

Cervical Spine Fractures

Cervical spine injuries are rare in children and are often difficult to diagnosis because of inability to obtain a clear history and the difficulty of imaging the immature spine. Therefore, a high index of suspicion is necessary to avoid missing the diagnosis and incurring associated sequelae. Unlike in adults, neurologic injury may be present despite negative imaging studies. The patterns of injury in children older than 10 years are similar to those in adults, with a greater incidence of subaxial injuries than in younger children, in whom injuries occur between the occiput and C2. The majority of injuries do not result in neurologic injury, and nonoperative treatment is usually effective. Children with spinal cord injuries (SCI) should be managed by a multidisciplinary team and must be followed by the orthopaedic surgeon for spinal deformity.

ANATOMY

The development of the atlas and axis with their ossification centers was well studied by Bailey.⁷ Three ossification centers are present in the immature atlas: one for the anterior ring, which usually appears by age 1 year, and one each for the posterior neural arches. The connection between the anterior and posterior arches is composed of the neurocentral synchondroses, which fuse at 7 years of age and can be mistaken for fracture prior to that. The posterior arch most often closes by age 3 but can remain open or partially closed (Fig. 40-1).

The ossification centers of the axis include one for the body, one for each neural arch, and one for the dens (Fig. 40-2). Fusion of the dens to the neural arches and the anterior body occurs between 3 and 6 years of age. During fetal development, the dens is formed from two ossification centers, which fuse during the seventh month of gestation. An ossification center at the tip of the odontoid appears between ages 4 and 6 years and fuses to the remaining odontoid by 12 years. The lower cervical vertebrae follow a similar pattern of development, with ossification centers at the body and each neural arch that close by the third year and the neurocentral synchondroses fusing between the fourth and sixth years.

The blood supply to the odontoid is derived from the anterior and posterior ascending arteries, which branch from the vertebral arteries at the level of the third cervical vertebrae and coalesce in the midline.⁹⁵ Anastomoses between

the carotid artery and the ascending arteries occur near the apex of the odontoid process.

MECHANISM OF INJURY

Cervical spine fractures in children account for a small percentage of all cervical spine fractures. A 20-year review of all cervical spine fractures at the Henry Ford Hospital found that only 12 (1.9 percent) of 631 patients were less than 15 years old.⁵² Other reports confirm the rarity of these injuries in children.^{19,57,74} The majority of spine injuries in children occur in the cervical region.⁵² Anderson and Schutt reported that 39 percent of 156 cases of spine injuries in children were in the cervical spine.⁴

In contrast to what is seen in adults, most cervical spine injuries in children occur between the occiput and C2, owing to increased ligamentous laxity and hypermobility together with a relatively increased head size, which results in the fulcrum of injury being above C3.^{13,52,89,90,102} As the child gets older and takes on a more adult body habitus, the incidence of cervical spine injuries resembles the adult pattern.⁸³ In a large series of adults, atlas and axis injuries accounted for 16 percent of cervical spine injuries, compared to 70 percent of cervical spine injuries in young children.⁴⁹

The mechanism of injury depends on the age of the child. In the neonatal period, birth injury with resultant cervical spine injury is not fully defined; however, up to 25 percent of all breech deliveries are associated with SCI.^{1,2,16} Child abuse, which should always be suspected in the young child with a cervical spine injury, is most often due to violent shaking of the child and may not produce radiographic abnormalities.^{24,66,114} In this age group a careful clinical evaluation is important, since a significant number of these injuries are the so-called spinal cord injury without radiographic abnormality.* In older children cervical spine injuries are more often due to motor vehicle accidents, pedestrian-motor vehicle encounters, falls from heights, and athletic injuries.^{6,13,52} Motor vehicle accidents are the most common cause of cervical spine injury in adolescents old enough to drive.⁷⁴

DIAGNOSTIC FEATURES

Every child evaluated in the emergency room after a traumatic event should be questioned about the exact mecha-

* See references 28, 48, 85, 86, 106, 114, 116, 123, 128.

ATLAS (C1) OSSIFICATION CENTERS

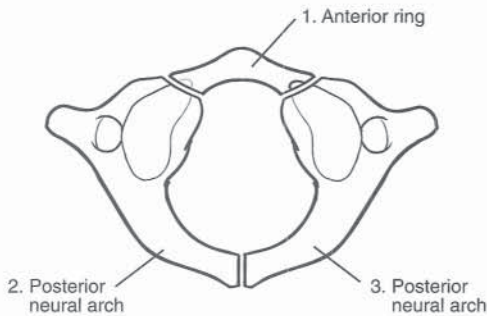


FIGURE 40-1 Ossification centers of the atlas. Note the neurocentral synchondrosis between the anterior ring and the posterior neural arches.

nism of injury and evaluated for injury to the cervical spine. Cervical spine injuries should be especially suspected in any child who arrives with facial abrasions or lacerations,⁷⁰ head trauma,⁹ clavicle fractures, a history of a high-speed motor vehicle accident, or a fall from a height. One of the most common presenting complaints when a cervical spine injury is present in an alert child is pain in the presence of torticollis.

The physical examination should include a full assessment, conducted from head to feet and including all organ systems. The appropriate trauma team should be present during the initial assessment. The head and face should be carefully inspected for lacerations and abrasions. The neck should be palpated to elicit tenderness, muscle guarding, or the presence of a gap in the spinous processes that would indicate a posterior ligamentous injury. A complete orthopaedic assessment of all four extremities together with the spine and pelvis should be performed. This should be followed by a complete neurologic examination, including a rectal examination, when neurologic injury is suspected.

The child who arrives in the emergency room unconscious is always considered to have a cervical spine injury. A cervical collar should be worn to stabilize the cervical spine until the patient is awake and can cooperate with the physical examination. When clonus is present in the extremities without decerebrate rigidity in a child with a closed head injury, a cervical spine injury should be strongly suspected.¹⁰⁴



FIGURE 40-3 Screening lateral radiograph of the cervical spine. The radiograph should show all seven cervical vertebrae, and should also include the C7-T1 level.

Radiographic evaluation should be performed when there is strong suspicion of a cervical spine injury. A review of 2,133 radiographs obtained in children less than 18 years old over a 7-year period disclosed cervical spine injury in only 1.2 percent of patients.⁹³ The two best predictors that cervical spine injury would be seen radiographically were involvement in a motor vehicle accident and complaints of neck pain. Although these two factors would have led to obtaining radiographs in all 25 patients who had cervical spine injuries, the authors did not recommend these predictors as the only indications for radiography. In the unstable patient a screening lateral radiograph of the cervical spine obtained in the emergency room is necessary to document obvious pathology (Fig. 40-3). This should be viewed as an

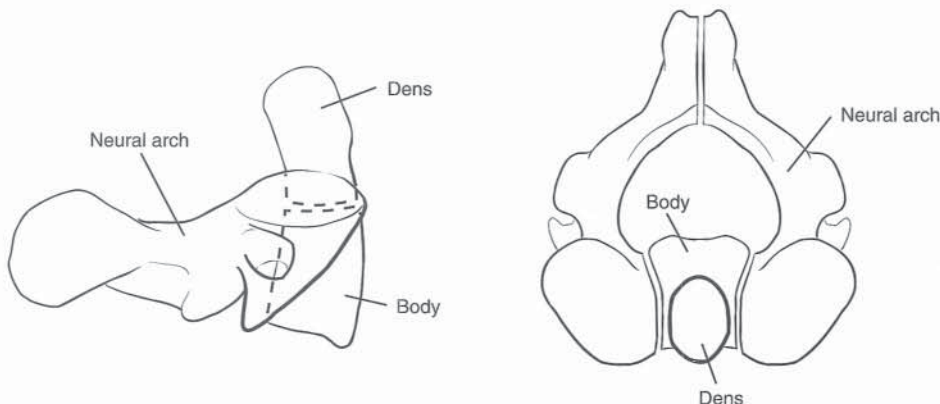


FIGURE 40-2 Ossification centers of the axis. The four centers of ossification are depicted. The anterior arch is composed of the body and the dens, while two neural arches compose the remaining centers of ossification.

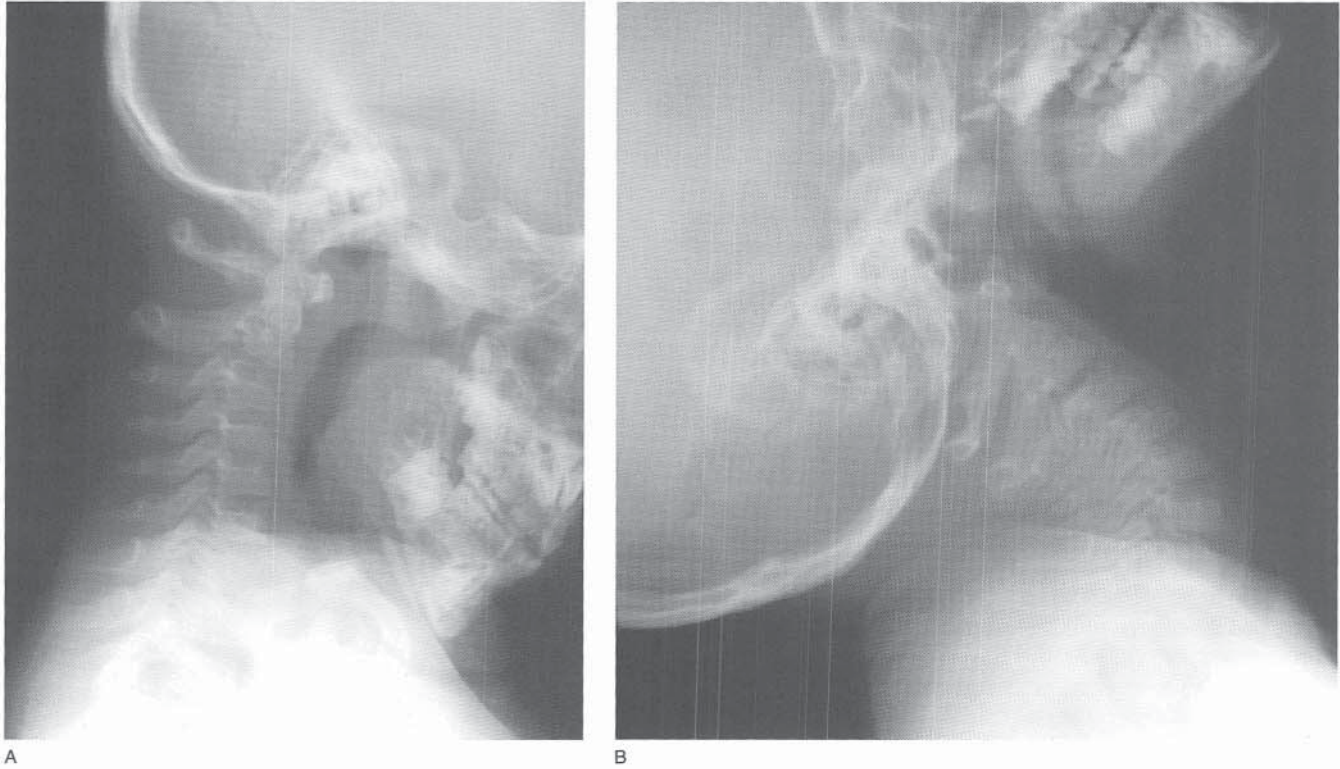


FIGURE 40-4 Flexion-extension lateral radiographs.

initial screening test, for up to 25 percent of radiographs obtained in such cases fail to demonstrate true pathology, and additional views must be obtained later when the condition of the patient allows.¹⁰¹ A complete radiographic examination should include anteroposterior (AP), lateral, open-mouth, and two oblique views. When injury is suspected despite normal-appearing radiographs, flexion-extension lateral radiographs should be obtained to help identify pathology (Fig. 40-4). It is very important to examine spine radiographs thoroughly and in their entirety without falling into the trap of focusing on the initial pathology, since multiple sites of injury may be present. In a study of 42 children with spine injuries seen over a 16-year period, 50 percent of injuries were in the cervical spine, and 35 percent of patients had more than one level of injury, usually contiguous segments.⁷⁹ McGrory and colleagues reported injuries in nonadjacent motion segments in six (4.2 percent) of 143 patients.⁷⁸

The lateral radiograph should be examined systematically, with the examiner looking first at spinal alignment. Alignment is checked by following the anterior and posterior lines of the vertebral bodies and the spinolaminar line as described by Swischuk (Fig. 40-5).^{113,114} This line is more accurate diagnostically than the line connecting the anterior and posterior lines of the vertebral bodies, which may exhibit a step-off, especially at the C2-4 levels. The line connecting the anterior edge of the spinous processes of C1, C2, and C3 should show these processes to be within 1 mm of each other. The classic pseudosubluxation at C2-3 has been well studied. Forward displacement of up to 4 mm is normal in children and is most commonly seen in children less than



FIGURE 40-5 Lateral radiograph demonstrating the spinal laminar line of Swischuk. This line is drawn by connecting the anterior edge of the spinous processes of C1, C2, and C3.

8 years old (Fig. 40–6).^{21,60,111} Second, the posterior interspinous process distance may help identify posterior ligamentous injury. Third, the prevertebral soft tissue width should be measured; it should be less than 5 to 6 mm anterior to the body of C2.²² Fourth, the normal cervical lordosis should be always assessed. Although a loss of cervical lordosis does not necessarily denote the presence of cervical spine injury, it is a good screening sign to indicate some muscle guarding and spasm. Posterior ligamentous instability is manifested on the lateral radiograph by an increase in the interspinous distance, loss of parallelism between the articular processes, and posterior widening of the disk space (Fig. 40–7).⁹⁰ Because children less than 11 years old have a higher incidence of injuries between the occiput and C3 than older children, it is important to evaluate this area well on the lateral radiograph and to obtain a good open-mouth view. Apple and colleagues reported that 10 of 11 patients less than 12 years old had injuries involving C1, C2, or the occipitoatlantal articulation, and in all, retropharyngeal swelling of more than 7 mm anterior to C2 was demonstrated.⁶ For the adolescent patient they recommend an initial cross-table lateral view, two 30-degree oblique trauma views with the patient supine,⁸¹ an AP view of the lower cervical spine, and an open-mouth view of the atlas and axis.⁶ Nitecki and Moir reported that 87 percent of patients less than 8 years had an injury at C3 or higher.⁸³

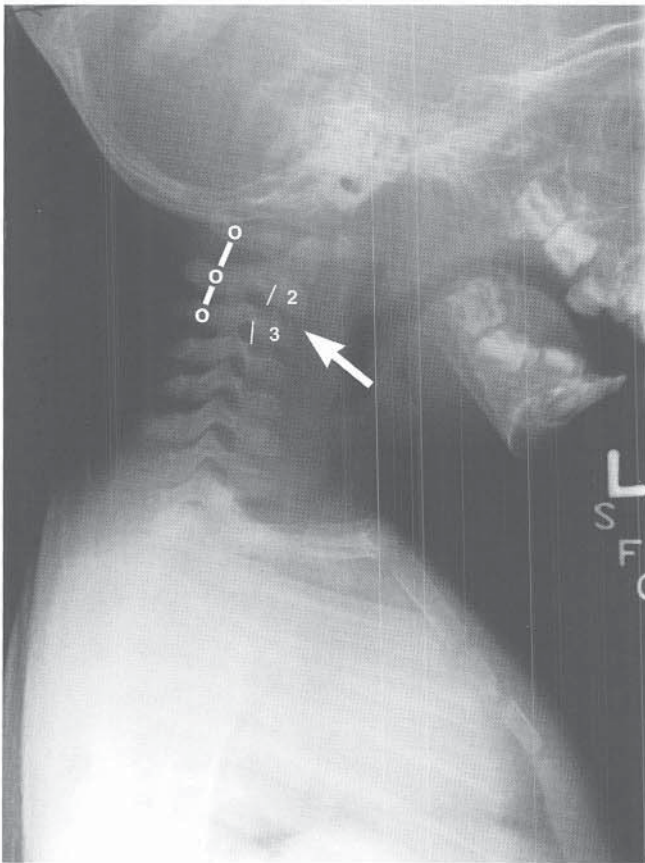


FIGURE 40–6 Pseudosubluxation of the cervical spine in children. On the lateral radiograph there is apparent subluxation of the vertebral body of C2 and C3. It appears that C2 is anteriorly subluxed on C3 (arrow). However, when the spinal laminar line of Swischuk is drawn, there is no true subluxation.

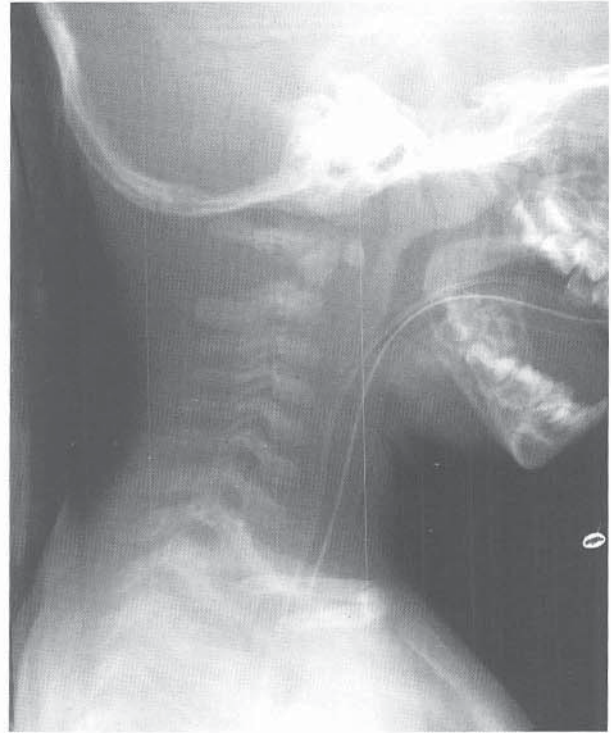


FIGURE 40–7 Posterior ligamentous instability. Lateral radiograph of a 2-year-old child demonstrating widening of the posterior elements between C1 and C2, indicating a posterior ligamentous injury.

