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SPECIFIC CATEGORIES OF INJURIES

Part of "Chapter 19 - Vertebral and Spinal Cord Injuries"

Soft-Tissue Injuries

Whiplash

Whiplash is a lay term for an acceleration injury involving hyperextension of the head during a rear-end vehicular collision. The ligaments and muscles of the neck sustain stress and strain injury. The usual signs and symptoms, which include stiff neck, pain in the neck and shoulder, limitation of movement, and muscle spasms, may not begin until 12 to 24 hours after injury. Other signs and symptoms may include headache, paresthesia, dizziness, vertigo, and tinnitus. The findings on physical examination are normal except for the previously listed signs and symptoms. The radiological examination is negative. The diagnosis is based on the history of injury and the presence of the characteristic signs and symptoms. This is a common injury causing much pain and suffering to the patient, even though no abnormalities are noted on radiographical examination.

The pain caused by whiplash is thought to be attributable to the tearing, stretching, microhemorrhage, and edema incurred by the anterior neck muscles (sternocleidomastoid, scalenus, and longus colli muscles). The muscles, and possibly the ligaments, are strained. Patients with preexisting cervical spondylosis and some with other conditions may have narrowing of the foramina and osteophytes and also increased rigidity of the spinal column. These conditions put them at greater risk of developing neurological problems if a whiplash injury occurs.

Treatment.

Treatment of whiplash is directed toward making the patient more comfortable. For less serious injuries treatment involves mild analgesics (e.g., nonsteroidal anti-inflammatory

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drugs [NSAIDs], acetaminophen), local ice, and rest. More severe injuries are treated with short-term use of a soft collar, heat and cold application, analgesics, muscle relaxants, and anti-inflammatory agents.

Narcotic analgesics are reserved for severe pain and should be used sparingly to prevent dependency. NSAIDs are commonly ordered both for their anti-inflammatory effect and for their ability to inhibit prostaglandin synthesis, a substance known to be related to pain. Muscle relaxants such as cyclobenzaprine hydrochloride (Flexeril) and methocarbamol (Robaxin) are also used. Extended use of a cervical collar is controversial. Some physicians believe that collars hinder recovery of involved muscles if worn for more than a

few days.

Other Soft-Tissue Injuries

The vertebral column depends on soft tissue for its stability. Therefore, any significant soft-tissue trauma (see Fig. 19-2) that occurs with vertebral injury can compromise the vertebral column. The other soft-tissue injuries with significance for vertebral stability are discussed in this chapter in conjunction with vertebral injuries and SCI.

Vertebral Injuries

Classification

Although fractures can occur singularly in any part of the vertebral arch, most injuries occur in combination with vertebral body injuries. The “ends” of the vertebral column, the cervical and lumbar portions, have the greatest built-in mobility, which predisposes them to injury. The thoracic region is less prone to trauma because of the rigidity imparted to it by the rib cage.

Vertebral injuries can be classified based on various perspectives of the injury:

- **Types of fractures:** simple or compression fractures
- **Fracture or dislocation:** pure fracture, pure dislocation, and fracture-dislocation
- **Stability or instability of injury:** according to a three-column framework
- **Segmental involvement:** upper cervical, subaxial cervical, thoracic, or lumbar and sacral

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Each category offers a special practical perspective and is discussed in the following section.

Type of Fractures.

For the purposes of this discussion, vertebral fractures are subdivided into simple and compression fractures (see Fig. 19-10 for a review of anatomical parts of a vertebra).



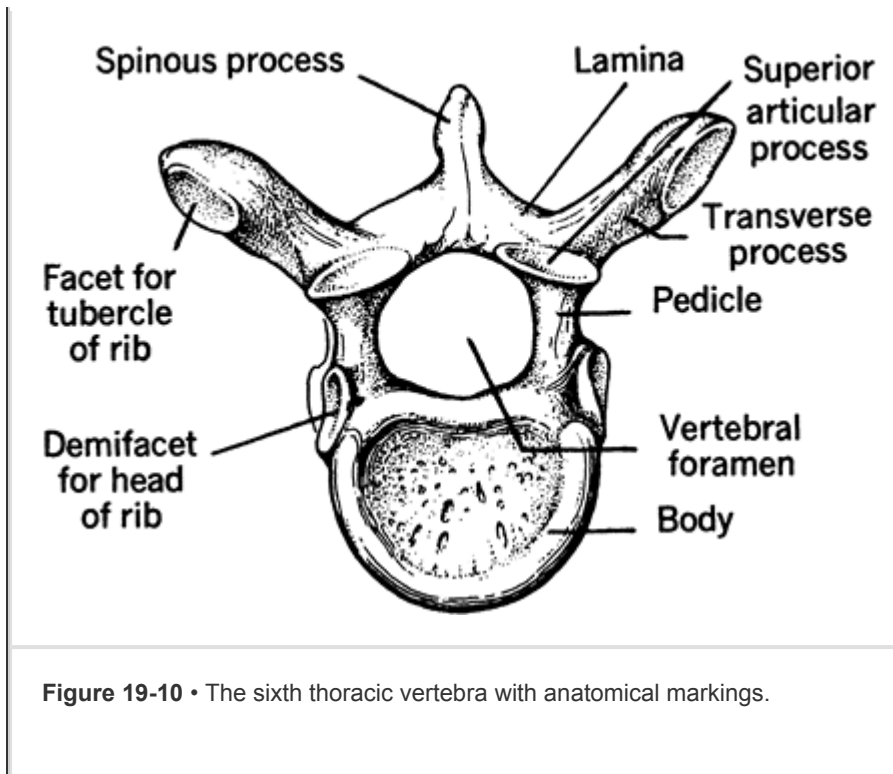


Figure 19-10 • The sixth thoracic vertebra with anatomical markings.

- **Simple fractures** appear as a singular break with the alignment of the vertebral parts remaining intact. These types of fractures usually occur to the spinous or transverse process, facets, pedicles, and vertebral body. There is usually no neural compression.
- **Compression fractures** are sometimes further subclassified as *simple wedge fractures*, *burst fractures*, and *teardrop fractures* (see Fig. 19-8). They are caused by axial loading and hyperflexion.
- **A simple (wedge) compression fracture** is caused by vertical compression when the cervical vertebral column is flexed. A burst fracture is caused by the same mechanical force, but the vertebral column is straight. Because the posterior ligaments are intact, the simple (wedge) compression fracture is stable. No surgery is required. These fractures heal well with hard collar immobilization for about 2 months.³ A halo jacket may also be used.
- **Burst fractures** are explosive fractures caused by severe axial loading on a straight cervical column. They shatter the vertebral body into several pieces. These fragments then can be driven into the spinal cord, resulting in serious injury. If there is no neurological damage and if the posterior ligaments are stable, wearing a hard collar for 2 months may be adequate therapy (Fig. 19-11). However, burst fractures often require a combined neurosurgical-orthopedic procedure for the removal of bone fragments, cord decompression, and vertebral column stabilization. Stabilization is accomplished by insertion of instrumentation, such as CD rods, a type of segmental rodding that can be accommodated to the individual's level of injury. See section on

surgical management for further discussion.

