CHAPTER 36

# Spinal Injuries

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## PRINCIPLES

### Background and Importance

According to the National Spinal Cord Injury Statistical Center, motor vehicle collisions (MVCs) account for 37% of all spinal injuries.[1] Speeding, alcohol intoxication, and failure to use restraints are major risk factors. The next most common cause of spinal cord injury (SCI) is falls, followed by acts of violence (primarily gunshot wounds) and sporting activities. Approximately 80% of victims are male, and the average age at injury is 42.6 years. The lifetime cost to care for SCI victims ranges from $1 million if older than 50 years, with incomplete motor function, to over $4 million for those younger than 25 years, with complete paraplegia. The total cost to society from lifelong medical expenses and lost productivity for all ages and types of spinal injuries is estimated to be more than $5 billion. The devastating emotional and psychological impact is incalculable.

Injuries of the soft tissues supporting the cervical spine can result in chronic pain and disability. The term *whiplash-associated disorder* (WAD) has been used to describe these injuries because of the flexion-extension movement of the neck that results from rear-end MVCs, the most common cause of a WAD. Due to the large number of people sustaining these injuries, the annual costs associated with a WAD exceed $230 billion, which is more than the combined costs associated with spinal cord and brain injuries caused by MVCs.[2]

### Anatomy and Physiology

The human spine consists of 33 bony vertebrae—7 cervical, 12 thoracic, 5 lumbar, 5 sacral (fused into one), and 4 coccygeal (usually fused into one; Fig. 36.1). These 26 individual units are separated from one another by flexible intervertebral disks and connected to form a single functioning unit by a complex network of ligaments (Fig. 36.2). The vertebral column protects the spinal cord, which extends from the midbrain to the level of the second lumbar vertebra.

Spinal injuries involve fractures in 85% of cases. Of these, 10% are ligamentous injuries without fracture, and 5% are SCIs without a radiographic abnormality (SCIWORA), in which the spinal cord is injured directly without radiographic evidence of bony or ligamentous injury. Stability of a spinal injury refers to the resistance to displacement of fracture fragments or, in the case of ligamentous injury, the entire vertebral unit. There are several classification systems for assessing the stability of subaxial spinal column injuries, including the Allen Ferguson classification, Association for Osteosynthesis classification, Dennis Classification, and thoracolumbar injury classification and severity score for thoracolumbar injuries. According to a survey of the members of Spine Trauma Study Group of the International Spinal Cord Society, practical implementation is evenly distributed among the classification systems.[3] The three parallel vertical column model proposed by Denis[2] depicts the anterior column as being formed by alternating vertebral bodies and intervertebral disks surrounded by the annulus fibrosus capsule and anterior longitudinal ligament. The middle column consists of the posterior part of the annulus fibrosus and posterior vertebral wall, posterior longitudinal ligament, spinal cord, paired laminae and pedicles, articulating facets, transverse processes, nerve roots, and vertebral arteries and veins. The posterior column consists of the spinous processes, nuchal ligament, interspinous and supraspinous ligaments, and ligamentum flavum. Disruption of a single column usually preserves stability but does not preclude an SCI from displaced fracture fragments. Disruption of two columns results in an injury that is stable in one direction but unstable in another (eg, stable in flexion but unstable in extension). Disruption of all three columns produces a highly multidirectional unstable injury.

### Pathophysiology

#### Classification of Spinal Column Injuries

Acute spinal injuries are classified according to the mechanism of trauma—flexion, flexion-rotation, extension, and vertical compression (Table 36.1).

**Flexion.** Pure flexion injuries involving the C1-C2 complex can cause unstable atlanto-occipital or atlantoaxial joint dislocation, with or without an associated fracture of the odontoid (Fig. 36.3). The basion-axial interval (BAI) and basion-dens interval (BDI) are normally less than 12 mm. A value greater than 12 mm is suggestive of an atlantoaxial joint dislocation (Fig. 36.4). Calculating the ratio of the distance from the basion to midvertical portion of the posterior laminar line of the atlas over the distance from the opisthion to midvertical portion of the posterior surface of the anterior ring of the atlas (Fig. 36.5) indicates subluxation if the ratio is greater than 1. These injuries are considered unstable because of their location and the relative lack of muscle and ligamentous support.

In pure flexion injuries below C2, a longitudinal pull is exerted on the strong nuchal ligament complex, which usually remains intact. Most of the force is expended on the vertebral body anteriorly, causing a simple wedge fracture. Radiographically, there is a diminished height and increased concavity of the anterior border of the vertebral body, increased density of the vertebral body resulting from bony impaction, and prevertebral soft tissue swelling (Fig. 36.6). Because the posterior column remains intact, this injury is usually stable. However, spinal instability may occur with severe wedge fractures (loss of more than half the vertebral height) or multiple adjacent wedge fractures.

A flexion teardrop fracture results when severe flexion forces cause anterior displacement of a wedge-shaped fragment (resembling a teardrop) of the anteroinferior portion of the involved vertebral body (Fig. 36.7). This injury, which is associated with neurologic injury, is highly unstable because the anterior and posterior ligaments are commonly disrupted.

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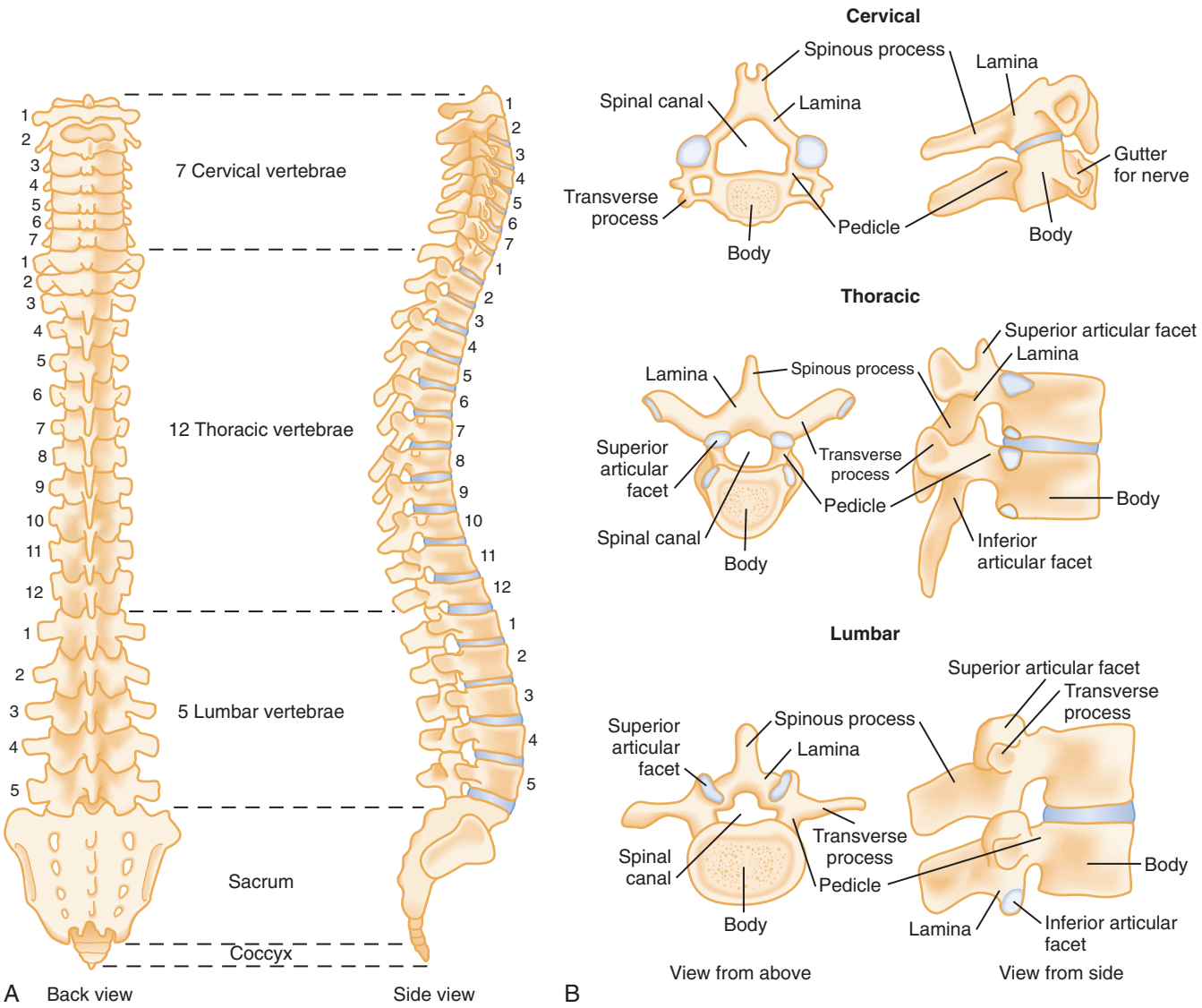



**Fig. 36.1.** **A,** Vertebral column. **B,** Typical vertebrae.

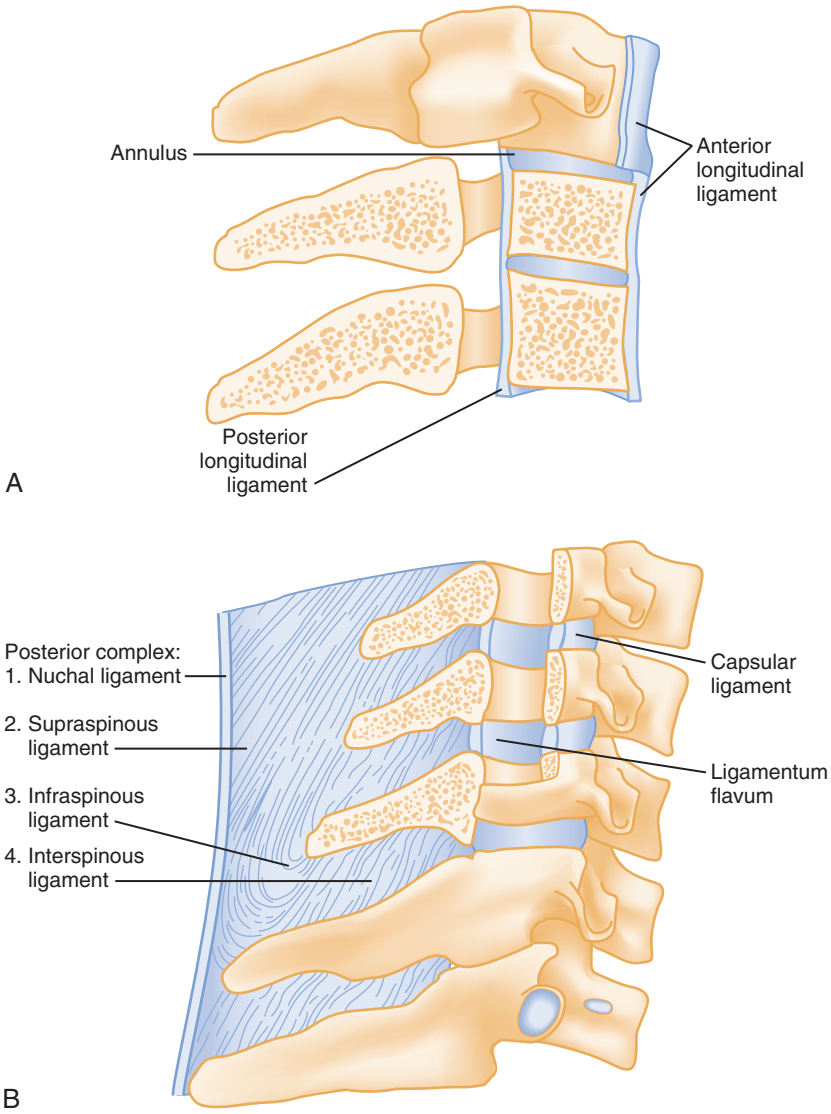



**Fig. 36.2.** **A,** Ligaments of the anterior column. **B,** Ligaments of the posterior column.

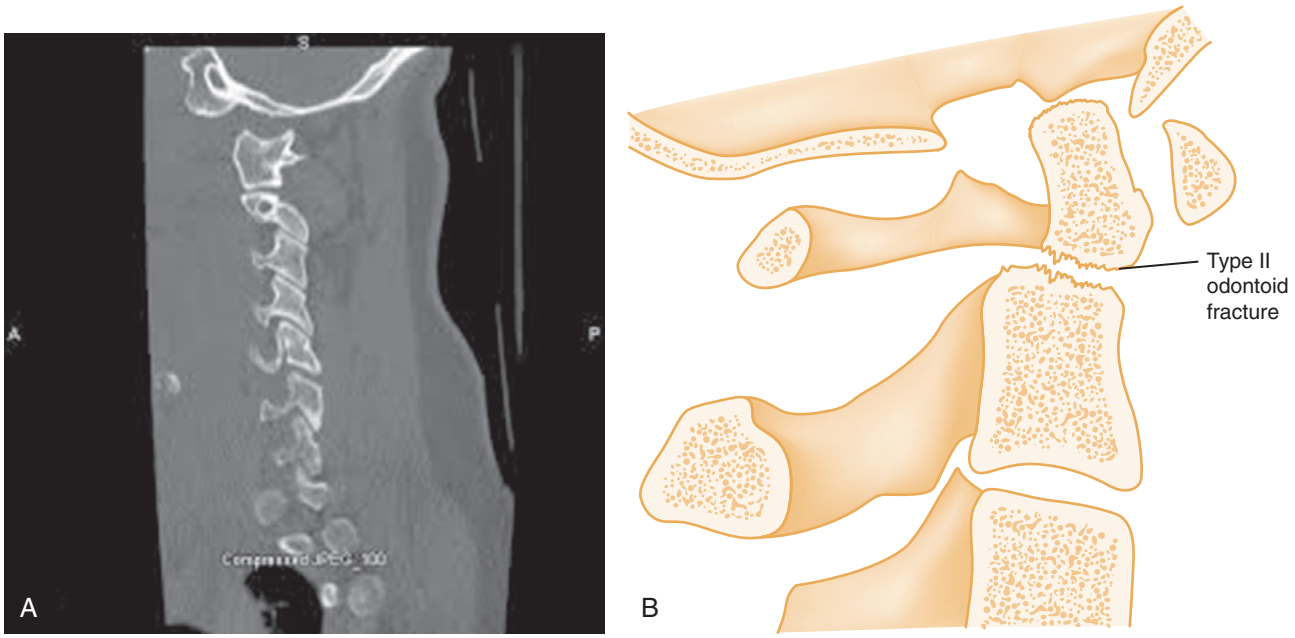



**Fig. 36.3. A, B,** Odontoid fracture with anterior dislocation. Mechanism—flexion with shearing; stability— unstable.

**TABLE 36.1**

## Classification of Spinal Injuries

| MECHANISM OF SPINAL INJURY | STABILITY |
|---|---|
| **FLEXION** | |
| Wedge fracture | Stable |
| Flexion teardrop fracture | Extremely unstable |
| Clay shoveler's fracture | Stable |
| Subluxation | Potentially unstable |
| Bilateral facet dislocation | Always unstable |
| Atlanto-occipital dislocation | Unstable |
| Anterior atlantoaxial dislocation with or without fracture | Unstable |
| Odontoid fracture with lateral displacement fracture | Unstable |
| Fracture of transverse process | Stable |
| **FLEXION-ROTATION** | |
| Unilateral facet dislocation | Stable |
| Rotary atlantoaxial dislocation | Unstable |
| **EXTENSION** | |
| Posterior neural arch fracture (C1) | Unstable |
| Hangman's fracture (C2) | Unstable |
| Extension teardrop fracture | Usually stable in flexion; unstable in extension |
| Posterior atlantoaxial dislocation, with or without fracture | Unstable |
| **VERTICAL COMPRESSION** | |
| Bursting fracture of vertebral body | Stable |
| Jefferson fracture (C1) | Extremely unstable |
| Isolated fractures of articular pillar and vertebral body | Stable |

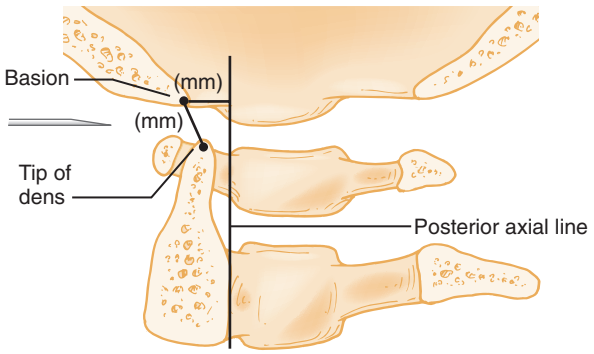



**Fig. 36.4.** The basion-axial interval (BAI) and basion-dens interval (BDI) are normally less than 12 mm.

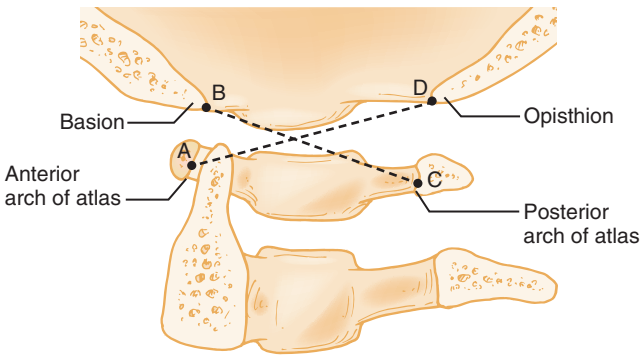



**Fig. 36.5.** The Power's ratio.

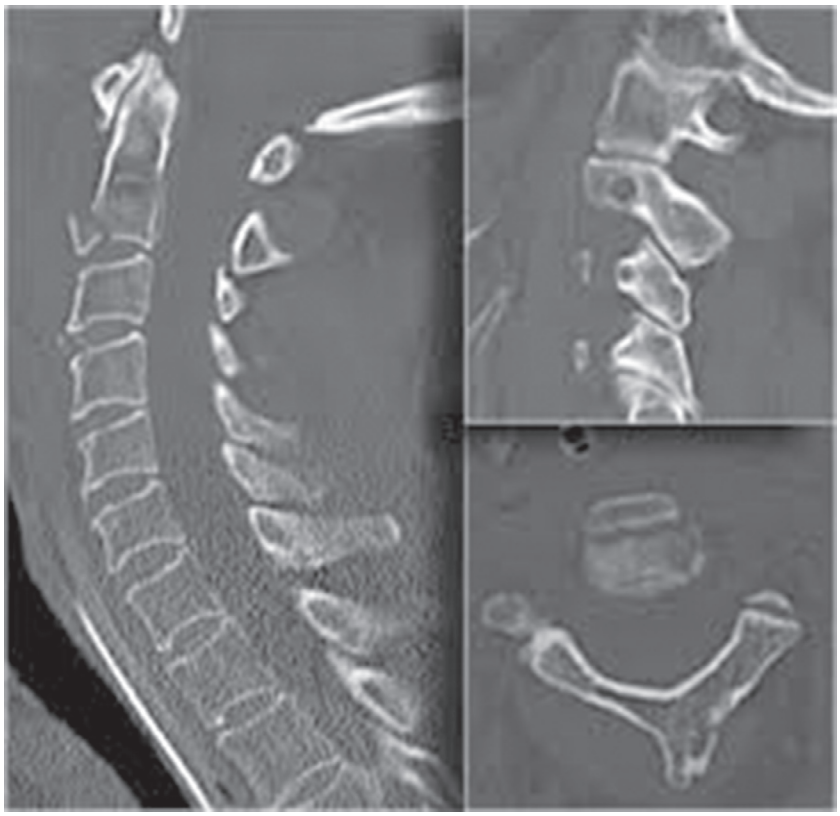


**Fig. 36.6.** **A,** Lateral view of a wedge fracture of C5 with angulation. Mechanism—flexion; stability—mechanically stable. **B,** Note the anterior wedging of the C4 vertebral body and angulation of C4 on C5.

