Likewise, spinal fractures have been reported in small case series[10,18] and in 0% to 4% of larger series of inflicted skeletal injury victims.[19,20] Injuries to the bony spine have been more common than cord damage, but the opposite

[c]*Comment on Case 3:* The discrepancy between normal leg and altered arm function was noted early, resulting in a rapid diagnosis of central cervical cord injury. Recognition of her diaphragmatic respiratory pattern, also due to cord injury, aided her diagnosis. The MRI was important for differentiating cord injury from brachial plexus injury.

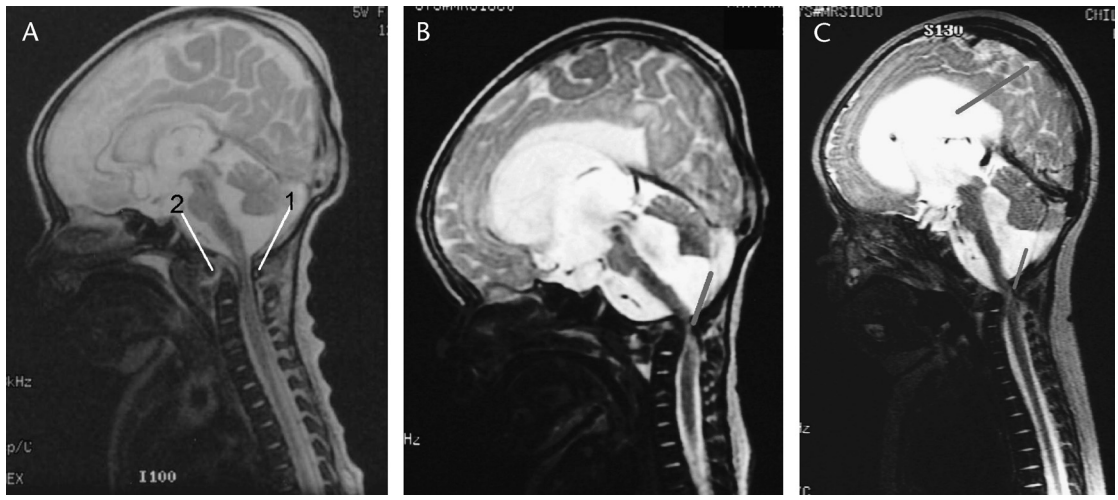

FIGURE 3. Case 4: sequential spinal sagittal T2 MRIs in a 6-month-old female infant with upper cervical cord injury and osteomyelitis. A, Two weeks after injury, the upper cervical cord is edematous and compressed by swelling from in front of and behind the cord by extradural tissues (line 1). Soft tissue edema is present anterior to the dens (line 2). B, Five weeks after injury, the vertebral column is unstable with significant spinal cord compression at C1-2 (line). The cord is attenuated. C, Two months after injury, marked spinal cord compression is noted (short line). Significant brain atrophy and ventriculomegaly are present (long line).

situation (SCIWORA) also has been noted in young children.[1,6–10] Inflicted bony spine injuries are more common at the thoracolumbar junction than in the cervical region.[17,20]

Autopsy series suggest that cervical epidural or subdural hemorrhage[16,21] and microscopic evidence of cervicomedullary junction injury[22] are more common than the clinically evident injuries. These cervicomedullary injuries may be a significant cause of the apnea commonly seen in victims of inflicted head injury.[22,23]

Our cases reinforce the previously reported delays and difficulties in diagnosing cervical cord injury in abused[2,5] and unintentionally injured[3] children. These children often have concurrent brain injury, which may require sedation, paralysis, and ventilation for treatment, thereby masking limb paralysis. Without careful examinations, symptoms and signs of cord dysfunction may be missed or attributed to the coexistent brain injuries.[2,5] Complicating this, serial examinations are required because cord dysfunction may develop subsequent to brain injury presentation.[1] The children may be too unstable to obtain technically adequate films of the bony spine, and SCIWORA may occur in the absence of bony changes.[1,6,7] Because children with incomplete cord injuries have a better prognosis for functional recovery than adults,[13,24] early recognition of spinal injury and instability is important to stabilize the spine and limit further cord damage