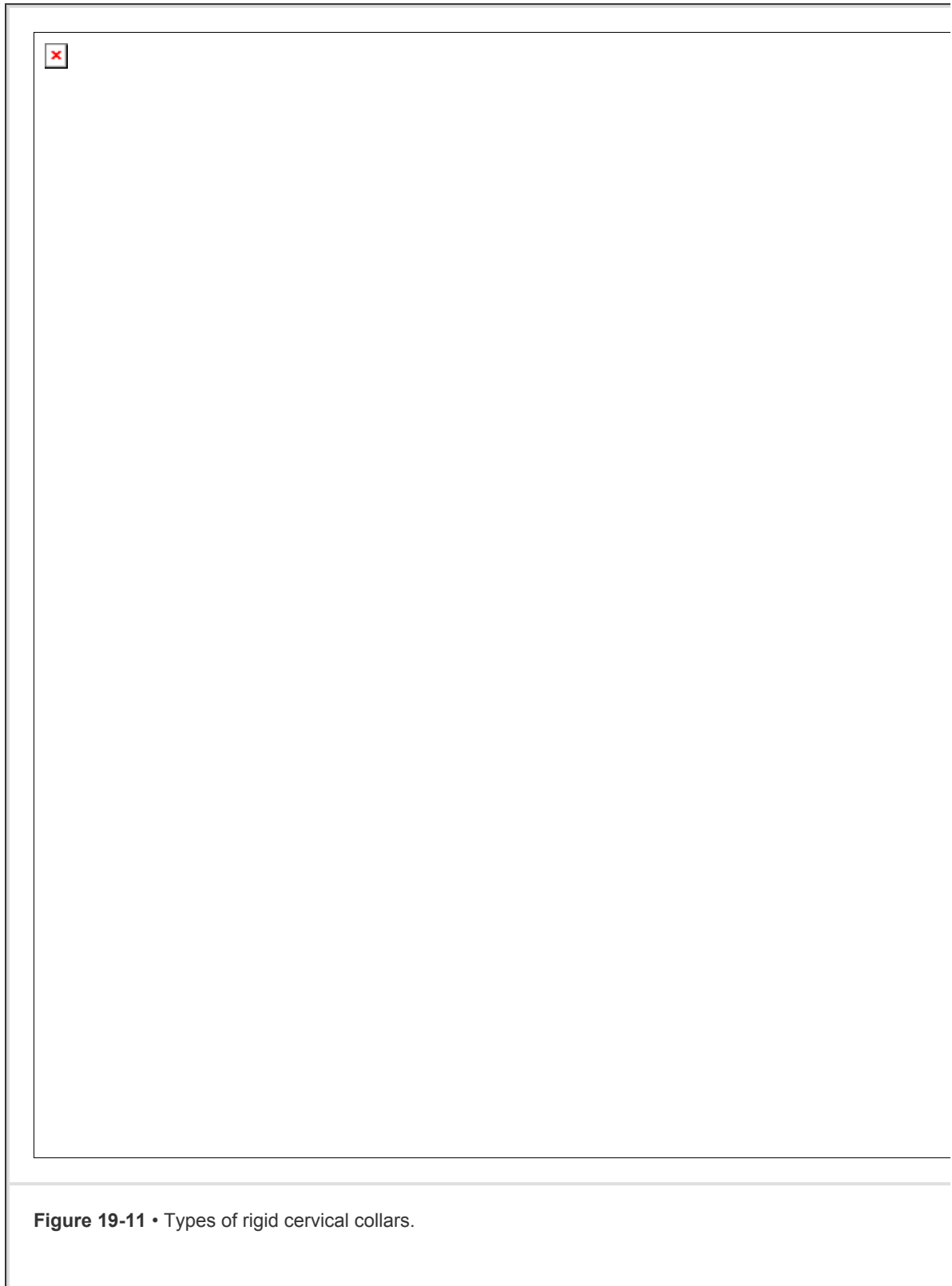


Figure 19-11 • Types of rigid cervical collars.

- **Teardrop fractures** are caused by extreme flexion with axial loading. With this

fracture a vertebral body is crushed by the vertebral body superior to it, causing the anterior portion of the compressed body to break away. These fractures, which are usually unstable (i.e., involving disruption of the posterior ligaments, resulting in forward dislocation), are managed with anterior decompression and fusion with halo immobilization.

Fracture or Dislocation.

From another perspective, a vertebral injury can be a fracture without a dislocation, a dislocation without a fracture or a fracture combined with a dislocation. **Dislocation** occurs when one vertebra overrides another, and there is unilateral or bilateral facet dislocation. Radiographic studies reveal a disruption in the established alignment of the vertebral column. Usually, the supporting ligaments are also injured, and the spinal cord may or may not be involved.

Subluxation is a partial or incomplete dislocation of one vertebra over another. Damage to the cord and supporting ligaments may or may not be present. With dislocation, reestablishment of alignment is necessary. This may be accomplished by traction followed by immobilization or by surgical stabilization (fusion or sometimes insertion of CD rods if the posterior ligaments are injured).

Fracture-dislocation, as the name implies, denotes a combined injury of a fracture and a dislocation that is usually accompanied by ligament and cord injury. As with simple dislocation, realignment is necessary. The fracture must be allowed to heal, and any bone fragments impinging on the cord must be removed. Therefore, surgery is indicated.

Stability or Instability of Injury.

From this perspective, it is critical when considering vertebral fractures to distinguish between stable and unstable fractures and dislocations. This distinction is often based on the posterior ligaments. If the posterior ligaments are intact, the injury is considered stable. If they have been torn, usually by a rotational force, they are considered unstable. Stability of the vertebral-spinal elements is also considered using a three-column theoretical framework. This approach is discussed in Chart 19-1. A stable fracture or dislocation is not apt to displace more than it was at the time of the injury, whereas an unstable fracture or dislocation is highly likely to displace further with extension of injury to the spinal cord. External immobilization or internal fixation may be unnecessary for stable injuries, whereas it is essential for unstable injuries. In addition, when ligaments heal, scar tissue forms. The scarred tissue, being weaker than the preinjury tissue, may result in chronic instability and lead to SCI.