Accepted criteria for instability of the cervical spine in children include more than 10 degrees of forward flexion of C1 on C2 and an atlanto-dens interval (ADI) of more than 4 mm.⁷¹ Pennecot and colleagues reported an upper limit of the ADI in children to be 3 ± 0.7 mm in flexion, with less than 0.5 mm of difference in the ADI occurring between flexion and extension radiographs.⁸⁹ In a classic article, Fielding and colleagues reported that in adults, when the ADI is between 3 and 5 mm the transverse ligament is ruptured, and the transverse and alar ligaments are ruptured when the ADI is 10 to 12 mm.³⁸ In the lower cervical spine no accepted criteria have been developed for children; however, White and colleagues reported that in adults, the accepted amount of angulation between the affected vertebra and the adjacent segment is 11 degrees.^{83,125,126}

Although plain radiographic assessment of the child is difficult, up to 98 percent of lateral cervical spine radiographs have been diagnostic when injury was present, and therefore careful scrutiny of good-quality radiographs, should allow the examiner at least to begin assessing and diagnosing these injuries.³⁰

Further tests, including computed tomography (CT) or magnetic resonance imaging (MRI), are indicated when abnormalities are seen on the initial plain radiographs and when cervical spine injury is suspected despite normal radiographs. CT is best used in children suspected of having osseous fractures, facet dislocations, or vertebral end-plate fractures.^{26,33} MRI is best used to evaluate soft tissue injuries, including posterior ligamentous injury, a herniated disk, encroachment of the neuroforamina, spinal cord lesions and edema, or a posttraumatic spinal cord cyst.⁴¹ MRI may have some prognostic value in distinguishing patients with spinal

cord edema, who generally recover neurologically, from patients with intraspinal hemorrhage, who often do not recover.²³ MRI may also be useful in demonstrating injuries to the spinal cord that are remote from the bony injury.¹¹

Because of the high energy required to produce these injuries, associated injuries are common and usually involve the face, head, abdomen, and other sites. Orthopaedic injuries have been reported to occur in up to 40 percent of all cases.³⁴ Closed head injuries are very common, having been reported in up to 58 percent of cases.³⁰

MANAGEMENT OF THE PATIENT WITH SUSPECTED CERVICAL SPINE INJURY

Because of the proportionally larger head compared to the body in the child, positioning the patient to prevent acute flexion of the neck is very important during transport, evaluation, and imaging studies. In 1989 Herzenberg and colleagues reported on 10 children less than 7 years old with unstable cervical spine injuries who were found to have an anterior angulation or translation on the lateral radiograph when the patient was positioned on the traditional backboard (Fig. 40–8).⁵⁴ They recommended using a bed or backboard with a posterior recess to allow the head to drop posterior and prevent anterior angulation at the neck. This is most important in young children, because the adult proportions begin to emerge in children 8 years old. At the initial presentation in the emergency room, the child should be examined with a cervical collar in place. Although a rigid collar provides some stability to the neck, residual motion can occur and can be limited with the use of tape and sandbags.⁵⁸ These devices should be gently removed while a second examiner applies a stabilizing force with mild in-line traction while the posterior elements are palpated. The

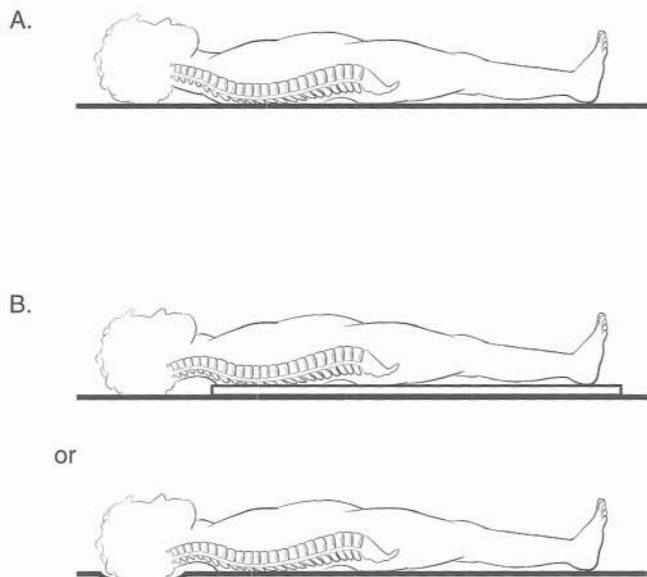


FIGURE 40–8 Proper transport of a child with a suspected cervical injury. A, Because of the proportionally large head of a child, a standard backboard will result in cervical spine flexion. B, A more appropriate transport backboard is one that includes a double mattress pad or a sunken headrest so that the head can fall back, providing a more normal lordotic position of the cervical spine.

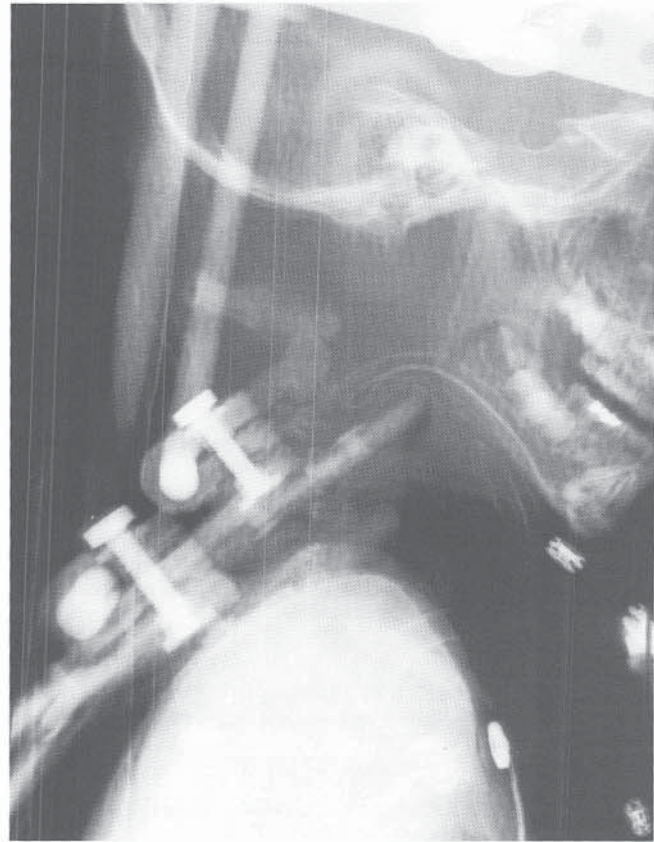


FIGURE 40–9 Atlantooccipital dislocation. Lateral radiograph of a 4-year-old child involved in a motor vehicle accident who sustained an atlantooccipital dislocation.

hard collar is then replaced and the appropriate imaging studies performed. When ventilatory support is required, the best method of intubation is controversial.^{14,55,62,73,110} It appears that gentle in-line traction with orotracheal intubation or nasotracheal intubation is safe and does not lead to further neurologic injury.

The recommendations for administration of steroids are discussed later, under Traumatic Injuries of the Thoracic and Lumbar Spine.

OCCIPITOATLANTAL DISLOCATION

This is a relatively rare injury. It usually occurs in motor vehicle accidents and is associated with a high mortality (Fig. 40–9). Bucholz and Burkhead reported findings in 112 postmortem specimens from victims of multiple trauma, of which nine (8 percent) showed occipitoatlantal dislocation.¹⁸ Their series included 20 children less than 18 years old, of whom three (15 percent) had this injury. The relatively low incidence of occipitoatlantal dislocation may be due to the horizontal orientation of the facet joints in children. Although these injuries are often fatal, children survive sometimes. Birney and Hanley reported findings in a 2-year-old child who sustained a complete craniocervical neurologic injury in a motor vehicle accident and underwent emergency C1–3 decompressive laminectomy.¹³ Apple and colleagues reported findings in one of three patients who survived this injury. The infant had sustained birth trauma, resulting in

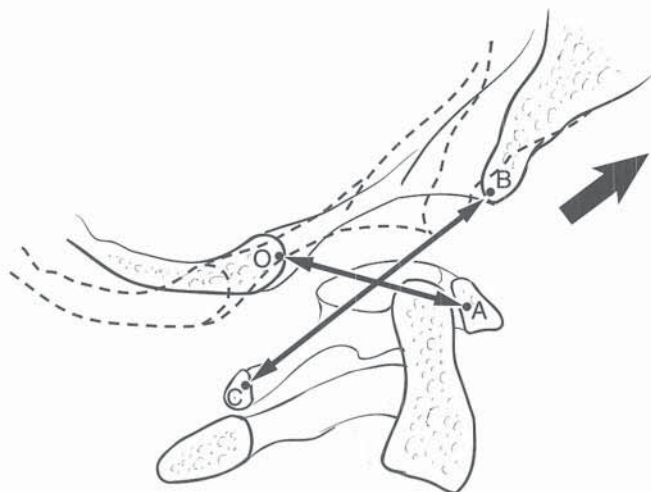


FIGURE 40-10 The Powers ratio. This ratio is determined by drawing a line from the posterior arch of the atlas (C) to the basion (B) and dividing this by the distance from the anterior arch of the atlas (A) to the opisthion (O). A normal Powers ratio is less than 0.9. A ratio greater than 1.0 is diagnostic of an atlantooccipital dislocation.

occipitoatlantal dislocation and quadriplegia.⁶ Others report patients who have survived with a variety of neurologic deficits.^{15,35,44,82,129}

Radiographic assessment of occipitoatlantal dislocations can be misleading because radiographs obtained in the emergency room often appear normal. The best tool for evaluating this injury is the Powers ratio, first described in 1979 (Fig. 40-10).⁹² If injury is strongly suspected in the absence of good radiographic evidence, very mild traction can be applied to the head to demonstrate abnormal distraction between the skull and C1.

Treatment consists of halo application and stabilization and posterior fusion from the occiput to usually C1 or C2.^{44,46,62} Postoperatively the patient is immobilized in a halo-vest jacket or halo cast (Fig. 40-11). Internal fixation in the young child may be difficult, although we have used sutures or metal wire placed around the posterior elements of C1 and C2 and through the base of the skull. The duration of immobilization should be 3 to 4 months, depending on the age of the child and the stability of fixation at the time of operation.

ATLAS FRACTURES

Fracture of the ring of C1, the so-called Jefferson fracture, is caused by an axial compressive force applied to the head, which results in direct compression of the ring of C1 by the occipital condyles. This is a very rare injury, accounting for less than 5 percent of all cervical spine fractures in children.^{6,13,42,75,80,118} The fracture may be at multiple sites within the ring of the atlas or it may be at the neurocentral synchondrosis. It is very difficult to detect a fracture of the atlas on plain radiographs, where it is best represented by displacement of the lateral masses. CT is the preferred imaging modality. When significant displacement of the fracture has occurred, the transverse ligament may become stretched and incompetent, resulting in C1-2 instability, which should be evaluated with flexion-extension radiographs.

Treatment of these injuries with external immobilization for 3 to 4 months is usually successful. We prefer halo-vest or halo-cast immobilization, although some prefer a Minerva cast. When instability of C1-2 is present, treatment requires fusion and stabilization of this joint, as outlined in the next section.

TRAUMATIC ATLANTOAXIAL INSTABILITY

In adults, instability of the atlantoaxial junction is most often a result of injury to the transverse ligament and the alar ligaments, resulting in an increased distance between the atlas and the dens. In children, this is most often true in the older child (Fig. 40-12) and in those who have underlying conditions such as Down syndrome, juvenile rheumatoid arthritis, Larsen's syndrome, and other bone dysplasias. In the younger child, the instability may be from injury to the synchondrosis at the base of the dens. Apple and colleagues described three patients who had sustained injuries to this area, which they described as an epiphysiolysis dentis. All patients were less than 5 years old.⁶

In the initial evaluation plain radiographs are obtained to assess the ADI on the true lateral radiograph and on flexion-extension views. The rule of thirds, first described by Steel, divides the area of the spinal canal at C1 into three equal areas (Fig. 40-13).¹⁰⁷ Most anterior is the odontoid, followed by the spinal cord, and finally the unoccupied area, which allows some "play" for the spinal cord. In the adult, when the odontoid is displaced posteriorly the distance of its diameter, the spinal cord is endangered, and therefore fixation of C1-2 is required to prevent neurologic injury. The indications in children are not as well defined; however, we prefer to perform surgical stabilization when anterior translation is greater than 8 to 10 mm or when neurologic deficits are present. The joint should be gently reduced to its anatomic location and fused. In the younger child, we prefer a posterior fusion followed by halo-vest or halo-cast immobilization. Internal fixation in the young child is often difficult; however, a Gallie or Brooks fusion provides additional stability to the C1-2 segment (Fig. 40-14). Halo immobilization should be maintained for approximately 2 to 4 months, depending on radiographic healing and the age of the child. In the older child (> 11 years), more stable fixation using transarticular screws between C1 and C2 allows the use of minimal external immobilization, generally a soft cervical collar that is worn for 8 weeks.^{20,43,61,72,108} We use 3.5-mm cortical screws placed under direct visualization to obtain a solid purchase in the anterior cortex of the anterior ring of the atlas. This technique can be supplemented with a Gallie or Brooks fusion (Fig. 40-15). If the presentation is delayed from the time of injury, it may be necessary to use halo traction to reduce the anterior translation before surgical stabilization is undertaken.

ODONTOID FRACTURES

Odontoid fractures account for approximately 10 percent of all cervical spine fractures and dislocations in children.* However, only about 10 percent of all odontoid fractures occur in children; the vast majority occur in adults.⁵ The

* See references 3, 6, 12, 13, 29, 36, 45, 47, 84, 99, 103.

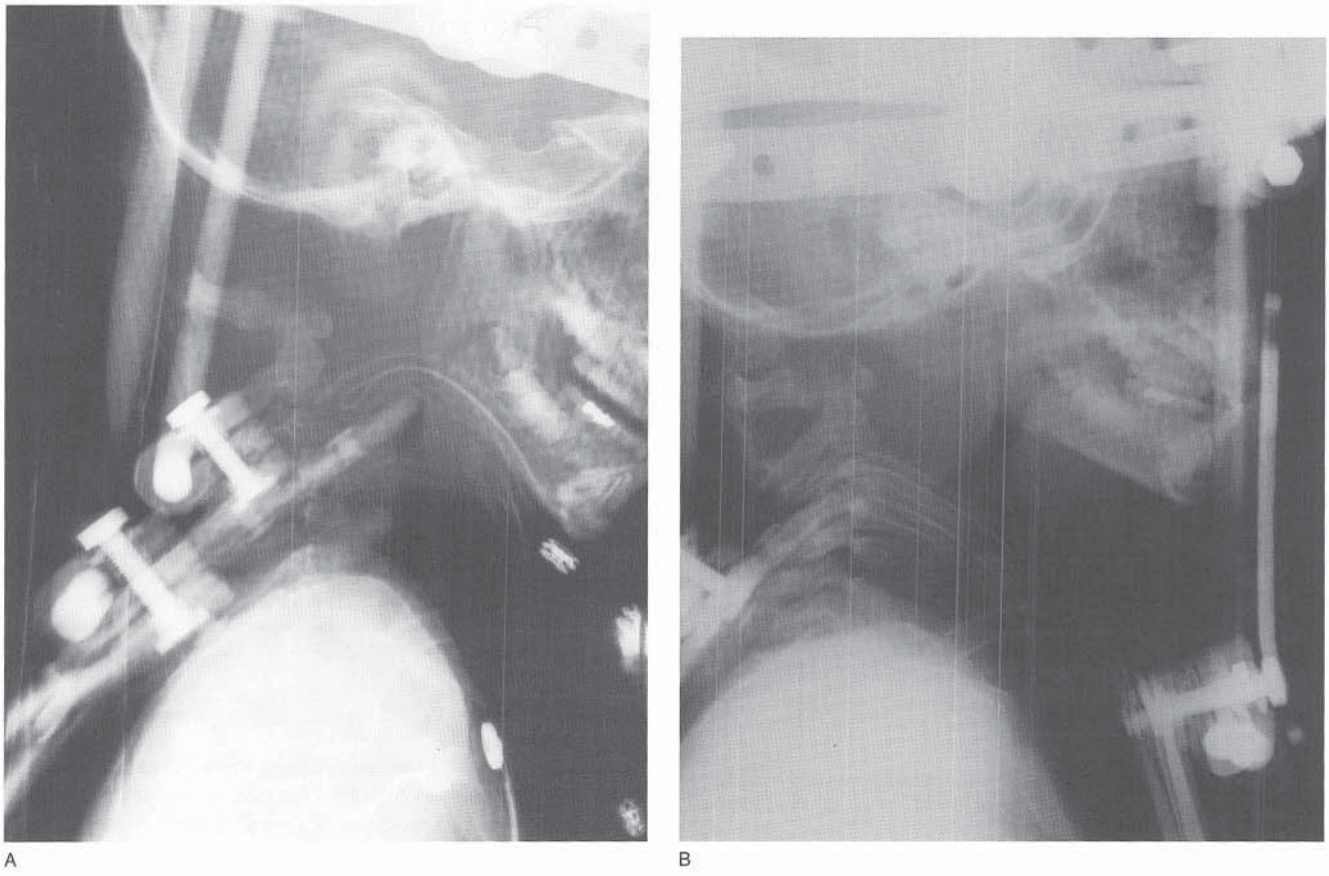


FIGURE 40-11 Treatment of an atlantooccipital dislocation. **A**, Lateral radiograph demonstrating an atlantooccipital dislocation. **B**, Lateral radiograph obtained following halo application with reduction. **C**, Lateral radiograph obtained 4 months after injury and fusion from occiput to C2, demonstrating fusion.

C

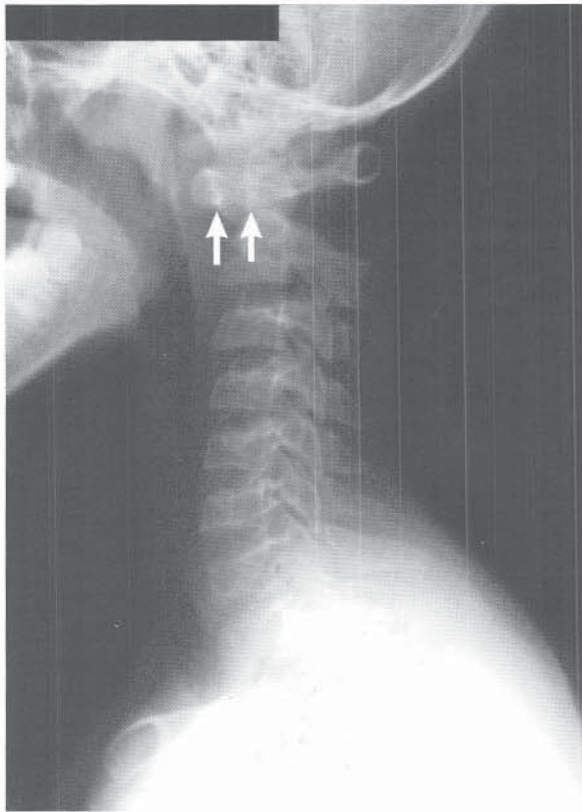


FIGURE 40-12 Lateral radiograph demonstrating atlantoaxial instability. The atlas is displaced anteriorly on the axis, with an atlanto-dens interval of 11 mm.

injury in the child occurs at the synchondrosis at the base of the dens and displaces anteriorly. The mechanism of injury is usually relatively severe, with falls from a significant height and motor vehicle accidents accounting for the majority of injuries.¹⁰³ However, in children less than 4 years old the mechanism of injury may be minor, such as a fall from a bed or a fence^{98,99} or a fall from a crib.³⁷ Associated injuries are rare; however, Odent and colleagues recently reported associated injuries in five of 15 children. Most commonly the injuries were facial fractures, but they also included a splenic injury, a pulmonary contusion, and a liver laceration.⁸⁴

The clinical examination should concentrate on associated facial trauma suggesting an acute flexion or extension injury, since other significant findings are rare. Traditionally

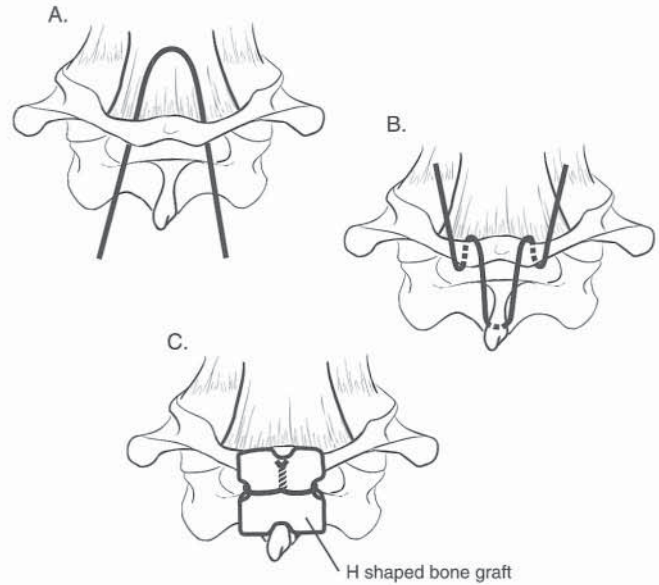


FIGURE 40-14 Posterior C1-2 fusion by a Gallie technique. A, A loop of wire is passed below the posterior arch of C1. B, The loop is then turned back on the posterior arch of C1 and looped around the inferior aspect of the spinous process of C2. C, An H-shaped iliac crest bone graft is then placed over the posterior arch of C1 and C2. The wire is twist-tied down to provide stable fixation.

it has been reported that neurologic injury is rare; however, Odent and colleagues reported neurologic injury in eight of 15 children, all of whom had complete lesions at the level of the cervicothoracic junction.⁸⁴ The patient may complain of neck pain and may have tenderness over the upper cervical spine. These injuries are often missed because of the innocuous nature of the original injury and the absence of impressive signs and symptoms.^{3,84,99} In the study by Odent and colleagues, the diagnosis was delayed up to 4 months from the time of injury in five of 15 children.⁸⁴ Persistent pain and neck irritability should alert the physician to injury. Seimon described a clinical sign that he felt correlated well with an odontoid fracture:⁹⁹ the patient strongly resists the examiner's attempts to extend the neck. The child will also resist attempts to be brought to either an erect or recumbent position unless the head is supported by the examiner.⁹⁹

The lateral radiograph usually shows the dens anteriorly displaced, usually more than 50 percent of its width. In approximately 10 to 15 percent of cases the displacement is posterior or there is no displacement. In such cases the

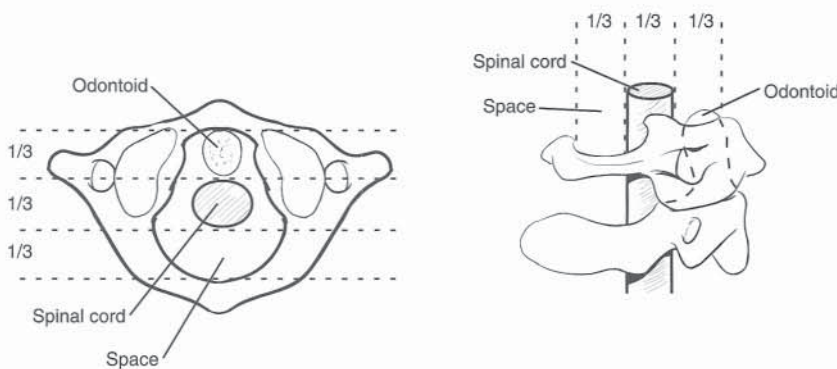


FIGURE 40-13 Steel's rule of thirds. One third of the space is occupied by the odontoid, one third by the spinal cord, and one third is space.