Harborview Medical Center (HMC) is the only level 1 pediatric trauma center in the region, and the Children's Hospital and Regional Medical Center (CHRMC) is the regional tertiary care pediatric facility. Although this series was not collected prospectively, the cases are memorable, and the authors, through their work at both the CHRMC and the HMC, are very likely to have been involved in the care of all children with spinal injury and suspicion of abuse. In our judgment, the 3 internal cases are likely to represent full ascertainment. The denominator of children with inflicted head injury is less certain. During a formal prospective ascertainment period of subdural hemorrhages from March 1995 to December 1998, slightly more than 1 child a month with inflicted head injury was identified at CHRMC and HMC (39/33 months).[25] During the 36-month period from January 2003 to December 2006, 31 inflicted head injury victims were identified at CHRMC. If the same, roughly 1 per month, rate for inflicted head injury was constant during the entire 23 years of this case series, approximately 1% of inflicted head injury victims had clinically evident cervical spinal cord injury. This estimate is obviously crude because of the lack of formal prospective data.

Some writers[26,27] have opined that infants cannot be shaken hard enough to cause intracranial and brain injury. A biomechanical argument has been made that if shaking could cause intracranial injury, cervical spine injury should universally accompany it.[28] However, that study's math was incorrect.[29] Extensive clinical experiences, such as the current case series, indicate that clinically significant cervical cord injury, coincident with indisputable inflicted head injury, occurs but is exceedingly rare. This is in contrast to the frequent and clinically unapparent but pathologically evident cord and cervicomedullary injury in fatal cases.[16,21,22] Two of our patients had MRI evidence of continuous nervous tissue injury from the medulla to the lower cervical cord and another sustained injury to the C1-2 articulation, affirming the significance of the craniospinal junction in these injuries. One of our 5 cases had no evidence of cranial impact (case 3), and 1 only had facial bruising as a sign of cranial impact (case 4). The assailant of the child who lacked evidence of impact confessed to shaking her.

TABLE 1. Attributes of 5 Children With Symptomatic Cervical Spinal Cord Injury Secondary to Child Abuse

Case	Age	Sex	Site/Type of Care	History	Spinal Injury	Diagnosis When/By What Means	Head Injury	Associated Injuries	Retinal Bleeding	Clinical Outcome
1	2 mo	Female	Study hospital	Fell off father's chest from the couch. Fussy, decreased tone, grunting respiration.	Posterior bulge C4 body, central cord hemorrhage C3-4. "central cord syndrome"	Day 4—loss of arm function recognized. Day 5—MRI.	Posterior medulla hemorrhage, acute R parietal, older L parietal skull Fxs	7 ribs, distal radius/ulna Fxs. CMLs L tibia, both femurs, both acromia, both proximal humeri, 3 L metatarsals.	No	Clinically well at 3 mo after injury
2	4 mo	Female	Study hospital	Fever, respiratory distress, flaccidity. No history of trauma. Father pled guilty—no admitted event.	C7 cord transection, arachnoid and dural tear, anterior longitudinal ligament avulsion, extradural cervical mass.	Day 5—sensorium improved but flaccid. CT widened C5-6. 1 mo after presentation—myelogram.	SDH R parietal, skull Fx	Bruises, burns, 19 rib Fxs. CMLs both distal femurs, both proximal and both distal tibias.	No	Ongoing quadriplegia
3	6 mo	Female	Study hospital	Decreased arousal and respiration. Father later confessed shaking.	C1-7 central cord hemorrhage "central cord syndrome"	Day 1—lack of arm function. Day 2—MRI.	SAH, medulla hemorrhage. R parietal Fx	Scar upper frenulum, tongue bite, 7 acute and old rib Fxs. Proximal L tibia Fx.	No	At age of 5 yrs, bladder dysfunction, bilateral weakness and spasticity
4 (prior report)[3]	3 1/2 wk	Female	Legal. Later neuro-surgical follow-up at study hospital	Fell off the couch. Later, father said shook to rouse her. Limp and cyanotic. Remained ventilated and sedated.	Dens Fx and osteomeyelitis C1-2. Compressed upper cervical cord.	2 wks—paralysis was recognized. Day 15—MRI after hospital arrival.	2 R SDHs, biparietal infarcts	8 acute rib Fxs. Abdomen bruise. Hepatic laceration. Staphylococcal sepsis with L hip osteomyelitis and endocarditis	No	Quadriplegia
5 (prior report)[12] (Fig. 3)	15 mo	Female	Legal	Fell off the couch onto a popcorn can. Later flaccid. Boyfriend convicted—no admission.	Briefly delayed paralysis. Swollen midcervical cord with hemorrhage.	Day 1—paralysis. Day 1—MRI.	Diffused edema, eventual cystic brain loss.	Lip and tongue bleeding. Head bruises. Face scald. Fx clavicle.	No	Vegetative quadriplegia. Died at 5 yrs.

