Spinal Injuries in Children: Role of MR

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PURPOSE: To define the clinical and prognostic role of MR in a pediatric population with spinal cord injury. METHODS: Fifteen children underwent MR 12 hours to 2 months postinjury using decoupled surface coils and ventilator support as needed. MR was correlated retrospectively with clinical, CT, and radiographic findings. RESULTS: On MR, of seven children with spinal cord neurologic deficits, four had hemorrhagic contusions, one had nonhemorrhagic contusion, one had extensive infarction, and one revealed a normal cord. All had persistent deficits on hospital discharge. Eight without cord neurologic deficit revealed no cord lesions on MR; this group included two with epidural hematoma, four with ligamentous disruption, and two with bone compression. CONCLUSIONS: Children may have extensive cord contusion and/or infarction with minor, remote, or no fracture dislocation. Because both hemorrhagic and nonhemorrhagic cord lesions found on MR were associated with significant, persistent cord deficits, the authors conclude that MR provides a practical tool for diagnosis/prognosis in children with acute/subacute spinal injury.

Index terms: Spinal cord, injuries; Spinal cord, magnetic resonance; Magnetic resonance, in infants and children; Pediatric neuroradiology

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In adults, magnetic resonance (MR) has proved useful for evaluation of the acutely injured spinal cord (1–8), and for late sequelae of spinal cord injury such as posttraumatic syringomyelia and myelomalacia (9, 10). Traumatic spinal injuries in children differ in clinical and radiographic findings and in prognosis from those in adults (11–15). Few series describe MR patterns of injuries encountered in children with spinal injury (13–15). In this paper, we describe our MR techniques for evaluating the child with spinal injury, and correlate MR results with the initial presentation, acute neurologic findings, and outcome in 15 children studied following spinal injury.

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Materials and Methods

A retrospective review of all pediatric patients referred for MR evaluation of acute/subacute spinal injury at our institution was completed, resulting in a group of 15 patients. The 15 patients were under 18 years of age and were studied from October 1988 to August 1991. The group included five girls and 10 boys ranging in age from 12 hours to 17 years. Mechanism of injury, neurologic deficits, radiographic findings, and MR findings obtained by chart and image review are described in Table 1. Bone stabilization was achieved by operative procedures in children with spinal instability. None of the children died as a result of these injuries. Imaging findings (MR, plain films, and computed tomography (CT)) were described by consensus of two experienced neuroradiologists. Cord abnormalities were categorized based on T1- and T2-weighted images as hemorrhagic contusion, nonhemorrhagic contusion, infarction, or normal. Other ligamentous, bony, and paraspinal abnormalities were noted. No unsuccessful studies were identified, although referral bias may have excluded children with insignificant injuries, those with injuries that were well explained by plain film or CT findings, and those who were extremely ill or too unstable for MR examination.

MR was performed at 0.5 (n = 2) or 1.5 (n = 13) T using a variety of circular and rectangular surface coils chosen based on the level(s) of suspected injury and the patient's physical size. The entire spinal cord was evaluated in children with clinical evidence of spinal cord injury; additionally, all sites of suspected or proved bone injury

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TABLE 1: Imaging and clinical findings in 15 children with spinal injuries