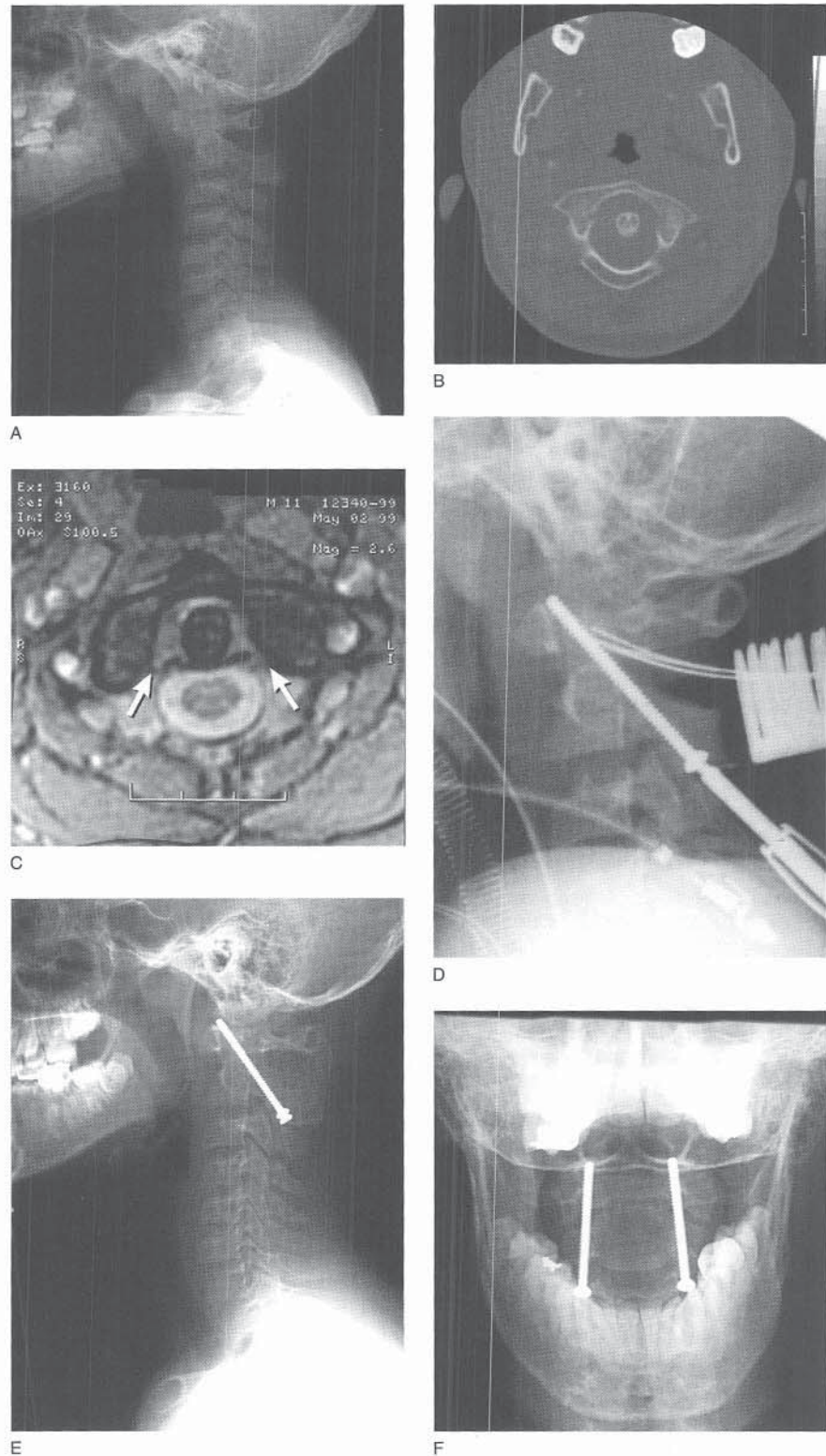


FIGURE 40-15 Transarticular screw fixation between C1 and C2. **A**, Lateral radiograph demonstrating atlantoaxial instability following a trampoline accident in a 12-year-old boy. **B**, CT scan demonstrating anterior subluxation of the atlas on the axis. **C**, MRI demonstrating avulsion of the transverse ligament (*arrows*), as well as fluid between the anterior aspect of the dens and the posterior arch of C1. **D**, Intraoperative fluoroscopic images demonstrating reduction of C1 on C2. Screws are placed after bone grafting of the articular surfaces. A modified Gallie fusion was also done posteriorly to supplement fixation. **E** and **F**, Six months after surgery there is solid healing of the anterior and posterior fusion. Note screws traversing articular facets of C1–2 on the AP radiograph.

injury is difficult to see on plain radiographs, and further imaging studies, such as CT with sagittal images or tomograms, may be necessary. In patients with neurologic injury, MRI may demonstrate SCI distal to C2, which is thought to be due to significant anterior displacement of the upper spine, leading to stretch of the spinal cord over the cervicothoracic junction.⁸⁴

Treatment of odontoid fractures in children is usually successful and most commonly does not require operative intervention.* Odent and colleagues reported successfully treating 11 patients with external immobilization, either in a Minerva jacket or in a halo cast; seven required a reduction and four did not. In one patient, the diagnosis was made 6 months following injury, at which time the fracture healed despite the fact that there had been no treatment. The three patients in this series treated operatively all had at least one complication.⁸⁴ We prefer closed reduction with a halo placed while the patient is under sedation to allow for constant neurologic assessment. This is followed by immobilization in a halo cast for 2 to 3 months until solid union is achieved. Before complete removal of the halo, flexion-extension radiographs should be obtained to identify any motion at the fracture site. Nonunion is extremely rare when this fracture is identified and treated early. Nonunion requires operative intervention that includes a posterior fusion of C1–2.

The os odontoideum is most likely an odontoid non-union, although some controversy exists as to whether this represents a congenital anomaly due to failure of fusion to the dens.† The importance of this entity is that the patient may be symptomatic, with pain, and may exhibit instability, which should be treated with a posterior C1–2 fusion.

PEDICLE FRACTURES OF C2 (HANGMAN'S FRACTURE)

Pedicle fractures of C2 are rare in children, with few cases reported in the literature.^{6,80,92,95,113,125,129} The mechanism of injury is usually extension and axial loading, with a high incidence of injuries of the face and head. The injury is usually incurred in motor vehicle accidents or a fall from a height. Neurologic injury is rare in these injuries, although some have reported deficits that resolved over the following year.^{6,91,124} Apple and colleagues described an 11-year-old girl who was thrown from a Go-cart, sustaining pedicle fractures at C2 with an associated mild upper extremity weakness.^{6,91,124} We prefer to perform CT in all suspected cases of hangman's fracture to fully define the extent of fracture and the amount of displacement (Fig. 40–16).

In a reliable patient an undisplaced fracture, or a fracture with less than 3 mm of anterior displacement of C2 on C3, can be treated with external immobilization in a collar. However, one should have a low threshold for using a halo cast when the child cannot be trusted. For fractures displaced more than 3 mm, gentle reduction should be performed to reduce the displacement, and immobilization in a halo cast for 2 to 3 months is necessary. Pizzutillo and colleagues reported successful treatment using a Minerva cast or a halo

cast, with union of the fracture occurring in four of five children. The fifth child required operative fusion after the fracture failed to unite with conservative treatment.⁹¹

FRACTURES AND DISLOCATIONS OF THE SUBAXIAL SPINE

Fractures and dislocations of the subaxial spine are relatively rare in young children; however, the incidence in children more than 8 years old is similar to the incidence in adults. Of 43 bony injuries in children, 24 (56 percent) involved the occiput to C2 (average patient age of 6.2 years) and 19 (44 percent) occurred between C3 and C7 (average patient age of 13.6 years).¹³ When all cervical spine injuries are included (including C1–2 rotatory subluxation and SCI without radiographic abnormalities), subaxial injuries account for only 23 percent of injuries.¹³ Apple and colleagues reported that in only one (9 percent) of 11 patients with a cervical spine injury was the injury below C2.⁶ However, McGrory and colleagues reported that 67 (47 percent) of 143 cervical spine fractures were between C4 and C7, and these injuries occurred predominantly in the 11- to 15-year-old age group.⁷⁸

These injuries can be subdivided into fracture-dislocations, burst fractures, compression fractures, posterior ligamentous injuries, unilateral or bilateral facet dislocations, and bilateral facet fractures.

The fracture-dislocation injury was the most common subaxial injury reported by Birney and Hanley (Fig. 40–17). Two patients were without neurologic injury, one had a transient incomplete injury, and one had a complete neurologic injury.¹³ This injury usually is a result of a motor vehicle accident or a fall with a direct blow to the head. The diagnostic workup should include an MRI of the spinal cord. Treatment generally includes a reduction maneuver and stabilization of the spine.

The burst fracture is due to axially applied loads to the head, usually with the head slightly flexed. The characteristic fracture pattern includes anterior displacement of the antero-inferior aspect of the body—the “teardrop” fracture. The danger occurs with the posterior aspect of the vertebral body, which fractures in the sagittal plane and can travel posteriorly into the canal. These injuries are most often associated with neurologic injury and are often the injury sustained by football players. Of six children with a burst fracture, two had a transient incomplete neurologic injury and one had a permanent complete injury.¹³ The canal should be assessed by CT, and posterior ligamentous injury and disk injury and herniation should be assessed by MRI. In the patient with a neurologic injury, gentle closed reduction with a halo should be performed, followed by halo-cast immobilization for 2 to 3 months. If neurologic injury is present and spinal cord compression persists despite realigning the patient with in-line traction, anterior decompression with removal of the retropulsed fragments should be performed, followed by strut grafting. Shackel and colleagues reported results in six children, age 3 to 14 years, who underwent anterior decompression with bony fusion for cervical spine fractures, with solid union and excellent results.¹⁰⁰ The anterior approach, however, should not be the first choice in young children. The canal is compromised, and continued posterior growth can lead to excess kyphosis.

* See references 12, 13, 36, 52, 84, 96, 99, 102.

† See references 25, 31, 32, 39, 40, 50, 51, 53, 56, 59, 63, 65, 68, 76, 97, 109, 115, 117, 119–121.

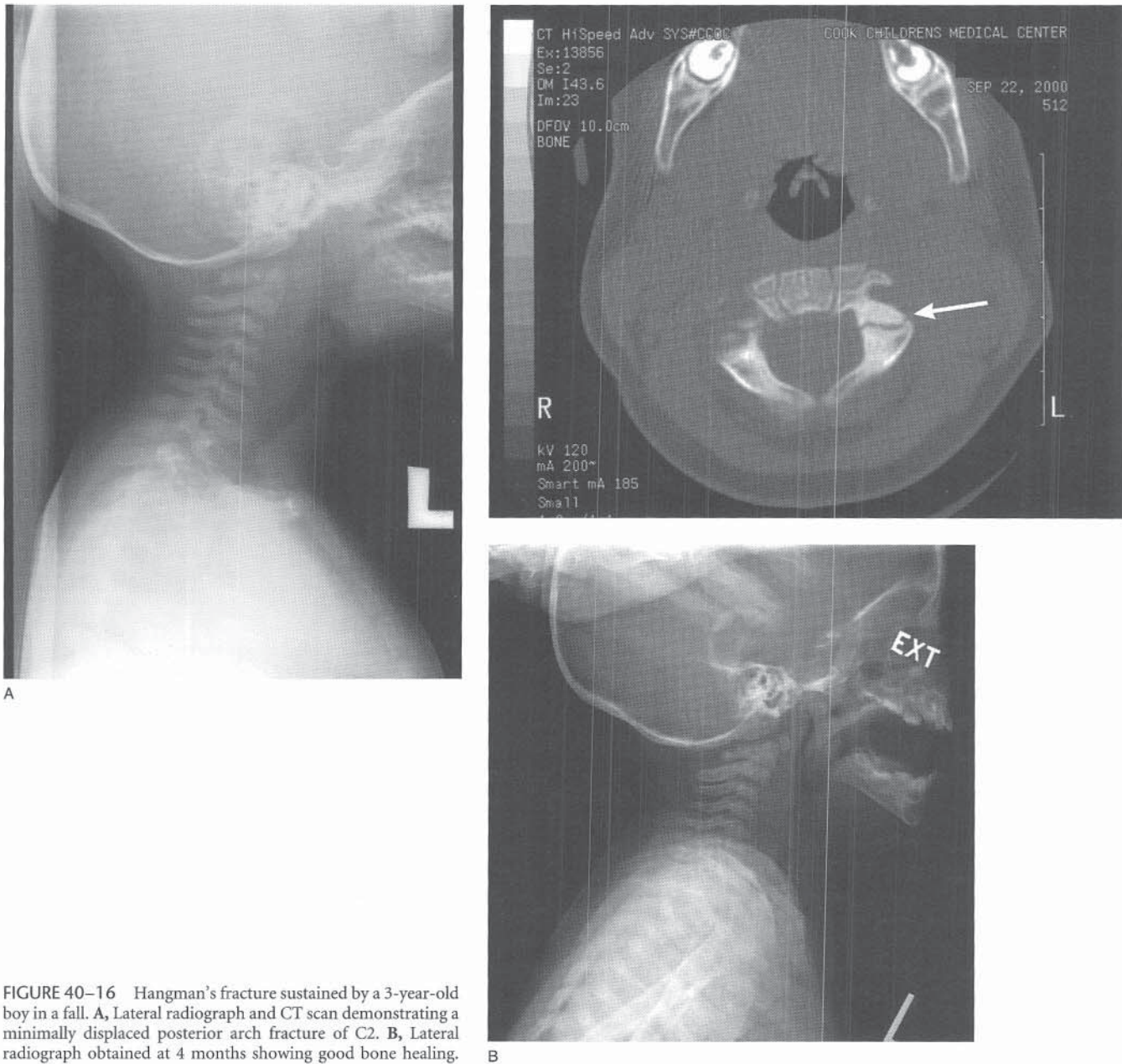


FIGURE 40-16 Hangman's fracture sustained by a 3-year-old boy in a fall. **A**, Lateral radiograph and CT scan demonstrating a minimally displaced posterior arch fracture of C2. **B**, Lateral radiograph obtained at 4 months showing good bone healing.

When an anterior burst fracture is associated with significant posterior ligamentous instability, a posterior stabilization procedure is necessary.¹⁰⁵

Compression fractures are due to a pure flexion moment without significant rotatory or axial loading. This leaves the posterior ligamentous structures intact and does not injure the posterior aspect of the vertebral body, and there is no bone or disk protrusion into the spinal canal. These injuries are relatively rare in children and usually do not result in neurologic injury. In 11 patients with cervical spine injuries, Apple and colleagues noted that only one, a 9-year-old girl, had compression fractures of the C4–6 bodies; she had been involved in a motor vehicle accident and had no neurologic injury.⁶ Henrys and colleagues did not describe any patient with this injury in their report on 18 patients.⁵² McGroary and colleagues reported that ten (7 percent) of 143 pediatric

patients had sustained a compression fracture; nine of the ten were more than 10 years old.⁷⁸ Neurologic injury is rare in these patients because of the lack of posterior body injury and therefore less risk of retropulsion into the canal.

Compression fractures are often difficult to diagnose because of the mild radiographic findings and the normal, anteriorly wedged shape of the vertebral body in children. Treatment consists of cervical spine immobilization in a cervical collar for 2 to 4 months, depending on the age of the child and the extent of injury. Surgical treatment is usually not necessary in these injuries.