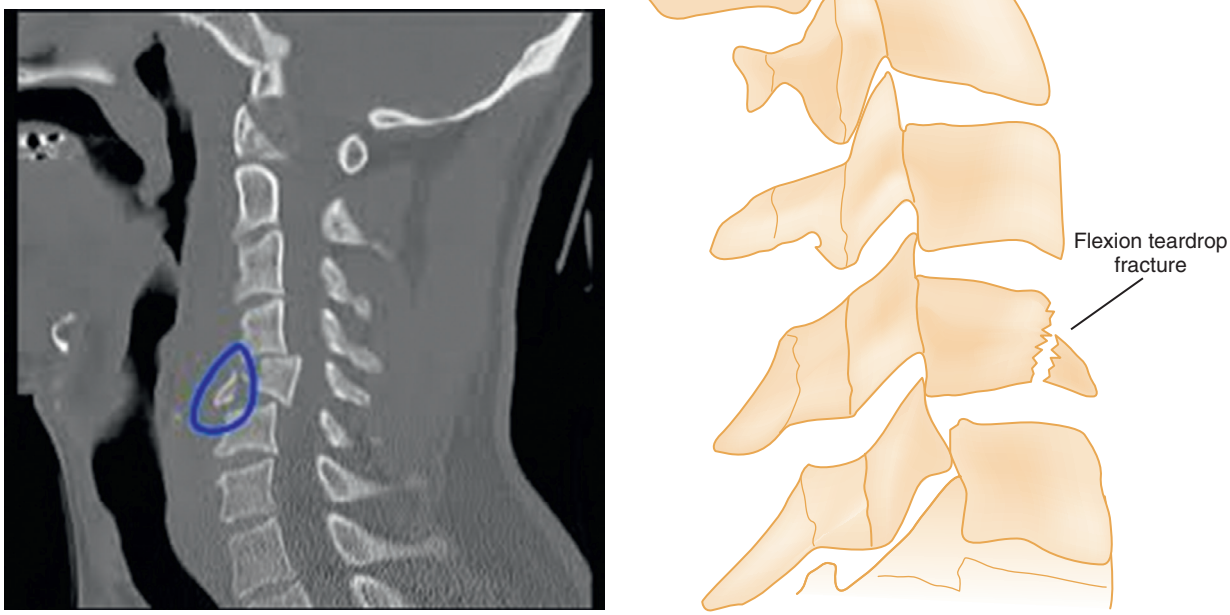



**Fig. 36.7.** **A, B,** Lateral view of a teardrop fracture. Mechanism—flexion; stability—unstable. The fractured fragment off the C5 body resembles a teardrop.

The clay shoveler's fracture is an oblique fracture of the base of the spinous process of one of the lower cervical vertebrae (Fig. 36.8). The injury derives its name from the fracture caused by the abrupt head flexion that clay miners experienced when lifting a heavy shovelful of clay and having the clay stick to the shovel. This force, transmitted through the supraspinous ligament, results in an avulsion fracture of the spinous process. Today, this fracture is seen after direct trauma to the spinous process and after sudden deceleration MVCs that result in forced neck flexion. Because this injury involves only the spinous process, it is stable and requires no treatment beyond symptomatic care.

Pure spinal subluxation occurs when the ligamentous complexes rupture without an associated bony injury. This injury begins posteriorly in the nuchal ligament and proceeds anteriorly to involve other ligaments (Fig. 36.9). Although rarely associated with neurologic damage, this injury is potentially unstable.

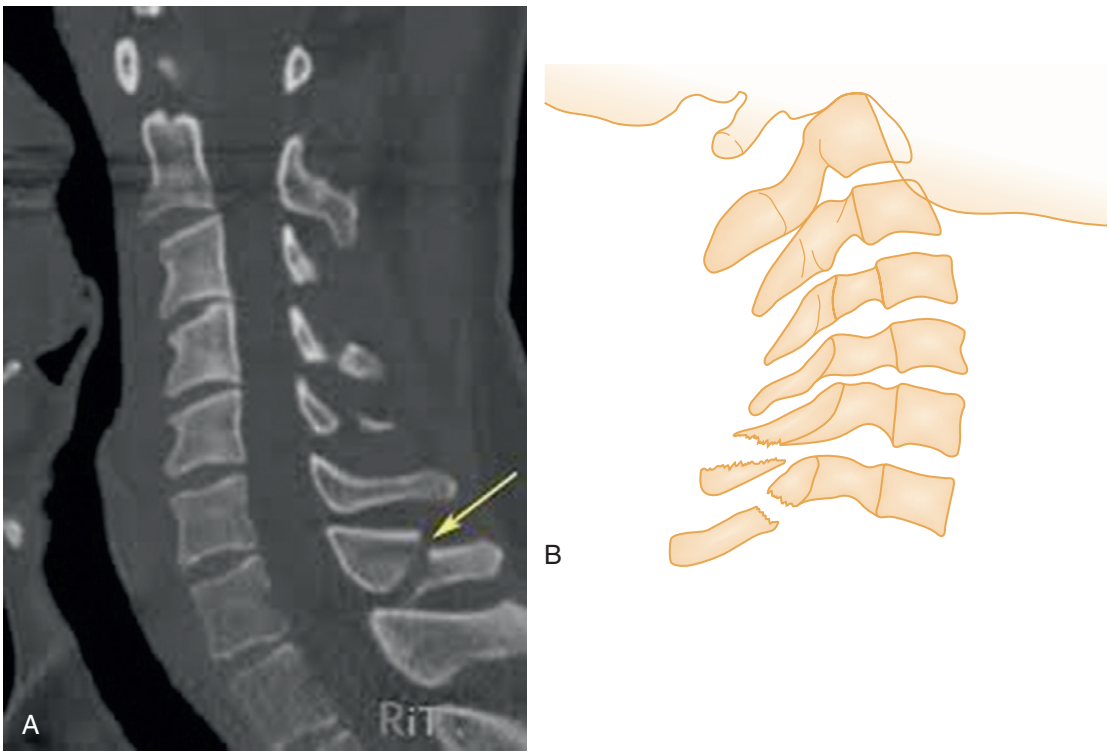



**Fig. 36.8. A, B,** Clay shoveler's fracture. Mechanism—flexion; stability—mechanically stable. Note the avulsed fragment off the tip of the C7 spinous process in an underpenetrated lateral view (*arrow*).

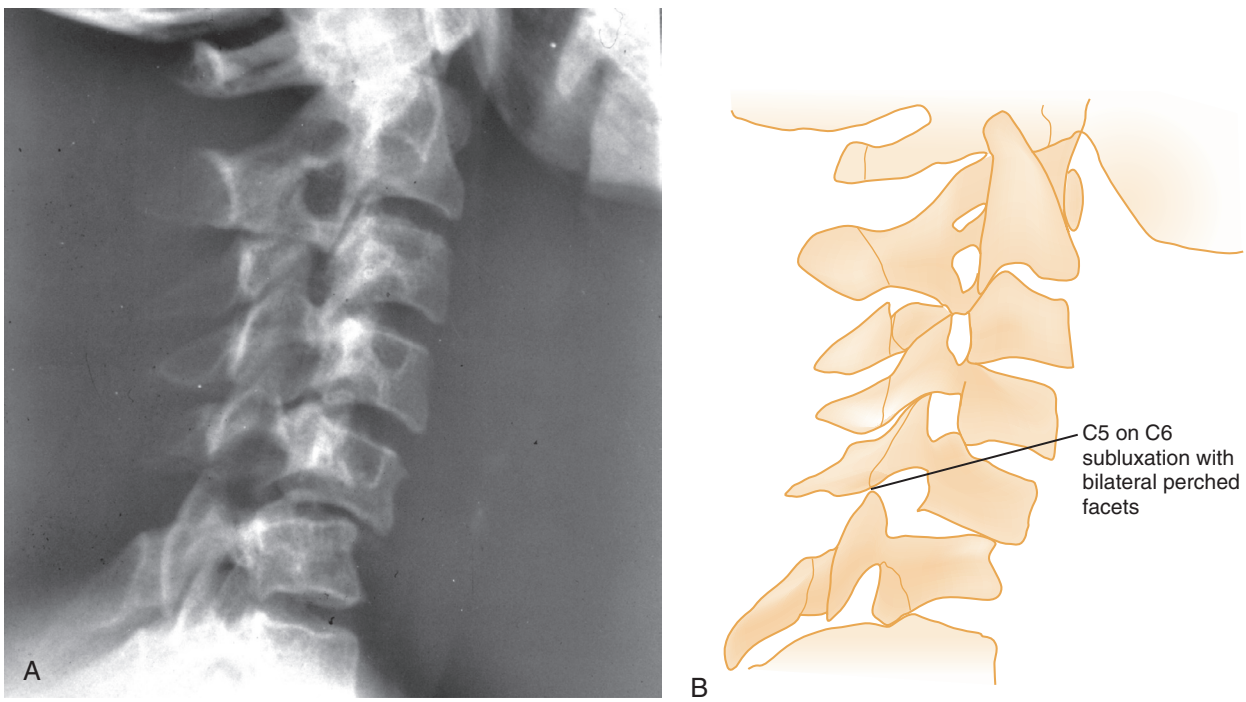



**Fig. 36.9. A, B,** Subluxation with bilateral perched facets at C5 and C6. Mechanism—flexion; stability—unstable. Lateral view shows severe subluxation of C5 on C6.

Bilateral facet dislocations occur when a greater force of flexion causes soft tissue disruption to continue anteriorly to the annulus fibrosis of the intervertebral disk and anterior longitudinal ligament, resulting in extreme instability. The forward movement of the spine causes the inferior articulating facets of the upper vertebra to pass upward and over the superior facets of the lower vertebra (Fig. 36.10), resulting in anterior displacement of the spine above the level of injury.

**Shear Injury.** Trauma to the head directed in an anteroposterior (AP) direction may result in fracture of the odontoid process above the transverse ligaments (type I) or, more commonly, at the base of the odontoid process where it attaches to C2 (type II; Fig. 36.11). Slight angulation of the force may result in extension of the fracture into the body of C2 (type III; Fig. 36.12). Type I odontoid fractures are usually stable because they are an avulsion injury to the odontoid tip. However, if traction forces injure the apical and alar ligaments, the fracture may be unstable. Type II odontoid fractures are, by definition, unstable and are often complicated by nonunion. Type III odontoid fractures are also mechanically unstable because they can extend laterally into the superior articular facet of the atlas.

**Flexion-Rotation.** Rotary atlantoaxial dislocation is an unstable injury visualized best on open-mouth odontoid radiographs (Fig. 36.13) or a computed tomography (CT) scan. When the x-ray image reveals symmetric basilar skull structures, a unilateral magnified lateral mass confirms a C1-C2 dislocation.

A unilateral facet dislocation is caused by both flexion and rotation. The rotational component of this injury occurs around one of the facet joints, which acts as a fulcrum. Simultaneous flexion and rotation cause the contralateral facet joint to dislocate, with the superior facet riding forward and over the tip of the inferior facet and coming to rest within the intervertebral foramen. In this position, the dislocated articular mass is mechanically locked in place, making this a stable injury even though the posterior ligament complex is disrupted.

Any cervical fracture or dislocation may cause torticollis however torticollis may also be caused by a benign process such as a muscle spasm. It may be difficult to differentiate the two

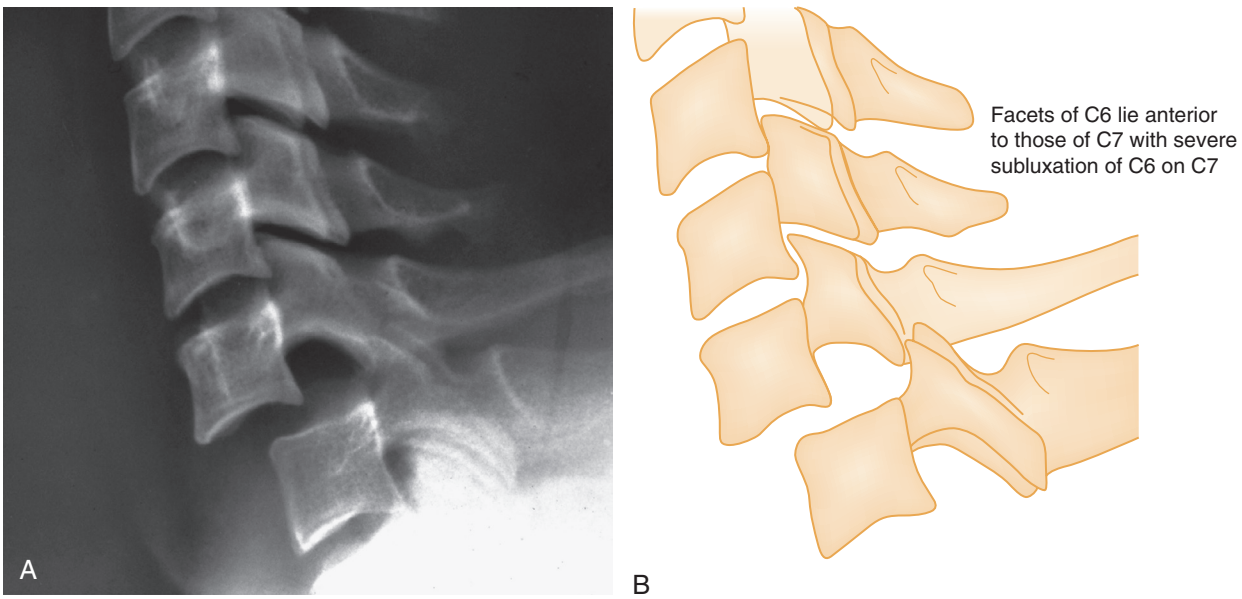



**Fig. 36.10. A, B,** Bilateral facet dislocation. Facets of C6 lie anterior to those of C7, with severe subluxation of C6 on C7.

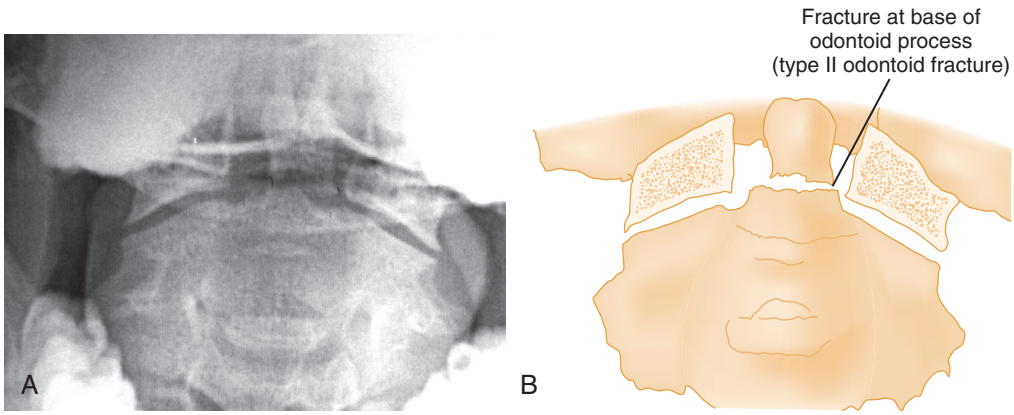



**Fig. 36.11. A, B,** Odontoid fracture with lateral displacement. Mechanism—flexion; stability—unstable. The tip of the odontoid process is laterally displaced in this lateral flexion injury.