CHART 19–1 • The Three-Column Framework: Spinal Stability and Instability*

Spinal stability refers to the ability of the vertebral support column to protect adequately the neural elements from injury during inactivity and activity. This determination is critical in managing patients because an unstable injury can result in extension of or new neurological deficits. Currently, criteria for determining spinal

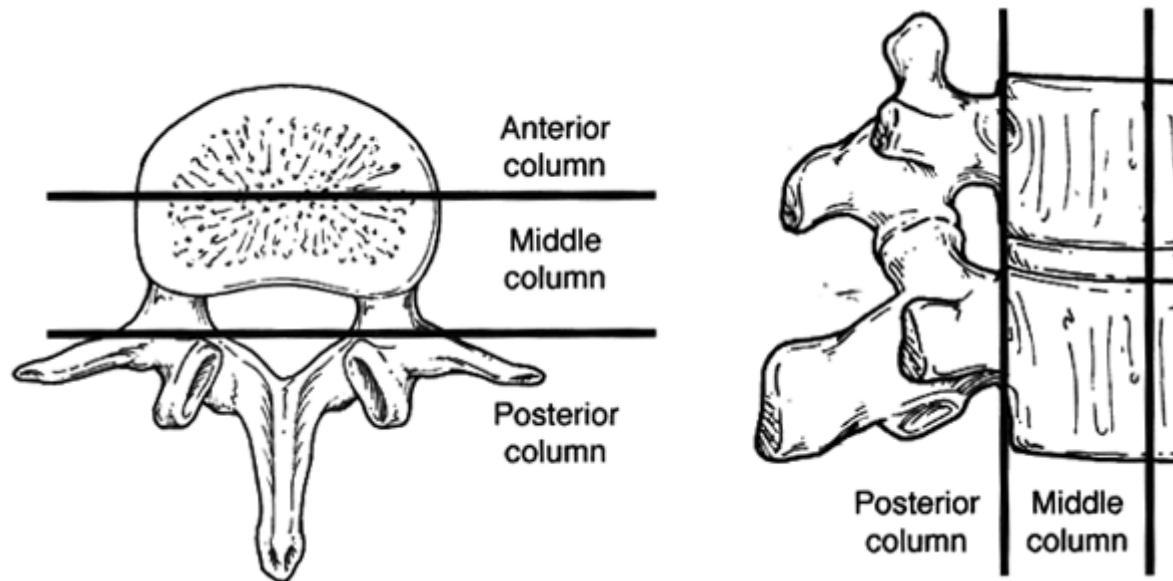
stability or instability are controversial.

Some physicians consider only the condition of the posterior ligaments when determining stability. Another approach is the three-column framework. The three-column approach provides an anatomical framework for considering stability. The cross-section of the spine is organized into three anatomical columns:

- The **anterior column** consists of the anterior longitudinal ligament, anterior half of the vertebral body, annulus fibrosis, and disc.
- The **middle column** consists of the posterior half of the vertebral body, annulus, disc, and posterior longitudinal ligament.
- The **posterior column** consists of the facet joints, ligamentum flavum, posterior elements, and interconnecting ligaments.

Applying this classification system to spinal injuries results in four classification categories, which are determined by the specific column(s) injured.

Type of Injury	Columns Injured		
	Anterior	Middle	Posterior
Compression fractures	Yes	No	No
Burst fractures	Yes	Yes	No
Flexion-distraction fractures	Yes/no	Yes	Yes
Fracture-dislocations	Yes	Yes	Yes



*The rule of thumb is that when one column is injured, the spine is usually stable; when two or three columns are the sustained injury is considered unstable.

Footnote

Lenke, L. G., O'Brien, M. F., & Bridwell, K. H. [1995]. Fractures and dislocations of the spine. In C. R. Perry, J. A. Elstrom, & A. M. Pankovich (Eds.). *The handbook of fractures* [pp. 157–189]. New York: McGraw-Hill

Vertebral Injuries According to Segmental Level

Vertebral injuries can be divided into four groups based on the involved segmental level: upper cervical, subaxial cervical, thoracic and lumbar, and sacral.⁴

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Upper Cervical Segment

The four most commonly encountered upper cervical segment injuries are atlas fractures, atlantoaxial subluxation, odontoid fractures, and so-called hangman's fractures. Four less common injuries are occipital condyle fractures, atlantooccipital dislocation, atlantoaxial rotary subluxation, and C-2 lateral mass fractures. A summary of upper cervical fractures and lower cervical fractures is presented in Table 19-1.