Posterior ligamentous injuries result from flexion and flexion-rotation mechanisms with tearing of the posterior ligaments and the facet joint capsule. When the flexion-rotation force is relatively mild, a posterior ligamentous injury occurs. A more serious injury to the ligamentous

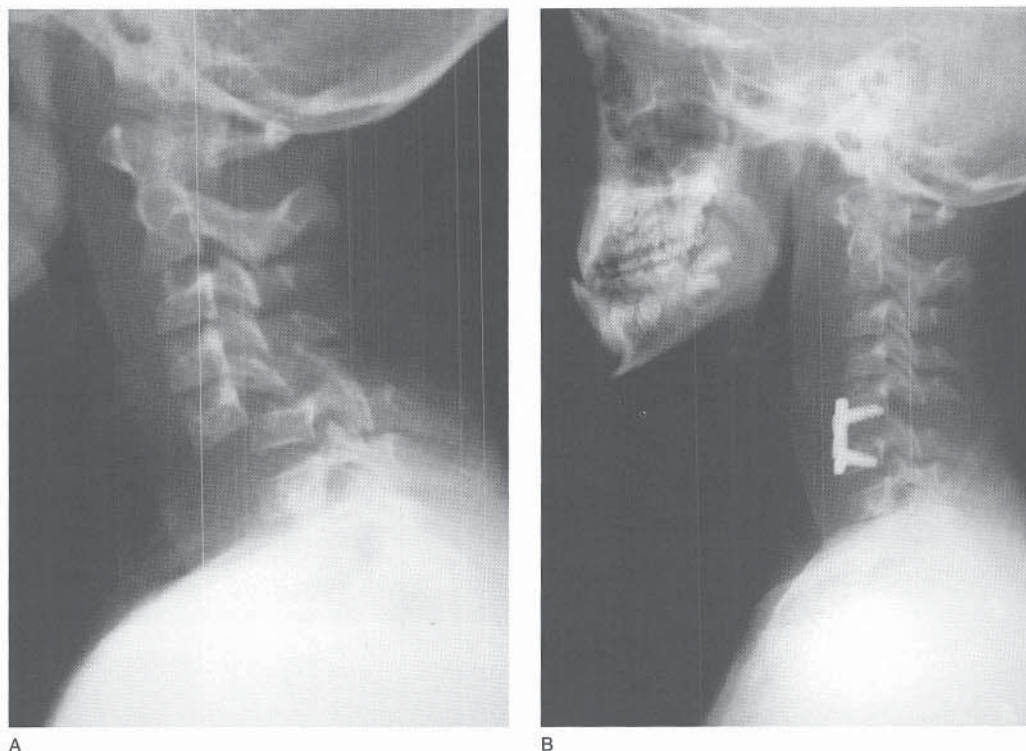


FIGURE 40-17 Fracture dislocation of the subaxial spine. **A**, Lateral radiograph demonstrating complete dislocation of C5 on C6. **B**, Lateral radiograph obtained after halo reduction and anterior plate fixation with an anterior strut graft.

structures is associated with unilateral or bilateral perched facets when greater forces are applied. A pure ligamentous injury in children is rare and is usually not associated with neurologic injury. Treatment is based on the degree of instability, but guidelines have not been fully defined in children. In adults, instability can be defined as angulation between adjacent vertebra in the sagittal plane of 11 degrees more than the adjacent normal segment or translation in the sagittal plane of 3.5 mm or more.^{87,88,125,126} Since the majority of these injuries occur in patients more than 10 years of age, similar criteria can be used in children. Significant posterior ligamentous injury requires posterior fusion with autologous bone graft and internal fixation with spinous process wires.

The unilateral or bilateral facet dislocation is a result of flexion and rotation of the cervical spine and may be associated with fractures of the facets. McGrory and colleagues reported one patient each with unilateral and bilateral facet dislocation and 11 patients with facet fracture with subluxation.⁷⁸ With a unilateral facet dislocation, there is anterior translation between vertebral bodies of 25 to 50 percent of the sagittal diameter, which may result in a unilateral nerve root compression or spinal cord lesion. Treatment should be acute reduction using halo traction with the patient under sedation. In a larger adolescent, it is necessary to treat the patient similar to the adult, with incrementally increasing weights followed by a lateral radiograph to determine whether reduction has occurred. The head should be in a slightly flexed position and then extended when the radiographs demonstrate that the facets are aligned and nearly reduced. Bilateral facet dislocations are very unstable injuries and at high risk for causing neurologic injury.¹³ These injuries should be reduced in a similar fashion as the unilateral

dislocation, with care to assess the neurologic status of the patient during the gradual distraction and reduction maneuvers. Following closed reduction of the unilateral or bilateral facet dislocation, most patients can be treated with 2 to 3 months of halo-cast immobilization. Failure to reduce a unilateral or bilateral facet dislocation requires an open reduction of the facet joint with primary fusion and stabilization with posterior wiring and halo-cast immobilization for 2 to 4 months.

LATE COMPLICATIONS FOLLOWING CERVICAL SPINE INJURIES

The patient with an SCI requires a multidisciplinary approach in order to manage the multiple needs and complications.^{8,10,122} The most common complications include pulmonary insufficiency, gastrointestinal hemorrhage (especially when steroids are used), deep venous thrombosis, urinary tract infection, and pressure sores.

The orthopaedic surgeon must realize that the incidence of late spinal deformity is very high and is most dependent on the age at which the patient sustained injury.^{17,27,64,69,77} Lancourt and colleagues reported that the incidence of scoliosis approached 100 percent if the injury occurred before age 10 years, was 19 percent in children age 10 and 16 years at the time of injury, and was only 12 percent of children older than 17 years.⁶⁹ Others, however, report a greater likelihood of the development of deformity, with those injured at 14 years having a 78 percent incidence of deformity and those injured at 16 years having a 50 percent incidence.¹⁷ Bracing early, while no spinal deformity is present, can assist in maintaining sitting balance, may prevent the development

of pressure sores, appears to lessen the incidence of the deformity, and may delay its progression approximately 1 year.¹⁷

Spinal deformity following SCI is treated by posterior spinal fusion. We prefer to fuse to the sacrum in all complete lesions when the curve reaches 40 to 50 degrees or when sitting balance and care of the child become difficult.

REFERENCES

Cervical Spine Fractures

- Abroms IF, Bresnan MJ, Zuckerman JE, et al: Cervical cord injuries secondary to hyperextension of the head in breech presentations. *Obstet Gynecol* 1973;41:369.
- Allen J: Birth injury to the spinal cord. *Northwest Med* 1970;69:323.
- Alp MS, Crockard HA: Late complication of undetected odontoid fracture in children. *BMJ* 1990;300:319.
- Anderson JM, Schutt AH: Spinal injury in children: a review of 156 cases seen from 1950 through 1978. *Mayo Clin Proc* 1980;55:499.
- Anderson LD, D'Alonzo RT: Fractures of the odontoid process of the axis. *J Bone Joint Surg* 1974;56-A:1663.
- Apple JS, Kirks DR, Merten DF, et al: Cervical spine fractures and dislocations in children. *Pediatr Radiol* 1987;17:45.
- Bailey DK: The normal cervical spine in infants and children. *Radiology* 1952;59:712.
- Bedbrook G: *The Care and Management of Spinal Cord Injuries*. New York, Springer-Verlag, 1981.
- Bertolami C, Kaban L: Chin trauma: a clue to associated mandibular and cervical spine injury. *Oral Surg* 1982;53:122.
- Betz RR: Unique management needs of pediatric spinal cord injury patients: orthopedic problems in the child with spinal cord injury. *J Spinal Cord Med* 1997;20:14.
- Betz RR, Gelman AJ, De Filipp GJ, et al: Magnetic resonance imaging (MRI) in the evaluation of spinal cord injured children and adolescents. *Paraplegia* 1987;25:92.
- Bhattacharyya SK: Fracture and displacement of the odontoid process in a child. *J Bone Joint Surg* 1974;56-A:1071.
- Birney TJ, Hanley EN Jr: Traumatic cervical spine injuries in childhood and adolescence. *Spine* 1989;14:1277.
- Bivins HG, Ford S, Bezmalinovic Z, et al: The effect of axial traction during orotracheal intubation of the trauma victim with an unstable cervical spine [see comments]. *Ann Emerg Med* 1988;17:25.
- Blauth M, Schmidt U, Lange U: [Injuries of the cervical spine in children]. *Unfallchirurg* 1998;101:590.
- Bresnan MJ, Abroms IF: Neonatal spinal cord transection secondary to intrauterine hyperextension of the neck in breech presentation. *J Pediatr* 1974;84:734.
- Brown JC, Swank SM, Matta J, et al: Late spinal deformity in quadriplegic children and adolescents. *J Pediatr Orthop* 1984;4:456.
- Buchholz RW, Burkhead WZ: The pathological anatomy of fatal atlanto-occipital dislocations. *J Bone Joint Surg* 1979;61-A:248.
- Burke DC: Traumatic spinal paralysis in children. *Paraplegia* 1974;11:268.
- Campanelli M, Kattner KA, Stroink A, et al: Posterior C1–C2 transarticular screw fixation in the treatment of displaced type II odontoid fractures in the geriatric population: review of seven cases. *Surg Neurol* 1999;51:596.
- Cattel HS, Filtzer DL: Pseudosubluxation and other normal variations in the cervical spine in children. *J Bone Joint Surg* 1965;47:1295.
- Clark W, Gehweiler J, Laib R: Twelve significant signs of cervical spine trauma. *Skeletal Radiol* 1979;3:201.
- Cotler HB, Kulkarni MV, Bondurant FJ: Magnetic resonance imaging of acute spinal cord trauma: preliminary report. *J Orthop Trauma* 1988;2:1.
- Cullen JC: Spinal lesions in battered babies. *J Bone Joint Surg* 1975;57-B:364.
- Dai L, Yuan W, Ni B, et al: Os odontoideum: etiology, diagnosis, and management. *Surg Neurol* 2000;53:106.
- Dake M, Jacob R, Margolin F: Computed tomography of posterior lumbar apophyseal ring fractures. *J Comput Assist Tomogr* 1985;9:730.
- Dearolf WW III, Betz RR, Vogel LC, et al: Scoliosis in pediatric spinal cord-injured patients. *J Pediatr Orthop* 1990;10:214.
- Dickman CA, Zabramski JM, Hadley MN, et al: Pediatric spinal cord injury without radiographic abnormalities: report of 26 cases and review of the literature. *J Spinal Disord* 1991;4:296.
- Diekema DS, Allen DB: Odontoid fracture in a child occupying a child restraint seat. *Pediatrics* 1988;82:117.
- Dietrich AM, Ginn-Pease ME, Bartkowski HM, et al: Pediatric cervical spine fractures: predominantly subtle presentation. *J Pediatr Surg* 1991;26:995.
- Dyck P: Os odontoideum in children: neurological manifestations and surgical management. *Neurosurgery* 1978;2:93.
- Edmunds IA, Cummine JL: Spontaneous bone graft resorption in os odontoideum. *Aust NZ J Surg* 1992;62:502.
- Epstein N, Epstein J: Limbus lumbar vertebral fractures in 27 adolescents and adults. *Spine* 1989;16:962.
- Evans DL, Bethem D: Cervical spine injuries in children. *J Pediatr Orthop* 1989;9:563.
- Evarts C: Traumatic occipito-atlantal dislocation: report of a case with survival. *J Bone Joint Surg* 1970;52-A:1653.
- Ewald FC: Fracture of the odontoid process in a seventeen-month-old infant treated with a halo: a case report and discussion of the injury under the age of three. *J Bone Joint Surg* 1971;53-A:1636.
- Fielding J: Disappearance of the central portion of the odontoid process: a case report. *J Bone Joint Surg* 1965;47-A:1228.
- Fielding JW, Cochran GvB, Lawsing JF III, et al: Tears of the transverse ligament of the atlas: a clinical and biomechanical study. *J Bone Joint Surg* 1974;56-A:1683.
- Fielding JW, Hensinger RN, Hawkins RJ: Os odontoideum. *J Bone Joint Surg* 1980;62-A:376.
- Forlin E, Herscovici D, Bowen JR: Understanding the os odontoideum. *Orthop Rev* 1992;21:1441.
- Gabriel KR, Crawford AH: Identification of acute posttraumatic spinal cord cyst by magnetic resonance imaging: a case report and review of the literature. *J Pediatr Orthop* 1988;8:710.
- Galindo MJ Jr, Francis WR: Atlantal fracture in a child through congenital anterior and posterior arch defects: a case report. *Clin Orthop* 1983;178:220.
- Gebhard JS, Schimmer RC, Jeanneret B: Safety and accuracy of transarticular screw fixation C1–C2 using an aiming device: an anatomic study. *Spine* 1998;23:2185.
- Georgopoulos G, Pizzutillo PD, Lee MS: Occipito-atlantal instability in children: a report of five cases and review of the literature. *J Bone Joint Surg* 1987;69-A:429.
- Godard J, Hadji M, Raul JS: Odontoid fractures in the child with neurological injury: direct anterior osteosynthesis with a cortico-spongiotic screw and literature review. *Childs Nerv Syst* 1997;13:105.
- Grantham S, Dick H, Thompson R, et al: Occipito-cervical arthrodesis: indications, techniques and results. *Clin Orthop Rel Res* 1969;65:118.
- Griffiths SC: Fracture of odontoid process in children. *J Pediatr Surg* 1972;7:680.
- 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.
- Hause M, Hoshino R, Omata S, et al: Cervical spine injuries in children. *Fukushima J Med Sci* 1974;20:114.
- Hawkins RJ, Fielding JW, Thompson WJ: Os odontoideum: congenital or acquired: a case report. *J Bone Joint Surg* 1976;58-A:413.
- Heick A: Juvenile avascular necrosis of the odontoid process: a review on the etiology of os odontoideum and absentia odontoidei. *Dan Med Bull* 1996;43:363.
- Henrys P, Lyne ED, Lifton C, et al: Clinical review of cervical spine injuries in children. *Clin Orthop* 1977;129:172.
- Hensinger RN, Fielding JW, Hawkins RJ: Congenital anomalies of the odontoid process. *Orthop Clin North Am* 1978;9:901.
- Herzenberg JE, Hensinger RN, Dedrick DK, et al: Emergency transport and positioning of young children who have an injury of the cervical spine: the standard backboard may be hazardous. *J Bone Joint Surg* 1989;71-A:15.
- Holley S, Jordan R: Airway management in patients with unstable cervical spine fractures. *Ann Emerg Med* 1989;18:1237.
- Hosono N, Yonenobu K, Ebara S, et al: Cineradiographic motion analysis of atlantoaxial instability in os odontoideum. *Spine* 1991;16:S480.

57. Hubbard DD: Injuries of the spine in children and adolescents. *Clin Orthop* 1974;100:56.
58. Huerta C, Griffith R, Joyce SM: Cervical spine stabilization in pediatric patients: evaluation of current techniques [see comments]. *Ann Emerg Med* 1987;16:1121.
59. Hukuda S, Ota H, Okabe N, et al: Traumatic atlantoaxial dislocation causing os odontoideum in infants. *Spine* 1980;5:207.
60. Jacobsen G, Bleeker H: Examination of the atlanto axial joint following injury with particular emphasis on rotational subluxation. *AJR Am J Roentgenol* 1959;76:1081.
61. Jeanneret B, Magerl F: Primary posterior fusion C1/2 in odontoid fractures: indications, technique, and results of transarticular screw fixation. *J Spinal Disord* 1992;5:464.
62. Joyce S: Cervical immobilization during orotracheal intubation in trauma victims. *Ann Emerg Med* 1988;17:88.
63. Juhl M, Seerup KK: Os odontoideum: a cause of atlanto-axial instability. *Acta Orthop Scand* 1983;54:113.
64. Kilfoyle R, Foley J, Norton P: Spine and pelvic deformity in childhood and adolescent paraplegia. *J Bone Joint Surg* 1965;47-A:659.
65. Kirlew KA, Hathout GM, Reiter SD, et al: Os odontoideum in identical twins: perspectives on etiology. *Skeletal Radiol* 1993;22:525.
66. Kleinman PK, Zito JL: Avulsion of the spinous processes caused by infant abuse. *Radiology* 1984;151:389.
67. Koop SE, Winter RB, Lonstein JE: The surgical treatment of instability of the upper part of the cervical spine in children and adolescents. *J Bone Joint Surg* 1984;66-A:403.
68. Kuhns LR, Loder RT, Farley FA, et al: Nuchal cord changes in children with os odontoideum: evidence for associated trauma. *J Pediatr Orthop* 1998;18:815.
69. Lancourt JE, Dickson JH, Carter RE: Paralytic spinal deformity following traumatic spinal-cord injury in children and adolescents. *J Bone Joint Surg* 1981;63-A:47.
70. Lewis V, Manson P, Morgan R: Facial injuries associated with cervical fractures: recognition, patterns and management. *J Trauma* 1985;25:90.
71. Locke GR, Gardner JI, Van Epps EF: Atlas-dens interval (ADI) in children: a survey based on 200 normal cervical spines. *AJR Am J Roentgenol* 1966;97:135.
72. Lu J, Ebraheim NA, Yang H, et al: Anatomic considerations of anterior transarticular screw fixation for atlantoaxial instability. *Spine* 1998;23:1229.
73. Majernick T, Bieniek R, Houston J: Cervical spine movement during oro-tracheal intubation. *Ann Emerg Med* 1986;15:417.
74. Mann DC, Dodds JA: Spinal injuries in 57 patients 17 years or younger. *Orthopedics* 1993;16:159.
75. Marlin AE, Williams GR, Lee JF: Jefferson fractures in children: case report. *J Neurosurg* 1983;58:277.
76. Matsui H, Imada K, Tsuji H: Radiographic classification of os odontoideum and its clinical significance. *Spine* 1997;22:1706.
77. Mayfield JK, Erkkila JC, Winter RB: Spine deformity subsequent to acquired childhood spinal cord injury. *J Bone Joint Surg* 1981;63-A:1401.
78. McGrory BJ, Klassen RA, Chao EY, et al: Acute fractures and dislocations of the cervical spine in children and adolescents. *J Bone Joint Surg* 1993;75-A:988.
79. McPhee IB: Spinal fractures and dislocations in children and adolescents. *Spine* 1981;6:533.
80. Mikawa Y, Watanabe R, Yamano Y, et al: Fracture through a synchondrosis of the anterior arch of the atlas. *J Bone Joint Surg* 1987;69-A:483.
81. Miller M, Gehweiler J, Martinez S, et al: Significant new observations on cervical spine trauma. *AJR Am J Roentgenol* 1978;130:659.
82. Montane I, Eismont FJ, Green BA: Traumatic occipitoatlantal dislocation. *Spine* 1991;16:112.
83. Nitecki S, Moir CR: Predictive factors of the outcome of traumatic cervical spine fracture in children. *J Pediatr Surg* 1994;29:1409.
84. Odent T, Langlais J, Glorion C, et al: Fractures of the odontoid process: a report of 15 cases in children younger than 6 years. *J Pediatr Orthop* 1999;19:51.
85. Osenbach RK, Menezes AH: Spinal cord injury without radiographic abnormality in children. *Pediatr Neurosci* 1989;15:168.
86. Pang D, Wilberger JE Jr: Spinal cord injury without radiographic abnormalities in children. *J Neurosurg* 1982;57:114.
87. Panjabi MM, White AA III, Johnson RM: Cervical spine mechanics as a function of transection of components. *J Biomech* 1975;8:327.
88. Panjabi MM, White AA III, Keller D, et al: Stability of the cervical spine under tension. *J Biomech* 1978;11:189.
89. Pennecot GF, Gouraud D, Hardy JR, et al: Roentgenographical study of the stability of the cervical spine in children. *J Pediatr Orthop* 1984;4:346.
90. Pennecot GF, Leonard P, Peyrot Des Gachons S, et al: Traumatic ligamentous instability of the cervical spine in children. *J Pediatr Orthop* 1984;4:339.
91. Pizzutillo PD, Rocha EF, D'Astous J, et al: Bilateral fracture of the pedicle of the second cervical vertebra in the young child. *J Bone Joint Surg* 1986;68-A:892.
92. Powers B, Miller M, Kramer R: Traumatic anterior atlanto-occipital dislocation. *Neurosurgery* 1979;4:12.
93. Rachesky I, Boyce WT, Duncan B, et al: Clinical prediction of cervical spine injuries in children: radiographic abnormalities. *Am J Dis Child* 1987;141:199.
94. Ruff SJ, Taylor TK: Hangman's fracture in an infant. *J Bone Joint Surg* 1986;68-B:702.
95. Schiff DC, Parke WW: The arterial supply of the odontoid process. *J Bone Joint Surg* 1973;55-A:1450.
96. Schippers N, Konings P, Hassler W, et al: Typical and atypical fractures of the odontoid process in young children: report of two cases and a review of the literature. *Acta Neurochir (Wien)* 1996;138:524.
97. Schuler TC, Kurz L, Thompson DE, et al: Natural history of os odontoideum. *J Pediatr Orthop* 1991;11:222.
98. Schwartz GR, Wright SW, Fein JA, et al: Pediatric cervical spine injury sustained in falls from low heights [published erratum appears in *Ann Emerg Med* 1998;31:141]. *Ann Emerg Med* 1997;30:249.
99. Seimon LP: Fracture of the odontoid process in young children. *J Bone Joint Surg* 1977;59-A:943.
100. Shacked I, Ram Z, Hadani M: The anterior cervical approach for traumatic injuries to the cervical spine in children. *Clin Orthop* 1993;292:144.
101. Shaffer M, Doris P: Limitation of the cross table lateral view in directing cervical spine injuries: a retrospective review. *Ann Emerg Med* 1981;10:508.
102. Sherk HH: Fractures of the atlas and odontoid process. *Orthop Clin North Am* 1978;9:973.
103. Sherk HH, Nicholson JT, Chung SM: Fractures of the odontoid process in young children. *J Bone Joint Surg* 1978;60-A:921.
104. Sneed R, Stover S: Undiagnosed spinal cord injuries in brain injured children. *Am J Dis Child* 1988;142:965.
105. Stauffer ES, Kelly EG: Fracture-dislocations of the cervical spine: instability and recurrent deformity following treatment by anterior interbody fusion. *J Bone Joint Surg* 1977;59-A:45.
106. Stauffer ES, Mazur JM: Cervical spine injuries in children. *Pediatr Ann* 1982;11:502.
107. Steel H: Anatomical and mechanical consideration of the atlanto-axial articulation. *J Bone Joint Surg* 1968;50-A:1481.
108. Stillerman CB, Wilson JA: Atlanto-axial stabilization with posterior transarticular screw fixation: technical description and report of 22 cases. *Neurosurgery* 1993;32:948.
109. Stillwell WT, Fielding JW: Acquired os odontoideum: a case report. *Clin Orthop* 1978;135:71.
110. Suderman V, Crosby E, Lui A: Elective oral tracheal intubation in spine injured adults. *Can J Anaesth* 1991;38:785.
111. Sullivan C, Bruwer A, Harris L: Hypermobility of the cervical spine in children: a pitfall in the diagnosis of cervical dislocation. *Am J Surg* 1958;95:636.
112. Sumchai AP, Sternbach GL: Hangman's fracture in a 7-week-old infant. *Ann Emerg Med* 1991;20:86.
113. Swischuk L, Rowe M: The upper cervical spine in health and disease. *Pediatrics* 1952;10:567.
114. Swischuk LE: Spine and spinal-cord trauma in the battered child syndrome. *Radiology* 1969;92:733.
115. Takakuwa T, Hiroi S, Hasegawa H, et al: Os odontoideum with vertebral artery occlusion. *Spine* 1994;19:460.
116. Taylor A: The mechanism of injury to the spinal cord in the neck without damage to the vertebral column. *J Bone Joint Surg* 1951;33-B:453.
117. Teng MM, Shoung HM, Chang CY, et al: CT and myelogram findings of os odontoideum. *Comput Med Imaging Graph* 1989;13:179.
118. Tolo VT, Weiland AJ: Unsuspected atlas fracture and instability associated with oropharyngeal injury: case report. *J Trauma* 1979;19:278.