Major MR Findings	MR Interval (days)	Spinal CT and Plain Film Findings	Age Sex	Event	Initial Deficit	Neurologic Outcome (Interval of Follow-up)
Hemorrhagic cord contusion						
 Medulla-upper thoracic contusion/infarction 	5	None	0.5 days F	Birth injury	Quadriplegia; no respi- ratory effort	Ventilator dependent, quadri- plegia (C5), (1.25 years)
Cervico-medullary hemorrhage	60	None	2 months M	? Birth injury	Palatal pharyngeal in- coordination and apnea	Stable with persistent episodic apnea (2 months)
Cord hemorrhagic contusions, C6–T6	5	None	6 years M	Pedestrian-MVA	Paraplegia (T2) CHI	Paraplegia (T2), mild right up- per extremity hyperflexia (2.6 years)
4. Conus contusion 2.5 levels above bone injury	<6	L3-L4 fracture/dislo- cation	4 years F	MVA	Paraplegia (T12); CHI	Paraplegia, no voluntary lower extremity motion (1 year)
Nonhemorrhagic cord contu- sion and infarction						
5. Cord contusion at T11 (nonhemorrhagic)	6	L4 transverse pedicle fracture, pseu- domeningocele	5 years M	MVA	Paraplegia, CHI	Paraplegia (L1) (3 months)
Cord infarction >9 seg- ments (thoracic)	1	None	4 years M	MVA	Paraplegia, CHI	Paraplegia (T8) (1.25 years)
Normal spinal cord 7. Normal cord	1	Type II odontoid fracture, 2-mm anterolisthesis	15 years M	MVA	Right body hemiparesis	Minor right arm weakness, markedly improved (6 months)
Normal conus, small epidural hemorrhage	3	L3–L4 Change frac- ture	5 years M	MVA, seat belt	Back pain, no deficit	Normal (1.25 years)
Normal cord with dis- rupted posterior liga- ments	<1	T12 fracture with re- tropulsion and posterior dias- tasis	8 years F	Fall into ditch	Back pain, no deficit	No deficit, slight ankle clonus (1.5 years)
Normal cord; C5–C6 posterior diastasis	60	C5–C6 posterior ele- ment diastasis	16 years M	Wrestling injury	Neck and right arm pain	No deficits, persistent shoul- der and right arm pain (6 months)
11. Normal cervical cord	2	C2–C3 pseudosub- luxation	5 years M	MVA	CHI	Stable (7 months)
12. Normal cervical cord	49	None	14 years M	MVA	Right brachial plexus injury	Persistent right triceps weak- ness (5 months)
13. Normal cervical cord	60	None	16 years M	Fall from bleacher	Radiculopathy	NA
14. Normal cervical cord	60	None	17 years F	MVA	Radiculopathy	NA
Lumbosacral epidural disk and hematoma	11	L5 fracture and bul- let fragment	1 year F	Gunshot	None	No deficit (8 weeks)

Note.—MVA = motor vehicle accident; CHI = closed head injury; NA = not available.

were studied. All examinations were performed using either surface coils or an open-ended head coil (infants), and included multisection T1-weighted images in a sagittal plane (469-666/20-30/2-4) (TR/TE/excitations). Sagittal T2-weighted images were completed using 3- to 5-mm section thickness (1700-2500/90-100/1-2 or gradient echo 200/21, flip angle 40°; or 308/6, flip angle 7°). The preferred plane of imaging was sagittal because it allowed for rapid study of long segments of the spinal column; additional pulse sequences and planes of imaging were chosen based on the sagittal image findings and the suspected levels of injury. These were completed only in clinically stable patients. Our MR system permits simultaneous

placement of several decoupled spinal surface coils within the magnet without coil or image interference. Thus, multiple spinal segments or the entire spinal column were imaged using a variety of surface coils without repositioning of the surface coils or the child, and without removing the child from the magnet.

Early during this study, patient monitoring was limited to electrocardiogram, chest wall motion, and direct observation. Improved MR monitoring for more recent studies included continuous pulse oximetry and end-tidal CO_2 evaluation. Ventilator-dependent patients were studied early using MR-compatible equipment, long tubing, and manual ventilation by a respiratory therapy, pediatric in-

tensive care, and/or pediatric anesthesiology team. Currently, an MR-compatible ventilator (Ohmeda Excel 210, BOC Health Care) is used for ventilator-dependent patients.

Results

Spinal cord injuries were suspected based on clinical deficits in seven patients; eight had spinal column injuries or neurologic symptoms without demonstrable spinal cord pathology on MR (Table 1).