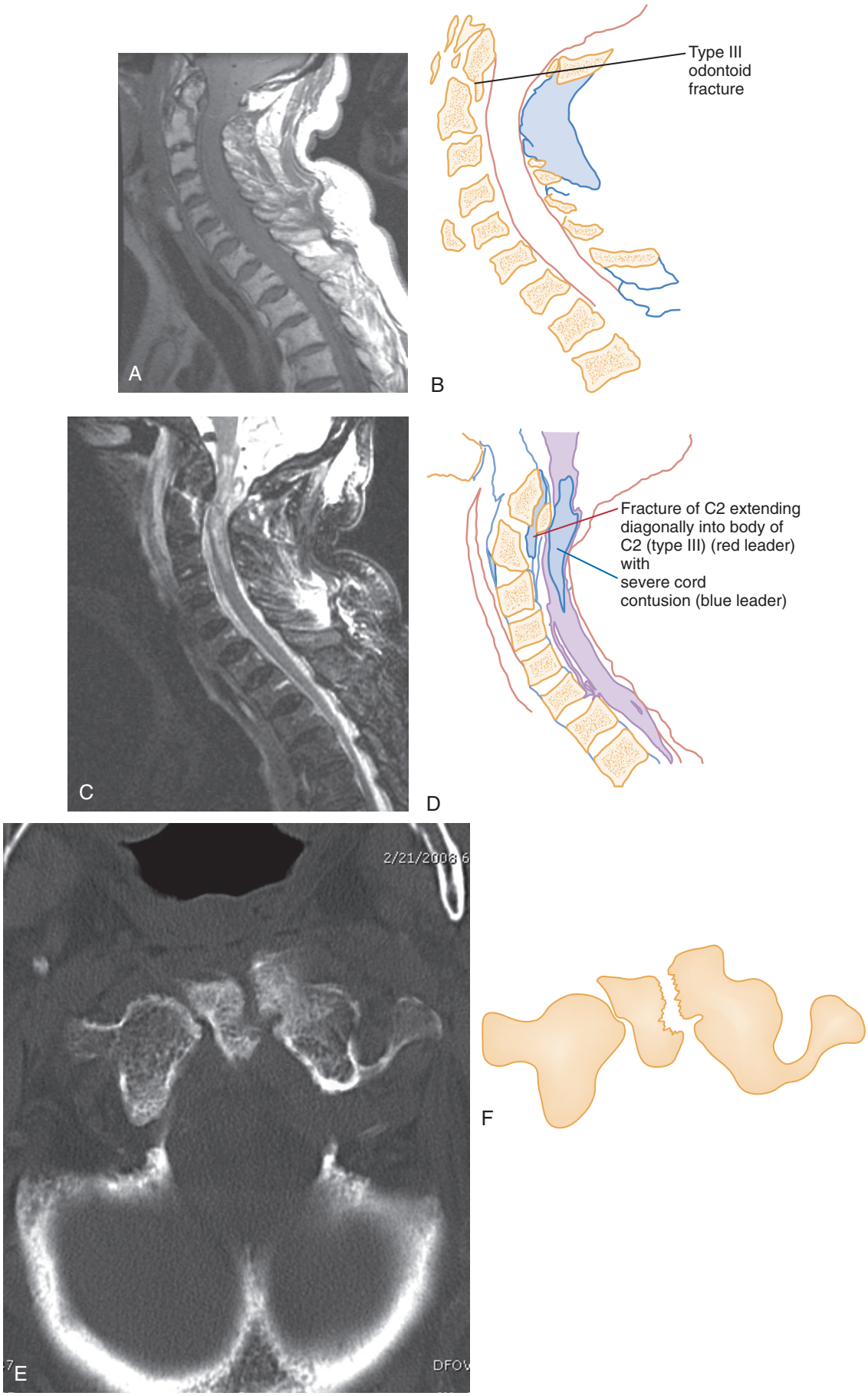



**Fig. 36.12.** **A–F**, Odontoid fracture, type III.

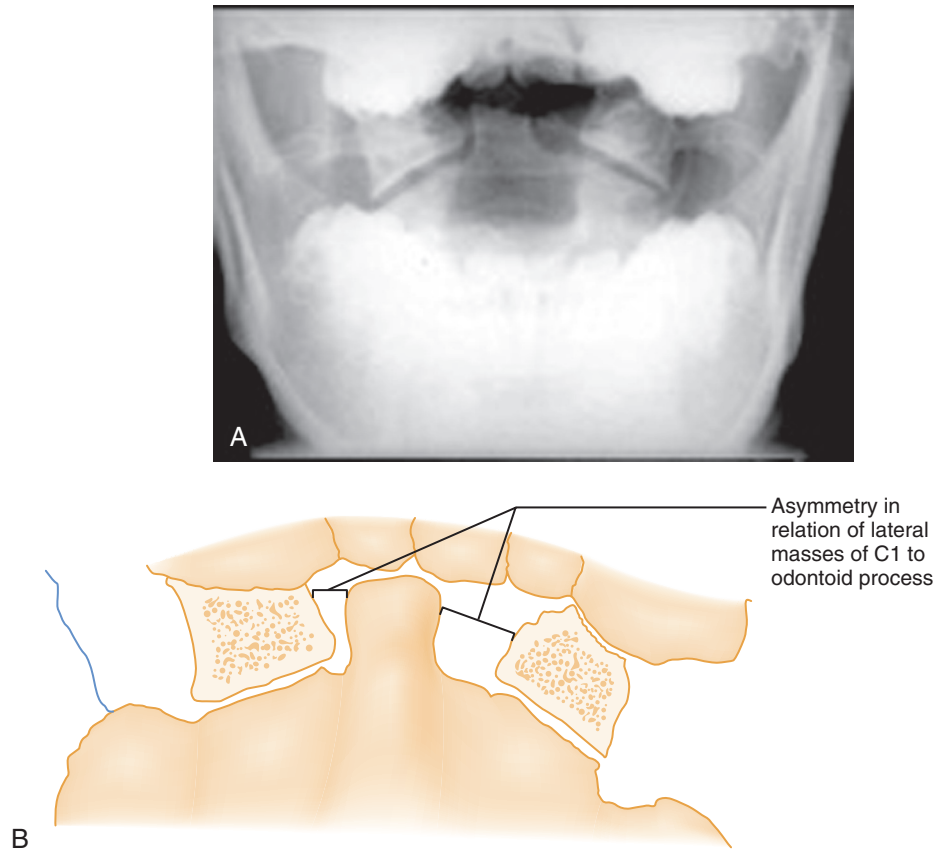



**Fig. 36.13.** **A, B,** Rotatory subluxation of C1 on C2. Mechanism—rotation; stability—unstable. There is marked asymmetry in the relationship of the lateral masses of C1 to the odontoid process. Rotation causes the right lateral mass to appear slightly larger (farther from the x-ray film) than the left (closer to the x-ray film).

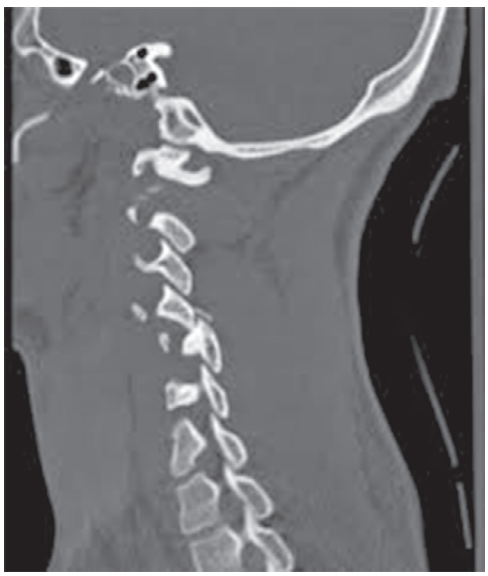


**Fig. 36.14.** Unilateral facet dislocation on CT.

and in the setting of trauma, CT (Fig. 36.14) or oblique radiographs may be necessary to demonstrate the dislocated facet joint (Fig. 36.15).

Due to the varying shapes of the articular processes, different types of flexion-rotation injuries result. In the cervical region, where articular processes are small and almost horizontal, unilateral facet dislocations occur, whereas in the lumbar region, in which articular processes are large and nearly vertical, unilateral facet dislocation is rare. Instead, one or both articular processes fracture, and the upper vertebra swings forward. Commonly seen in the thoracolumbar and lumbar regions, this rotation fracture-dislocation is unstable (Fig. 36.16).

**Extension.** Fracture of the posterior neural arch of the atlas (C1) results from compression of the posterior elements between the occiput and spinous process of the axis (C2) during forced neck extension (Fig. 36.17). Although the anterior arch and transverse ligament remain intact, this fracture is potentially unstable because of its location.

The hangman's fracture, or traumatic spondylolysis of C2, occurs when the cervicocranium—the skull, atlas, and axis functioning as a unit—is hyperextended as a result of abrupt deceleration. Bilateral fractures of the pedicles of the axis occur with or without dislocation (Fig. 36.18). Although unstable, cord damage is often minimal because the AP diameter of the neural canal is greatest at C2, and the bilateral pedicular fractures permit spinal canal decompression. Originally described in victims of hanging injury, today it is most often the result of head-on MVCs.

The extension teardrop fracture occurs when abrupt extension of the neck causes the anterior longitudinal ligament to pull the anteroinferior corner of a vertebral body away from the remainder of the vertebra, producing a triangular fracture that is radiographically similar to the flexion teardrop fracture. Often occurring in lower cervical vertebrae (C5–C7) from diving accidents, this injury may be associated with a central cord syndrome (see later) and is caused by the ligamentum flavum buckling into the spinal cord. Because the posterior elements remain intact, this injury is stable in flexion but potentially unstable in extension.

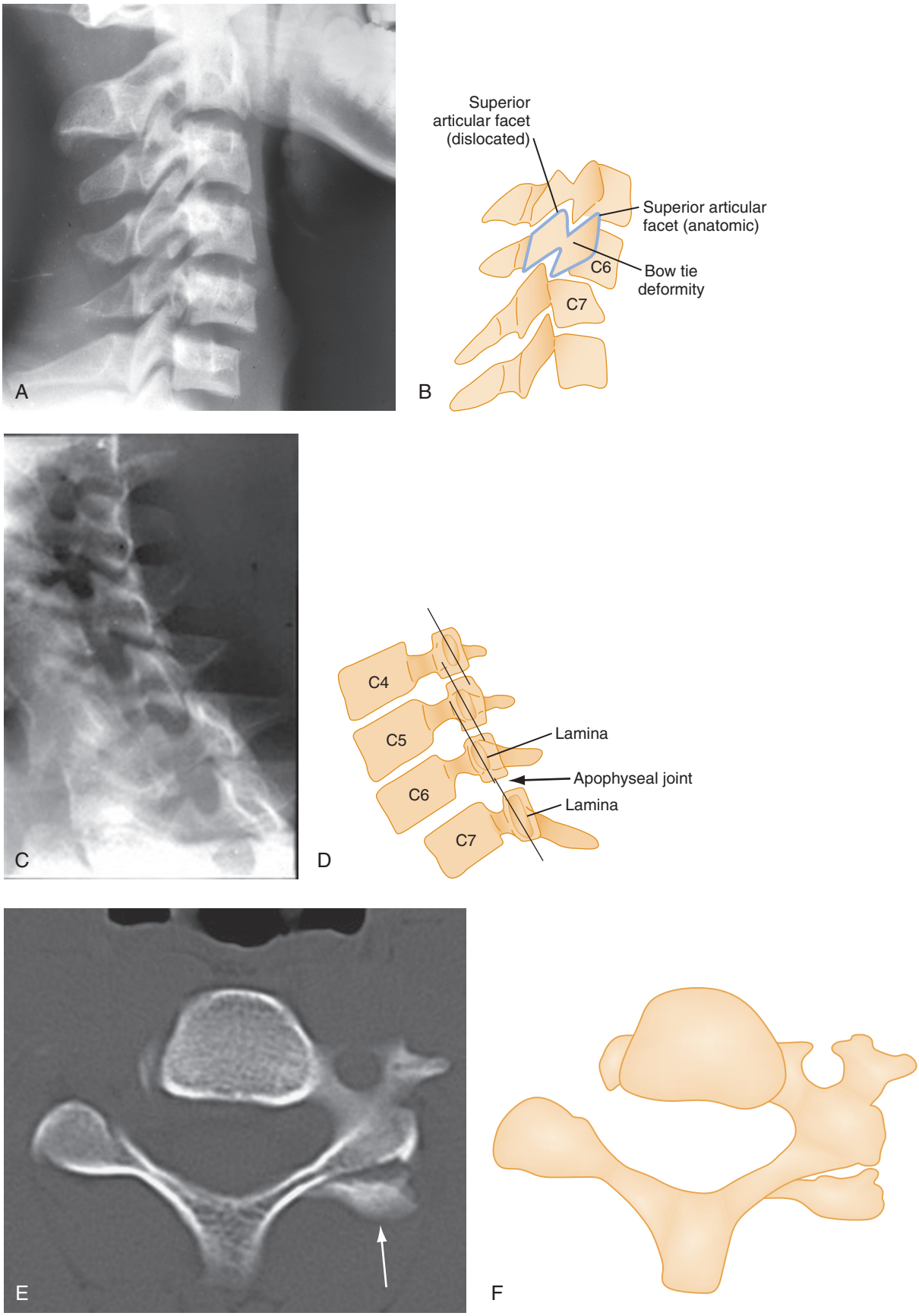



**Fig. 36.15.** Unilateral facet dislocation. Mechanism—flexion and rotation; stability—stable. **A, B,** Lateral view showing one dislocated articular facet of C5 lying anterior to the corresponding facet of C6 and creating a bowtie deformity. The C5 vertebral body is subluxed anteriorly on C6. **C, D,** Oblique view of unilateral facet dislocation with the lamina of C6 projecting into the neural foramen. **E, F,** CT scan showing facet dislocation. The inferior facet (*arrow*) lies posterior to the superior facet.

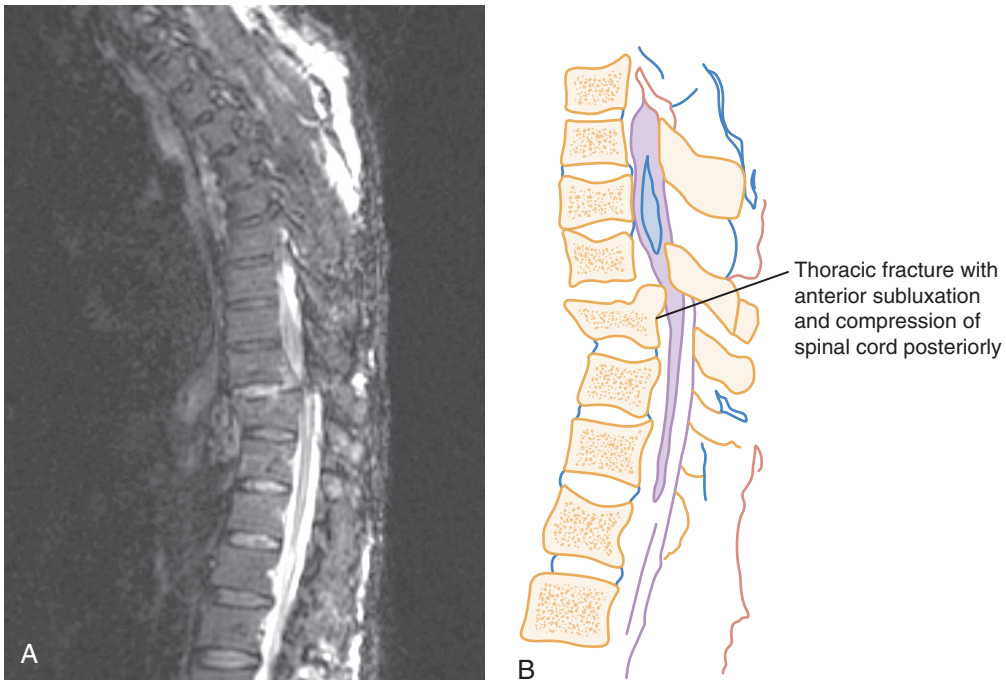



**Fig. 36.16.** **A**, **B,** MRI scan showing fracture-dislocation of the thoracic spine.

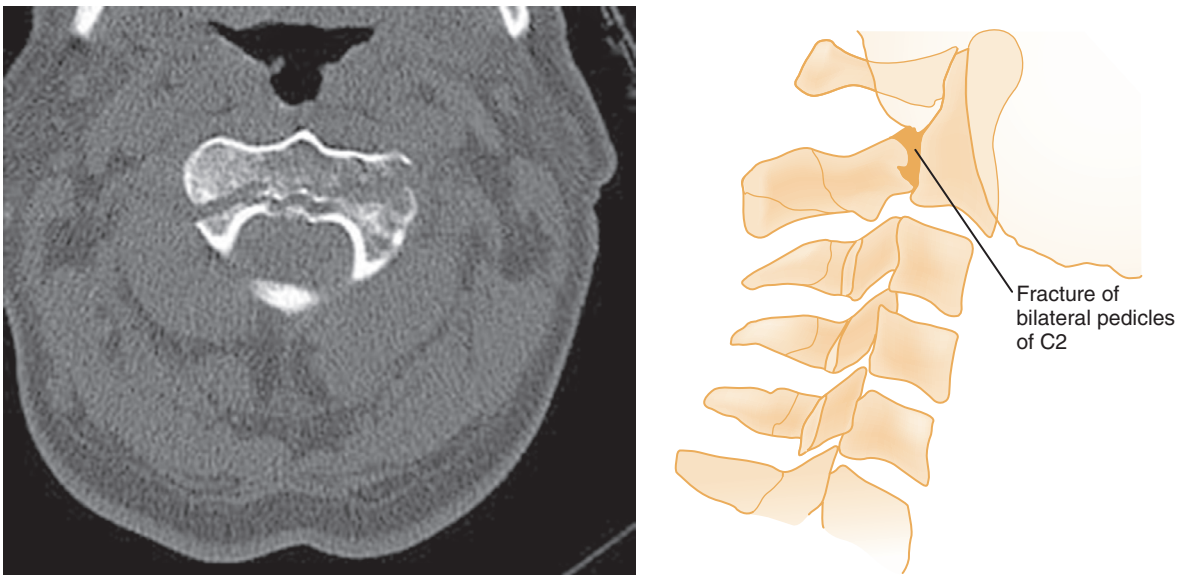



**Fig. 36.17.** **A, B,** CT scan of posterior neural arch fracture of C1. Mechanism—extension; stability—unstable. The fracture line is well visualized.

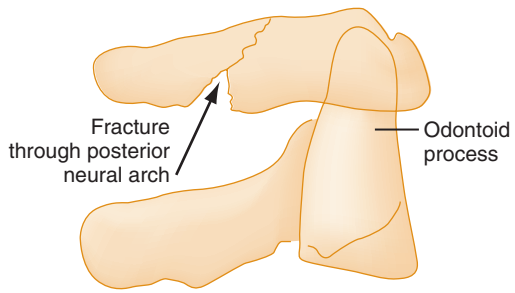



**Fig. 36.18.** Hangman's fracture. Mechanism—extension; stability—unstable. Fracture lines extending through the pedicles of C2 are well visualized. Retropharyngeal soft tissue swelling is apparent.