119. Tsou HK, Shen CC, Wang YC: Os odontoideum: a case report and review. *Chung Hua I Hsueh Tsa Chih (Taipei)* 1998;61:741.
120. Verska JM, Anderson PA: Os odontoideum: a case report of one identical twin. *Spine* 1997;22:706.
121. Vickers ED: Atlantoaxial anomalies with particular emphasis on os odontoideum. *J Manipulative Physiol Ther* 1990;13:471.
122. Vogel L, Mulcahy MJ, Betz RR: The child with a spinal cord injury. *Dev Med Child Neurol* 1997;39:202.
123. Walsh JW, Stevens DB, Young AB: Traumatic paraplegia in children without contiguous spinal fracture or dislocation. *Neurosurgery* 1983;12:439.
124. Weiss MH, Kaufman B: Hangman's fracture in an infant. *Am J Dis Child* 1973;126:268.
125. White AA III, Johnson RM, Panjabi MM, et al: Biomechanical analysis of clinical stability in the cervical spine. *Clin Orthop* 1975;109:85.
126. White AA III, Panjabi MM: The basic kinematics of the human spine: a review of past and current knowledge. *Spine* 1978;3:12.
127. Yngve DA, Harris WP, Herndon WA, et al: Spinal cord injury without osseous spine fracture. *J Pediatr Orthop* 1988;8:153.
128. Zanette G, Ori C, Zadra N, et al: Hangman's fracture in a paediatric patient: considerations for anaesthesia. *Paediatr Anaesth* 1997;7:473.
129. Zigler J, Waters R, Nelson R: Occipito-cervico-thoracic spine fusion in a patient with occipito-cervical dislocation with survival. *Spine* 1986;11:645.

Traumatic Injuries of the Thoracic and Lumbar Spine

In children, injuries of the thoracic and lumbar spine are less common than cervical spine injuries. Patients in the first decade of life are more likely to sustain upper thoracic (T4 to T10) injuries and are more likely to be injured from falls or motor vehicle–pedestrian collisions. They may also be injured from abuse.* Patients in the second decade of life are more likely to sustain injuries at the thoracolumbar junction and are commonly injured in motor vehicle collisions or during recreational events.† Neurologic injury occurs in approximately half of patients, with a slight predominance of incomplete lesions.^{33,58,74,84,118} It is important for the surgeon treating these patients to remember these are high-energy injuries that are frequently associated with other visceral or orthopaedic injuries, including multiple injuries of the spinal column.‡

The nomenclature for thoracic and lumbar spine fractures is somewhat confusing, as both thoracic and lumbar injuries as well as injuries at the thoracolumbar junction are frequently referred to as “thoracolumbar” injuries. We reserve the phrase “thoracolumbar injuries” for injuries occurring between T12 and L1.

ANATOMY

An understanding of the anatomy of the immature spine is important in evaluating and treating children with spinal injuries. The pediatric spine is more flexible than the adult spine, which may contribute to the frequency of neurologic injury as well as the finding of SCI without radiographic abnormality, or SCIWORA. Several factors contribute to the flexibility of the child's spine. First, the soft tissues are more forgiving, the ligaments are more elastic, the muscles are

smaller, and the intervertebral disks are healthy and well hydrated. Second, there is a higher ratio of cartilage to bone. Finally, the facets are more horizontal, allowing greater motion.^{4,69,79,87,116,120} Vertebral growth occurs equally from the superior and inferior apophyses, which develop within the cartilaginous end-plate. These apophyses are wider peripherally than centrally, which gives them a ring appearance, the origin of the term ring apophysis. They are similar to the epiphysis of a long bone. Ring apophyses appear radiographically between 8 and 12 years of age and fuse with the body between 21 and 25 years of age.

Management of children with thoracic and lumbar spinal injuries requires an understanding of the “three-column spine,” a concept introduced by Denis in 1983.³¹ This anatomic description provides the basis for the most efficient means of classification as well as a foundation for a rational approach to treatment. Denis realized that complete rupture of the posterior ligamentous structures did not produce instability. Rather, instability in flexion required not only rupture of the posterior ligaments, but also disruption of what he termed the “middle column”—the posterior longitudinal ligament, the posterior annulus fibrosus, and the posterior wall of the vertebral body (Fig. 40–18). The anterior column consists of the anterior longitudinal ligament, the anterior annulus fibrosus, and the anterior vertebral body. The posterior arch and the posterior ligamentous complex (the supraspinous and interspinous ligaments, facet joint capsules, and ligamentum flavum) make up the posterior column.

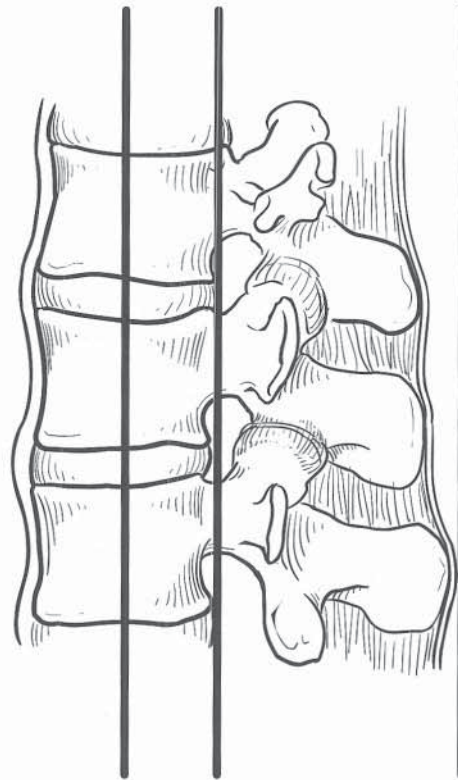


FIGURE 40–18 The three-column spine. The anterior column consists of the anterior longitudinal ligament, the anterior annulus fibrosus, and the anterior vertebral body. The middle column consists of the posterior wall of the vertebral body, the posterior longitudinal ligament, and the posterior annulus fibrosus. The posterior column consists of the posterior arch and the posterior ligamentous complex (supraspinous and interspinous ligaments, facet joint capsules, and ligamentum flavum).

* See references 4, 18, 32, 50, 61, 65, 92, 122.

† See references 9, 61, 62, 75, 101, 108, 126, 138.

‡ See references 3, 5, 7, 14, 23, 51, 52, 60, 61, 64, 85, 88, 94, 114, 115, 120, 124, 132, 133, 136, 139.

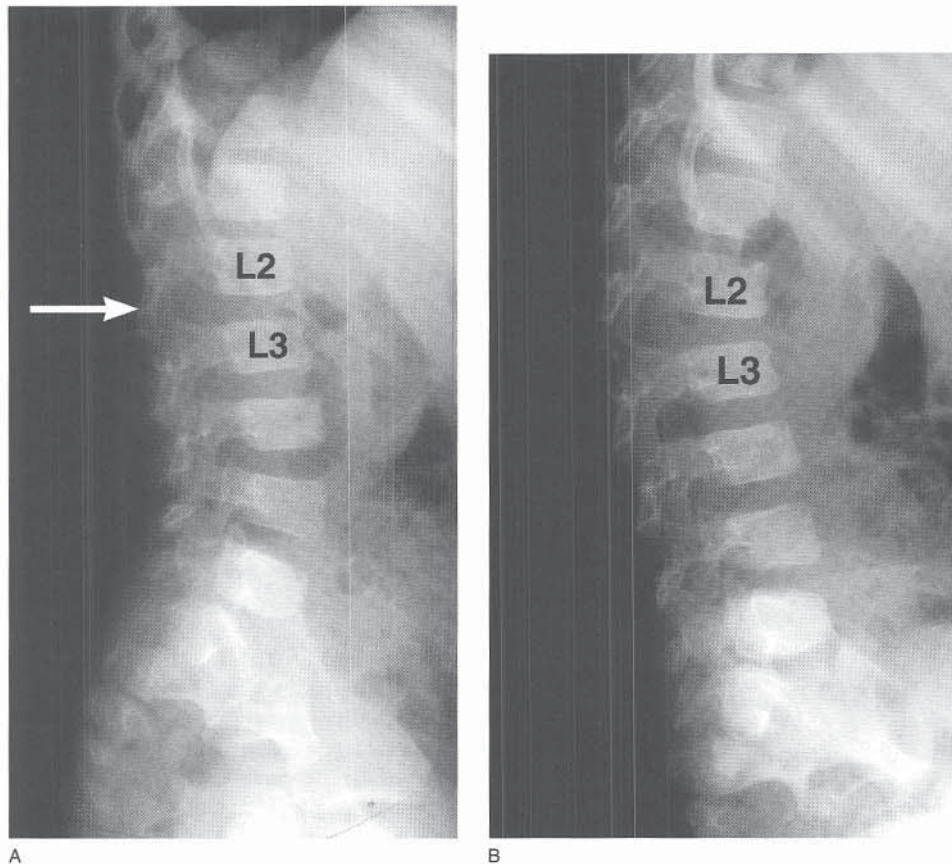


FIGURE 40-19 Initial imaging studies in thoracic and lumbar spine injuries may be nondiagnostic. **A**, Lateral radiograph of a 4-year-old girl involved in a high-speed motor vehicle accident. The films were initially interpreted as normal. **B**, Because of persistent back pain with localized tenderness in the lumbar spine, a flexion stress view was obtained that revealed an acute kyphosis due to soft tissue disruption between L2 and L3. In retrospect, the initial lateral radiograph shows some widening of the disk space and foramen.

MECHANISM OF INJURY

Thoracic and lumbar spine injuries are most commonly the result of high-energy forces. Motor vehicle–related injuries are the most common, although falls, recreational activities, child abuse, obstetric injury, and gunshots have all been reported as mechanisms.* The force that produces the injury is most commonly flexion, which may be combined with compression, distraction, or shear forces.† Extension injuries have been described but are extremely uncommon.⁹⁷

Roaf in 1960 reported on the events that lead to spinal fracture. As a vertical load is applied, the end-plate bulges toward the vertebral body; there is little change in either the annulus or the nucleus of the disk. As the load increases, the deformation of the end-plate forces blood out of the cancellous bone of the vertebral body, decreasing its energy-absorbing ability. Eventually the elastic limit of vertebral body is exceeded, and fracture occurs.¹¹⁶ The elasticity of the pediatric spine allows these forces to be distributed over multiple levels, which explains why multiple compression fractures are seen more commonly in children. If a distraction or shear force exists concurrently, it may also produce deformity, usually through the end-plate rather than the disk.^{21,65,68,75,116,126}

Neurologic injury is classified as primary or secondary. Primary injuries are the result of direct injury to the neural elements. They may be the result of contusion, stretch, compression, or laceration. Contusion injuries are most com-

mon and have a poor prognosis for recovery. Compression produces injury both primarily, through direct neuronal damage, as well as secondarily, by altering vascular perfusion. Secondary injuries are the result of ischemia. Secondary injuries are most common in the “watershed” area of the thoracic spine (T7 to T10). Ischemic injury is a mechanical and biochemical cycle. The initial injury produces a mechanical ischemia, which results in cell death and the release of vasoactive substances. The substances produce both vasoconstriction and edema. The edema produces further mechanical compression, and the cycle continues.¹⁴⁰ Because of their cyclic nature, ischemic injuries may evolve over time, and the delayed presentation of neurologic injury is not uncommon.^{22,67,95,103} Ischemic SCIs may be exacerbated by systemic hypotension associated with shock from other traumatic injuries. In fact, paraplegia has been reported in both children and adults with hypotensive episodes and no injury to the spinal cord.^{1,26,67,77,123,135}

DIAGNOSIS

Thoracic and lumbar spine injuries may be difficult to diagnose. These patients frequently have multiple injuries and an altered state of consciousness. Occasionally the elasticity of the pediatric spine allows it to “recoil” into a more normal position. If this occurs, the displacement at the time of injury and subsequently the amount of instability may not be appreciated on initial x-rays (Fig. 40-19). Thus, all patients with significant traumatic injuries should be assumed to have spinal column instability until such an injury is

* See references 24, 32, 61, 80, 86, 97, 114, 119, 136.

† See references 2, 8, 20, 39, 43, 65, 68, 114, 120, 128.

excluded.^{23,94,104} All trauma patients should be “log-rolled” during the initial assessment, and the entire spine should be inspected and palpated for ecchymosis, soft tissue swelling, “step-offs,” and tenderness. Obviously, inability to move the extremities heightens the suspicion of spinal column injury, as should significant abdominal injuries and the “lap belt” sign—a large ecchymosis over the abdomen. Once one injury of the spine has been identified, the entire spine must be imaged, as injuries may have occurred at multiple levels.*

Every patient with a spinal injury requires a careful and thorough neurologic examination. If there is a neurologic deficit, it is important to determine whether the lesion is complete or incomplete. A complete lesion is defined as the absence of both motor and sensory function below the SCI. Spinal shock must have resolved before an injury can be classified as complete. Return of the bulbocavernosus reflex indicates that the S3–4 region of the conus medullaris of the spinal cord is both physiologically and anatomically functional and spinal shock has resolved. In 99 percent of patients the bulbocavernosus reflex returns within 24 hours.¹²⁹ The presence of some neurologic function below the level of injury defines the injury as incomplete. Incomplete lesions have a better prognosis for recovery. Sacral sparing may be the only evidence of an incomplete lesion at the time of initial examination. Sacral sparing is evidenced by perianal sensation, voluntary rectal motor function, and great toe flexor activity. These findings indicate continued function of the lower sacral motor neurons and their connections to the cerebral cortex, and improve the prognosis for recovery. Conversely, absence of these sacral nerve functions may be the only finding in a patient with an injury to the conus medullaris or cauda equina. Thus, a complete examination of the patient with an SCI must include an assessment of these functions. The American Spinal Injury Association (ASIA) has produced an evaluation form to help ensure a complete initial assessment for the patient with an SCI (Fig. 40–20). Another important evaluation at the time of initial assessment is the degree of functional deficit. The ASIA recommends using a modified version of the scale, described by Frankel (Fig. 40–21).⁴¹

Thoracic or lumbar spine injury from minor trauma should raise the suspicion of pathologic fracture. These injuries are most typically compression fractures, and the bone is usually obviously pathologic. Gaucher’s disease, all of the mucopolysaccharidoses, osteogenesis imperfecta, idiopathic osteoporosis, metastatic neuroblastoma, Ewing’s sarcoma, and leukemia may all manifest with back pain and multiple compression fractures (Fig. 40–22).^{102,127}

RADIOGRAPHIC FINDINGS

Radiographic imaging begins with a careful assessment of AP radiographs for clues to spinal column injury, such as shortening of vertebral height, interpedicular widening, or asymmetry of the spinous process. Lateral radiographs often reveal the nature of the injury. They are particularly helpful for identifying injuries sustained from extension forces. CT defines the three-dimensional anatomy, including the extent of canal involvement. CT with sagittal reconstructions can be helpful in assessing areas that are difficult to see on plain

radiographs, such as the cervicothoracic junction and upper thoracic spine (Fig. 40–23). However, CT without sagittal reconstructions may be of limited value in many injuries, including seat belt injuries, as the injury is in the axial plane and can be difficult to appreciate on axial CT scans.⁵¹ MRI is the single best imaging tool for the traumatically injured spine. It provides direct information regarding the cord, canal, intervertebral disk, and posterior ligamentous structures. It is important to realize, however, that false positive and false negative MRI studies do occur. Recently, MRI findings at the time of injury have been correlated with functional neurologic outcome.^{11,57,89}

CLASSIFICATION

We use Denis’s five-part classification of spinal column injuries (Table 40–1).³¹ He classified spinal injuries as minor or major, and then subdivided major injuries into four classes. Minor injuries include fractures of the spinous and transverse processes, facets, and pars interarticularis. Major injuries include compression fractures, burst fractures, seat belt injuries, and fracture dislocations. Compression fractures represent failure of only the anterior column. There is no loss of posterior vertebral body height. The intact middle column is pathognomonic for compression fractures. Burst fractures result from failure of the anterior and middle columns in flexion; the posterior column remains intact. A lateral radiograph will reveal fracture of the posterior wall, loss of posterior vertebral body height, and tilting of one or both end-plates. Retropulsion of fragments into the canal may be difficult to appreciate on the lateral radiograph and is best seen on CT scans. The AP radiograph will show loss of vertebral body height and a widened interpedicular distance. Seat belt injuries are the result of a flexion-distraction force. Both the posterior and middle columns fail in tension; the anterior column may remain intact or may fail in compression. Seat belt injuries are further subdivided based on the location (through bone or ligament) of the posterior and middle column injury and on whether both columns are injured at the same level or at adjacent levels (Fig. 40–24). Fracture dislocations compose the last and most unstable class of thoracic and lumbar injuries. These injuries represent failure of all three columns in compression, tension, rotation, or shear.