Hemorrhagic Contusion

Four children with spinal cord neurologic deficits had MR findings of intramedullary hemorrhage. Nonhemorrhagic foci of infarction and/or edema were present adjacent to the hematomyelia in three of the four children with acute injuries. The fourth child had a subacute injury (2 months), and had evidence of previous hemorrhage only. All had significant persistent cord deficits on follow-up examination 6 weeks to 1 year after injury. Two were infants with spinal cord deficits which dated from birth (cases 1 and 2); neither child had a bone or ligamentous abnormality. One infant with absent respiratory effort and quadriplegia underwent MR with ventilatory support at 12 hours of age. MR demonstrated marked acute spinal cord hemorrhage and edema/infarction, with marked cord atrophy on follow-up (case 1, Fig. 1). A second infant had a small hypointense lesion on both T1- and T2-weighted images at the cervicomedullary junction (case 2, Fig. 2) compatible with a birth-related hemorrhage. A vascular malformation was not demonstrated by conventional angiography or flow-sensitive gradient-echo MR.

Hemorrhagic cord contusions were identified in two older children. One 6-year-old child (case 3) was paraplegic following pedestrian-motor vehicle accident, without radiographic evidence of bone or ligamentous spinal abnormalities (SCI-WORA). Extensive hemorrhagic spinal cord contusion with associated edema involved at least nine vertebral segments; a central localized area of profound signal loss over three spinal segments correlated with intracord hemorrhage, as described in previous reports (1–4, 7, 16, 17) (Fig. Although his acute injury was described as a paraplegia, follow-up neurologic examination also revealed a mild upper extremity hyperreflexia. A 4-year-old child with a seat belt-type fracture at L3-L4 had neurologic deficits that localized clinically to a T12 cord injury. MR revealed a thoracic

cord hemorrhagic contusion from vertebral level T8 to T11 (case 4).

Nonhemorrhagic Cord Contusion and Infarction

Two children had nonhemorrhagic cord lesions and spinal cord deficits. One 5-year-old child with acute paraplegia sustained multiple injuries including an L4 fracture and thecal sac rent (case 5, Fig. 4). MR revealed a focal conus/lower thoracic spinal cord area of high signal intensity on T2-weighted image; no adjacent ligamentous or bone abnormality was apparent on plain films. Myelography confirmed a posttraumatic pseudomeningocele that was difficult to differentiate on MR from posterior interspinous and intraspinous ligament disruption without dural tear. CT and plain films demonstrated transverse fractures bilaterally through the L4 pedicles and lamina with no radiographically detectable vertebral body fracture. The L4 vertebral body had abnormally high signal on T2-weighted image, suggesting vertebral body injury below the detectable threshold of CT. The second child, a 3½-year-old unrestrained passenger, sustained multiple injuries and presented acutely with paraplegia with no spinal fracture on plain films. MR demonstrated extensive high signal intensity without hemorrhage on T2-weighted image from the T4-T5 disk space to the conus; this correlated clinically with an extensive spinal cord infarction. His spinal cord neurologic level of T8 corresponded anatomically with the MR abnormalities extending cephalad to about the T5 vertebral body level. Aortography was normal, with no evidence of aortic laceration or dissection (case 6, Fig. 5).

Normal Spinal Cord

A 15-year-old with a pure right motor hemiparesis following a type II odontoid fracture had a normal spinal cord on two sequential MR studies despite high-resolution techniques (3-mm images, 1.5 T, sagittal T1, and T2-weighted image, axial gradient-echo images, field of view 230 to 280 mm, case 7). His persistent deficits excluded the diagnosis of spinal cord concussion, although he eventually had a good neurologic recovery (18). With the exception of this case, the other cord-injured patients with minimal or no bone compromise were children less than 7 years old.