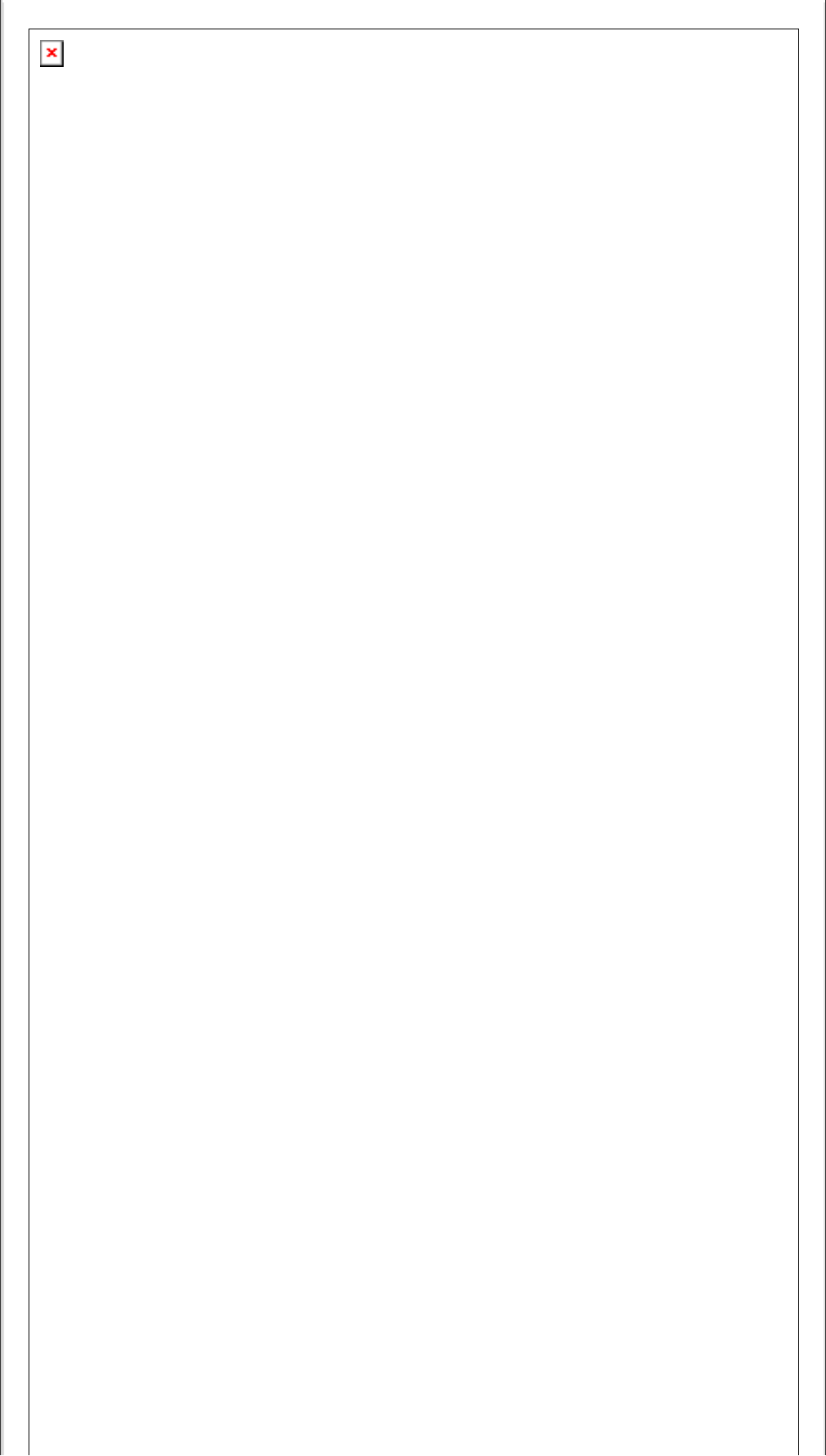


Table 19-1 • CERVICAL VERTEBRAL INJURIES

Subaxial Cervical Segments

Compared with the upper cervical segment, there is an increased risk of cervical cord damage in injuries to the lower cervical vertebrae (subaxial, i.e., below C-2). Two factors account for this: the size of the spinal canal is decreased in the lower spine, and there is an increased prevalence of injuries that narrow rather than expand the canal. Five types of subaxial cervical vertebrae injuries are discussed:

- **Isolated posterior element fractures** of the lamina, articular process, or spinous process occur when there is impact of the posterior elements on one another and compression-extension results.
- **Minor avulsion and compression fractures** of the subaxial cervical vertebrae include anterior compression or avulsion injuries of the vertebral body. Additionally, anterior and posterior concurrent bone injuries with minimal displacement and angulation can occur.
- **Vertebral body-burst fractures**, common in diving accidents, result from axial loading and flexion. The anterior and middle columns are involved, creating instability. Bone may protrude into the spinal canal.
- **With teardrop fractures** flexion and axial loading result in a teardrop fragment on the anteroinferior aspect of the affected body. SCI and three-column instability are usual.
- **Facet injuries causing spinal malalignment** result from a variety of biomechanical forces. Reduction may have to be done in stages to prevent additional injury. If traction is not possible, surgery is necessary. Traumatic disc herniation may also be present and usually requires an anterior discectomy and fusion.

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The treatment options for subaxial cervical vertebral injuries include immobilization with sternal-occipital-mandibular orthotic device; halo vest; posterior fusion and stabilization with wires or instrumentation; anterior approaches for decompression; fusion with or without instrumentation; or a combination of these therapies. Treatment choices depend on the specifics of the fracture or malalignment and on the stability of the ligaments.

Thoracic and Lumbar Segments

Vertebral injuries in the thoracic and lumbar regions account for paralysis of the trunk and lower extremities. Compared with the lumbar region, there is normally little movement

possible in the vertebrae of the thoracic region because of the inherent structural stability provided by the rib cage. The spinal cord ends at the upper border of the first lumbar vertebra. The cord gradually tapers, beginning at the lower two thoracic vertebrae. As the cord tapers, it forms a cone called the *conus medullaris*, which continues at the filum terminale. The nerve roots coming off the lower segments of the spinal cord, termed the *cauda equina*, hang loosely and are susceptible to injury. However, injury to these nerves is less likely to be permanent than is injury to the spinal cord. In addition, the emergency procedures of decompression are less likely to be required because the nerve roots tolerate trauma far better than does the spinal cord itself.

There are four general categories of vertebral fractures of the thoracic, thoracolumbar, and lumbar spine:

- **Compression fractures**, caused by axial loading and hyperflexion, are common in the thoracic and upper lumbar regions of the vertebral column. Significant direct force must be applied to produce a fracture in the thoracic area. Injury is usually the result of a direct force applied to one vertebra, which is hyperflexed. The force is then transferred to the underlying cord, putting it at risk of injury. A compression fracture in the thoracic or lumbar region may be compressed anteriorly with or without subluxation of the vertebra. The other possibility is total compression of the vertebral body with anteroposterior protrusion.
- **Burst fractures**, which include injury to the anterior and middle columns and possibly the posterior column, are unstable. Axial loading with flexion creates the biomechanical force responsible for the injury. The vertebral body explodes or “bursts” as a result of the energy associated with injury, and often the vertebral body protrudes into the spinal canal.
- **Flexion-distraction injuries** (also called **Chance fractures**) involve three columns and are thus unstable. The fracture extends through the posterior elements, pedicle, and vertebral body. The mechanism of injury is acute flexion of the torso while restrained with only a lap belt. The flexion-distraction injuries (Chance fractures; Fig. 19-12) are classified according to the involvement of bone and soft tissue components.

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Figure 19-12 • Types of flexion-distraction (Chance) fracture. (A) Disruption through the entire bony element (B) Disruption through the entire ligamentous elements. (C and D) Disruption through bony and ligamentous elements.

- **Fracture-dislocations** of the thoracic and lumbar areas are of three general categories: anterior or posterior dislocation of the whole vertebral body with fracture of the bony parts; comminuted fractures of the vertebral body with anterior or posterior displacement and rotation so that the rotational force usually tears the supporting ligaments; and lateral dislocation of the vertebra with fracture. All three

columns are involved so that the fracture is unstable, placing the patient at high risk for neurological injury.

Sacral Segments

Fractures of the sacrum and coccyx usually result from direct trauma, most frequently caused by falls. Any fall in the sitting position, such as falling on ice or being thrown from a horse and landing on the buttocks, can result in such a fracture. Nerve injury in this region can cause bladder, bowel, or sexual dysfunction and saddle anesthesia.

Lesions of the *conus medullaris* can occur with fractures in the lumbar region. These lesions can have confusing clinical presentations. Injury to the conus usually results in lower motor neuron symptoms (muscle flaccidity, muscle atrophy, hyporeflexia) because of the disruption of the anterior gray horn cells. A decompression laminectomy may be necessary if there is pressure on the neural elements. Lesions involving the cauda equina produce selected root syndromes. A decompression laminectomy may also be necessary.