TREATMENT

Treatment options for thoracic and lumbar spine injuries include symptomatic treatment with reassurance, brace or cast immobilization, and spinal fusion with or without decompression.

Nonoperative Treatment. Nonoperative treatment is appropriate for all minor injuries (fractures of the spinous and transverse processes, facets, and pars interarticularis), all compression fractures, “bony” seat belt injuries, and many burst fractures. Minor fractures usually require nothing more than symptomatic treatment. The most important aspect of these injuries is to realize they are frequently the result of high-energy trauma and consequently may be associated with other, often intra-abdominal, injuries. Patients with minor fractures may be treated with a few days of

* See references 14, 23, 51, 60, 94, 115, 136.

STANDARD NEUROLOGICAL CLASSIFICATION OF SPINAL CORD INJURY

MOTOR

KEY MUSCLES

	R	L
C2		
C3		
C4		
C5		
C6		
C7		
C8		
T1		
T2		
T3		
T4		
T5		
T6		
T7		
T8		
T9		
T10		
T11		
T12		
L1		
L2		
L3		
L4		
L5		
S1		
S2		
S3		
S4-5		

Elbow flexors

Wrist extensors

Elbow extensors

Finger flexors (distal phalanx of middle finger)

Finger abductors (little finger)

0 = total paralysis

1 = palpable or visible contraction

2 = active movement, gravity eliminated

3 = active movement, against gravity

4 = active movement, against some resistance

5 = active movement, against full resistance

NT = not testable

Voluntary anal contraction (Yes/No)

TOTALS + = **MOTOR SCORE**

(MAXIMUM) (50) (50) (100)

SENSORY

KEY SENSORY POINTS

LIGHT TOUCH

	R	L
C2		
C3		
C4		
C5		
C6		
C7		
C8		
T1		
T2		
T3		
T4		
T5		
T6		
T7		
T8		
T9		
T10		
T11		
T12		
L1		
L2		
L3		
L4		
L5		
S1		
S2		
S3		
S4-5		

PIN PRICK

	R	L
C2		
C3		
C4		
C5		
C6		
C7		
C8		
T1		
T2		
T3		
T4		
T5		
T6		
T7		
T8		
T9		
T10		
T11		
T12		
L1		
L2		
L3		
L4		
L5		
S1		
S2		
S3		
S4-5		

0 = absent
1 = impaired
2 = normal
NT = not testable

Any anal sensation (Yes/No)

TOTALS + = **PIN PRICK SCORE** (max: 112)

+ = **LIGHT TOUCH SCORE** (max: 112)

(MAXIMUM) (56) (56) (56) (56)

NEUROLOGICAL LEVELS

The most caudal segment with normal function

R L

SENSORY

MOTOR

COMPLETE OR INCOMPLETE?

Incomplete = Any sensory or motor function in S4-5

ASIA IMPAIRMENT SCALE

ZONE OF PARTIAL PRESERVATION

Partially innervated segments

R L

SENSORY

MOTOR

This form may be copied freely but should not be altered without permission from the American Spinal Injury Association.

FIGURE 40-20 ASIA form for documentation of acute spinal cord injury. (From Standard Neurological Classification of Spinal Cord Injury, Version 4p, GHC 1996, published by the American Spinal Injury Association.)

bedrest, followed by a gradual return to normal activities. Bracing is not required, although a simple lumbar corset may afford significant pain relief. If the patient is treated on an outpatient basis, it is important to realize that the retroperitoneal hematoma associated with these injuries in the lumbar spine may produce a significant ileus. Patients should be advised accordingly.^{49,61}

Compression fractures can also be treated with simple conservative measures. Most patients with compression fractures are more comfortable with an extension brace.^{51,61,73} Studies have shown no difference between bedrest and casting. Regardless of treatment, most patients are symptom-free within 2 weeks.^{65,68,92} Anterior vertebral height may be restored through remodeling, particularly in younger children.^{4,66,73,102} Chance fractures that are entirely through bone

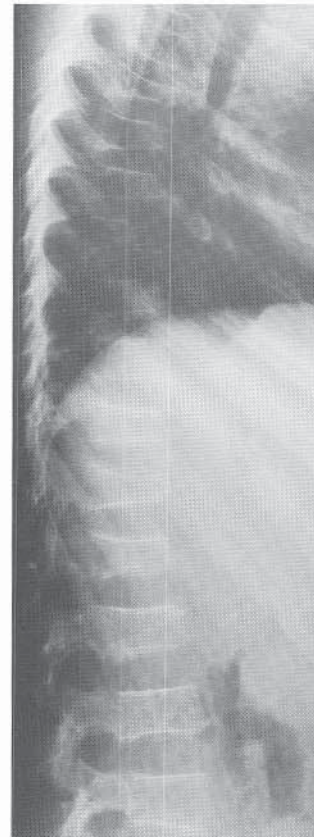


FIGURE 40–22 Lateral radiograph of a patient who presented with back pain. Note the multiple compression fractures. A complete blood cell count revealed acute lymphoblastic leukemia.

Functional Independence Measure (FIM)		
7	Complete Independence (Timely, Safely)	No Helper
6	Modified Independence (Device)	No Helper
LEVELS	Modified Dependence	Helper
	5 Supervision	
	4 Minimal Assist (Subject = 75%+)	
	3 Moderate Assist (Subject = 50%+)	
	Complete Dependence	
2 Maximal Assist (Subject = 25%+)		
1	Total Assist (Subject = 0%+)	
		ADMIT DISCH
Self Care		
A. Eating	<input type="checkbox"/>	<input type="checkbox"/>
B. Grooming	<input type="checkbox"/>	<input type="checkbox"/>
C. Bathing	<input type="checkbox"/>	<input type="checkbox"/>
D. Dressing-Upper Body	<input type="checkbox"/>	<input type="checkbox"/>
E. Dressing-Lower Body	<input type="checkbox"/>	<input type="checkbox"/>
F. Toileting	<input type="checkbox"/>	<input type="checkbox"/>
Sphincter Control		
G. Bladder Management	<input type="checkbox"/>	<input type="checkbox"/>
H. Bowel Management	<input type="checkbox"/>	<input type="checkbox"/>
Mobility		
Transfer:		
I. Bed, Chair, Wheelchair	<input type="checkbox"/>	<input type="checkbox"/>
J. Toilet	<input type="checkbox"/>	<input type="checkbox"/>
K. Tub, Shower	<input type="checkbox"/>	<input type="checkbox"/>
Locomotion		
L. Walk/wheelChair	W <input type="checkbox"/> C <input type="checkbox"/>	W <input type="checkbox"/> C <input type="checkbox"/>
M. Stairs	<input type="checkbox"/>	<input type="checkbox"/>
Communication		
N. Comprehension	A <input type="checkbox"/> V <input type="checkbox"/> V <input type="checkbox"/> N <input type="checkbox"/>	A <input type="checkbox"/> V <input type="checkbox"/> V <input type="checkbox"/> N <input type="checkbox"/>
O. Expression	<input type="checkbox"/>	<input type="checkbox"/>
Social Cognition		
P. Social Interaction	<input type="checkbox"/>	<input type="checkbox"/>
Q. Problem Solving	<input type="checkbox"/>	<input type="checkbox"/>
R. Memory	<input type="checkbox"/>	<input type="checkbox"/>
Total FIM	<input type="text"/>	<input type="text"/>
NOTE: Leave no blanks; enter 1 if patient not testable due to risk.		

FIGURE 40–21 Frankel scale of neurologic injury. (From Frankel HL, Hancock DO, Hyslop G, et al: The value of postural reduction in the initial management of closed injuries of the spine with paraplegia and tetraplegia. *Paraplegia*. 1969;7:179.)

(types A and C in Fig. 40–24) will heal with immobilization in a hyperextension cast.^{86,103,114,136,137}

Burst fractures in children occur most commonly in adolescents, and their management is similar to that for adults. Unfortunately, the management of burst fractures in adults continues to be debated.^{19,25,48,63,76,99} We treat the majority of burst fractures in neurologically intact patients with a period of bedrest, followed by 6 to 12 weeks in a cast or thoracolumbosacral orthosis. Each fracture must be treated on an individual basis, taking into consideration the patient's age and associated injuries as well as the amount of kyphosis, anterior collapse, and canal compromise. In general, more than 25 degrees of kyphosis (15 degrees if there is greater than 50 percent collapse of the anterior vertebral body) or 50 percent canal compromise are thought to preclude conservative treatment.¹³⁰ If canal compromise is the only surgical indication, it is important to bear in mind that several studies have documented reconstitution of the spinal canal with the conservative treatment of burst fractures.*

Operative Treatment. Indications for operative treatment include the presence of neurologic deficits, seat belt injuries with posterior ligamentous injuries, burst fractures not amenable to conservative treatment, and fracture dislocations. Operative treatment consists of spinal fusion with or without decompression. We recommend decompression for all patients with incomplete neurologic injury. We rarely perform

* See references 27, 29, 56, 71, 72, 81, 121, 141.

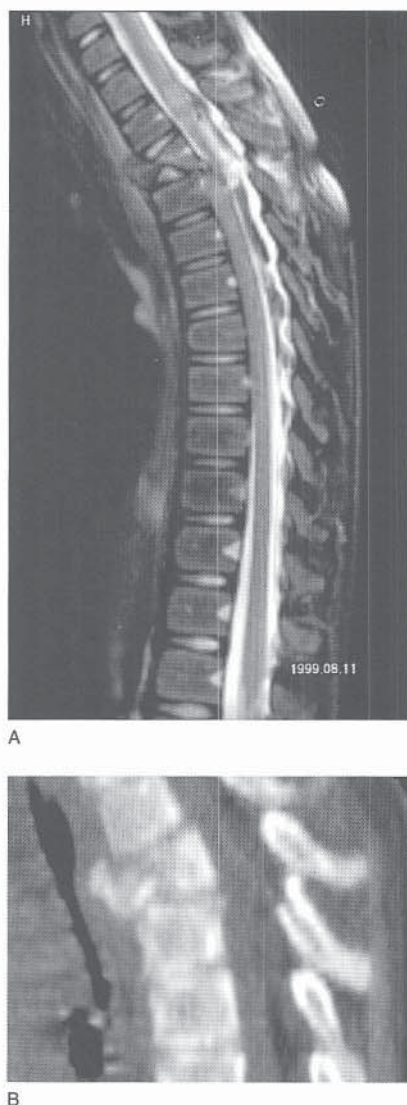


FIGURE 40-23 A, Sagittal MRI of a patient with T3-4 fracture dislocation. Plain lateral radiographs of this area are often difficult to interpret. B, CT scan with sagittal reconstruction demonstrates the bony deformity.

decompression in neurologically intact patients (aside from the decompression that occurs during reduction and stabilization). The decision to perform decompression in patients with complete lesions is made on an individual basis, realizing these patients have little potential for neurologic recovery. The optimal timing of surgical decompression is un-

TABLE 40-1 **Denis's Classification of Thoracic and Lumbar Spine Injuries**

Minor injuries
Articular process fracture
Transverse process fracture
Spinous process fracture
Pars interarticularis fracture
Major injuries
Compression fractures
Burst fractures
Seat belt injuries
Fracture dislocation

known. Ideally, decompression should be performed in the first 8 hours after injury. However, this is rarely possible. Advocates for early surgery stress the importance of prompt decompression, while others express concern that the surgical trauma can contribute to the edema-ischemia cycle.^{35,98,105,134} The surgical approach is determined by the nature of the fracture and the necessity to decompress the canal. The technique for the operative approach and instrumentation is the same as that previously described for anterior or posterior fusion for scoliosis (see Chapter 11, Scoliosis).

Most ligamentous seat belt injuries can be treated with simple posterior fusion. If the patient is large enough, we prefer to perform an instrumented fusion. If the patient is too small for even pediatric-size hook-and-rod systems, we perform a spinous process wiring and place the patient in a cast. (We also routinely immobilize patients treated with pediatric-size instrumentation with a cast. Older patients can frequently be managed with no immobilization or with a removable brace.) The length of the fusion is determined by the age of the patient and the extent of the injury. Young patients with a single-level injury may be treated with a two-level posterior fusion. Older patients with two-level injuries may require extension of the fusion two levels above and below (Fig. 40-25).

Burst fractures not amenable to conservative treatment and fracture dislocations may be managed with either anterior or posterior fusion. In general we prefer a posterior approach for reduction, decompression, and stabilization, although when circumstances dictate we will perform decompression and fusion through an anterior approach. Again, the fusion levels are determined by the age of the patient and the magnitude and location of the injury. Advocates of short-segment fusion argue that this technique alters less of the "normal" spine. The trade-off is increased stress within the fused segment and an increased risk of loss of correction and pseudarthrosis. Thus, the benefits of a shorter fusion segment must be weighed against the increased risk of non- or malunion. We believe restitution of appropriate sagittal balance is a more important factor in the long-term prognosis than the length of the fusion.^{10,13,37,82} Thus, we will extend the fusion to whatever level is required to provide a stable construct that can maintain sagittal balance (Fig. 40-26). Regardless of the surgical plan, it is important to realize that pathology that was not appreciated preoperatively is occasionally uncovered intraoperatively. Subtle laminar, transverse process, or facet fractures discovered intraoperatively will force the surgeon to be flexible with her preoperative plan. Additionally, the traumatically injured spine should be approached cautiously, as these subtle injuries may put undamaged neural elements at risk during exposure.

Pharmacologic Treatment of Neurologic Injuries. A number of pharmacologic agents have been used in an attempt to improve neurologic recovery after SCI. The goal of these agents is to interrupt the cycle of edema and ischemic injury. A number of drugs have shown promise in animal studies, including methylprednisolone, thyrotropin-releasing hormone, naloxone, and GM₁ ganglioside. However, only methylprednisolone has received widespread clinical attention. (GM₁ ganglioside has also shown clinical

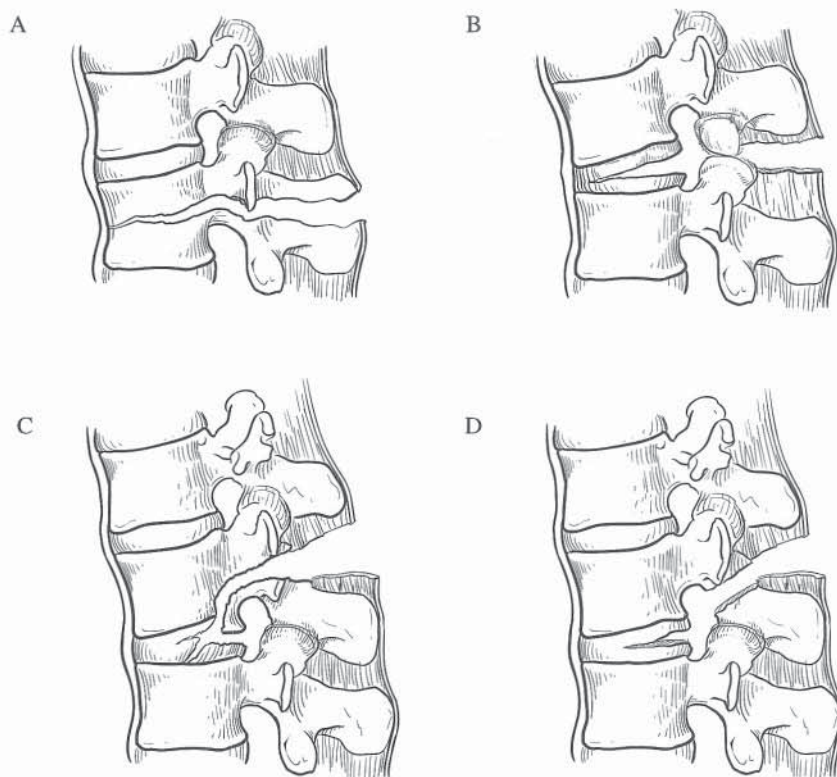


FIGURE 40-24 Flexion-distraction injuries commonly associated with seat belts. **A**, Single-level injury entirely through bone. **B**, Single-level injury entirely through soft tissue. **C**, Two-level injury primarily through bone. While the supra- and interspinous ligaments are disrupted, there is a bony component of the injury in both the posterior and middle columns. This fracture will heal with cast immobilization. **D**, Two-level soft tissue injury. Again, the supra- and interspinous ligaments are disrupted. Although there is a fracture through the pars intra-articularis, this injury is unlikely to heal with cast immobilization because of the soft tissue nature of the medial column injury.

success in a smaller study.*) In 1990, the Second National Acute Spinal Cord Injury Study (NASCIS-II) was the first multicenter study to report improved recovery in patients treated with a pharmacologic agent. Patients who received methylprednisolone within 8 hours of either complete or incomplete SCI had a better neurologic outcome than patients given placebo or naloxone. This study has been criticized for flaws in experimental design and incomplete data. Perhaps the most significant criticism of NASCIS-II is the lack of a functional outcome measure, making it impossible to determine whether the measured improvements were clinically relevant.^{30,42,46,47,100,143} In 1997 the results of NASCIS-III were published. All patients in this study received the 30 mg/kg bolus of methylprednisolone that was shown to be useful in NASCIS-II. Patients were then randomized to receive either 24 or 48 hours of methylprednisolone at 5.4 mg/kg/hr or to receive tirilazad, a lazeroid (an antioxidant), every 6 hours for 48 hours. Patients who received the initial bolus of methylprednisolone within 3 hours of injury had similar rates of motor recovery. In patients treated 3 to 8 hours after injury, those receiving methylprednisolone for 48 hours had the highest rates of recovery, statistically greater than those who received only 24 hours of methylprednisolone. Patients given tirilazad recovered at a rate between the 48- and 24-hour methylprednisolone groups.

Although the NASCIS-II investigators reported no difference in morbidity or mortality between groups, several authors have expressed concerns about the potentially adverse effects of massive steroid doses in polytraumatized patients.^{30,47,100} Despite concerns, most studies assessing the NASCIS-II protocol are similar to the report of Gerndt and

colleagues, who noted an increased incidence of pneumonia and a longer ICU stay, but no change in mortality and a decrease in the rehabilitation period.⁴⁷ The findings of NASCIS-III were similar: patients receiving 48 hours of methylprednisolone had higher rates of severe sepsis and pneumonia but no difference in mortality.¹⁶

We follow the recommendations of NASCIS-III. Patients with SCI who receive methylprednisolone within 3 hours of injury are maintained on the treatment regimen (5.4 mg/kg/hr) for 24 hours. When methylprednisolone therapy is begun 3 to 8 hours after injury, we continue it for 48 hours.^{16,30}

COMPLICATIONS

Complications following thoracic or lumbar spine injuries without neurologic deficit are uncommon. Growth arrest or deformity is unusual in children less than 10 years old because of their great remodeling capacity.^{4,61,65,68-70,113} This ability may be compromised if the end-plate is damaged, because it contains the physis. As Roaf has shown, end-plate damage is most likely to occur from the nucleus pulposus during axial loading.¹¹⁶ Patients treated operatively may develop any of the complications associated with spinal fusion, including infection (early or delayed), instrumentation failure, loss of correction, and pseudarthrosis.

Patients with spinal injuries producing a neurologic deficit frequently develop complications. Acute complications include pneumonia, sepsis, and pulmonary embolism. In a review of 28,692 pediatric trauma patients, deep vein thrombosis developed in six and pulmonary embolism in two. Both patients with pulmonary embolism had a spinal cord injury. The overall incidence of pulmonary embolism was 0.000069 percent. However, in patients with SCI the inci-

* See references 15, 30, 42-46, 59, 91, 100, 142, 143.