Three patients had bone and/or ligamentous spinal column injuries without clinical or radiographic evidence of spinal cord injury (cases 8–

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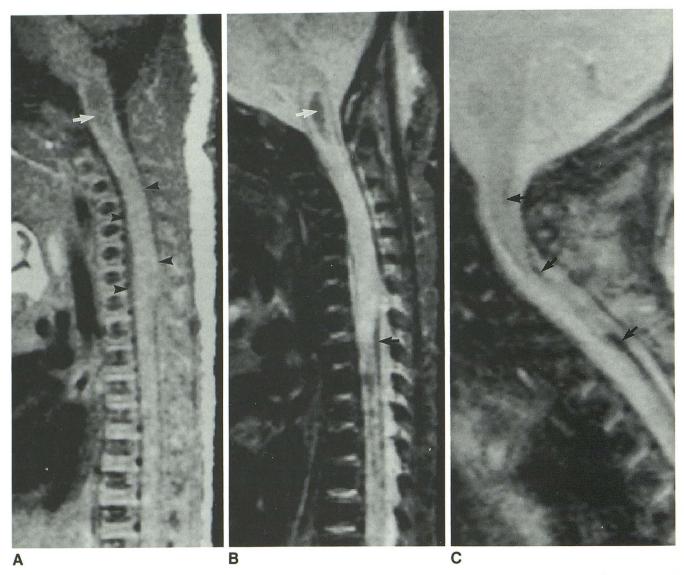


Fig. 1. A, The cervical spinal cord and medulla are enlarged (*arrowheads*) in this 12-hour-old infant with no spontaneous respiratory effort and quadriplegia following a difficult breech delivery (case 1A; 602/20). At 0.5 T, the acute hemorrhage into the medulla (*arrow*) results in a subtle central area of hypointensity. The enlarged cervical cord fills the entire vertebral canal. Note the hypointense vertebra of this newborn infant.

B, The focal area of hypointensity within the medulla and upper cervical spinal cord is confirmed, compatible with a 12-hour-old hemorrhage (white arrow). The remainder of the cervical spinal cord is hyperintense and enlarged to the level of the cervicothoracic junction (2500/90). They hypointensity of the dorsal and ventral surfaces of the lower spinal cord is of uncertain etiology (black arrow). Possibilities include cerebrospinal fluid flow accentuated due to the marked spinal cord enlargement or areas of peripheral preserved spinal cord with central cord edema or infarction.

C, Follow-up MR 1 month later demonstrates marked spinal cord atrophy and small intramedullary foci of hypointensity in the medulla, cervical, and thoracic spinal cord (*arrows*). These hypointense foci may represent gliosis, residual hemosiderin from intramedullary hemorrhage that was too acute or petechial in nature to recognize on the initial MR, or sequelae of hemorrhage that progressed in severity and extent following the initial MR (2000/90).

10). Another child with closed head injury was initially thought to have a significant traumatic subluxation at C2-C3, confidently diagnosed as pseudosubluxation after a normal MR study and supervised flexion and extension plain films (case 11). One patient with a left hemiparesis resulting from closed head injury underwent cervical MR

because of an unexplained right brachial plexus injury (case 12). While no MR abnormality was identified, a posttraumatic nerve root avulsion was not confidently excluded on the basis of MR. Two adolescents with radicular pain without deficit suggestive of nerve root compression had normal MR studies (cases 13 and 14). No trau-



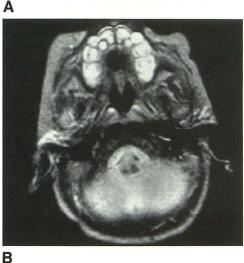


Fig. 2. A, A small focus of hypointensity on T1-weighted image (arrow) is seen at the cervicomedullary junction in this 2-month-old infant with palatal and pharyngeal incoordination dating from birth (case 2, 694/29).

B, The hypointense focus in the dorsal medulla/cervical cord compatible with old hemorrhage is confirmed on T2-weighted image (2200/90).

matic disk herniation was apparent, although nerve root avulsion could not be confidently excluded based on MR.