Spinal Cord Injuries

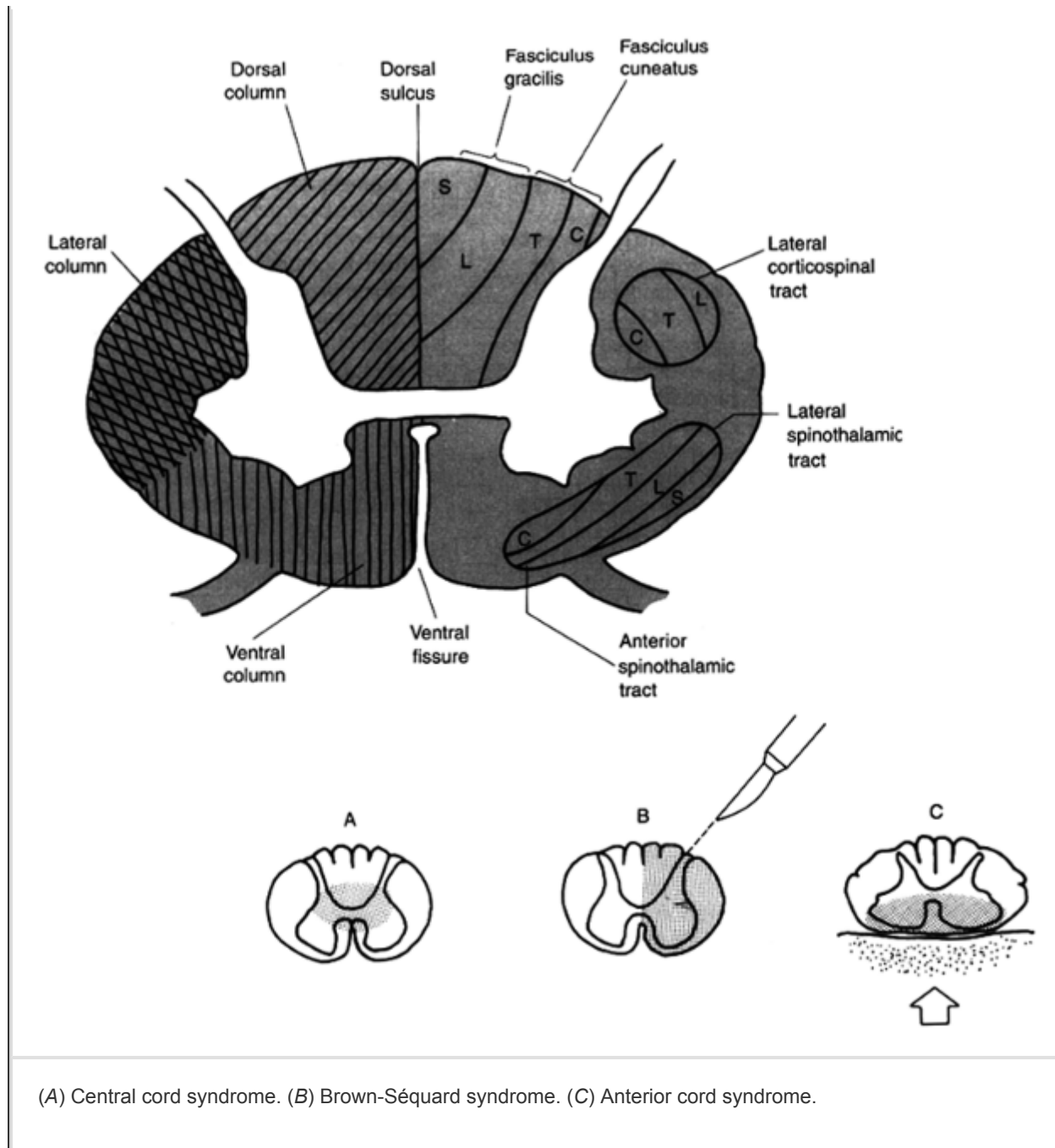
Injury to the spinal cord can be devastating because the resulting loss of body function also involves the loss of independence. The loss of function may be permanent or temporary, depending on the type of injury. The several syndromes related to SCI are summarized in Chart 19-2.

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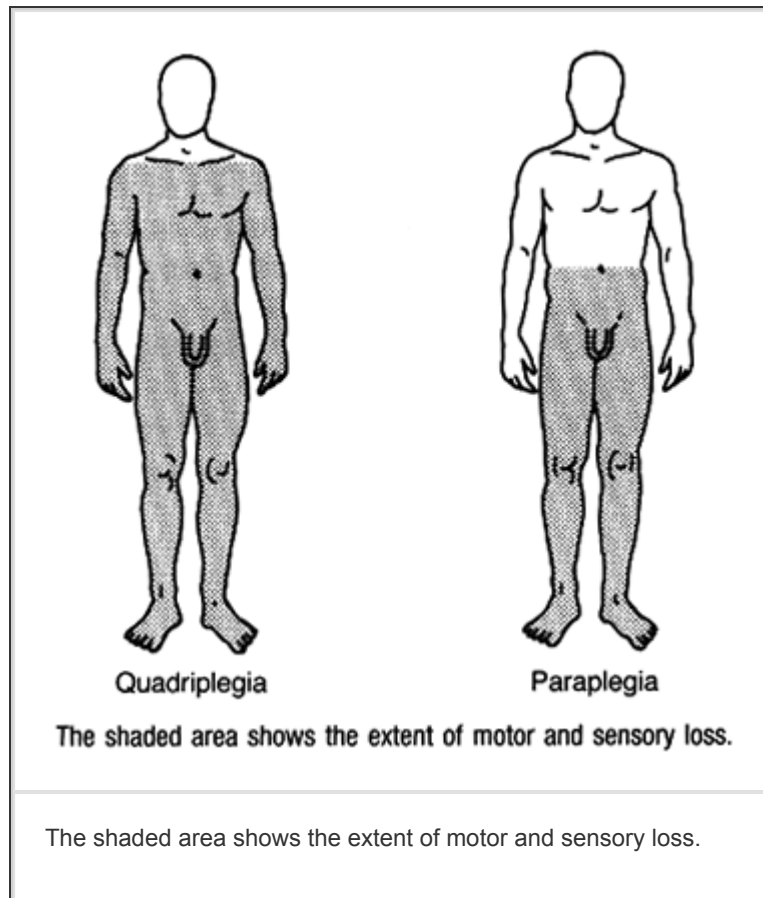
CHART 19-2 • Selected Syndromes Related to Spinal Cord Injury



Several terms related to spinal cord injury are defined.