**Vertical Compression.** Vertical compression injuries occur in the cervical and lumbar regions, which are capable of straightening at the time of impact. When forces are applied from above (skull) or below (pelvis or feet), one or more vertebral body endplates may fracture. The nucleus pulposus of the intervertebral disk is forced into the vertebral body, which is shattered outward, resulting in a burst fracture (Fig. 36.19). Sagittal CT cuts and a lateral radiograph will demonstrate a comminuted vertebral body, and there will typically be greater than 40% compression of the anterior vertebral body, which helps differentiate it from the simple wedge fracture. Coronal CT cuts and a frontal radiograph demonstrate a characteristic vertical fracture of the vertebral body. This is a stable fracture because all the ligaments remain

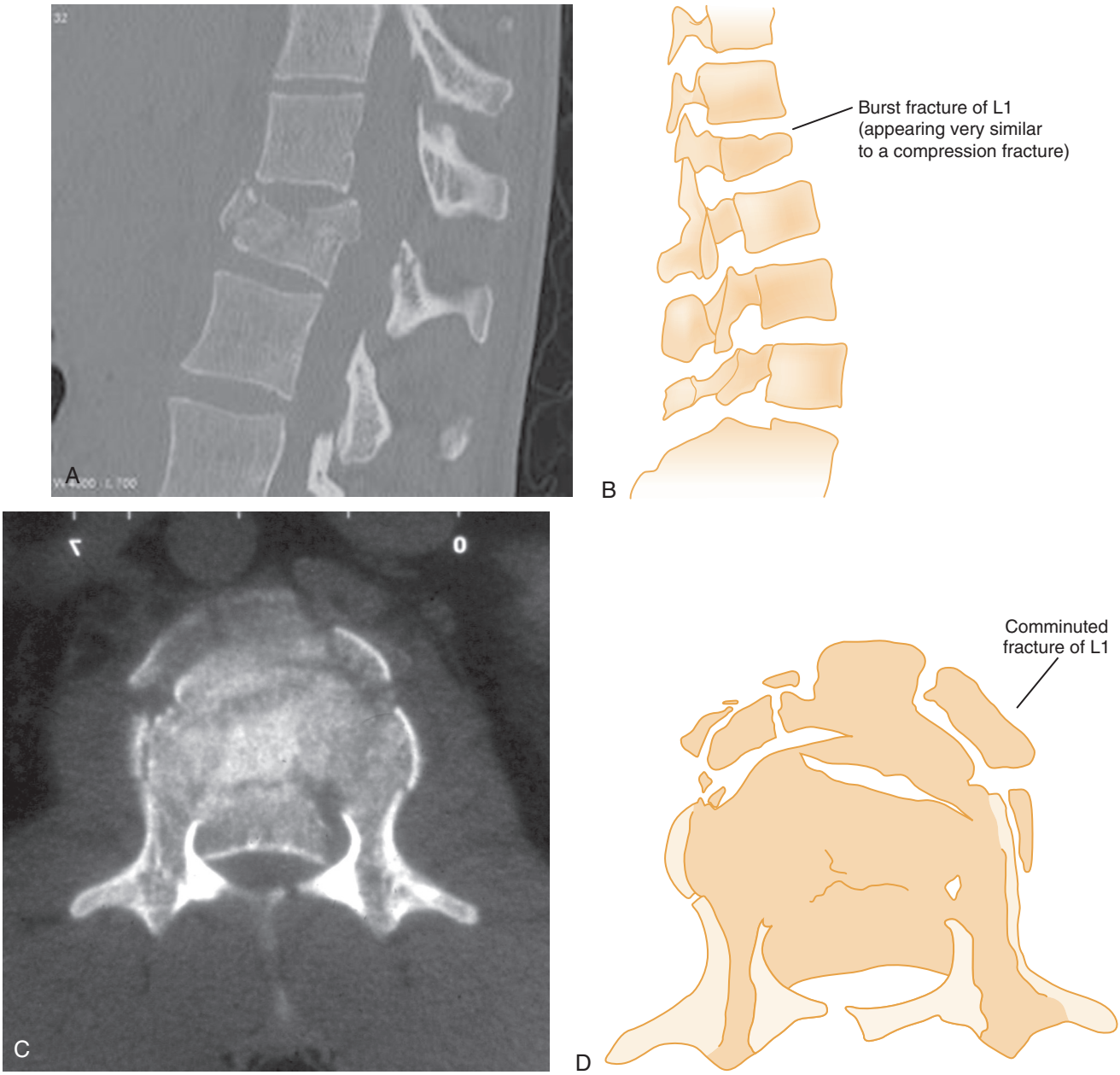



**Fig. 36.19.** Burst fracture of a vertebral body. Mechanism —vertical compression and flexion; stability—unstable**. in the right place? A**, **B,** Lateral CT scan showing a burst fracture of L1, appearing very similar to a compression fracture. Mechanism—flexion; stability—usually stable. **C, D,** CT scan of L1 in the same patient showing comminution of the fracture and retropulsion of fragments into the spinal canal.

intact. However, fracture fragments may impinge on or penetrate the ventral surface of the spinal cord and cause an anterior cord syndrome (Fig. 36.20).

An extremely unstable injury, the C1 Jefferson fracture occurs when a vertical compression force is transmitted through the occipital condyles to the superior articular surfaces of the lateral masses of the atlas, driving the lateral masses outward, disrupting the transverse ligament and resulting in fractures of the anterior and posterior arches of the atlas (Fig. 36.21). The lateral film may demonstrate a widening of the predental space between the anterior arch of C1 and the odontoid, or dens. The open-mouth view will demonstrate a bilateral offset of right and left lateral masses of C1 relative to the lateral masses of C2. A fracture should be diagnosed when the sum of the offset distances from the right and left sides exceeds 7 mm. However, when the fragments are minimally displaced, the Jefferson fracture is difficult to recognize.

Rarely, vertical compression fractures may result in isolated fractures of the articular pillar or vertebral body, exhibiting vertical and oblique lines of fracture.

## Classification of Spinal Cord Injuries

**Primary Spinal Cord Injury.** The spinal cord may be injured by three broad categories of injury patterns. First, penetrating trauma or massive blunt trauma with disruption of the vertebral column causes transection of neural elements. Because neurons within the central nervous system do not regenerate, such injuries are irreversible. Less severe blunt trauma may have similar effects resulting from a displaced bony fragment or herniated disk injuring the cord.

Second, when patients with cervical osteoarthritis and spondylosis, particularly older adults, are subjected to forcible cervical

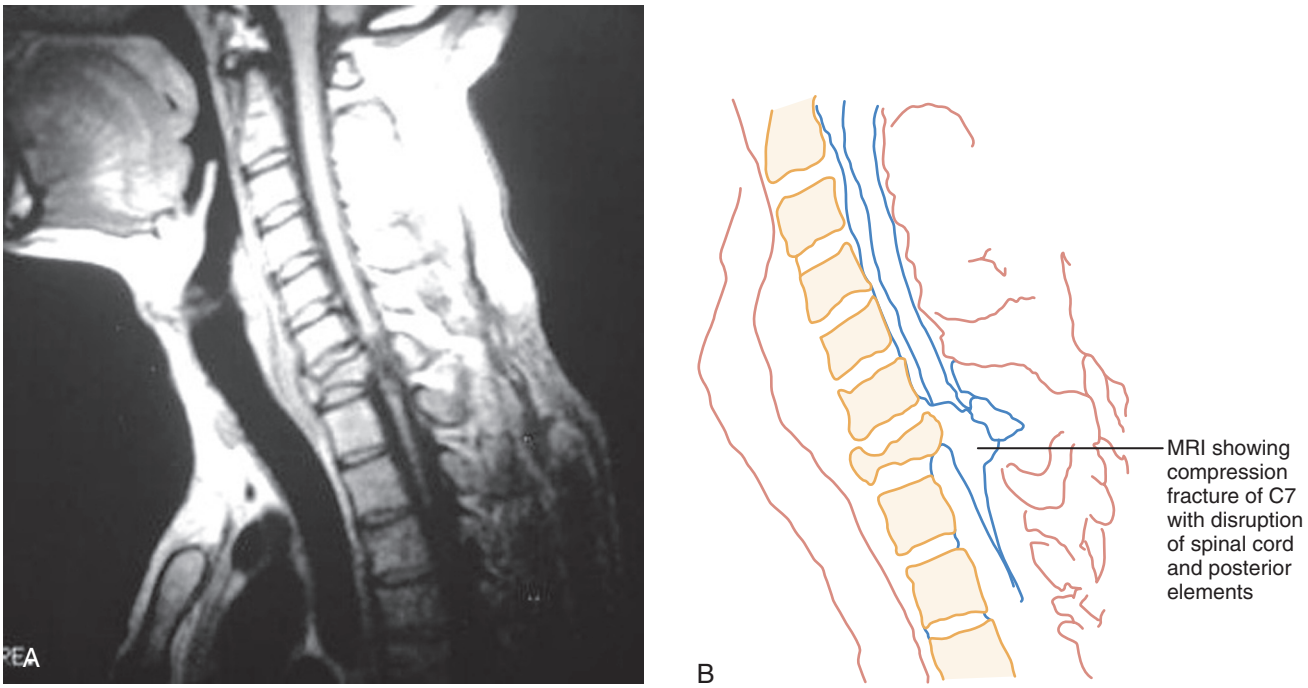



**Fig. 36.20. A, B,** MRI scan showing a burst fracture of C7 with complete spinal cord disruption.

spine extension, the spinal cord may be injured secondary to compression between an arthritically enlarged anterior vertebral ridge and a posteriorly located hypertrophic ligamentum flavum (Fig. 36.22). This injury frequently results in a central cord syndrome.

The third mechanism is primary vascular damage to the spinal cord. The spinal cord may be compressed by an extradural hematoma, particularly in patients who are on anticoagulants or have bleeding disorders. Vascular injuries should also be suspected when there is a discrepancy between the clinically apparent neurologic deficit and known level of spinal injury. For example, a lower cervical dislocation may compress the vertebral arteries as they travel within the spinal foramina of the vertebrae, resulting in thrombosis of the anterior spinal artery that originates from both vertebral arteries at C1 (Fig. 36.23). On physical examination, such an injury may erroneously appear to be localized to the level of C1 or C2. Also, the great radicular artery of Adamkiewicz, originating from the aorta and entering the spinal canal at the level of L1, sends branches as cephalad as T4. Therefore, a lumbar fracture or dislocation can produce a neurologic deficit as high as T4.

**Secondary Spinal Cord Injury.** The maximum neurologic deficit after blunt spinal cord trauma is often not seen on initial examination and may, instead, progress over many hours. Studied extensively in animal models, the histopathology of secondary SCI is now thought to be due to a complex cascade of biochemical events that result in progressive ischemia of gray and white matter during the postinjury period (Fig. 36.24). Other factors, such as hypoxia, hypotension, hyperthermia, and hypoglycemia, also affect the ultimate extent of SCI.

### Classification of Cervical Soft Tissue Injuries

Blunt force trauma can injure one or more of the soft tissues of the neck, including ligaments, muscles, intervertebral disks, zygapophysial facet joints, dorsal root ganglia, and vertebral artery. Although injuries of these tissues have been documented in biomechanical, animal, and human autopsy studies, a validated diagnostic test is only available for facet injuries.[4,5] The cardinal symptom of a WAD is neck pain, but neck stiffness, neck and arm paresthesias, and dizziness are commonly reported. Table 36.2 shows the Quebec Task Force classification of WADs, the most common classification used worldwide.[2]

**TABLE 36.2**

**Quebec Task Force Classification of Whiplash-Associated Disorders**

| GRADE | DESCRIPTION |
|---|---|
| 0 | Whiplash injury but no pain, symptoms, or signs |
| 1 | Delayed neck pain, minor stiffness, nonfocal tenderness only, no physical signs |
| 2 | Early onset of neck pain, focal neck tenderness, spasm, stiffness, radiating symptoms |
| 3 | Early onset of neck pain, focal neck tenderness, spasm, stiffness, radiating symptoms and signs of neurologic deficit |
| 4 | Neck complaint (grade 2 or 3 above) and fracture dislocation |

(Adapted from Sterling S: Physiotherapy management of whiplash-associated disorders [WAD]. J Physiother 60:5–12, 2014.)

## CLINICAL FEATURES

### Neurologic Evaluation

The initial neurologic evaluation of a patient with a suspected spinal injury should begin with observation. Careful inspection, beginning with the head and proceeding downward, may reveal signs of possible spinal involvement. Significant head and facial trauma have a 5% to 10% incidence of associated cervical spine injuries. Scapular contusions suggest a rotation or flexion-rotation injury of the thoracic spine. Chest and neck abrasions from

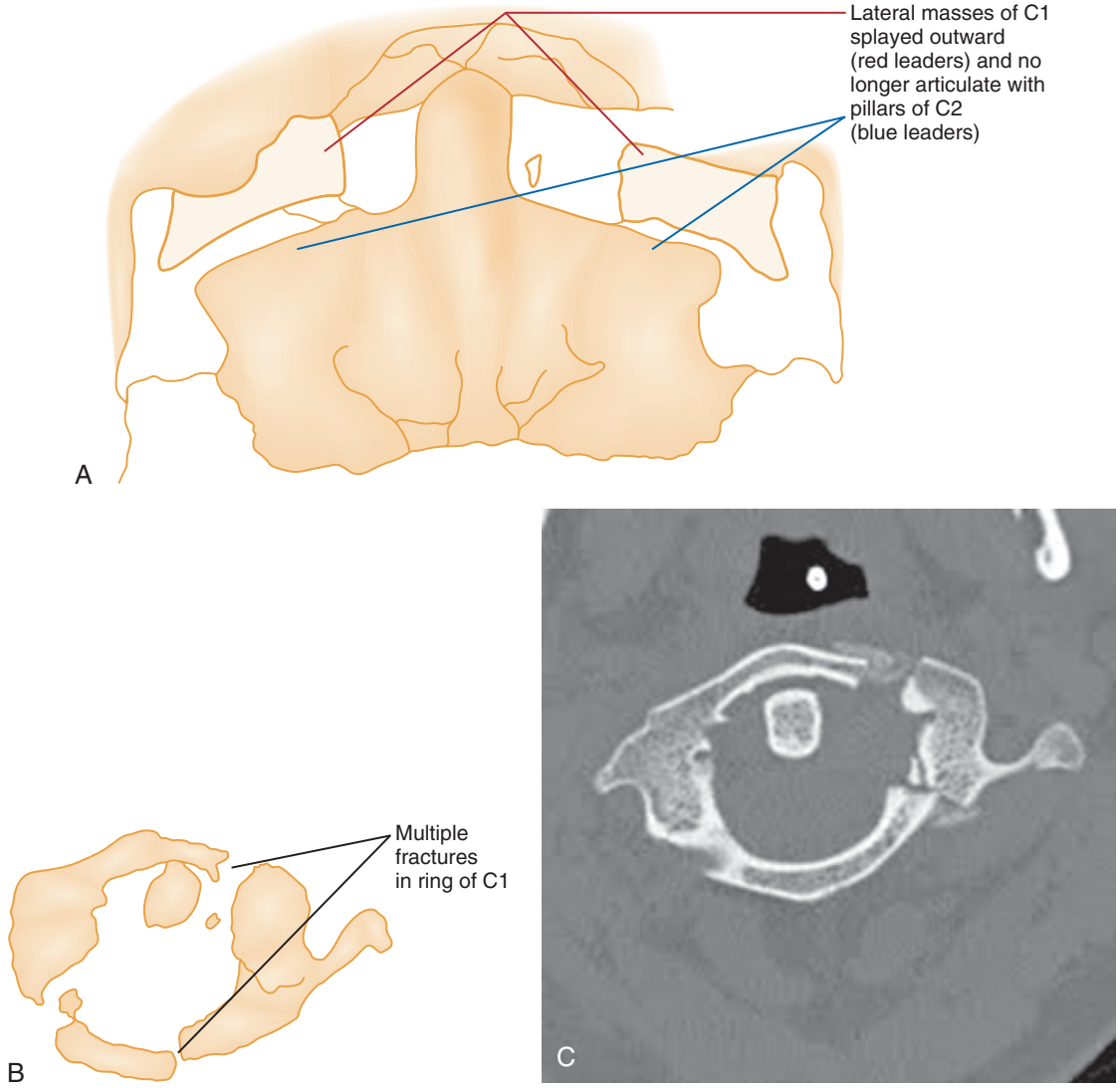



**Fig. 36.21.** Jefferson fracture. Mechanism— vertical compression; stability— unstable. **A, B,** Bilateral lateral displacement of the lateral masses of C1 with respect to the articular pillars of C2 confirms a Jefferson fracture and differentiates it from fracture of the posterior neural arch of C1 on an anteroposterior view. **C,** CT scan of C1 showing two fracture sites in the ring of C1, with lateral displacement of the lateral mass on the left.

automobile shoulder belts and lower abdominal markings from lap belts indicate possible blunt carotid and vertebral injuries, as well as spinal, intrathoracic, and intra-abdominal injuries. As occurs with falls from considerable heights, injuries to the gluteal region, calcaneal fractures, and severe ankle fractures suggest a compression type of spinal injury.

Because the diaphragm is innervated by the phrenic nerve, which originates at C3-C4, an abdominal breathing pattern may provide an important clue to an upper cervical injury. The presence of Horner's syndrome, characterized by unilateral ptosis, miosis, and anhidrosis, may result from disruption of the cervical sympathetic chain, usually between C7 and T2. Priapism may occur with severe SCI, and it is often associated with spinal shock, which is a transient reflex depression of the spinal cord below the level of the injury.

The emergency clinician should speak with the patient during the examination because it provides the patient with reassurance and the emergency clinician with valuable information. Patients may experience pain in the sensory dermatome corresponding to the injured spinal level. For example, a C2 lesion may cause occipital pain, whereas discomfort in the trapezius muscle, particularly in the absence of signs of local trauma, suggests a C5 injury. The past medical history is important because certain conditions predispose patients to cervical injury. For example, Down syndrome patients are predisposed to atlanto-occipital dislocation, whereas rheumatoid arthritis patients are prone to rupture of the C2 transverse ligament.

Palpation of the entire spine and paraspinal musculature may reveal areas of tenderness, deformity, or muscle spasm. A step-off may be appreciated with severe subluxation. Widening of an interspinous space indicates a tear in the posterior ligament complex and a potentially unstable spinal injury.

The motor activity of the body is complex. Because a single motion is often governed by muscles innervated by multiple spinal segments, localizing a spinal lesion based solely on motor function is extremely difficult. Testing the presence and strength of those motions outlined in Table 36.3, however, provides a rapid baseline assessment. When a deficit is noted, the motor and neurologic examination should be repeated because progression of dysfunction may occur. Even the most minimal of motor

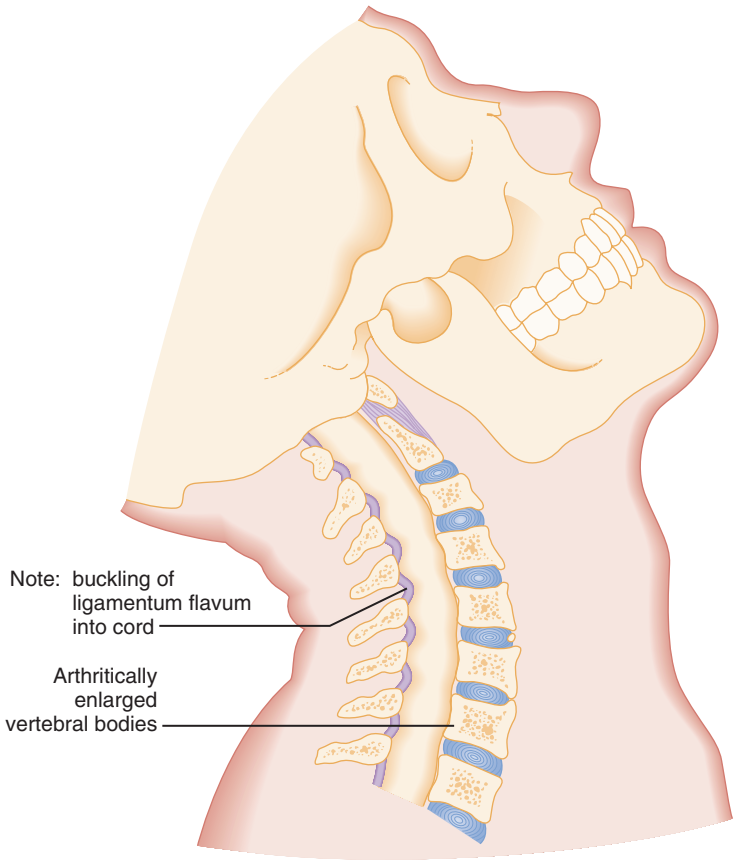



**Fig. 36.22.** Older patients subjected to extension forces can sustain cervical spinal cord injury as a result of compression of the spinal cord between the posterior hypertrophic ligamentum flavum and arthritically enlarged anterior vertebral bodies.