MR was performed in a 1-year-old child with a "through and through" abdominal penetrating injury (gunshot wound). It demonstrated a mixed high- and isointense extradural soft-tissue collection in the ventral spinal canal at the injured disk space (T1-weighted image), suggesting a combination of extradural disk fragment and hematoma (case 15, Fig. 6). Bone and metallic fragments in the same area were not well demonstrated on

MR. Following an abdominal exploratory laparotomy, cerebrospinal fluid drainage from the dorsal exit wound required surgical exploration and closure.

Discussion

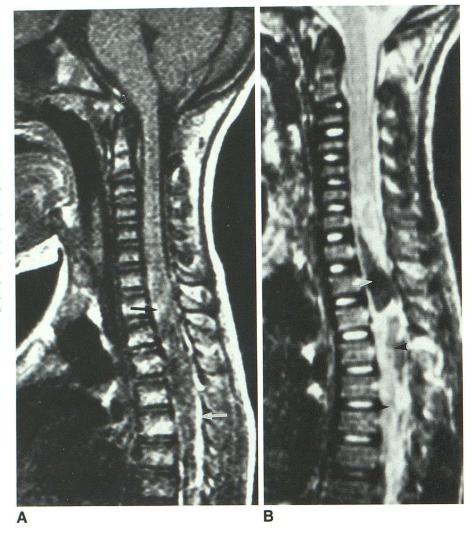
Spinal injury patterns in children are significantly different in level of injury, type of injury, and possibly in outcome compared with those in adults. Younger children are more subject to high cervical spine and cord injuries (occiput to C3) than older children and adults (11-13, 19). Children with spinal injury are reported to have greater susceptibility to delayed neurologic deficits and more frequent complete neurologic deficits than adults (12). Spinal cord injury without radiographic abnormality demonstrable by plain film and/or myelography (SCIWORA) is generally confined to children, and is accompanied by significant neurologic deficits (12-13, 19). Lap belt injuries in children involve spinal, bowel, and bladder injuries because of the greater flexibility of the young spine and frequency of improper lap belt positioning in young children (20, 21).

Reasons for these differences include less mineralization of the child's vertebral column, greater flexibility of the nonbone musculoskeletal structures, poorly developed musculature, and the greater proportion by weight of the cranium relative to the rest of the body (11-13, 19, 22-24). The mechanisms of spinal injury in children differ from those of adults, and include a greater percentage of falls, pedestrian-motor vehicle accidents, sports-related events, or difficult delivery (11-13, 19, 22-25). Recognition of the level and type of injury, exclusion of surgically treatable spinal cord or root compression, and prediction of neurologic recovery are all important goals of MR imaging in the spinal-injured child. The transition from the spinal injury patterns of childhood to those of adults clinically occurs at about 8 to 12 years of age, corresponding with the progressive mineralization and habitus changes of late childhood.

MR of the potentially unstable child necessitates a suite equipped with MR-compatible monitoring devices, oxygen, respiratory support equipment, and personnel experienced in the care of sick pediatric patients. With appropriate personnel and equipment, critically ill and/or ventilator-dependent children can undergo MR, and spinal alignment in patients with potential or actual spinal instability can be maintained (4, 6).

Fig. 3. A, MR was performed 5 days after a motor vehicle accident in a child (case 3) with no spinal injury on plain films. The T1-weighted image demonstrates marked lower cervical and thoracic cord enlargement with small foci of intra- and extramedullary hyperintensity (arrows, 363/30/4).