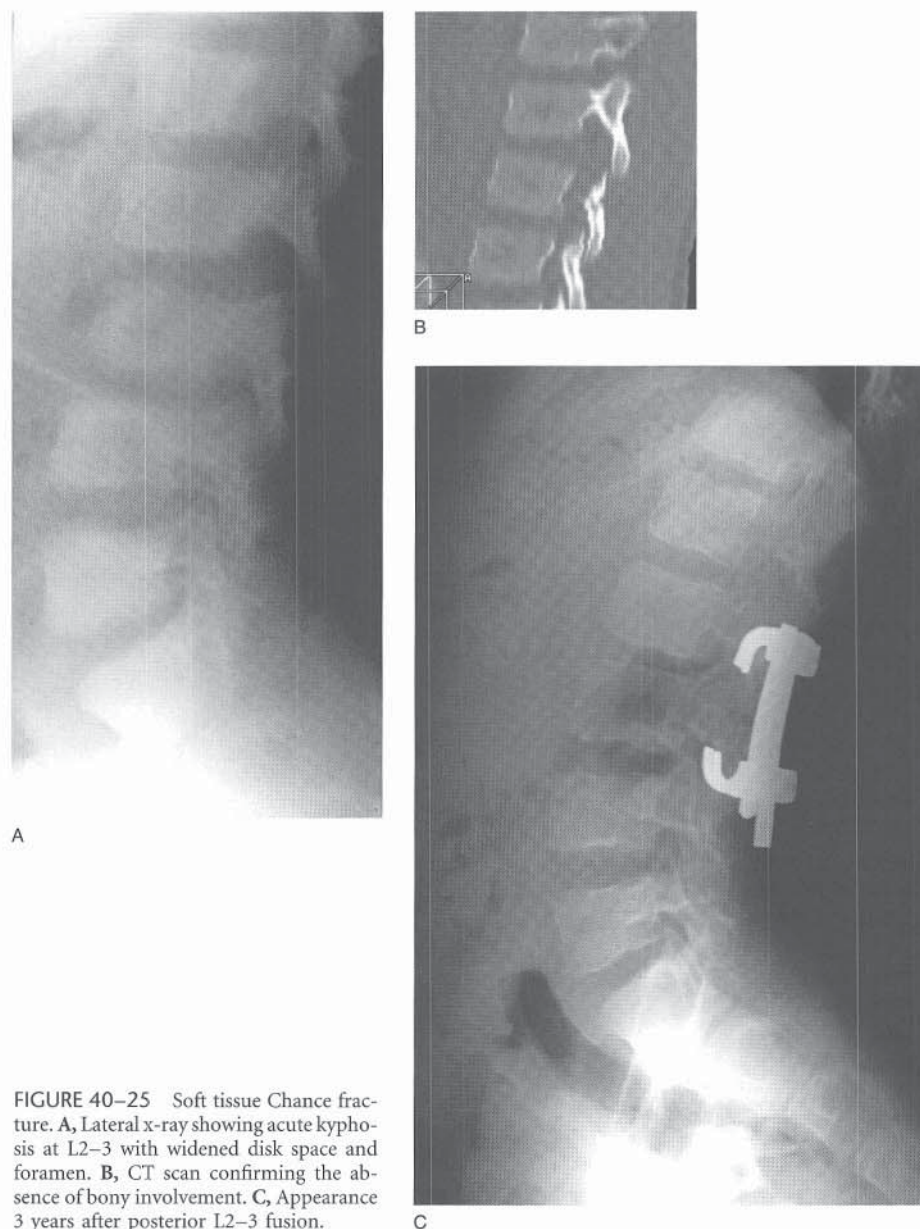


FIGURE 40-25 Soft tissue Chance fracture. A, Lateral x-ray showing acute kyphosis at L2-3 with widened disk space and foramen. B, CT scan confirming the absence of bony involvement. C, Appearance 3 years after posterior L2-3 fusion.

dence was 1.85 percent.⁹⁰ Although deep vein thrombosis and pulmonary embolism are rarely seen in children, some authors treat children with SCI with prophylaxis.⁹³

The long-term complications of patients with SCI are severe. These children potentially can develop all of the complications associated with myelomeningocele; in fact, we routinely refer all patients with SCI to our multidisciplinary spina bifida clinic. In addition to pulmonary and urologic problems, pressure sores, syringomyelia, and scoliosis may develop. Since the advent of MRI, syringomyelia has been noted quite frequently following SCI. Syringomyelia may present in the first few months after SCI or decades later. It is more common in patients with complete lesions. The most common presenting symptoms include pain, dyesthesia, increased tone, and weakness.* Scoliosis is the most

common complication of SCI in children. The incidence has been reported between 85 and 100 percent and is inversely correlated with age at the onset of paralysis and directly correlated with the location of the lesion (i.e., patients with higher, more cephalad lesion are more likely to develop scoliosis). Brace treatment is ineffective in managing paralytic scoliosis but may help delay surgical treatment in the younger patient. If untreated, paralytic scoliosis can lead to sitting imbalance and pulmonary problems. It is best treated with posterior spinal fusion from the high thoracic spine to the sacrum. Younger patients in whom the crankshaft phenomenon is a concern and patients with large, stiff curves may benefit from anterior spinal release and fusion (Fig. 40-27).*

* See references 36, 40, 53, 78, 109, 110, 117, 125.

* See references 6, 12, 17, 28, 38, 83, 96, 112, 131.

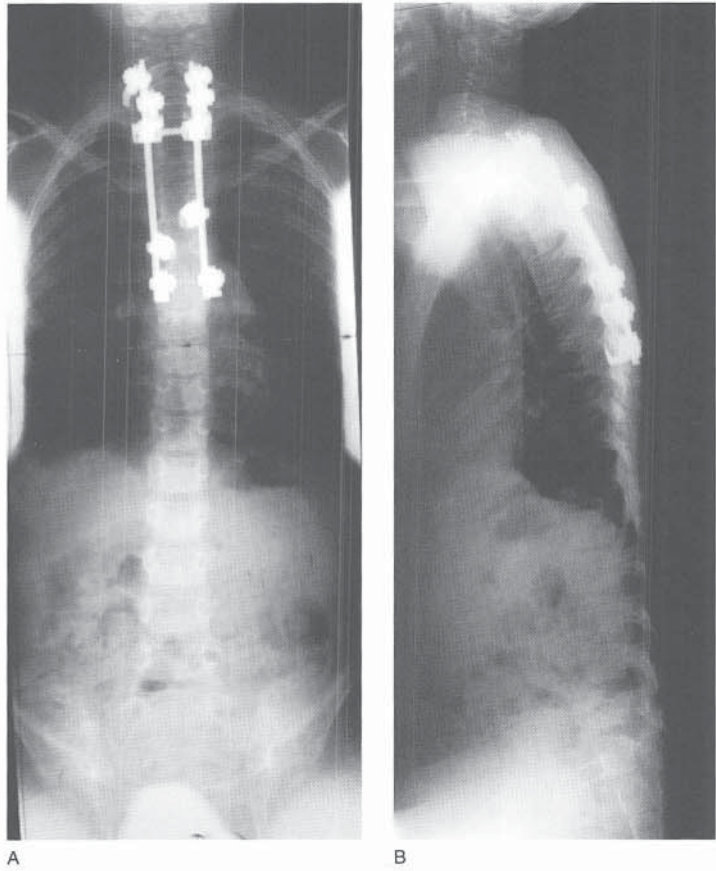


FIGURE 40-26 A and B, Postoperative sitting AP and lateral radiographs of the patient with T3-4 fracture dislocation shown in Figure 40-23. He was treated with posterior fusion from C7 to T8.

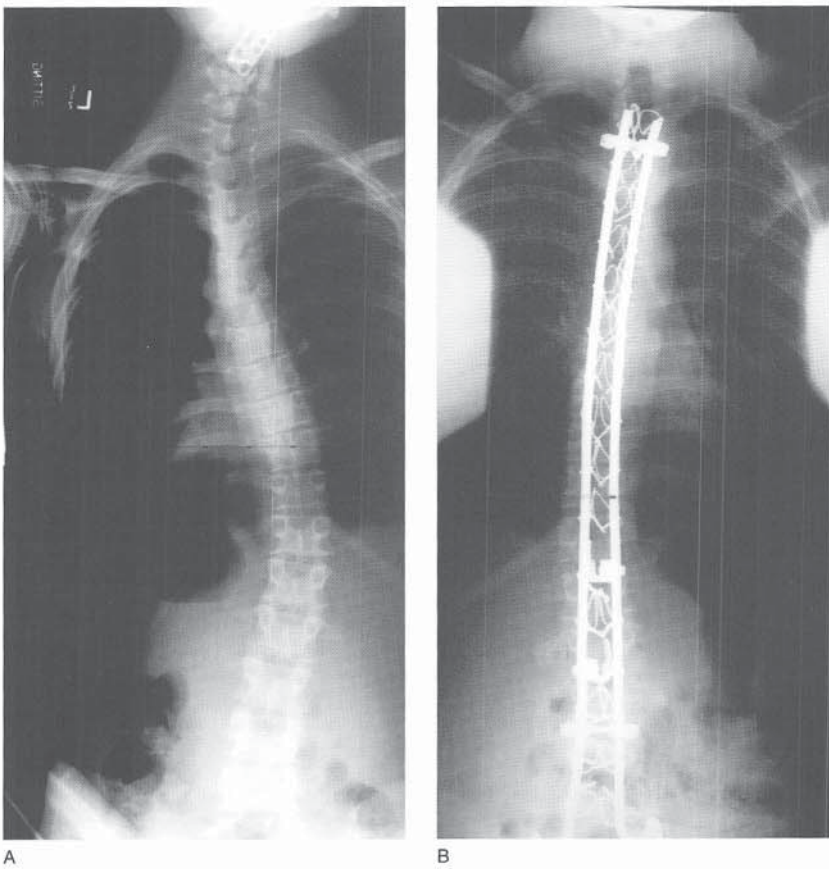


FIGURE 40-27 A, Sitting AP radiograph of a patient several years following C3-4 fracture dislocation. Note the long neuromuscular scoliosis and the anterior cervical plate. B, AP radiograph following posterior spinal fusion from T2 to the sacrum.

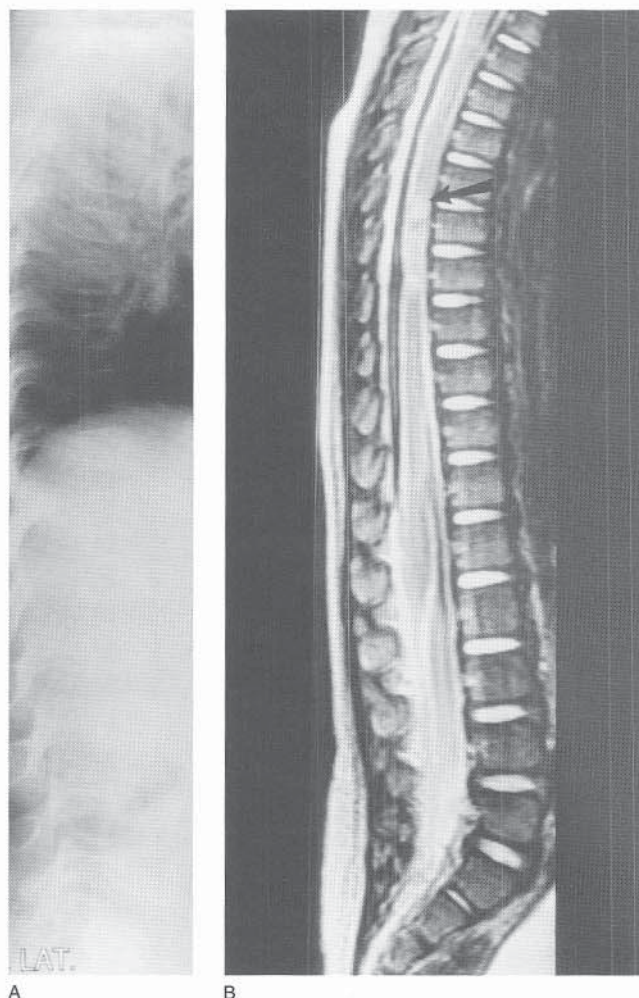


FIGURE 40–28 SCIWORA (spinal cord injury without radiographic abnormalities). A, Lateral radiograph of a patient involved in a high-speed motor vehicle accident who presented with bilateral lower extremity paralysis. There is no abnormality. B, Midsagittal MRI. Note the increased signal within the spinal cord (arrow). There is no ligamentous injury.

SPINAL CORD INJURY WITHOUT RADIOGRAPHIC ABNORMALITY

SCI without radiographic abnormality (SCIWORA) usually occurs only in children. (Adults may sustain cord injury without fracture but usually have a ligamentous injury noted on MRI.⁵⁵) The term SCIWORA as applied to children was coined by Pang and Wilberger in 1982.¹⁰⁷ The pathogenesis of SCIWORA lies in the fact that the spinal column is more elastic than the spinal cord. Thus the spinal column can stretch beyond the elastic limit of the neural elements.^{79,104,106,107} When the deforming force is removed, the spinal column returns to its normal state, but the cord is left permanently damaged (Fig. 40–28A). SCIWORA has been reported to account for 15 to 35 percent of SCIs in children.^{34,58,79,104,118} SCIWORA may occur at any age and at any location, but it is most common in the cervical spine. In Pang and Wilberger's original series, half of the children presented with a delayed onset of paralysis of up to 4 days.¹⁰⁷

The characteristics of SCIWORA vary according to age.

SCIWORA is more common in young children, who frequently have complete lesions of the cervical spine, with a poor prognosis for neurologic recovery. The disproportionately large head of young children probably serves as the force that deforms the cervical spine beyond the physiologic limit of the cervical cord. Adolescents are more likely to have incomplete lesions, with a better prognosis for recovery.^{34,55,104,106,107}

SCIWORA is a diagnosis of exclusion. Therefore, the initial evaluation and management are the same as for any child with an SCI. Following initial assessment and resuscitation, plain radiographs and, if indicated, CT scans are obtained. If the preliminary studies fail to reveal pathology, MRI is usually performed. MRI is diagnostic, revealing abnormal signal in the cord in the absence of changes in the spinal column (see Fig. 40–28B).³⁴ Once the diagnosis has been established, the entire spine should be imaged and the patient should be treated with spine precautions until awake and alert. Patients seen within 8 hours of injury should be treated with methylprednisolone per the NASCIS-III guidelines. Once the child is awake, alert, and cooperative, dynamic flexion-extension radiographs should be obtained to ensure there is no subtle ligamentous pathology. Immobilization of patients with SCIWORA may seem unnecessary, as there is, by definition, no abnormality of the spinal column. However, Pang and Pollack noted that eight (15 percent) of 55 children with SCIWORA suffered a second SCIWORA 3 days to 10 weeks after their initial injury.¹⁰⁶ They hypothesized that the initial injury made the spine “incipiently unstable” and susceptible to additional, often more severe, neurologic trauma. We believe that the high incidence of “secondary SCIWORA” warrants immobilization of these patients for 3 months following injury.^{104,106,107,111}

REFERENCES

Traumatic Injuries of the Thoracic and Lumbar Spine

1. Abad C, Marti M, Marrero L, et al: [Paraplegia following surgical repair of a ductus and of a coarctation of the aorta in childhood]. *Cir Pediatr* 1993;6:84.
2. Abel MS: Transverse posterior element fractures associated with torsion. *Skeletal Radiol* 1989;17:556.
3. Agran PF, Dunkle DE, Winn DG: Injuries to a sample of seatbelted children evaluated and treated in a hospital emergency room. *J Trauma* 1987;27:58.
4. Akbarnia BA: Pediatric spine fractures. *Orthop Clin North Am* 1999;30:521.
5. Anderson PA, Henley MB, Rivara FP, et al: Flexion distraction and Chance injuries to the thoracolumbar spine. *J Orthop Trauma* 1991;5:153.
6. Apple DF Jr, Anson CA, Hunter JD, et al: Spinal cord injury in youth. *Clin Pediatr (Phila)* 1995;34:90.
7. Athey AM: A 3-year-old with spinal cord injury without radiographic abnormality (SCIWORA). *J Emerg Nurs* 1991;17:380.
8. Aufdermaur M: Spinal injuries in juveniles: necropsy findings in twelve cases. *J Bone Joint Surg* 1974;56-B:513.
9. Banniza von Bazan UK, Paeslack V: Scoliotic growth in children with acquired paraplegia [proceedings]. *Paraplegia* 1977;15:65.
10. Boachie-Adjei O, Dendrinos GK, Ogilvie JW, et al: Management of adult spinal deformity with combined anterior-posterior arthrodesis and Luque-Galveston instrumentation. *J Spinal Disord* 1991;4:131.
11. Bondurant FJ, Cotler HB, Kulkarni MV, et al: Acute spinal cord injury: a study using physical examination and magnetic resonance imaging. *Spine* 1990;15:161.
12. Bonnett C, Brown JC, Perry J, et al: Evolution of treatment of paralytic