- Quadriplegia refers to a lesion involving one of the cervical segments of the spinal cord that results in dysfunction of both arms, both legs, bowel, and bladder.
- Paraplegia refers to a lesion involving the thoracic lumbar or sacral regions of the spinal cord that results in dysfunction of the lower extremities, bowel, or bladder.

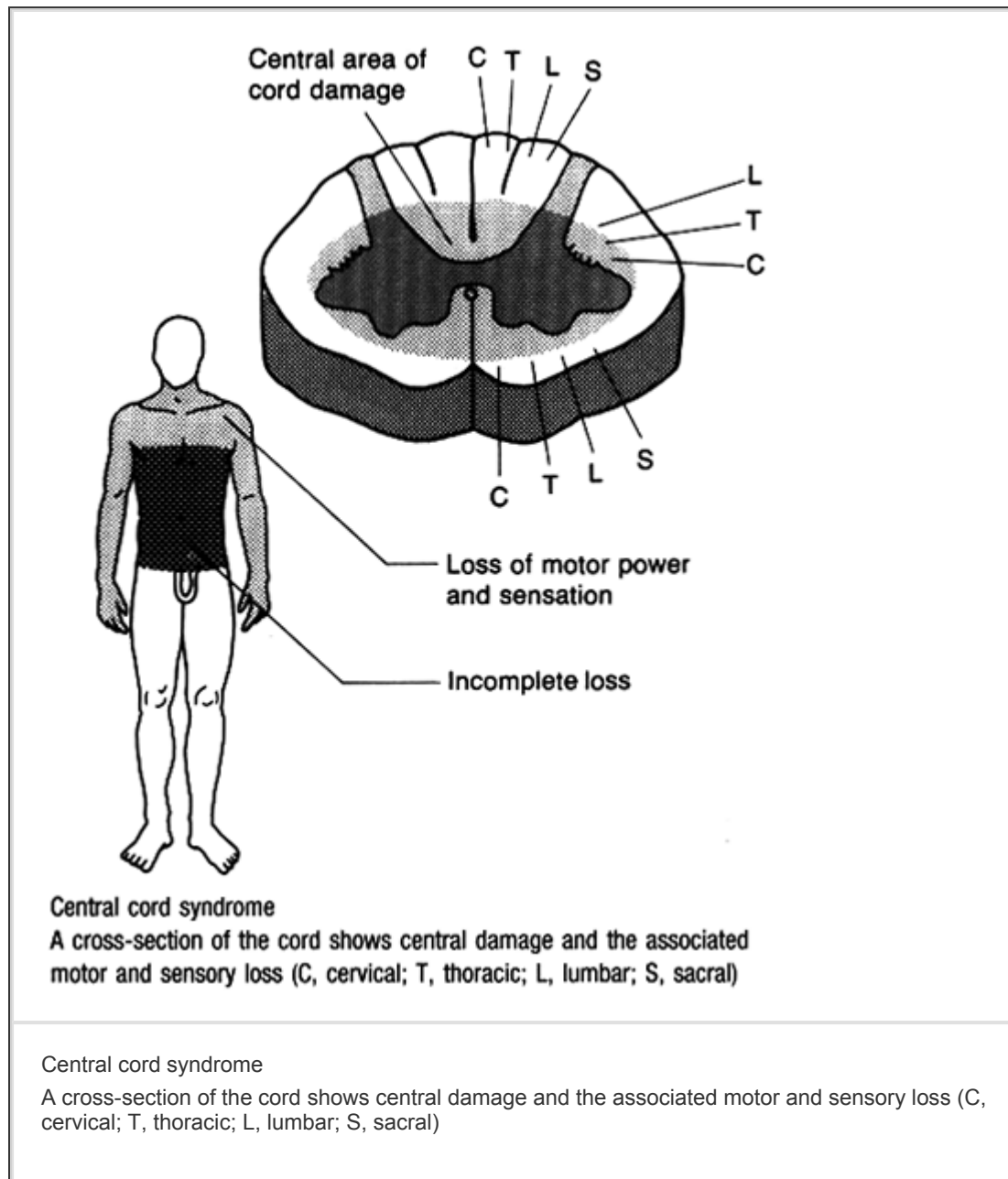
- A complete lesion (e.g., complete quadriplegia or complete paraplegia) implies total loss of sensation and voluntary muscle control below the injury.
- An incomplete lesion implies preservation of the sensory or motor fibers, or both, below the lesion. Incomplete lesions are classified according to the area of damage: central, lateral, anterior, or peripheral.



CENTRAL CORD SYNDROME

- Presentation: There are more motor deficits in the upper extremities than the lower extremities; sensory loss varies but is more pronounced in the upper extremities; bowel/bladder dysfunction is variable, or function may be completely preserved.
- Cause: Injury or edema of the central cord, usually of the cervical area, is the underlying cause; hyperextension injuries, particularly if bony spurs are noted, can be causative.
- Result: Edema in the central cord exerts pressure on the anterior horn cells. The cervical fibers of the corticospinal tract are located in a more central position in the cord than the sacral fibers, which are located in the periphery. As a result, motor deficits are less severe in the lower extremities than in the upper extremities.

- Treatment: High-dose steroid (methylprednisolone) protocol for acute cord injury (see pp. 426–427); immobilization or bed rest is the treatment of choice. Flexion-extension radiographs are usually obtained. The prognosis varies.