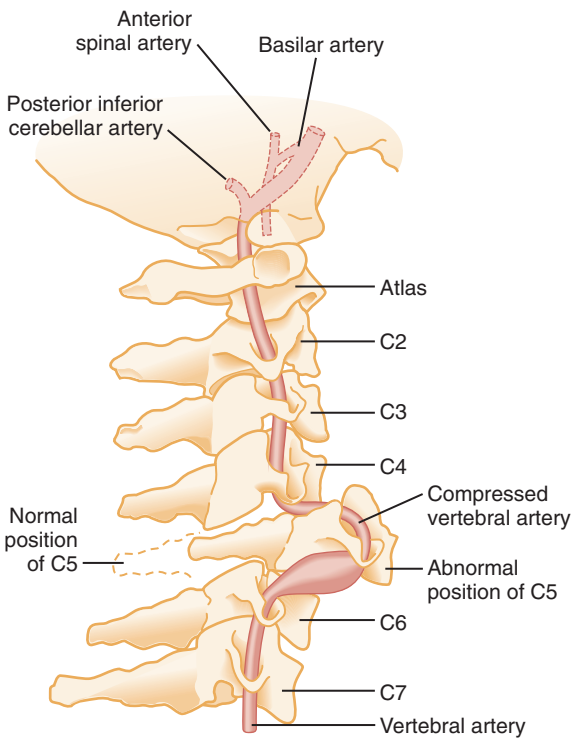



**Fig. 36.23.** Mechanism of vascular injury of the spinal cord resulting from cervical vertebral injury.

**TABLE 36.3**

## Spinal Motor Examination

| LEVEL OF LESION | RESULTING LOSS OF FUNCTION |
|---|---|
| C4 | Spontaneous breathing |
| C5 | Shrugging of shoulders |
| C6 | Flexion at elbow |
| C7 | Extension at elbow |
| C8-T1 | Flexion of fingers |
| T1-T12 | Intercostal and abdominal muscles[a] |
| L1-L2 | Flexion at hip |
| L3 | Adduction at hip |
| L4 | Abduction at hip |
| L5 | Dorsiflexion of foot |
| S1-S2 | Plantar flexion of foot |
| S2-S4 | Rectal sphincter tone |

[a]Localization of lesions in this area is best accomplished with the sensory examination.

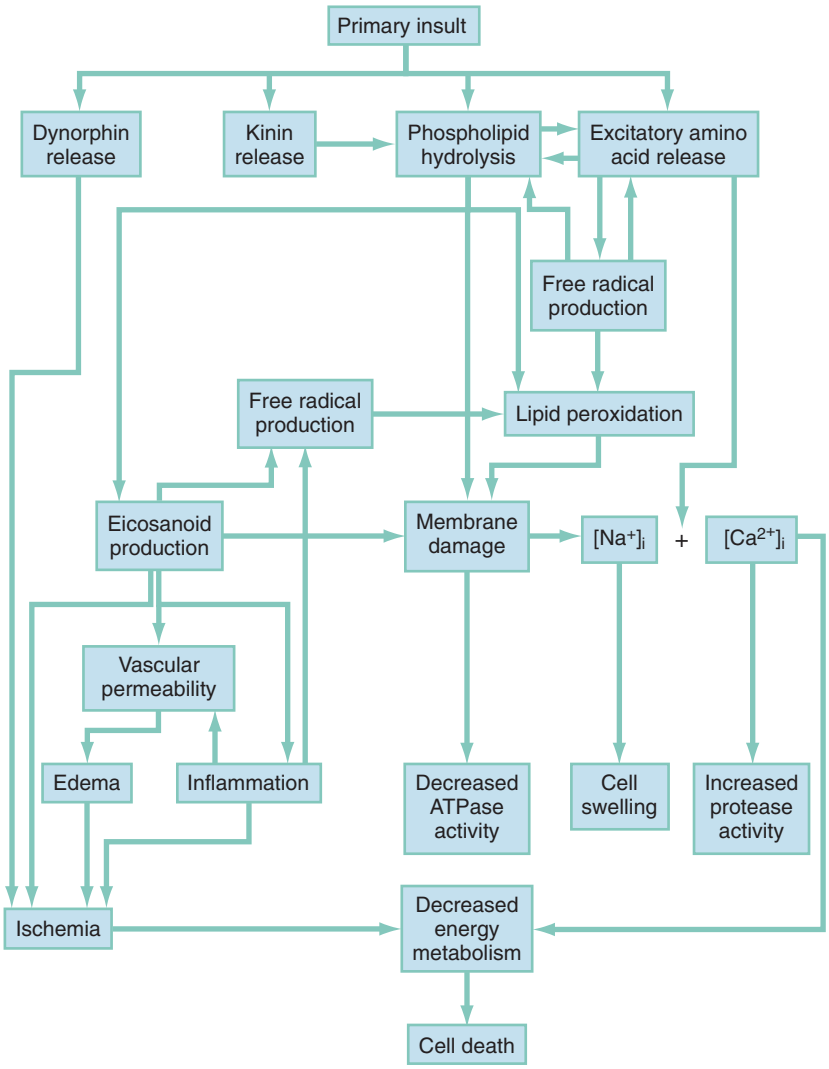



**Fig. 36.24.** Speculative paradigm of secondary pathophysiologic events after primary traumatic injury to the spinal cord. $Ca^{2+}$, Calcium ion; $Na^{+}$, sodium ion.

**TABLE 36.4**

## Spinal Reflex Examination

| LEVEL OF LESION (AT OR ABOVE) | RESULTING LOSS OF REFLEX |
|---|---|
| C6 | Biceps |
| C7 | Triceps |
| L4 | Patellar |
| S1 | Achilles |

response should be elicited and documented, because any response improves prognosis. A slight toe flicker in an otherwise paralyzed individual indicates that the patient may again eventually walk unassisted.

The presence of cord-mediated deep tendon reflexes can be helpful as a localizing diagnostic aid (Table 36.4). Typically, muscle paralysis associated with intact deep tendon reflexes indicates an upper motor neuron (spinal cord) lesion, whereas paralysis associated with absent deep tendon reflexes indicates a lower motor neuron (nerve root or cauda equina) lesion. This differentiation is important because the latter condition may be caused by a surgically correctable lesion. After the initial period of areflexia, reflexes gradually return after 1 to 3 days and, after 1 to 4 weeks, patients with SCI will manifest characteristic hyperreflexia and spasticity. Reflexes are typically absent during the initial phase of spinal shock in the emergency department (ED), however.

Sensory function can be quickly evaluated through the use of a structured approach (Table 36.5) or graphic dermatome chart (Fig. 36.25). After locating an area of hypesthesia, one should move the sensory stimulus from areas of decreased sensation outward, rather than the reverse, because patients are more sensitive to the appearance of sensation than to its disappearance. This test should be performed first with a cotton swab to assess sensitivity to light touch, a posterior column function. A pin should be used to assess pain, which is an anterior spinothalamic tract function. Even in the presence of complete motor paralysis, the presence of islands of preserved sensation within an affected dermatome or below the level of dysfunction indicates potential for functional recovery. An accurate baseline sensory examination is imperative because cephalad progression of hypesthesia is the most sensitive indicator of deterioration. When this is observed in the cervical region, one should anticipate impending respiratory failure and preemptively secure the airway.

**TABLE 36.5**

## Spinal Sensory Examination

| LEVEL OF LESION | RESULTING LEVEL OF LOSS OF SENSATION |
|---|---|
| C2 | Occiput |
| C3 | Thyroid cartilage |
| C4 | Suprasternal notch |
| C5 | Below clavicle |
| C6 | Thumb |
| C7 | Index finger |
| C8 | Small finger |
| T4 | Nipple line |
| T10 | Umbilicus |
| L1 | Femoral pulse |
| L2-L3 | Medial aspect of thigh |
| L4 | Knee |
| L5 | Lateral aspect of calf |
| S1 | Lateral aspect of foot |
| S2-S4 | Perianal region |

## Spinal Cord Lesions

### Complete Spinal Cord Lesions

A complete spinal cord lesion is defined as total loss of motor power and sensation distal to the site of an SCI. Functional motor recovery is rare with a complete cord syndrome that persists for longer than 24 hours. Before making the diagnosis of a complete cord syndrome, however, two points should be considered. First, any evidence of minimal cord function, such as sacral sparing, excludes the patient from this group. Signs of sacral sparing include perianal sensation, preserved rectal sphincter tone, and flexor toe movement. Any of these signs indicates a partial lesion, usually a central cord syndrome, and the patient ultimately may have substantial functional recovery, including bowel and bladder control and eventual ambulation.

Second, a complete spinal cord lesion may be mimicked by a condition termed *spinal shock,* which may persist for a few weeks. Spinal shock results from a concussive injury to the spinal cord that causes total neurologic dysfunction distal to the site of injury. The end of spinal shock is heralded by the return of the bulbocavernosus reflex, which is a normal cord-mediated reflex elicited by placing a gloved finger in the patient's rectum and then squeezing the glans penis or clitoris or by tugging gently on the Foley catheter. An intact reflex results in rectal sphincter contraction. Absence of this reflex indicates the presence of spinal shock, during which time the patient's prognosis cannot be accurately assessed.

### Incomplete Spinal Cord Lesions

Approximately 90% of incomplete spinal injuries can be classified as one of three clinical syndromes—the central cord syndrome, Brown-Séquard syndrome, and anterior cord syndrome (Fig. 36.26). The most common is the central cord syndrome, often seen in patients with degenerative arthritis who suffer neck hyperextension. The ligamentum flavum buckles into the cord, resulting in a concussion of the central gray matter in the pyramidal and spinothalamic tracts. Because fibers innervating distal structures are located in the spinal cord periphery, the upper extremities are more severely affected than the lower extremities. The prognosis is variable, but more than 50% of patients with central cord syndrome become ambulatory and regain bowel and bladder control, as well as some hand function.

The Brown-Séquard syndrome, or hemisection of the spinal cord, usually results from penetrating trauma but may also be seen after lateral mass fractures of the cervical spine. Patients with this lesion have ipsilateral loss of position and vibration sense, as well as motor paralysis, but also have contralateral loss of pain and temperature sensation distal to the level of injury. Because the fibers of the lateral spinal thalamic tract cross at a different level, the pain and temperature loss may be found variably one or two segments above the lesion. Virtually all patients maintain bowel and bladder function and unilateral motor strength, and most become ambulatory.

The anterior cord syndrome results from hyperflexion injuries causing cord contusion by the protrusion of a bony fragment or herniated disk into the spinal canal or by laceration or thrombosis of the anterior spinal artery. This syndrome is characterized by paralysis and hypalgesia below the level of injury, with preservation of posterior column functions, including position, touch, and vibratory sensations. Suspicion for an anterior cord syndrome warrants prompt neurosurgical consultation because it is a potentially surgically correctable lesion. After surgical intervention, patients have variable degrees of recovery during the first 24 hours but little improvement thereafter.

Several less common spinal cord syndromes may result from direct injury to the cervicomedullary junction and upper cervical segments or from vertebral artery occlusion resulting from severe hyperextension (Fig. 36.27). The posteroinferior cerebellar artery syndrome may produce dysphagia, dysphonia, hiccups, nausea, vomiting, dizziness or vertigo, and cerebellar ataxia. The Dejeune onion skin pattern of analgesia of the face is caused by damage to the spinal trigeminal tract. Horner's syndrome results from damage to the cervical sympathetic chain and is characterized by ipsilateral ptosis, miosis, and anhidrosis. Injuries below the L2 level can result in an acute cauda equina syndrome, characterized by perineal or bilateral leg pain, bowel or bladder dysfunction, perianal anesthesia, diminished rectal sphincter tone, and lower extremity weakness.

The syndrome of SCIWORA is seen primarily in younger children but may occur in any age group. In fact, there is increasing evidence that SCIWORA has been underreported in adults.[6] The mechanism is unclear but has been ascribed to the increased ligamentous elasticity seen in the young, leading to transient spinal column subluxation, stretching of the spinal cord, and vascular compromise. Patients often experience a brief episode of upper extremity weakness or paresthesias followed by neurologic deficits that appear hours to days later. The prognosis for patients with SCIWORA is variable, depending on the degree of neurologic impairment and rate of resolution

## DIFFERENTIAL DIAGNOSIS

The differential diagnosis of spinal injuries includes peripheral nerve injuries that may mimic sensory or motor deficits from a central lesion. For example, compression of the superficial peroneal nerve from a fibular fracture may result in a foot drop, but impingement of a lumbar spinal nerve root from a lumbar vertebral fracture could also result weakness in dorsiflexion. As noted, ligamentous injury in SCIWORA is also a consideration, especially if no fractures are found on imaging. Muscle contusions and strains around the neck, thorax, and lumbosacral regions would also be part of the differential diagnosis. Finally, a diagnosis of exclusion, conversion disorder can result in apparent

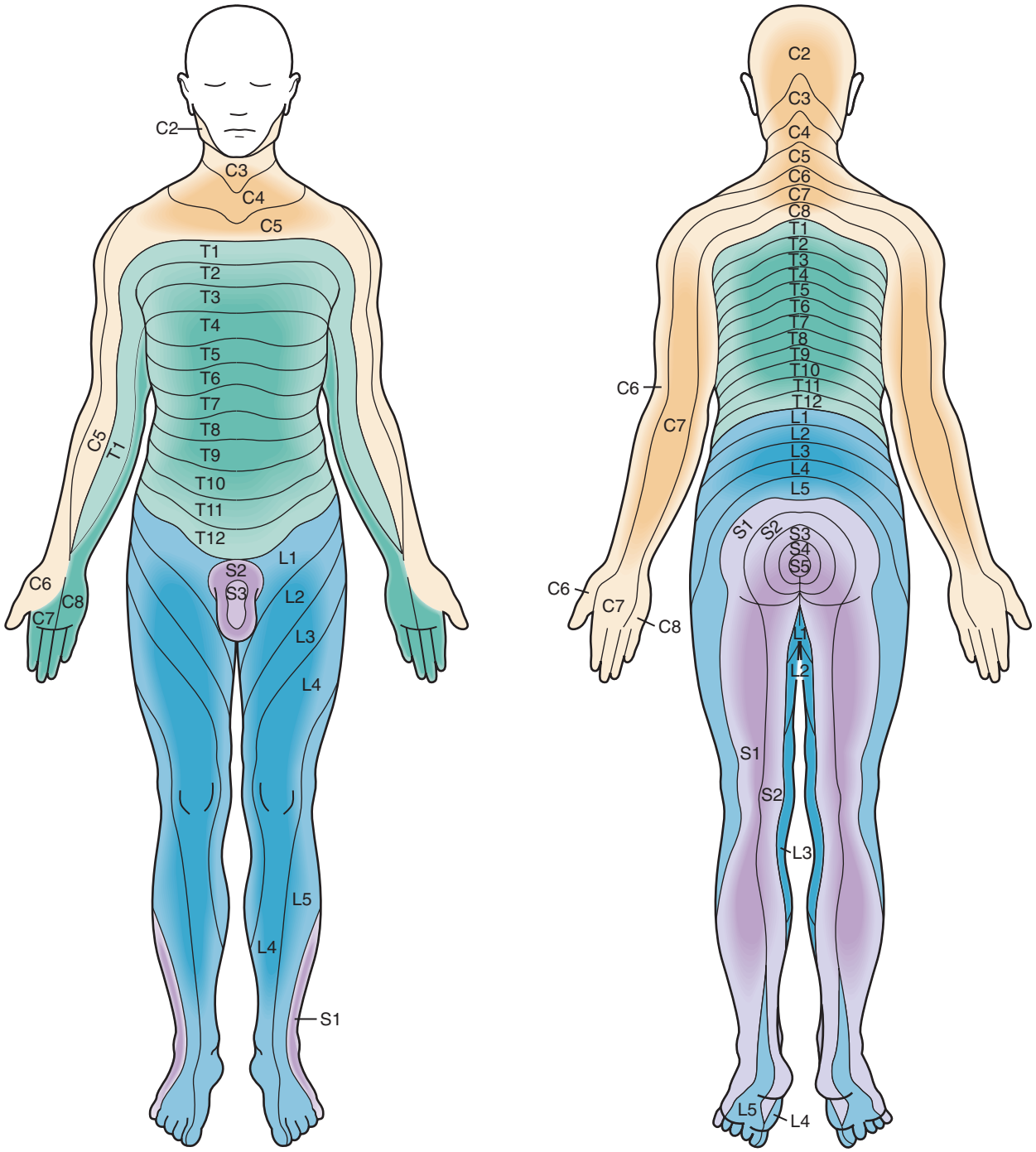



**Fig. 36.25.** Sensory dermatomes.

manifestations of sensory and motor deficits that may initially be confused and attributed to spinal injuries.