CML indicates classic metaphyseal lesion; Fx, fracture; L, left; R, right; RH, retinal hemorrhage; SAH, subarachnoid hemorrhage; SDH, subdural bleeding.

However, shaking was also confessed in 1 child who had a skull fracture as evidence of cranial impact.

The lack of retinal hemorrhages in these cases contrasts with the usual 70% to 80% frequency of retinal hemorrhages found with inflicted head injury.[30] This could imply a difference in the mechanics of these injuries. It has been suggested that some children with cervical cord injury may have been shaken while grasped by their head[31] instead of by their chest or upper extremities.[21]

Early diagnosis of cervical spine and cord injuries is critical to optimal treatment and outcome. Evaluation with MRI is essential to evaluate spinal cord and ligamentous injury. Spinal stabilization may be required for 3 to 6 months. Rehabilitation medicine follow-up is essential for children with neurological injury.

CONCLUSIONS

Clinically evident cervical spinal cord injury is infrequent in children with inflicted head injury. Because of this rarity and presentations that may be subtle, masked by coexistent injuries or supportive treatments, or accompanied by delayed symptom onset, diagnosis may be difficult. Rigorous serial examinations, supplemented by appropriate imaging, are necessary for early diagnosis and management.

REFERENCES

1. Pang D, Wilberger JE. Spinal cord injury without radiologic abnormalities in children. *J Neurosurg*. 1982;57:114–129.
2. Sneed RC, Stover SL. Undiagnosed spinal cord injuries in brain-injured children. *Am J Dis Child*. 1988;142:965–967.
3. Piatt JH Jr, Steinberg M. Isolated spinal cord injury as a presentation of child abuse. *Pediatrics*. 1995;96:780–782.
4. Chen LS, Blaw ME. Acute central cord syndrome caused by minor trauma. *J Pediatr*. 1986;108:96–97.
5. Orenstein JB, Klein BL, Ochsenschlager DW. Delayed diagnosis of pediatric cervical spine injury. *Pediatrics*. 1992;89:1185–1188.
6. Kriss V, Kriss TC. SCIWORA (Spinal cord injury without radiologic abnormality) in infants and children. *Clin Pediatr*. 1996;35:119–124.
7. Grabb PA, Pang D. Magnetic resonance imaging in the evaluation of spinal cord injury without radiologic abnormality in children. *Neurosurgery*. 1994;35:406–414.
8. Brown RL, Brunn MA, Garcia V. Cervical spine injuries in children: a review of 103 patients treated consecutively at a level 1 pediatric trauma center. *J Pediatr Surg*. 2001;36:1107–1114.
9. Cirak B, Ziegfeld S, Knight VM, et al. Spinal injuries in children. *J Pediatr Surg*. 2004;39:607–612.
10. Osenbach RK, Menezes AN. Pediatric spinal cord and vertebral column injury. *Neurosurgery*. 1992;30:385–390.