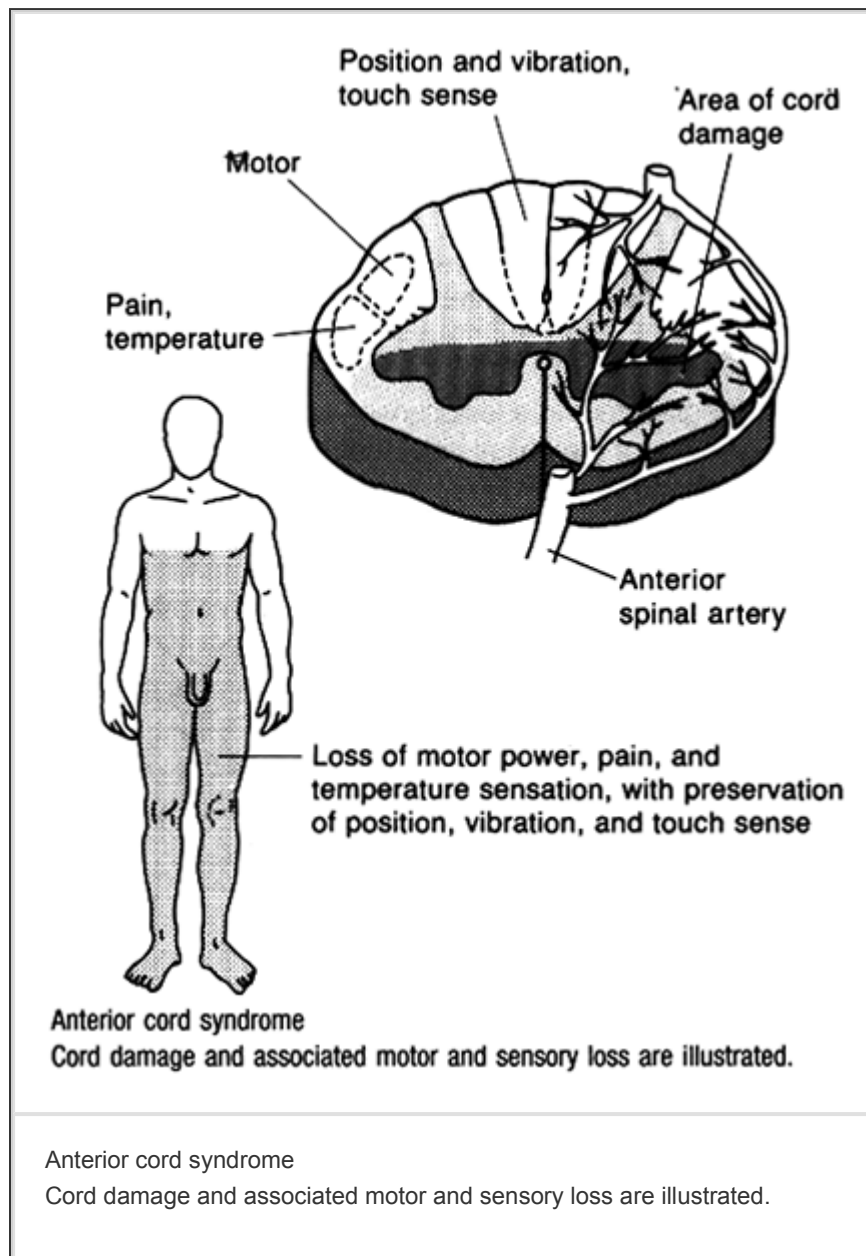


ANTERIOR CORD SYNDROME

- Presentation: Loss of perception pain and temperature, and motor function is noted below the level of the lesion; light touch, position, and vibration sensation remain intact.
- Cause: The syndrome may be caused by acute disc herniation or hyperflexion

injuries associated with fracture-dislocation of a vertebra. It also may occur as a result of injury to the anterior spinal artery, which supplies the anterior two thirds of the spinal cord.

- Result: Injury to the anterior part of the spinal cord, which includes the spinothalamic tracts (pain), corticospinal tracts (temperature), and anterior gray horn motor neurons, is noted.
- Treatment: High-dose steroid (methylprednisolone) protocol for acute cord injury (see pp. 426–427); surgical decompression is usually necessary to manage fracture-dislocation. The prognosis varies.



BROWN-SÉQUARD SYNDROME (LATERAL CORD SYNDROME)

- Presentation: Ipsilateral paralysis or paresis is noted, together with ipsilateral loss of touch, pressure, and vibration and contralateral loss of pain and temperature.
- Cause: Transverse hemisection of the cord (half the cord is transected from north to south), usually as a result of a knife or missile injury, fracture-dislocation of a unilateral articular process, or possibly an acute ruptured disc.
- Result: With right-sided cord transection, for example, the following would occur: paralysis of all voluntary muscles below the level of injury on the right side of the body (lateral corticospinal tract); loss of perception of touch, vibration, and position on the right side of the body below the level of injury (posterior columns, which include the fasciculus gracilis and fasciculus cuneatus); and loss of pain and temperature perception on the left side of the body below the injury (lateral spinothalamic tracts). Fibers that carry pain and temperature cross to the opposite side of the cord immediately after entering the cord and then ascend. The other tracts mentioned do not cross until they reach the brain stem.
- Treatment: High-dose steroid (methylprednisolone) protocol for acute cord injury (see pp. 426–427); no specific treatment is undertaken except fracture-dislocation management.

POSTERIOR CORD SYNDROME

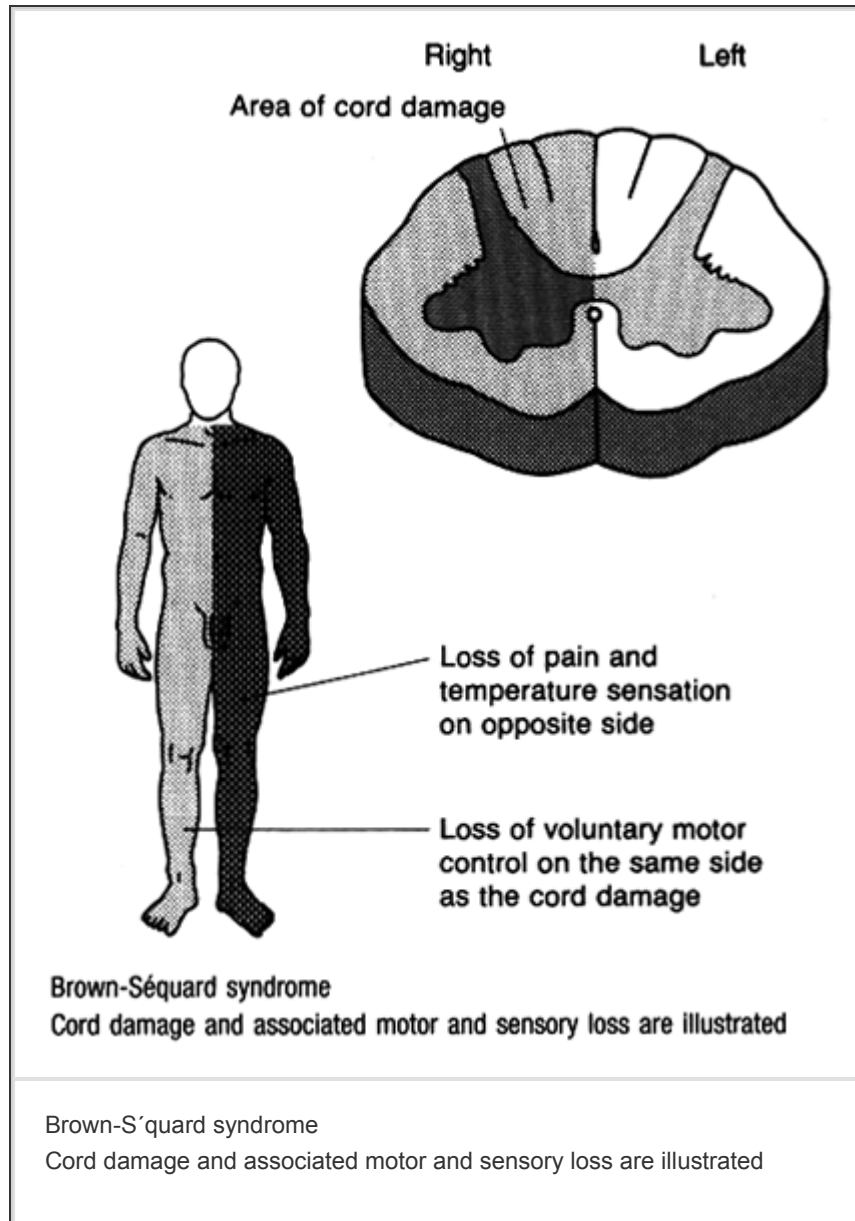
- This is a rare syndrome in which the position and vibration senses of the posterior columns are involved.