## DIAGNOSTIC TESTING

### Radiographic Evaluation

#### Indications

Emergency clinicians have historically taken a liberal approach to imaging the cervical spine in the setting of trauma because failure to recognize an SCI may result in devastating neurologic consequences. In an effort to standardize clinical practice and guide emergency clinicians to be more selective in radiographic imaging without jeopardizing patient care, two clinical decision rules have been developed. Use of selective but safe imaging modality may decrease overall health care costs, reduce radiation exposure, and decrease complications (eg, aspiration and pressure trauma to skin) associated with the patients lying flat on a backboards with a rigid collar. The first rule to be developed, the National Emergency X-Radiography Utilization Study (NEXUS) Low-Risk Criteria (NLC), was based on a multicenter prospective observational study involving almost 35,000 trauma patients seen at 21 EDs in the United States. The decision instrument required patients to meet five criteria to be classified as having a low probability of injury: (1) no midline cervical tenderness; (2) no focal neurologic deficit; (3) normal alertness; (4) no intoxication; and (5) no painful, distracting injury. The decision rule identified all but 8 of

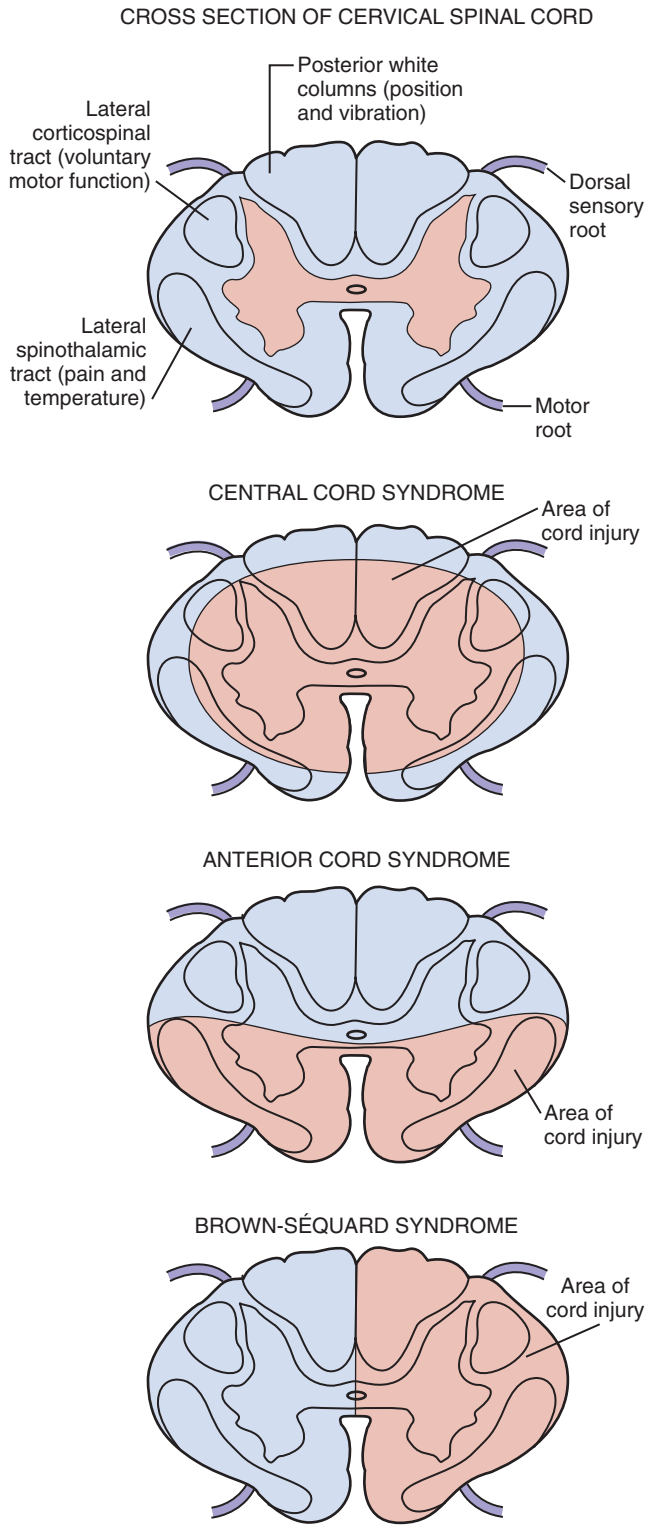



**Fig. 36.26.** Incomplete spinal cord syndromes.

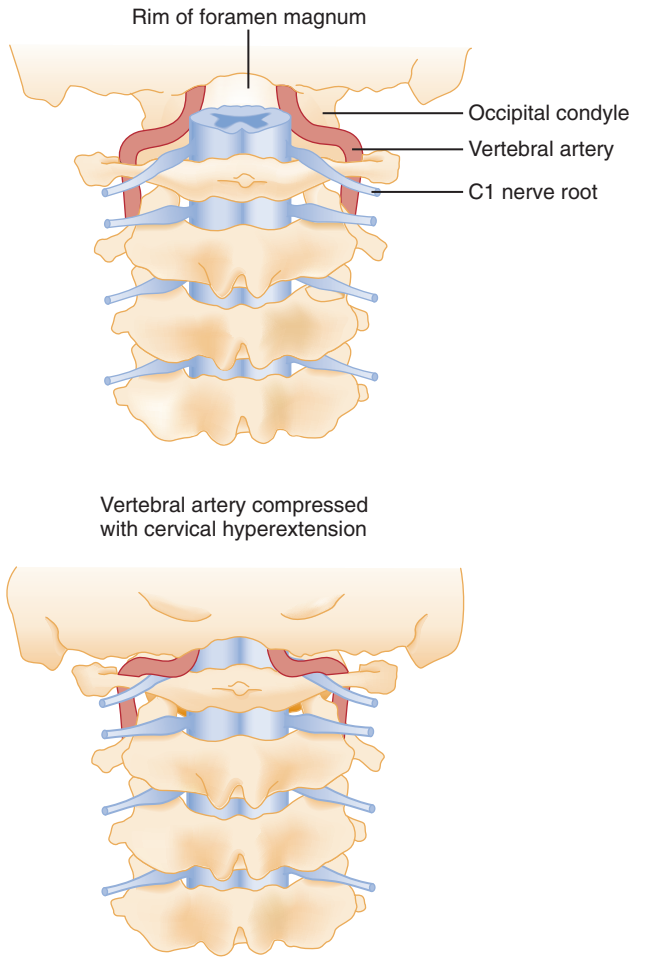



**Fig. 36.27.** Mechanism of vertebral artery injury in extension injuries of the cervical spine.

the 818 patients who had spinal injuries. Two of these patients had a clinically significant injury, only one of whom required surgical stabilization, and neither sustained a permanent neurologic injury. Sensitivity, specificity, and negative predictive value of the NLC were 99.6%, 12.9%, and 99.8%, respectively.

Owing to concerns about the low specificity of the NLC, the Canadian C-Spine Rule (CCR) was developed using 25 selected clinical predictor variables associated with spine injury. In 2003, the CCR was prospectively studied and compared with the NLC in nine Canadian tertiary care hospitals. Of 8283 patients, 162 were found to have clinically significant injuries, and the sensitivity, specificity, and negative predictive values of the CCR were, respectively, 99.4%, 45.1%, and 100%.The CCR is composed of the following three questions:

1. Are there any high-risk factors that mandate radiography?
2. Are there any low-risk factors that allow safe assessment of range of motion?
3. Is the patient able to rotate his or her neck actively 45 degrees to the left and right?

According to the CCR, patients with no high-risk factors, any low-risk factor, and the ability to rotate the neck do not require radiographic evaluation. High-risk factors include age older than 65 years, a dangerous mechanism of injury (eg, fall from a height >1 m, axial loading injury, high-speed MVC [>100 km/hr], rollover, ejection, motorized recreational vehicle or bicycle collision), or the presence of paresthesias. Low-risk factors include simple rear-end vehicle crashes, to a sitting position in the ED, ambulatory at any time, delayed onset of neck pain, and absence of midline neck tenderness. Although the NEXUS criteria are more widely used in the United States, there is controversy regarding which of the two rules to implement; a systematic review demonstrated better diagnostic accuracy for the CCR.[7] There are methodologic differences in the respective study designs, such as different inclusion and exclusion criteria.[8] Nonetheless, both rules have been well-validated and are sensitive, and the use of either rule decreases the number of unnecessary radiographs while rarely missing clinically significant injuries.

## Cervical Plain Radiographs

Due to the widespread availability and superior test characteristics of CT in the United States, spinal plain radiographs are now rarely obtained, especially when CT is ordered to visualize a different body part. Furthermore, plain radiographs have been shown to be inadequate to visualize the entire cervical spine in up to 72% of cases, thus necessitating CT. However, plain radiographs are often used outside the United States, and there is increasing concern regarding cost and exposure to medical radiation from CT. When compared to plain radiographs, CT respectively confers a 10- to14-fold increase in radiation exposure to the skin and thyroid.

Thus, in light of cost and radiation exposure, plain radiographs of the cervical spine may be preferentially obtained in patients who sustain a relatively minor mechanism of injury but fail the NLC and CCR criteria, and do not warrant CT of the head or other body parts. On plain radiographs, the C7-T1 vertebrae may be obscured in muscular or obese patients, as well as in patients with spinal lesions causing paralysis of the muscles that act to depress the shoulders. In this case, a swimmer's view of the lower cervical vertebrae, or CT, is often needed. The cross-table lateral view of the cervical spine is the most helpful x-ray, but its inadequacy as the sole view is well documented. The diagnostic yield is significantly increased when the AP and odontoid views are included. The NLC has shown that a technically adequate three-view trauma series will fail to diagnose significant spinal injury in only 0.07% of patients with injuries and in only 0.008% of patients with unstable injuries. Note that once CT is performed, however, plain radiographs do not add any further clinically relevant information and should not be obtained.

**Cross-Table Lateral View.** The inspection of the lateral cervical spine film should be methodical and complete. It is helpful to remember the ABCs of interpreting the lateral film, where A stands for alignment, B for bony abnormalities, C for cartilage space assessment, and s for soft tissues.

To check alignment, two imaginary lines are drawn that connect the anterior and posterior margins of the vertebral bodies, the anterior and posterior contour lines. A third line, the spinolaminal line, connects the bases of the spinous processes extending to the posterior aspect of the foramen magnum (Fig. 36.28). All three lines should form a smooth, continuous lordotic curve, and any disruption of these lines suggests a bony or ligamentous injury. An exception to this rule is the pseudosubluxation of C2 and C3, which is commonly seen in infants and children. This phenomenon is attributed to immature muscular development and a hypermobile spine. Thus, if a high cervical injury is suspected in a child, the posterior cervical line, which connects the points bisecting the bases of the spinous processes of C1 and C3, should be used (Fig. 36.29). If the base of C2 lies more than 2 mm anterior or posterior to the posterior cervical line, an injury at that level should be suspected. On the lateral view, the predental space, which is the distance between the anterior aspect of the odontoid process and posterior aspect of the anterior ring of C1, should not exceed 3 mm in an adult or 5 mm in a child (Fig. 36.30). A widening of this space may indicate a Jefferson fracture of C1.

Subtle signs of cervical subluxations and dislocations can be identified through cartilage space assessment. A slight anterior or posterior widening of the intervertebral or interspinous space may be the only clue to an unstable dislocation.

Finally, the soft tissues of the retropharyngeal space should be assessed for prevertebral swelling and hemorrhage, often the only radiographic signs of spinal injury. The retropharyngeal space, measured from the anterior border of the body of C2 to the posterior wall of the pharynx, should not exceed 6 mm in children or adults. At the level of C3 and C4, this should not exceed 5 mm or should be less than half the width of the vertebral body at that level (see Fig. 36.30). Below the level of C4, the prevertebral soft tissue space is widened by the esophagus and cricopharyngeal muscle. The retrotracheal space, measured from the anterior border of the body of C6 to the posterior wall of the trachea, should not exceed 22 mm in adults or 14 mm in children younger than 15 years. In children younger than 2 years, the retropharyngeal space may normally appear widened during expiration; therefore, inspiratory films should be obtained. Air in the prevertebral space may indicate rupture of the esophagus or some portion of the respiratory tree, and anterior bulging of the prevertebral fat stripe is an excellent sign of an underlying bony or soft tissue injury.

**Odontoid View.** The open-mouth or closed-mouth view of the atlas and axis can be helpful in diagnosing Jefferson and odontoid fractures. Nonfusion of the odontoid in children and congenital anomalies of the odontoid in adults may mimic fractures.

**Anteroposterior View.** The AP spinal film completes the spinal series. Connecting imaginary dots placed at the base of each spinous processes should form a straight line, and the laryngeal and tracheal air shadows should be midline. The regular outline of the lateral masses should be verified, and the pedicles viewed

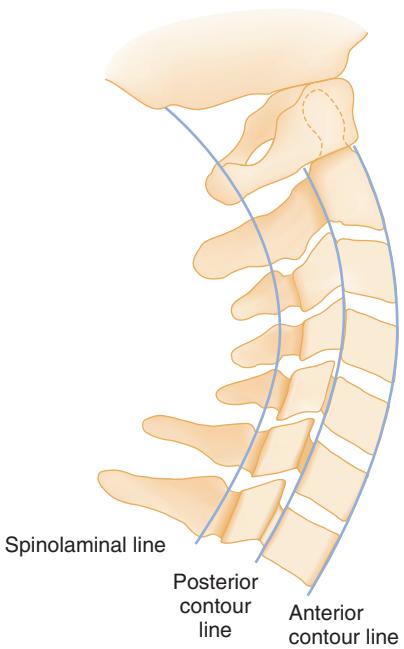



**Fig. 36.28.** Normal structural relationships of the lateral cervical spine.

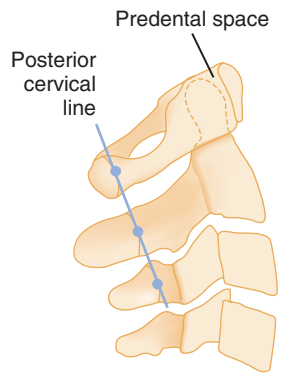



**Fig. 36.29.** Posterior cervical line of a normal lateral spine.

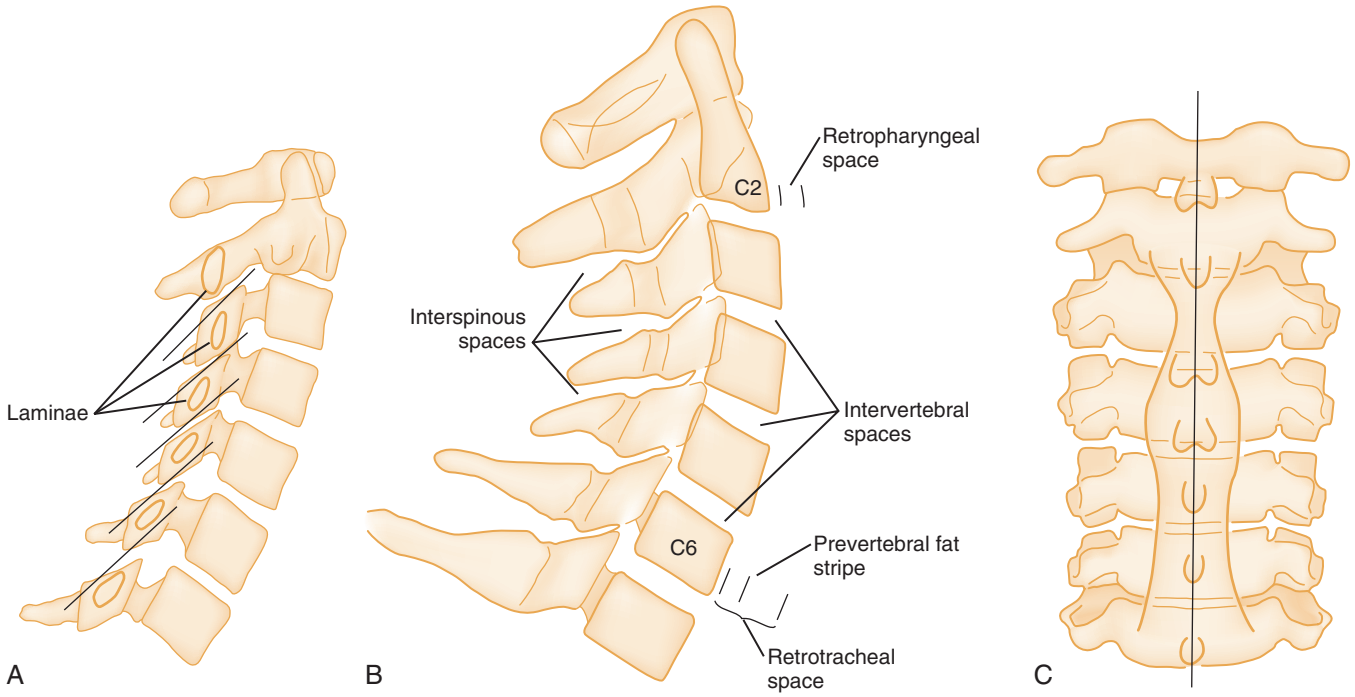



**Fig. 36.30.** **A**, Normal structural relationships of the cervical spine laminae in an oblique view form a so-called shingles on a roof appearance. **B,** In the lateral view, the intervertebral spaces and interspinous spaces should be compared with the spaces above and below for asymmetry and important clues in flexion and extension injuries. The retropharyngeal and retrotracheal soft tissues are measured at the C2 and C6 levels for swelling. **C,** Normal relationship between soft tissues and bony structures of the cervical spine in the lateral and anteroposterior (AP) views. **C,** In the AP view, the tracheal and laryngeal air shadows should be within the midline. A straight line should connect points bisecting the spinous processes. If such is not the case, rotatory injuries are suspected.

end-on can be checked for fracture. Widening of the interpedicular distance compared with adjacent vertebrae suggests a burst fracture (Fig. 36.31). Bulging of the mediastinal stripe may be the only evidence of a thoracic vertebral body fracture, which may cause hemorrhage that produces mediastinal widening on the chest x-ray.

Flexion and Extension Views. Flexion-extension (F/E) views are rarely indicated in the acute evaluation of a patient presenting to the ED after acute trauma, but may be useful when there is concern for ligamentous injury and magnetic resonance imaging (MRI) is not available. F/E views should be obtained only in patients who are alert and able to articulate the presence of pain, numbness, or paresthesias, because such symptomatology may indicate instability. The NEXUS investigators demonstrated that 86 of 818 patients(10.5%) ultimately found to have cervical injury underwent F/E testing. Although two patients had bony injuries and four patients had subluxations demonstrated only on F/E views, all six patients had other injuries apparent on routine radiographs.

F/E views are also deemed inadequate for interpretation in nearly one-third of studies.[8] A more recent review of 1000 F-E radiographs revealed that 80% of the films did not demonstrate the C7-T1 junction or had less than a 30-degree range of motion.[9] In the acute setting, F/E radiographs have been reported to have unacceptably high false-positive and false-negative rates because of concomitant muscle spasm. Delayed F/E views obtained 1 week after injury may be helpful, but they have little value in the ED when the CT scan is negative.[10] Thus, we do not recommend obtaining F/E radiographs in the ED unless there is concern for ligamentous instability in an alert evaluable patient, and MRI is not available. Such evaluation should occur in consultation with, and images should be obtained under the supervision of, a spine or trauma surgeon.