11. Jenny C, Hymel KP, Ritzen A, et al. Analysis of missed cases of abusive head trauma. *JAMA*. 1999;281:621–626.
12. Ghatan S, Ellenbogen RG. Pediatric spine and spinal cord injury after inflicted trauma. *Neurosurg Clin N Am*. 2002;13:227–233.
13. Hamilton MG, Myles ST. Pediatric spinal injury: a review of 174 hospital admissions. *J Neurosurg*. 1992;77:700–704.
14. Ruge JR, Sinson GP, McLone DG, et al. Pediatric spinal injury: the very young. *J Neurosurg*. 1988;68:25–30.
15. Keiper MD, Zimmerman RA, Bilaniuk LT. MRI assessment of the supportive soft tissues of the cervical spine in acute trauma in children. *Neuroradiology*. 1998;40:359–363.
16. Khanna G, El-Khoury GY. Imaging of cervical spine injuries of childhood. *Skeletal Radiol*. 2007;36:477–494.
17. Feldman KW, Weinberger E, Milstein JM, et al. Cervical spine MIR in abused infants. *Child Abuse Negl*. 1997;21:199–205.
18. Swischuk LE. Spine and spinal cord trauma in the battered child syndrome. *Radiology*. 1969;92:733–738.
19. Merten DF, Radkowski MA, Leonidis JC. The abused child: a radiological reappraisal. *Radiology*. 1983;14:377–381.
20. Kleinman PC. Spinal trauma. In: Kleinman PK, ed. *Diagnostic Imaging of Child Abuse*. 2nd ed. St Louis, MO: Mosby; 1998: 149–167.
21. Hadley MN, Sonntag VKH, Rekate HL, et al. The infant whiplash-shake injury syndrome: a clinical and pathological study. *Neurosurgery*. 1989;24:536–540.
22. Geddes JF, Hackshaw AK, Vowles GH, et al. Neuropathology of inflicted head injury in children I. Patterns of brain damage. *Brain*. 2001;124:1290–1298.
23. Kemp AM, Stoodley N, Cobley C, et al. Apnoea and brain swelling in non-accidental head injury. *Arch Dis Child*. 2003;88:472–476.
24. Hadley MN, Zabramski JM, Browner CM, et al. Pediatric spinal trauma: review of 122 cases of spinal cord and vertebral column injuries. *J Neurosurg*. 1988;68:18–24.
25. Feldman KW, Bethel R, Shugerman RP, et al. The cause of infant and toddler subdural hemorrhage: a prospective study. *Pediatrics*. 2001; 108:636–646.
26. Leestma JE. "Shaken baby syndrome": do confessions by alleged perpetrators validate the concept? *J Am Phys Surg*. 2006;11:14–21.
27. Miller M, Leestha J, Barnes P, et al. A sojourn in the abyss: hypothesis, theory, and established truth in infant head injury. *Pediatrics*. 2004;114:326.
28. Bandak FA. Shaken baby syndrome: a biomechanical analysis of injury mechanisms. *Forensic Sci Int*. 2005;151:71–79.
29. Margulies S, Prange M, Myers BS, et al. *Forensic Sci Int*. 2006; 164:278–279.
30. Levin AV. Retinal hemorrhages and child abuse. In: David TJ, ed. *Recent Advances in Pediatrics*. London: Churchill Livingstone; 2000: 151–219.
31. Case MES, Graham MA, Wood JE. Spinal cord injury in abuse by shaking. Second National Conference on Shaken Baby Syndrome; September 1998; Salt Lake City, Utah.