- scoliosis at Rancho Los Amigos Hospital. *J Bone Joint Surg* 1975; 57-A:206.
13. Booth KC, Bridwell KH, Lenke LG, et al: Complications and predictive factors for the successful treatment of flatback deformity (fixed sagittal imbalance). *Spine* 1999;24:1712.
 14. Born CT, Ross SE, Iannaccone WM, et al: Delayed identification of skeletal injury in multisystem trauma: the "missed" fracture. *J Trauma* 1989;29:1643.
 15. Bracken MB: Pharmacological treatment of acute spinal cord injury: current status and future projects. *J Emerg Med* 1993;1:43.
 16. Bracken MB, Shepard MJ, Holford TR, et al: Administration of methylprednisolone for 24 or 48 hours or tiludazone mesylate for 48 hours in the treatment of acute spinal cord injury: results of the Third National Acute Spinal Cord Injury Randomized Controlled Trial. National Acute Spinal Cord Injury Study. *JAMA* 1997;277:1597.
 17. Bridwell KH, O'Brien MF, Lenke LG, et al: Posterior spinal fusion supplemented with only allograft bone in paralytic scoliosis: does it work? *Spine* 1994;19:2658.
 18. Campbell J, Bonnett C: Spinal cord injury in children. *Clin Orthop* 1975;112:114.
 19. Carl AL, Tranmer BI, Sachs BL: Anterolateral dynamized instrumentation and fusion for unstable thoracolumbar and lumbar burst fractures. *Spine* 1997;22:686.
 20. Chance GQ: Note on a type of flexion fracture of the spine. *Br J Radiol* 1948;21:432.
 21. Cheng JC, Aguilar J, Leung PC: Hip reconstruction for femoral head loss from septic arthritis in children: a preliminary report. *Clin Orthop* 1995;314:214.
 22. Choi JU, Hoffman HJ, Hendrick EB, et al: Traumatic infarction of the spinal cord in children. *J Neurosurg* 1986;65:608.
 23. Cramer KE: The pediatric polytrauma patient. *Clin Orthop* 1995; 318:125.
 24. Crawford AH: Operative treatment of spine fractures in children. *Orthop Clin North Am* 1990;21:325.
 25. Cresswell TR, Marshall PD, Smith RB: Mechanical stability of the AO internal spinal fixation system compared with that of the Hartshill rectangle and sublaminar wiring in the management of unstable burst fractures of the thoracic and lumbar spine. *Spine* 1998;23:111.
 26. Darwish H, Archer C, Modin J: The anterior spinal artery collateral in coarctation of the aorta: a clinical angiographic correlation. *Arch Neurol* 1979;36:240.
 27. de Klerk LW, Fontijne WP, Stijnen T, et al: Spontaneous remodeling of the spinal canal after conservative management of thoracolumbar burst fractures. *Spine* 1998;23:1057.
 28. Dearolf WW III, Betz RR, Vogel LC, et al: Scoliosis in pediatric spinal cord-injured patients. *J Pediatr Orthop* 1990;10:214.
 29. Deburge A, Blamoutier A: [Remodeling of the spinal canal after comminuted fracture of the spine: apropos of a case]. *Rev Chir Orthop Reparatrice Appar Mot* 1992;78:124.
 30. Delamarter RB, Coyle J: Acute management of spinal cord injury. *J Am Acad Orthop Surg* 1999;7:166.
 31. Denis F: The three column spine and its significance in the classification of acute thoracolumbar spinal injuries. *Spine* 1983;8:817.
 32. Diamond P, Hansen CM, Christofersen MR: Child abuse presenting as a thoracolumbar spinal fracture dislocation: a case report. *Pediatr Emerg Care* 1994;10:83.
 33. Dickman CA, Rekte HL, Sonntag VK, et al: Pediatric spinal trauma: vertebral column and spinal cord injuries in children. *Pediatr Neurosci* 1989;15:237.
 34. Dickman CA, Zabranski JM, Hadley MN, et al: Pediatric spinal cord injury without radiographic abnormalities: report of 26 cases and review of the literature. *J Spinal Disord* 1991;4:296.
 35. Duh MS, Shepard MJ, Wilberger JE, et al: The effectiveness of surgery on the treatment of acute spinal cord injury and its relation to pharmacological treatment. *Neurosurgery* 1994;35:240.
 36. el Masry WS, Biyani A: Incidence, management, and outcome of post-traumatic syringomyelia. In memory of Mr Bernard Williams. *J Neurol Neurosurg Psychiatry* 1996;60:141.
 37. Farcy JP, Schwab FJ: Management of flatback and related kyphotic decompensation syndromes. *Spine* 1997;22:2452.
 38. Farley FA, Hensinger RN, Herzenberg JE: Cervical spinal cord injury in children. *J Spinal Disord* 1992;5:410.
 39. Ferrandez L, Usabiaga J, Curto JM, et al: Atypical multivertebral fracture due to hyperextension in an adolescent girl: a case report. *Spine* 1989;14:645.
 40. Foo D, Bignami A, Rossier AB: A case of post-traumatic syringomyelia: neuropathological findings after 1 year of cystic drainage. *Paraplegia* 1989;27:63.
 41. Frankel HL, Hancock DO, Hyslop G, et al: The value of postural reduction in the initial management of closed injuries of the spine with paraplegia and tetraplegia. I. Paraplegia 1969;7:179.
 42. Gabler C, Maier R: [Clinical experiences and results of high-dosage methylprednisolone therapy in spinal cord trauma 1991 to 1993]. *Unfallchirurgie* 1995;21:20.
 43. Geisler FH: GM-1 ganglioside and motor recovery following human spinal cord injury. *J Emerg Med* 1993;1:49.
 44. Geisler FH: Clinical trials of pharmacotherapy for spinal cord injury. *Ann NY Acad Sci* 1998;845:374.
 45. Geisler FH, Dorsey FC, Coleman WP: Recovery of motor function after spinal-cord injury: a randomized, placebo-controlled trial with GM-1 ganglioside. *N Engl J Med* 1991;324:1829.
 46. George ER, Scholten DJ, Buechler CM, et al: Failure of methylprednisolone to improve the outcome of spinal cord injuries. *Am Surg* 1995;61:659.
 47. Gerndt SJ, Rodriguez JL, Pawlik JW, et al: Consequences of high-dose steroid therapy for acute spinal cord injury. *J Trauma* 1997;42:279.
 48. Ghanayem AJ, Zdeblick TA: Anterior instrumentation in the management of thoracolumbar burst fractures. *Clin Orthop* 1997;335:89.
 49. Gilsanz V, Miranda J, Cleveland R, et al: Scoliosis secondary to fractures of the transverse processes of lumbar vertebrae. *Radiology* 1980;134:627.
 50. Glasauer FE, Cares HL: Traumatic paraplegia in infancy. *JAMA* 1972;219:38.
 51. Glass RB, Sivit CJ, Sturm PF, et al: Lumbar spine injury in a pediatric population: difficulties with computed tomographic diagnosis. *J Trauma* 1994;37:815.
 52. Glassman SD, Johnson JR, Holt RT: Seatbelt injuries in children. *J Trauma* 1992;33:882.
 53. Goldstein B, Hammond MC, Stiens SA, et al: Posttraumatic syringomyelia: profound neuronal loss, yet preserved function. *Arch Phys Med Rehabil* 1998;79:107.
 54. Grabb PA, Pang D: Magnetic resonance imaging in the evaluation of spinal cord injury without radiographic abnormality in children. *Neurosurgery* 1994;35:406.
 55. Gupta SK, Rajeev K, Khosla VK, et al: Spinal cord injury without radiographic abnormality in adults. *Spinal Cord* 1999;37:726.
 56. Ha KI, Han SH, Chung M, et al: A clinical study of the natural remodeling of burst fractures of the lumbar spine. *Clin Orthop* 1996;323:210.
 57. Hackney DB, Finkelstein SD, Hand CM, et al: Postmortem magnetic resonance imaging of experimental spinal cord injury: magnetic resonance findings versus in vivo functional deficit. *Neurosurgery* 1994;35:1104.
 58. Hadley MN, Zabranski JM, Browner CM, et al: Pediatric spinal trauma: review of 122 cases of spinal cord and vertebral column injuries. *J Neurosurg* 1988;68:18.
 59. Hamilton AJ, McBlack P, Carr D: Contrasting actions of naloxone in experimental spinal cord trauma and cerebral ischemia: a review. *Neurosurgery* 1985;17:845.
 60. Harrington T, Barker B: Multiple trauma associated with vertebral injury. *Surg Neurol* 1986;26:149.
 61. Hegenbarth R, Ebel KD: Roentgen findings in fractures of the vertebral column in childhood examination of 35 patients and its results. *Pediatr Radiol* 1976;5:34.
 62. Herkowitz HN, Samberg LC: Vertebral column injuries associated with tobogganing. *J Trauma* 1978;18:806.
 63. Hitchon PW, Torner JC, Haddad SF, et al: Management options in thoracolumbar burst fractures. *Surg Neurol* 1998;49:619.
 64. Hoffman MA, Spence LJ, Wesson DE, et al: The pediatric passenger: trends in seatbelt use and injury patterns. *J Trauma* 1987;27:974.
 65. Horal J, Nachemson A, Scheller S: Clinical and radiological long term follow-up of vertebral fractures in children. *Acta Orthop Scand* 1972;43:491.
 66. Horsley MW, Taylor TK: Spontaneous correction of a traumatic kyphosis after posterior spinal fusion in an infant. *J Spinal Disord* 1997;10:256.
 67. Howell JM, McFarling DA, Chisholm CD: Ischemic injury to the spinal cord as a cause of transient paraplegia. *Am J Emerg Med* 1987;5:217.

68. Hubbard DD: Injuries of the spine in children and adolescents. *Clin Orthop* 1974;100:56.
69. Hubbard DD: Fractures of the dorsal and lumbar spine. *Orthop Clin North Am* 1976;7:605.
70. Jackson RW: Surgical stabilization of the spine. *Paraplegia* 1975;13:71.
71. Johnsson R, Herrlin K, Hagglund G, et al: Spinal canal remodeling after thoracolumbar fractures with intraspinal bone fragments: 17 cases followed 1–4 years. *Acta Orthop Scand* 1991;62:125.
72. Karlsson MK, Hasserius R, Sundgren P, et al: Remodeling of the spinal canal deformed by trauma. *J Spinal Disord* 1997;10:157.
73. Kathrein A, Huber B, Waldegger M, et al: [Management of injuries of the thoracic and lumbar vertebrae in children]. *Orthopade* 1999;28:441.
74. Kewalramani LS, Kraus JF, Sterling HM: Acute spinal-cord lesions in a pediatric population: epidemiological and clinical features. *Paraplegia* 1980;18:206.
75. Kewalramani LS, Tori JA: Spinal cord trauma in children: neurologic patterns, radiologic features, and pathomechanics of injury. *Spine* 1980;5:11.
76. Kifune M, Panjabi MM, Liu W, et al: Functional morphology of the spinal canal after endplate, wedge, and burst fractures. *J Spinal Disord* 1997;10:457.
77. Knight RQ, Devanny JR: Spinal cord ischemia and paraplegia in the multiple-trauma patient with aortic arch injury: case report. *Spine* 1987;12:624.
78. Kramer KM, Levine AM: Posttraumatic syringomyelia: a review of 21 cases. *Clin Orthop* 1997;334:190.
79. Kriss VM, Kriss TC: SCIWORA (spinal cord injury without radiographic abnormality) in infants and children. *Clin Pediatr (Phila)* 1996;35:119.
80. Kumar A, Wood GW II, Whittle AP: Low-velocity gunshot injuries of the spine with abdominal viscus trauma. *J Orthop Trauma* 1998;12:514.
81. Kuner EH, Schlickewei W, Hauser U, et al: [Reconstruction of open width of the spinal canal by internal fixator instrumentation and remodeling]. *Chirurg* 1996;67:531.
82. Lagrone MO, Bradford DS, Moe JH, et al: Treatment of symptomatic flatback after spinal fusion. *J Bone Joint Surg* 1988;70-A:569.
83. Lancourt JE, Dickson JH, Carter RE: Paralytic spinal deformity following traumatic spinal-cord injury in children and adolescents. *J Bone Joint Surg* 1981;63-A:47.
84. Le Blanc HJ, Nadell J: Spinal cord injuries in children. *Surg Neurol* 1974;2:411.
85. Le Gay DA, Petrie DP, Alexander DI: Flexion-distraction injuries of the lumbar spine and associated abdominal trauma. *J Trauma* 1990;30:436.
86. Letts M, Davidson D, Fleuriu-Chateau P, et al: Seat belt fracture with late development of an enterocolic fistula in a child: a case report. *Spine* 1999;24:1151.
87. Leventhal HR: Birth injuries of the spinal cord. *J Pediatr* 1960;56:447.
88. Lim LH, Lam LK, Moore MH, et al: Associated injuries in facial fractures: review of 839 patients. *Br J Plast Surg* 1993;46:635.
89. Mascalchi M, Dal Pozzo G, Dini C, et al: Acute spinal trauma: prognostic value of MRI appearances at 0.5 T. *Clin Radiol* 1993;48:100.
90. McBride WJ, Gadowski GR, Keller MS, et al: Pulmonary embolism in pediatric trauma patients. *J Trauma* 1994;37:913.
91. McIntosh TK, Faden AI: Opiate antagonists in traumatic shock. *Ann Emerg Med* 1986;15:1462.
92. McPhee IB: Spinal fractures and dislocations in children and adolescents. *Spine* 1981;6:533.
93. Merli G, Crabbe S, Paluzzi RG, et al: Etiology, incidence, and prevention of deep vein thrombosis in acute spinal cord injury. *Arch Phys Med Rehabil* 1993;74:1199.
94. Metak G, Scherer MA, Dannohl C: [Missed injuries of the musculoskeletal system in multiple trauma: a retrospective study]. *Zentralbl Chir* 1994;119:88.
95. Meuli M, Sacher P, Lasser U, et al: Traumatic spinal cord injury: unusual recovery in 3 children. *Eur J Pediatr Surg* 1991;1:240.
96. Miladi LT, Ghanem IB, Draoui MM, et al: Iliosacral screw fixation for pelvic obliquity in neuromuscular scoliosis: a long-term follow-up study. *Spine* 1997;22:1722.
97. Miller JA, Smith TH: Seatbelt induced Chance fracture in an infant: case report and literature review. *Pediatr Radiol* 1991;21:575.
98. Mirza SK, Krengel WF III, Chapman JR, et al: Early versus delayed surgery for acute cervical spinal cord injury. *Clin Orthop* 1999;359:104.
99. Muller U, Berlemann U, Sledge J, et al: Treatment of thoracolumbar burst fractures without neurologic deficit by indirect reduction and posterior instrumentation: bisegmental stabilization with monosegmental fusion. *Eur Spine J* 1999;8:284.
100. Nesathurai S: Steroids and spinal cord injury: revisiting the NASCIS 2 and NASCIS 3 trials. *J Trauma* 1998;45:1088.
101. Odom JA, Brown CW, Messner DG: Tubing injuries. *J Bone Joint Surg* 1976;58-A:733.
102. Oliveri MB, Mautalen CA, Rodriguez Fuchs CA, et al: Vertebral compression fractures at the onset of acute lymphoblastic leukemia in a child. *Henry Ford Hosp Med J* 1991;39:45.
103. O'Neill MJ: Delayed-onset paraplegia from improper seat belt use. *Ann Emerg Med* 1994;23:1123.
104. Osenbach RK, Menezes AH: Spinal cord injury without radiographic abnormality in children. *Pediatr Neurosci* 1989;15:168.
105. Owen JH, Naito M, Bridwell KH, et al: Relationship between duration of spinal cord ischemia and postoperative neurologic deficits in animals. *Spine* 1990;15:846.
106. Pang D, Pollack IF: Spinal cord injury without radiographic abnormality in children: the SCIWORA syndrome. *J Trauma* 1989;29:654.
107. Pang D, Wilberger JE Jr: Spinal cord injury without radiographic abnormalities in children. *J Neurosurg* 1982;57:114.
108. Paulson JA: The epidemiology of injuries in adolescents. *Pediatr Ann* 1988;17:84.
109. Perrouin-Verbe B, Lenne-Aurier K, Robert R, et al: Post-traumatic syringomyelia and post-traumatic spinal canal stenosis: a direct relationship. Review of 75 patients with a spinal cord injury. *Spinal Cord* 1998;36:137.
110. Perrouin-Verbe B, Robert R, Lefort M, et al: [Post-traumatic syringomyelia]. *Neurochirurgie* 1999;1:58.
111. Pollina J, Li V: Tandem spinal cord injuries without radiographic abnormalities in a young child. *Pediatr Neurosurg* 1999;30:263.
112. Pouliquen JC, Beneux J, Pennecot GF: [The incidence of progressive scoliosis and kyphosis after fractures and dislocations of the spine in children]. *Rev Chir Orthop Reparatrice Appar Mot* 1978;64:487.
113. Povaz F: Behandlungsergebnisse und Prognose von Wirbelbrüchen bei Kindern. *Chirurg* 1969;40:30.
114. Reid AB, Letts RM, Black GB: Pediatric Chance fractures: association with intra-abdominal injuries and seatbelt use. *J Trauma* 1990;30:384.
115. Rinaldi I, Mullins WJ, Kretz WK, et al: Missed spinal fractures: a serious problem in the patient with multiple injuries. *Va Med Mon* 1975;102:305.
116. Roaf R: A study of the mechanics of spinal injuries. *J Bone Joint Surg* 1960;42-B:810.
117. Robinson LR, Little JW: Motor-evoked potentials reflect spinal cord function in post-traumatic syringomyelia. *Am J Phys Med Rehabil* 1990;69:307.
118. Ruge JR, Sinson GP, McLone DG, et al: Pediatric spinal injury: the very young. *J Neurosurg* 1988;68:25.
119. Ruggieri M, Kmarason AK, Pike M: Spinal cord insults in the prenatal, perinatal, and neonatal periods. *Dev Med Child Neurol* 1999;41:311.
120. Rumball K, Jarvis J: Seat-belt injuries of the spine in young children. *J Bone Joint Surg* 1992;74-B:571.
121. Scapinelli R, Candiottio S: Spontaneous remodeling of the spinal canal after burst fractures of the low thoracic and lumbar region. *J Spinal Disord* 1995;8:486.
122. Scher AT: Trauma of the spinal cord in children. *S Afr Med J* 1976;50:2023.
123. Schossberger P: Vasculature of the spinal cord: a review. II. Clinical considerations. *Bull Los Angeles Neurol Soc* 1974;39:86.
124. Scalfani SJ, Florence LO, Phillips TF, et al: Lumbar arterial injury: radiologic diagnosis and management. *Radiology* 1987;165:709.
125. Sgouros S, Williams B: Management and outcome of posttraumatic syringomyelia. *J Neurosurg* 1996;85:197.
126. Shrosbree RD: Spinal cord injuries as a result of motorcycle accidents. *Paraplegia* 1978;16:102.
127. Sinha AK, Seki JT, Moreau G, et al: The management of spinal metastasis in children. *Can J Surg* 1997;40:218.
128. Smith WS, Kaufer H: Patterns and mechanisms of lumbar injuries associated with lab seat belts. *J Bone Joint Surg* 1969;51-A:239.

129. Stauffer ES: Diagnosis and prognosis of acute cervical spinal cord injury. *Clin Orthop* 1975;112:9.
130. Stauffer ES: The management of thoracolumbar junction fractures. In Stauffer ES (ed): *Thoracolumbar Spine Fractures Without Neurologic Deficit*, p 60. Rosemont, IL, American Academy of Orthopaedic Surgeons, 1993.
131. Stricker U, Moser H, Aebi M: Predominantly posterior instrumentation and fusion in neuromuscular and neurogenic scoliosis in children and adolescents. *Eur Spine J* 1996;5:101.
132. Sturm JT, Hines JT, Perry JF Jr: Thoracic spinal fractures and aortic rupture: a significant and fatal association. *Ann Thorac Surg* 1990; 50:931.
133. Sturm JT, Perry JF: Injuries associated with fractures of the transverse processes of the thoracic and lumbar vertebrae. *J Trauma* 1984; 24:597.
134. Tator CH, Fehlings MG, Thorpe K, et al: Current use and timing of spinal surgery for management of acute spinal surgery for management of acute spinal cord injury in North America: results of a retrospective multicenter study. *J Neurosurg* 1999;91:12.
135. Vanhulle C, Durand I, Tron P: [Paraplegia due to medullary ischemia after repair of coarctation of the aorta in an infant]. *Arch Pediatr* 1998;5:633.
136. Vedantam R, Crawford AH: Multiple noncontiguous injuries of the spine in a child: atlantooccipital dislocation and seat-belt injury of the lumbar spine. *Acta Orthop Belg* 1997;63:23.
137. Voss L, Cole PA, D'Amato C: Pediatric chance fractures from lapbelts: unique case report of three in one accident. *J Orthop Trauma* 1996; 10:421.
138. Wholey MH, Bruwer AJ, Baker HL Jr: The lateral roentgenogram of the neck, with comments on the atlanto-odontoid-basion relationship. *Radiology* 1958;71:350.
139. Woelfel GF, Moore EE, Cogbill TH, et al: Severe thoracic and abdominal injuries associated with lap-harness seatbelts. *J Trauma* 1984; 24:166.
140. Yashon D: Pathogenesis of spinal cord injury. *Orthop Clin North Am* 1978;9:247.
141. Yazici M, Atilla B, Tepe S, et al: Spinal canal remodeling in burst fractures of the thoracolumbar spine: a computerized tomographic comparison between operative and nonoperative treatment. *J Spinal Disord* 1996;9:409.
142. Yoon DH, Kim YS, Young W: Therapeutic time window for methylprednisolone in spinal cord injured rat. *Yonsei Med J (Korea)* 1999; 40:313.
143. Young W, Bracken MB: The Second National Acute Spinal Cord Injury Study. *J Neurotrauma* 1992;9:S397.