## Advanced Imaging: Computed Tomography and Magnetic Resonance

The CT scan is the technique of choice for the evaluation of acute cervical spine trauma because of its superior test characteristics and time efficiency in the radiology department when compared to plain radiography. CT permits examination without moving the patient from the supine position and is thus preferable in terms of fracture stabilization, airway control, and other life support measures. CT can also identify bony fragments, acute disk herniation, foreign body, paraspinal hematoma, or extramedullary hematoma. Thus, routine plain radiographs in many centers are reserved for the alert patient with minor trauma. In addition to those undergoing CT imaging of other body parts, CT may be preferred when plain radiographs are difficult to interpret because of abnormal anatomy, such as in older adults with degenerative disease or the patient with rheumatoid arthritis. Additionally, rotational and distraction injuries resulting in atlanto-occipital dislocations may be missed on plain x-ray. For patients who have a severe mechanism of injury, unless CT is not available, we support the practice guidelines from the Eastern Association for the Surgery of Trauma, which recommend that CT from the occiput to T1 be used as the primary screening. Because fractures in contiguous and noncontiguous vertebrae are fairly common, CT scans should be obtained to visualize the entire cervical spine.

Fractures involving the transverse foramina or C1-C3 are associated with vertebral artery dissection or thrombosis in up 22% of cases, as well as basilar artery stroke. When such fractures are identified, we recommend further study by magnetic

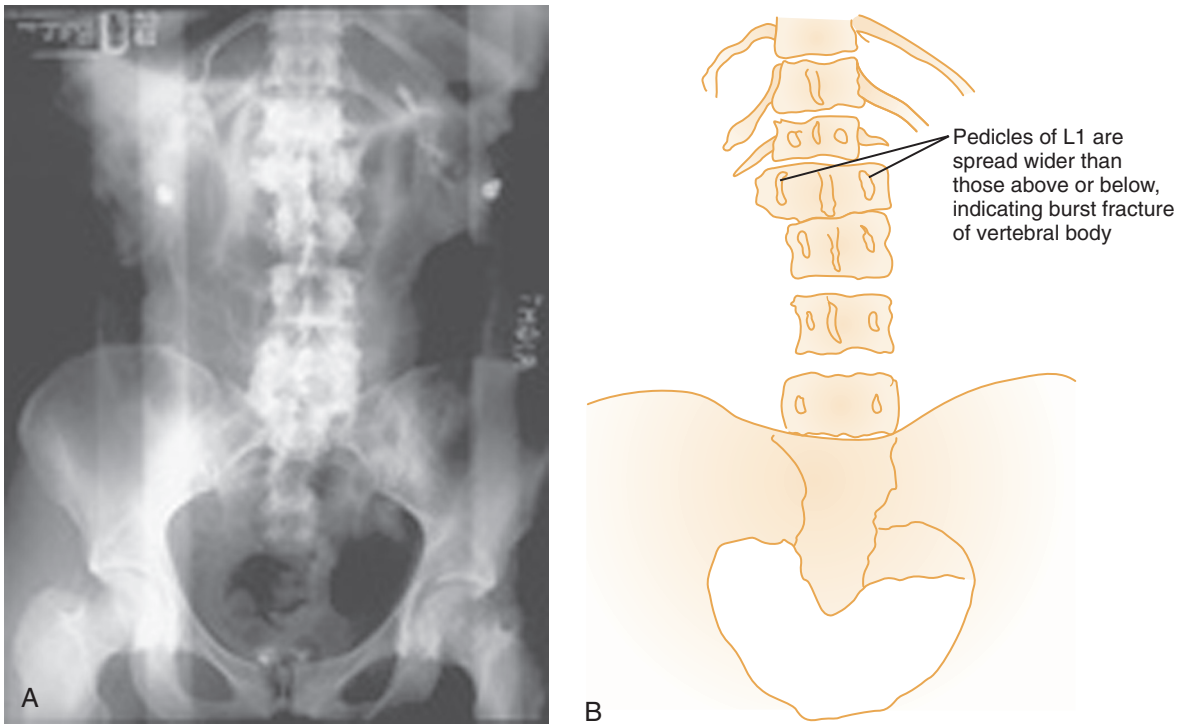



**Fig. 36.31.** **A**, **B,** Burst fracture of L1. An anteroposterior radiograph shows increased distance between the pedicles of L1 in comparison to adjacent vertebrae. An intravenous pyelogram showed renal injury on the left.

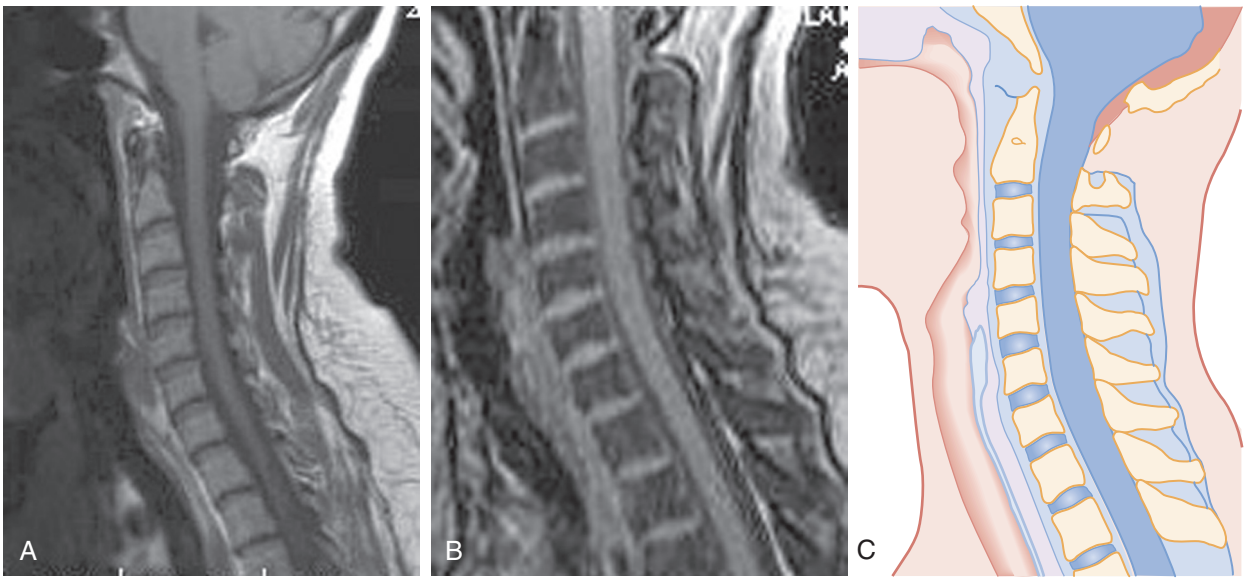



**Fig. 36.32.** Normal sagittal magnetic resonance images of the cervical spine. **A,** T1-weighted and flip angle (**B**) scans. **C,** Cervical spine.

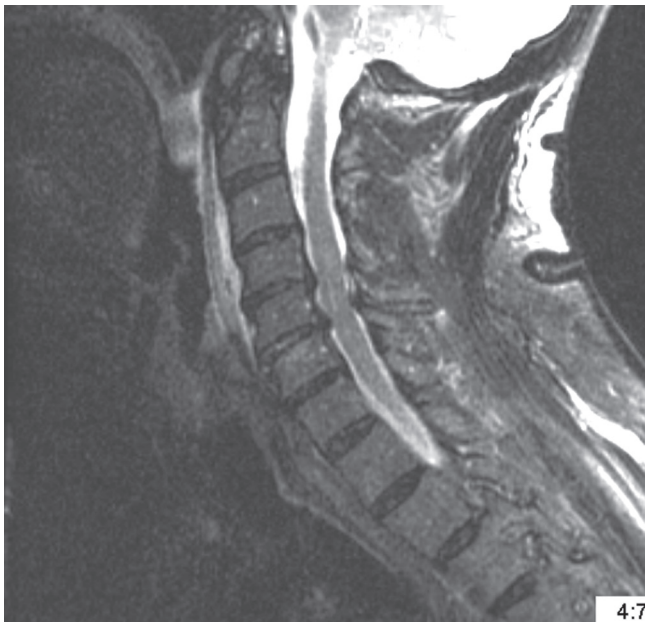
resonance angiography (MRA), CT angiography (CTA), or four-vessel angiography.

Vertebral images reconstructed from CT scans of the abdomen and pelvis obtained for the evaluation of chest and abdominal injuries provide sufficient data to screen for spinal fractures. CT is also thought to be adequate to clear cervical spines, even in the obtunded blunt trauma patient; in fact, a meta-analysis of 10 studies involving 1850 obtunded trauma patients has demonstrated a negative predictive value and specificity greater than 99%,[11] whereas a single-center cohort study of 83 patients demonstrated a sensitivity and specificity of 100% for CT in detecting unstable cervical spine injuries compared to MRI.[12,13]

Although CT has a higher sensitivity than MRI to detect fractures and dislocations at the craniocervical junction, as well as fractures of the posterior elements, MRI, with its superior resolution and lack of ionizing radiation, has the primary advantage of the ability to image nonosseous structures directly, including intramedullary and extramedullary spinal abnormalities that potentially cause neurologic deficit (Fig. 36.32). Its major impact has therefore been in demonstrating potentially surgically correctable lesions, including acute disk herniation, ligamentous injury, bony compression, epidural and subdural hemorrhages, and vertebral artery occlusion. MRI can identify three separate patterns of SCI, including acute cord hemorrhage, cord edema or

contusion, and mixed cord injury. Patients with cord edema or contusion show significant neurologic improvement, whereas those with cord hemorrhage (Fig. 36.33) fare far worse. MRI can also diagnose a developing intramedullary (posttraumatic) syrinx or subarachnoid cystic changes (Fig. 36.34). MRI is also the best diagnostic imaging modality for SCIWORA. Thus, a patient who demonstrates neurologic deficit or persistent neck pain suggesting ligamentous injury or an occult spine injury, should undergo an expedited MRI, regardless of a normal CT scan or plain radiograph (Fig. 36.35).

There are risks to performing an MRI, however, such as aspiration, secondary brain injury, and the difficulty of monitoring and resuscitation in the MRI suite. In addition, MRI cannot be used when MRI-incompatible life support, monitoring systems, pacemakers, cerebral aneurysm clips, and cervical traction devices are used, although MRI-compatible support systems exist. In the obtunded or unreliable patient, MRI may not be necessary to exclude unstable injuries if the CT scan is normal. A recent prospective study of the use of cervical spine CT in 402 obtunded patients reported a sensitivity of greater than 99%.[14]



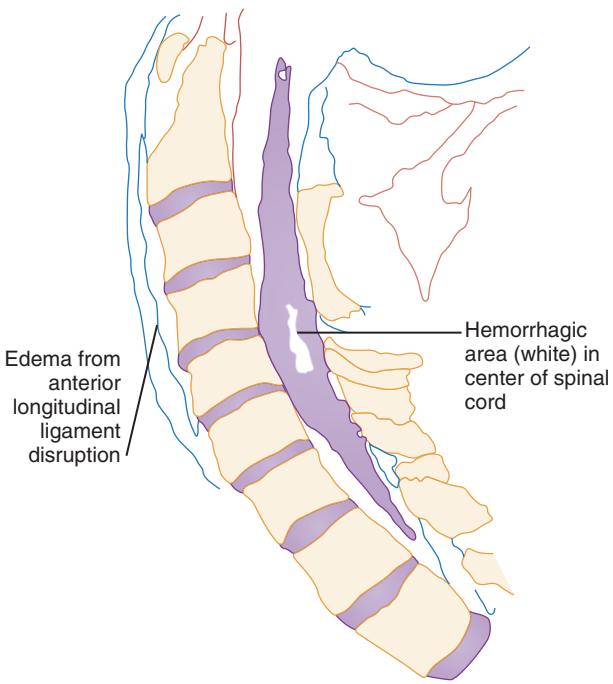




**Fig. 36.33.** MRI scan showing a small area of central cord hemorrhage and anterior and posterior ligamentous disruption.

## MANAGEMENT

Spinal injury should be suspected in all trauma victims with an unknown or suggestive mechanism of injury associated with complaints of neck or back pain, evidence of significant head or facial trauma, spinal tenderness, signs of focal neurologic deficit, impaired consciousness, potentially distracting injuries, or unexplained hypotension (Fig. 36.36).

### Spinal Column Stabilization

#### Out-of-Hospital Care

Prehospital personnel are well versed in the care of the patient with a potentially traumatized spine, and all emergency medical services (EMS) incorporate these principles. The traditional approach to immobilization requires the use of a backboard, rigid cervical collar, and supportive blocks on both sides of the head. In the past, a concerning mechanism of injury called for automatic and routine initiation of such spinal immobilization at the scene. However, it has been noted that many trauma patients are unnecessarily immobilized by EMS, and immobilization is not a benign intervention. For example, in addition to resulting in prolonged on-scene time and delayed transport to definitive care, the backboard can lead to pressure ulcers, increased pain, and decreased functional respiratory residual capacity. Also, the cervical collars can hide other injuries, such as lacerations and hematomas, and have even been found to result in worsening vertebral distraction injuries.[15] There is also ample evidence that EMS providers can safely apply spinal assessment guidelines, such as NEXUS.

#### Emergency Department

Trauma victims are assessed as described in Chapter 33 while maintaining immobilization. If the patient's spine can be clinically cleared by use of the NEXUS criteria or CCR, the immobilization device may be removed. If the trauma victim was wearing a helmet and the helmet was not removed in the field, the face mask, helmet, and any sports padding (eg, shoulder pads on hockey or football players) may be carefully removed while immobilization is maintained. Ideally, at least two or three providers should be present to perform the task of helmet removal. Once the helmet and shoulder pads have been removed, a rigid collar should be placed if the patient's cervical spine cannot be cleared by use of the NEXUS criteria or CCR.

Patients with probable spinal injury who are conscious and cooperative should be immobilized until imaging has been performed. Patients who are uncooperative because of head injury, drug or alcohol intoxication, hypotension, or presence of multiple painful injuries require a deliberate approach, including the use of chemical and mechanical restraints. Suspected thoracic and lumbar spinal injuries are best managed by keeping the patient supine and immobile. The goal of stabilization in cervical spine trauma is to immobilize the neck and body because any movement may extend the initial injury. If the patient is not already immobilized on a backboard, the torso should be firmly anchored to the examining table by straps or rolled sheets. Sedation, drug-induced paralysis, and intubation may be required for patients who pose a danger to themselves because of excessive movement and whose injuries otherwise will likely require intubation. Paralysis and intubation are not used simply to control patient

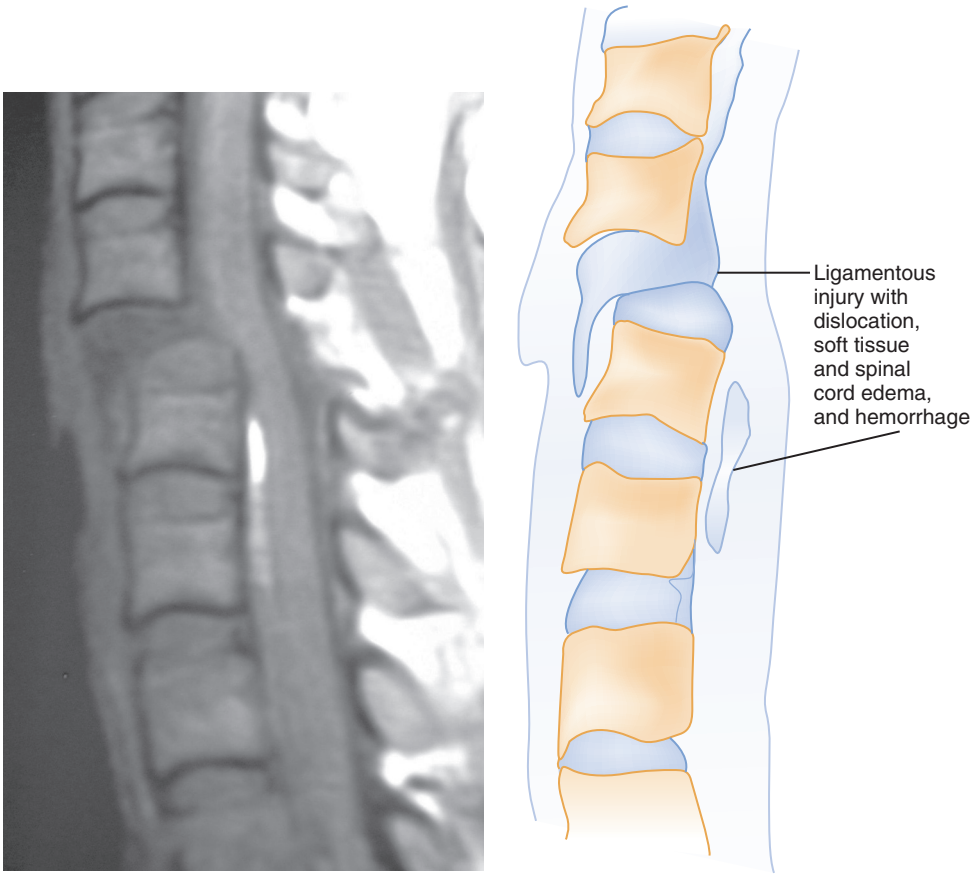



**Fig. 36.34.** Anteroposterior longitudinal ligament disruption. A sagittal MRI scan demonstrates ligamentous disruption between C4 and C5, with blood tracking in the anterior spinal canal.

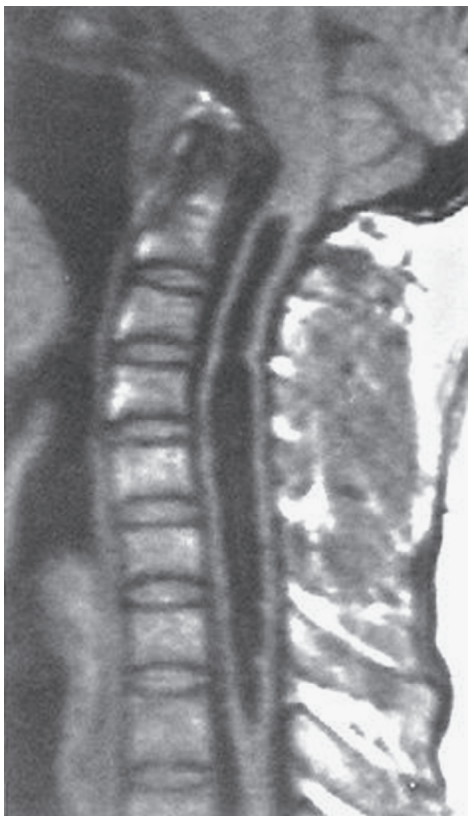


**Fig. 36.35.** MRI scan showing posttraumatic syrinx of the spinal cord.

movement or lack of cooperation. Spinal precautions should be maintained in patients with an altered sensorium until the presence of an injury can be excluded clinically or radiographically. Suctioning should be readily available to prevent aspiration. Vomiting patients should be placed on their side by logrolling while spinal alignment is maintained.