ROOT SYNDROMES (PERIPHERAL SYNDROMES)

- Presentation: Root syndromes cause tingling, pain, motor weakness of an isolated muscle or muscle group, and absent or diminished reflexes in the involved area. The spinal cord terminates at T-12 or L-1. The nerve roots that extend from the conus medullaris are collectively called the *cauda equina*. Lesions of L-1 through L-5 denote paraplegia. Patients with this level of involvement can move readily and walk with the assistance of various types of bracing. Lesions of the lumbrosacral region may involve multiple roots of the cauda equina with a varying pattern of motor and sensory loss. Deep tendon reflexes are usually diminished or absent. Isolated nerve root involvement is common in the lumbrosacral region so that saddle hypalgesia—diminished or absence of sensation in the saddle region—is possible. Therefore, careful examination of the patient is necessary to identify the erratic sensory or motor loss. When the sacral roots are involved, the patient may experience bladder or bowel dysfunction. If the cervical region is involved, there is usually tingling in the arm, muscle weakness in the arm or shoulder, and pain radiating down the arm and into the shoulder.
- Cause: Compression secondary to intervertebral disc herniation or vertebral subluxation is the cause of these syndromes. Any area of the cord can be

involved.

- Result: Compression of one or more nerve roots coming off the spinal cord, rather than compression or injury to the cord itself, occurs. The compression can also cause edema.
- Treatment: Nonsurgical treatment with traction is initiated to release the compressed nerve roots, and drugs may be prescribed to control the associated edema, pain, and muscle spasms. In some cases, surgery for decompression of the nerve roots may be necessary.



HORNER'S SYNDROME

- Presentation: Horner's syndrome, which may be seen with partial spinal cord

transection at the level of T-1 or above, includes miosis, ptosis, and loss of sweating on the ipsilateral side.

- **Cause:** The syndrome is caused by a lesion involving either the preganglionic sympathetic trunk or the cervical postganglionic sympathetic neurons.

Footnote

Cross-sections of spinal cord in this chart are from Kitt, S. & Kaiser, J. [1990]. *Emergency nursing: A physiological and clinical perspective*. Philadelphia: W. B. Saunders.

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Injuries to the spinal cord can be classified by type of injury and by syndrome produced.

Classification by Cause

The spinal cord can be injured by concussion, contusion, compression, laceration, transection, hemorrhage, damage to the blood vessels that supply the cord, or damage to blood vessels within the cord.

- **Concussion:** A jarring, that is, severe shaking, of the spinal cord can result in a temporary loss of function (i.e. spinal shock [see subsequent discussion]) lasting hours to weeks. No identifiable macro-neuropathological changes are noted on examination of the cord.
- **Compression:** As part of the distraction, (i.e., distortion of the normal curvatures), of the spinal cord at the moment of injury, the neural element can be compressed and contused, lacerated, or transected.
- **Contusion:** Contusion is bruising, which can lead to bleeding into the cord, subsequent edema, and possible necrosis from the compression caused by edema or direct damage to the tissue. The extent of the neurological deficits depends on the severity of the contusion and the amount of necrosis, if any. Fractures, dislocations, and direct trauma to the cord can cause a contusion.
- **Laceration:** An actual tear in the cord results in permanent injury to the cord. Contusion, edema, and cord compression accompany a laceration.
- **Transection:** A severing of the cord can be complete or incomplete. Actual complete transection is rare. However, clinical presentations, which mimic complete transections, are frequently seen.
- **Hemorrhage:** Blood in or around the spinal cord is an irritant to the delicate tissue. Changes in the neurochemical environment, edema, and neurological deficits can result.
- **Damage to the blood vessels that supply the cord:** Interference with or damage to

the vessels that supply the spinal cord, the anterior spinal artery, or the two posterior spinal arteries results in ischemia and possible necrosis. Episodes of ischemia can cause temporary neurological deficits. Prolonged ischemia and necrosis causes permanent deficits.

Pathophysiology

Spinal cord injury results from both the **primary injury**, which occurs at the time of the impact, and **secondary injury**, which occurs in the first few hours after primary injury. The primary injury is the result of compression, laceration, or disruption of the spinal cord or surrounding vascular components. These disruptions of neural elements and/or injury to the vascular supply within the spinal cord or supplying the spinal cord cause *ischemic injury*. The degree of injury depends on the magnitude of the force applied to the spinal cord and the angle of impact.

Several chemical and vascular changes occur following primary injury, which cause the spinal cord to initiate an intrinsic process of additional injury and self-destruction. These processes are collectively called **secondary injury**. Within minutes of injury, a cascade of concurrent events (including alterations in blood flow, edema, hemorrhage, electrolyte abnormalities, membrane injury, and release of cytotoxic mediators and excitotoxic neurotransmitters) occurs and contributes to cellular membrane injury and posttraumatic ischemia.⁵ The secondary injury results from vascular and neuronal pathological changes and the release of vasoactive agents and cellular enzymes. The shift in intracellular calcium activates calcium-dependent proteases and impairs mitochondrial and other intracellular functions. This injury to the blood vessels results in ischemia, increased vascular permeability, and edema. Hypoxia of the gray matter stimulates the release of catecholamines, which contribute to the hemorrhage and necrosis and cause further spinal cord dysfunction. The release of catecholamines and vasoactive substances (norepinephrine, serotonin, dopamine, and histamine) from the injured tissue can cause vasospasm and impede microcirculation. These events further extend necrosis of blood vessels and neurons. The release of proteolytic and lipolytic enzymes from the injured cells causes delayed swelling and necrosis in the spinal cord.⁶ The function of highly specialized central nervous system cells is disrupted by ischemia and hypoxia within 30 minutes of injury. Irreversible nerve damage develops as a result of the replacement of normal neural elements with glial and fibrotic scar tissue. As a result, neurological deficits become permanent.