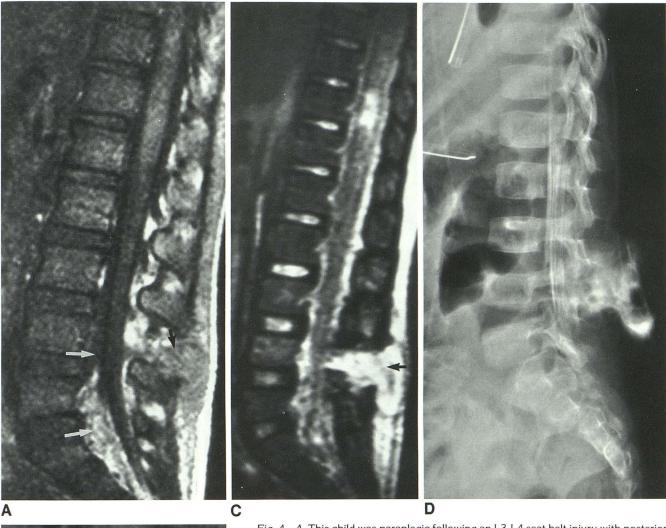
B, A T2-weighted image (2000/100/ 2) reveals multiple foci of marked hypointensity; the largest of these spanned the cord from C7 to T3, and correlated with clinical paraplegia associated with intracord hemorrhage and contusion (arrow). The complex foci more caudally (arrowheads) probably represent multicompartmental hemorrhage (intramedullary, subarachnoid, subdural, or epidural) and/or flow (cerebrospinal fluid or enlarged epidural vessels below the level of cord enlargement). Surgical decompression was deemed of little benefit because of the extensive hematomyelia and clinical deficit.



The ability to evaluate the entire spinal column without changing surface coils or patient positioning is essential for the requisite speed of examination and image resolution. The need to visualize multiple segments is emphasized by the two children in our series with cord lesions remote from the bone injury seen on plain films. In this study, we used MR technology which permitted simultaneous positioning of multiple surface coils without interference between the coils themselves. An alternative approach might be use of a segmented spine coil, although the commercially available segmented coils are generally sized for the adult spinal column and are not optimal for use in the pediatric patient. In our experience, large field-of-view images obtained using a body coil have not reliably or adequately demonstrated subtle intramedullary pathology. Preliminary reports suggest that phased array surface coil technology may be an alternative

technique for obtaining rapid, high-resolution images of the entire spinal column (26).

In the acute setting, MR offers a noninvasive technique for exclusion of significant cord compression requiring surgical decompression. In this series, no patient underwent emergency neurosurgical intervention for decompression; however, this possibility has often been the cause for emergency imaging procedures. Our study suggests that, although acute traumatic herniated nucleus pulposus is a frequent finding in adults with spinal trauma, this is not a common sequelae of closed spinal injury in a pediatric population. Our results also confirm the paucity of spinal cord injury with no or minimal spinal column injury (ie, SCIWORA) in older children and adolescents, and support the clinical and radiographic descriptions of a transition in late childhood to early teen years to an adult-type pattern of spinal injury.



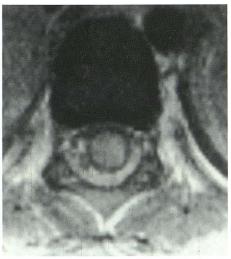


Fig. 4. A, This child was paraplegic following an L3-L4 seat belt injury with posterior element diastasis and fractures (case 5). On T1-weighted image, a focal signal abnormality is visible in the posterior soft tissues at L3-L4 (*black arrow*) (469/30). The ventral spinal canal has a complex signal intensity below this lesion, suggesting subarachnoid hemorrhage and an epidural collection of hemorrhage, fat, and extravasated cerebrospinal fluid (*white arrows*).