## Airway Management

Cervical spine injuries often require early intubation as part of the resuscitation. Lesions above C3 may rapidly progress to respiratory paralysis, and the spread of edema from a lower injury may cause delayed phrenic nerve paralysis, as well as ascension of the neurologic injury above the level of C3. Cervical injuries may be associated with airway obstruction from retropharyngeal hemorrhage or edema or maxillofacial trauma.

Airway management of the trauma patient, including those with suspected spine injury, is discussed in Chapter 1.

## Spinal Shock

Spinal shock is characterized by the temporary loss of neurologic function and autonomic tone below the level of an acute spinal cord lesion. Patients usually exhibit flaccid paralysis with loss of sensation, deep tendon reflexes, and urinary retention, along with bradycardia, hypotension, hypothermia, and intestinal ileus. Recovery from spinal shock, which may last from less than 24 hours to more than 2 weeks, is heralded by the return of the bulbocavernosus reflex.

Neurogenic hypotension, caused by loss of vasomotor tone and lack of reflex tachycardia, is a diagnosis of exclusion in the trauma victim. It should not be considered the cause of hypotension

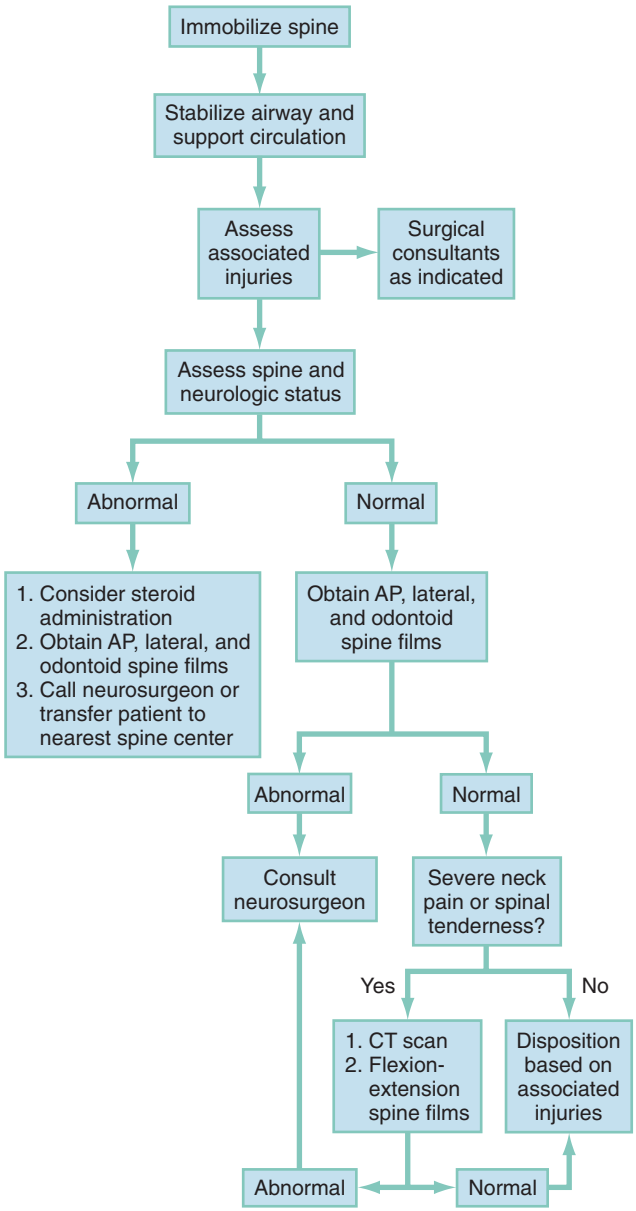



**Fig. 36.36.** Approach to a patient with suspected cervical spine injury. *AP,* Anteroposterior; *CT,* computed tomography.

unless the patient is flaccid and areflexic, reflex tachycardia and peripheral vasoconstriction are absent and, most important, the possibilities of coexisting hemorrhagic shock, cardiac tamponade, or tension pneumothorax have been eliminated.

Although there is no evidence for an optimal mean arterial pressure (MAP), we recommend initiating the resuscitation of hypotensive trauma victims with a balanced crystalloid fluid infusion, as outlined in Chapter 33. Most cases of pure neurogenic hypotension are mild (eg, systolic blood pressure > 90 mm Hg) and may not require fluid resuscitation or will respond to modest amounts of fluid. Severe neurogenic hypotension(eg, systolic blood pressure < 70 mm Hg), seen in 20% to 30% of cases, usually occurs with high cervical injuries associated with total or near-total loss of neurologic function. Because hypotension can lead to hypoperfusion and secondary spinal cord ischemia, prolonged severe hypotension (systolic blood pressure < 70 mm Hg) should be prevented and treated. Fluid resuscitation is often ineffective in such patients and may result in fluid overload. Thus, when there is persistent hypotension despite fluids, we recommend vasopressor support with norepinephrine to be started at 0.05 μg/kg/min and titrated upward to a maximum dose of 1 μg/kg/min to achieve an MAP of 85 mm Hg.[16]

## Pharmacologic Treatment for Incomplete Cord Injury

Delayed biochemical damage contributes to ongoing tissue loss and worsening neurologic function in SCI. Thus, numerous neuroprotective and neuroregenerative treatment strategies, including pharmacologic treatment, hypothermia, and decompression,[17-19] have been investigated in laboratory animal studies and human clinical trials. Substantial media attention was prompted by case reports of athletes, such as the Buffalo Bills tight end Kevin Everett, who underwent therapeutic hypothermia and was subsequently able to walk just 3 months after his treatment. Since 2010, there has been one prospective case series of 20 patients,[17] two retrospective case series,[20,21] and one case report.[19] In all these studies, the patients had surgical decompression in addition to the hypothermia treatment (32°–34° C [89.6°–93.2° F) for 6 to 48 hours and, although there appeared to be some association of hypothermia with improvement in the American Spinal Injury Association Impairment Scale, this cannot be considered evidence in support of the use of therapeutic hypothermia for acute spinal cord injury. Reported complications from hypothermia induction include pneumonia, thrombocytopenia, and atrial fibrillation. The Miami Project to Cure Paralysis is a phase 1 study currently being conducted at the University of Miami and should be able to help delineate the risks and benefits better, as well as the duration of hypothermia.[22] At this time, hypothermia should be considered experimental.

Methylprednisolone, once widely recommended for use on the basis of extremely weak evidence, has been found to have no benefit and is likely, on balance, to be harmful. It is no longer recommended or used for acute spinal cord injury.

## Associated Injuries

### Cardiopulmonary

Although cardiopulmonary deterioration in a trauma victim is usually the result of hemorrhagic shock or direct injury to the heart or lungs, pulmonary edema may also occur in response to brain injury and SCI. Spinal cord trauma stimulates an intense sympathetic discharge with two subsequent effects. First, pulmonary capillary endothelial cells are disrupted, leading to the pulmonary capillary leak syndrome, in which pulmonary edema occurs in the presence of normal pulmonary artery pressures (<18 mm Hg). Second, marked increases in afterload may lead to pulmonary edema associated with high pulmonary artery pressures (>18 mm Hg) from ventricular dysfunction. Excessive fluid resuscitation can also contribute to pulmonary edema. Later in the recovery period, many SCI patients suffer from alternating episodes of low and high blood pressure, often with labile heart rates, termed *autonomic dysreflexia*.[23] The treatment for this is primarily supportive by addressing causative factors, such as bladder distention, pain, and hydration status.

### Gastrointestinal and Genitourinary

If SCI renders the abdominal examination unreliable, an abdominal CT scan or ultrasound is often necessary. In the acute stages of SCI, the gastrointestinal tract and bladder become atonic. Thus, a nasogastric tube should be placed to prevent gastric distention and a Foley catheter inserted to prevent bladder distention and monitor fluid output. Because gastrointestinal bleeding from

stress ulcers occurs in 2% to 20% of spinal trauma patients, ulcer prophylaxis with histamine H2 receptor antagonists or proton pump inhibitors should be initiated.

### Skin

Denervated skin is extremely susceptible to pressure necrosis, and sores can develop in less than 1 hour on unpadded spinal carts. Therefore, backboards should be removed as soon as possible. Padding pressure areas with sheepskin or foam can help minimize decubitus ulcers.

## Definitive Treatment and Prognosis

The role of prompt surgical intervention in the management of spinal injuries is currently limited to relieving spinal cord impingement caused by foreign bodies, herniated disks, bony fracture fragments, or epidural hematoma. Surgery may be necessary later to stabilize severe bony injuries or reduce spinal dislocations. The timing of surgical intervention is controversial because there are no well-designed studies that have determined whether early (<12 hours) versus late decompression is beneficial.

Once almost uniformly fatal, major spinal injury caused death from pulmonary complications or sepsis from skin necrosis or urinary infection. The advent of antibiotic therapy made long-term survival not only possible but also expected. Today, patients with SCIs are best managed at a regional spine injury center, where a team of neurosurgeons, orthopedic surgeons, psychologists, and physical therapists can initiate rehabilitation. Specialized SCI treatment centers offer patients a chance to return to a productive life within the limits of their disability. With the exception of patients with high cervical lesions (above C5), most patients attain sufficient independence to live outside of high-level care environments.

# DISPOSITION

## Cervical Soft Tissue Injuries

Patients with cervical soft tissue injuries of the spine who have only mild to moderate discomfort without neurologic impairment or abnormal radiographic findings (WAD class 1 or 2) are best managed as outpatients. Discharge instructions should include educating the patient that pain often increases over the first 24 to 48 hours but that the symptoms will begin to dissipate thereafter. We recommend treatment with analgesics, such as acetaminophen, 650 to 1000 mg/dose, up to qid. Although analgesic doses of a nonsteroidal antiinflammatory drug, such as ibuprofen, 400 to 600 mg/dose, is also reasonable, there is ample evidence that acetaminophen, which has fewer adverse effects on the gastrointestinal and renal systems, is equally effective, and there is no indication for antiinflammatory treatment. Additionally, we do not recommend medications with purported muscle relaxant properties, such as cyclobenzaprine, because they have not been found to provide additional benefit and have an adverse side effect profile (principally anticholinergic effects and drowsiness.).[24] Finally, referral for follow-up with a primary care physician is indicated because up to 50%of patients experiencing neck pain after trauma will continue to have symptoms at 1 year. This is more likely in patients with WAD class 3 but can occur in patients with class 2 and rarely class 1. Box 36.1 lists the prognostic indicators of poor functional recovery in patients with a WAD.

**BOX 36.1**

### Prognostic Indicators of Poor Functional Recovery Following Whiplash-Associated Disorders

**FACTORS WITH CONSISTENT EVIDENCE FOR BEING PROGNOSTIC INDICATORS FOR POOR RECOVERY**

- Initial pain levels > 5.5/10
- Initial disability levels: NDI > 29%
- Symptoms of posttraumatic stress
- Negative expectations of recovery
- High pain catastrophizing
- Cold hyperalgesia

**FACTORS WITH CONSISTENT EVIDENCE OF NOT BEING PROGNOSTIC INDICATORS**

- Accident-related features (eg, collision awareness, position in vehicle, speed of accident)
- Findings on imaging
- Motor dysfunction

**FACTORS WITH INCONSISTENT EVIDENCE**

- Older age
- Female gender
- Neck range of movement
- Compensation-related factors

*NDI,* Neurological Disability Index.
Adapted from Sterling S: Physiotherapy management of whiplash-associated disorders (WAD). J Physiother 60:5–12, 2014.

## Minor Fractures

Most patients with spinal fractures require hospitalization. Patients with isolated cervical vertebral body compression or spinous process fractures may be managed as outpatients if there is no evidence of neurologic impairment or associated ligamentous instability, and the degree of patient distress is not severe. Appropriate follow-up should be arranged for all patients because even minor spinal injuries may be associated with prolonged disability from chronic pain.

For patients with minor wedge fractures (<10% compression of the anterior vertebral body height) who do not have an associated ileus or neurologic deficit, outpatient management may be possible. However, most wedge fractures of the thoracic and lumbar spines are usually best managed in the hospital for several reasons. First, patients with these injuries usually have marked discomfort, often requiring parenteral opioid analgesia. Second, significant force is generally required to fracture thoracic or lumbar vertebrae, and associated intrathoracic or abdominal injuries are common. Third, lower thoracic and lumbar fractures are associated with prolonged and occasionally delayed gastrointestinal ileus, requiring continuous nasogastric suction. Finally, older adults who have vertebral fractures and only minor associated injuries may require admission to facilitate assessment of fall risks and expedite rehabilitation.

## KEY CONCEPTS

- The anterior cord syndrome, characterized by paralysis and hypalgesia below the level of injury, with preservation of position, touch, and vibration, results from hyperflexion injuries causing cord contusion, by the protrusion of a bony fragment or herniated disk into the spinal canal, or by laceration or thrombosis of the anterior spinal artery. Suspicion for an anterior cord syndrome warrants prompt neurosurgical consultation because it is a potentially surgically correctable lesion.
- In the awake, evaluable trauma patient, the NEXUS or CCR decision rules may be used to determine the need for radiographic imaging.
- In the awake, evaluable trauma patient, unless the patient has a very minor trauma mechanism (or CT is not available), CT is preferred over plain radiography, especially if CT is being performed on other body parts.
- Neurogenic hypotension, caused by loss of vasomotor tone and lack of reflex tachycardia, is a diagnosis of exclusion in the trauma victim. It should not be considered the cause of hypotension unless the patient is flaccid and areflexic, reflex tachycardia and peripheral vasoconstriction are absent and, most importantly, the possibilities of coexisting hemorrhagic shock, cardiac tamponade, or tension pneumothorax have been eliminated.
- Because neurogenic hypotension can lead to hypoperfusion and secondary spinal cord ischemia, prolonged, severe hypotension (systolic blood pressure < 70 mm Hg) should be prevented and treated. When there is persistent hypotension despite fluid resuscitation, we recommend vasopressor support with norepinephrine to be started at 0.05 μg/kg/min and titrated upward to a maximum dose of 1 μg/kg/min to achieve an MAP of 85 mm Hg.
- Methylprednisolone or any other steroid is not beneficial in the treatment of acute spinal cord injury and should not be used.
- Emergency department management of SCI includes care to prevent pressure ulcers, bladder distention, and gastric distention.

*The references for this chapter can be found online by accessing the accompanying Expert Consult website.*

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## CHAPTER 36: QUESTIONS & ANSWERS

**36.1.** Which of the following is a stable cervical spine fracture?
- **A.** Anterior atlantoaxial dislocation without fracture
- **B.** Bilateral facet dislocation
- **C.** Flexion teardrop
- **D.** Jefferson fracture
- **E.** Unilateral facet dislocation

**Answer: E.** See Table 36.1 for a classification of spinal injuries according to stability.

**36.2.** A 28-year-old man is brought to the emergency department (ED) after a rollover motor vehicle collision (MVC). He is moderately hypotensive, unable to flex his elbows, and has diffuse lower extremity paralysis. What is the likely site of the lesion?
- **A.** C4
- **B.** C5
- **C.** C6
- **D.** C7
- **E.** C8

**Answer: C.** See Table 36.2.

**36.3.** Which of the following statements regarding high-dose methylprednisolone after spinal cord injury is true?
- **A.** Dexamethasone is superior to methylprednisolone if a concurrent closed head injury is present.
- **B.** High-dose methylprednisolone should not be used to treat spinal cord injury.
- **C.** It is efficacious after penetrating injury if given within 4 hours.
- **D.** It is efficacious in cases of spinal shock.
- **E.** It is more efficacious after thoracic than after lumbar injuries.

**Answer: B.** Evidence that high-dose methylprednisolone is a clinically efficacious intervention in the management of acute, blunt, partial spinal cord injury is lacking and, because of severe side effects, should not be used.

**36.4.** A 27-year-old man presents after a high-speed rollover MVC. The physical examination is remarkable for T8 motor-sensory deficit and a moderately distended abdomen that is nontender. Vital signs are heart rate, 108 beats/min, blood pressure, 88/40 mm Hg, respiratory rate, 22 breaths/min, temperature, 35°C (95°F), and oxygen ($O_2$) saturation, 96%. Which of the following tests or treatments is indicated?
- **A.** Baseline laboratory tests and observation
- **B.** Computed tomography (CT) scan of the abdomen
- **C.** Intravenous phenylephrine infusion
- **D.** Packed red blood cell transfusion
- **E.** Thoracolumbar spine films

**Answer: B.** Spinal cord injury often renders the abdominal examination unremarkable. CT, ultrasonography, diagnostic peritoneal lavage, or some combination is necessary to rule out intra-abdominal injury.

**36.5.** A 23-year-old man presents after a rollover MVC. He is brought by emergency medical services (EMS) from the scene fully restrained on a backboard, with a cervical collar. The physical examination is remarkable for moderate symmetric numbness below the neck, symmetric arm and leg weakness, intact reflexes, and diminished rectal tone. Vital signs are heart rate, 94 beats/min, blood pressure, 80/46 mm Hg, respiratory rate, 24 breaths/min, and $O_2$ saturation, 96%. Which of the following treatment sequences is most indicated?
- **A.** Crystalloid to CT scan to phenylephrine
- **B.** Crystalloid to focused assessment with sonography for trauma (FAST) examination to transfusion
- **C.** Crystalloid to phenylephrine to transfusion
- **D.** Dopamine to crystalloid to CT scan
- **E.** Transfusion to phenylephrine

**Answer: B.** Spinal shock should not be considered the cause of hypotension unless the patient is flaccid and areflexic. Crystalloid

is the first step regardless of the traumatic hypotensive cause. The possibilities of coexisting hemorrhagic shock, cardiac tamponade, tension pneumothorax, or other life-threatening injuries should first be addressed. The absence of vasomotor activity in patients with neurogenic hypotension may mask the usual presentation of these life-threatening injuries. In this case, the lack of flaccidity and presence of reflexes argues for a nonneurogenic cause for the hypotension.