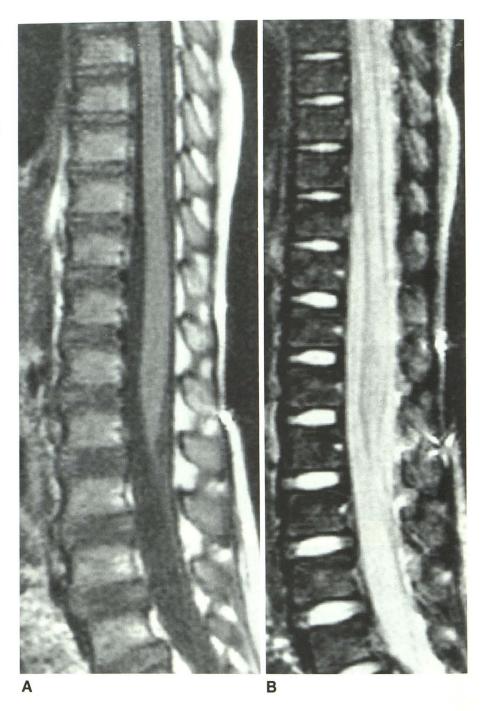
 $\it B$, A transverse T1-weighted image at T11 demonstrates subtle cord enlargement (575/30). The circumferential higher signal intensity of the extramedullary spaces was not confirmed on sagittal images and probably represents entry flow phenomenon.

C, A hyperintense focus is apparent on T2-weighted image within the distal thoracic spinal cord. Note that this cord lesion is many levels remote from the site of bone injury (1800/100). The L4 vertebral body is hyperintense, although no vertebral body fracture could be identified on plain films or CT. A complex high signal intensity traumatic pseudomeningocele presumably containing a mixture of blood and cerebrospinal fluid is visible in the soft tissues posteriorly at the L3-L4 diastasis (*arrow*), and the complex ventral hemorrhagic and CSF collections are again seen below L4.

D, Myelography demonstrates marked extravasation posteriorly into a pseudomeningocele at L3-L4. The transverse pedicle fracture of L4 was visible only in retrospect on MR. The cord contusion was not well demonstrated myelographically in part because of rapid egress of contrast into the pseudomeningocele. Even with better myelographic contrast it could have been overlooked because of the \$\frac{1}{2}\$bsence of significant cord enlargement.

Fig. 5. A, A T1-weighted image in this acutely paraplegic child with chest and closed head injuries (case 6) is unrevealing (498/20).

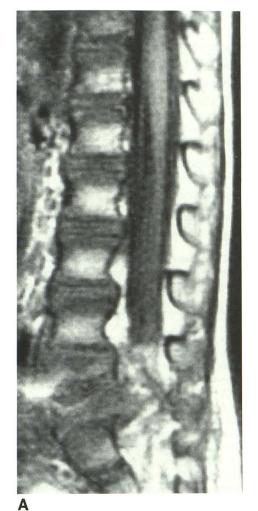
B, A T2-weighted image reveals diffuse hyperintensity of the thoracic spinal cord without cord enlargement or hemorrhage. This correlated clinically with extensive spinal cord infarction (2000/90).



MR demonstration of the presence, extent, and pattern of spinal cord injury offers diagnostic and potentially prognostic information not readily obtainable by other means. Better characterization of acute cord injuries may influence therapeutic management as newer medical and pharmacologic interventions are evaluated (27, 28). The correlations between spinal cord level and extent of abnormality on MR and level of clinical deficit proved imprecise, in part related to the difficulty in assigning exact clinical levels of injury and

difficulty in differentiation of edema, nonhemorrhagic contusion, and infarction. In this series, cord abnormalities in children who previously would have been classified as having SCIWORA based on plain films and/or myelography were demonstrated on MR. MR also provided direct visualization of cord injuries remote from sites of bone injury in children with otherwise unexplained spinal cord deficits.

In a series composed largely of adults, Kulkarni and colleagues described three patterns of MR







signal abnormalities that correlated well with recovery or lack of resolution of neurologic deficits at 1 year after injury (3, 4). According to this classification, a type 1 injury (inhomogeneous on T1-weighted image, large central hypointensity with thin hyperintense rim on T2-weighted image) has the poorest prognosis for partial or complete recovery. Type II injuries (normal T1-weighted image, hyperintense cord on T2-weighted image) were intermediate in severity, and type III injuries (normal T1-weighted image, isointense center with thick hyperintense rim on T2-weighted image) have a good prognosis for significant recovery or resolution of deficits (3, 4).

In this series, the four children with hemorrhagic contusions had MR patterns that best fit a Kulkarni type I injury. All had significant deficits that persisted and generally required rehabilitation upon discharge. Two were sequelae of birth injuries; little or no return of neurologic function was documented in these children during hospitalization and early follow-up. Indeed many children with extensive cord injury at birth die as a result of this lesion (14, 29, 30). While this poor recovery of neurologic function correlates reasonably well with the Kulkarni study, longer followup is required before this can be stated conclusively.

Two children had MR findings that best fit a Kulkarni type II pattern. One clinically had an extensive spinal cord infarction with persistent paraplegia on follow-up. Spinal cord infarction has been described with minor or major trauma in children, and is one of the proposed pathologic entities underlying SCIWORA and birth-related cord injuries (29-32). Although the signal intensity pattern is similar to that of a nonhemorrhagic contusion, our experience and the reported severe neurologic deficits with SCIWORA suggest that these extensive injuries may not have as favorable a prognosis for neurologic recovery as those described by Kulkarni with a more focal nonhemorrhagic contusion. Tator et al suggest that vascular spinal cord injury/infarction in association with systemic hypotension and dimin-

Fig. 6. A, Marked bone and soft-tissue injury obscures the lower thecal sac in this young child with a gunshot injury (case 15, 498/20). The hyperintense lesion ventral to L4 represents small residual metallic bullet fragments on CT.

B and *C*, Axial images at L5 (640/20) demonstrate central and leftward hyperintense hematoma and presumed disk fragments (this material was isointense to disk on all sequences) (*B*, *arrow*). The path of the bullet posteriorly through the left L5 lamina is recognizable (*C*, *arrow*).

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ished cardiac output may exacerbate acute spinal trauma, and the midthoracic spinal cord is particularly at risk with hypotension because of its relatively poor blood supply (33).

No Kulkarni type III MR patterns of injury were identified in this series. One child with a persistent pure motor hemiparesis with facial sparing had no convincing cord abnormality on MR. He eventually regained significant neurologic function. This suggests that significant cord injuries with minimal or no MR findings carry a better prognosis for recovery of neurologic function than those with MR abnormalities.

Seat belt injuries or chance fractures deserve special mention (34). In children these injuries occur at a lower spinal level more often than in adults (20, 21, 35). Detection may be delayed due to associated abdominal or head injuries (20, 21). In this series, three children had seat belt injuries at L3 or L4, not at the thoracolumbar junction as might be expected based on adult studies. These injuries may be subtle on plain films and CT, and the posterior element fractures associated with this injury are readily overlooked on MR (20, 21). Two of three children with this type of injury in our series had clinically significant lower spinal cord contusions and suffered persistent paraplegia as a result. In this group, MR proved particularly useful in explaining deficits that did not correlate with the level of bone injury.

In summary, our experience with MR for children with acute or subacute spinal trauma suggests that MR is a reasonable, practical imaging modality for demonstration of intramedullary lesions and extramedullary cord compression. We stress that spinal column stability should be assessed prior to the MR examination with conventional radiographic studies (ie, plain films, flexionextension films, and/or CT). With appropriate technology and support personnel, pediatric patients with spinal instability and those requiring ventilatory assistance can successfully undergo MR. MR uniquely provides direct visualization of the level and pattern of cord abnormality in children who otherwise would be classified as SCIWORA, and in those whose deficits are not explained by location or extent of bone injuries. Although longer follow-up is needed, our preliminary data suggest that children with traumatic hematomyelia, like adults, are likely to have persistent significant neurologic sequelae. The prognosis of nonhemorrhagic lesions may be variable. perhaps depending on the degree of spinal cord

infarction versus nonhemorrhagic contusion and edema as the underlying cause for MR signal abnormalities. The neurologic improvement in our one patient with a significant cord injury and normal MR suggests that this injury may have a favorable prognosis.

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