Tintinalli, Judith E., Kelen, Gabor D., Stapczynski, J. Stephan
Emergency Medicine: A Comprehensive Study Guide, 6th Edition

256

Spinal Cord Injuries

Bonny J. Baron

Thomas M. Scalea

EPIDEMIOLOGY

Spinal injuries are probably the most devastating of all trauma-related injuries. The incidence of traumatic spinal cord injury (SCI) in the United States is estimated to be 30 cases per million population at risk. Thus, 8000 to 10,000 new cases can be expected annually. The actual incidence is probably higher. Minor injuries are often not reported, and those associated with trauma fatalities may go unnoticed. SCI is predominantly a disease of young men. The most comprehensive data available indicates the mean age has been reported as 33.5 years,1 with a male-to-female predominance of 4 to 1. Spinal injury occurs more frequently on weekends and holidays and during summer months. The majority (90 percent) of cases are caused by blunt trauma with most of these from motor vehicle crashes.

P.1570

FUNCTIONAL ANATOMY

The vertebral column serves as the central supporting structure for the head and trunk and provides bony protection for the spinal cord. It consists of 33 vertebrae: 7 cervical, 12 thoracic, 5 lumbar, 5 sacral (fused to form the sacrum), and 4 coccygeal, which are usually fused.

Vertebrae

In accordance with their weight-bearing function, the vertebrae become larger toward the lower end of the column, but all are built on the same fundamental plan. A typical vertebra is composed of a body anteriorly and a vertebral arch posteriorly (Figure 256-1). Between the body and arch is the vertebral foramen through which the spinal cord runs. The vertebral arch is made up of two pedicles, two laminae, and seven processes (one spinous, two transverse, and four articular). The spine has the potential to move in flexion, extension, lateral flexion, rotation, or circumduction (combination of all movements). The articular processes form synovial joints that act as pivots of the spinal column. The orientation of these articular facet joints changes at different levels of the spine. Differences in orientation of the facet joints account for variations in motion of specific regions of the vertebral column. A series of ligaments serve to maintain the alignment of the spinal column. The anterior and posterior longitudinal ligaments run along the vertebral

bodies. Surrounding the vertebral arch are the ligamentum flavum and the supraspinous, interspinous, intertransverse, and capsular ligaments. The intervertebral disks lie between adjacent vertebral bodies. Each disk consists of a peripheral annulus fibrosus and a central nucleus pulposus. The annulus fibrosus is composed of fibrocartilage. The nucleus pulposus is a semifluid, gelatinous structure made up of water and cartilage fibers. With advancing age, the proportion of water decreases and fibrocartilage increases. The disks act as shock absorbers to distribute axial load. When compressive forces exceed the absorptive capacity of the disk, the annulus fibrosus ruptures, allowing the nucleus pulposus to protrude into the vertebral canal. This may result in spinal nerve or spinal cord compression.

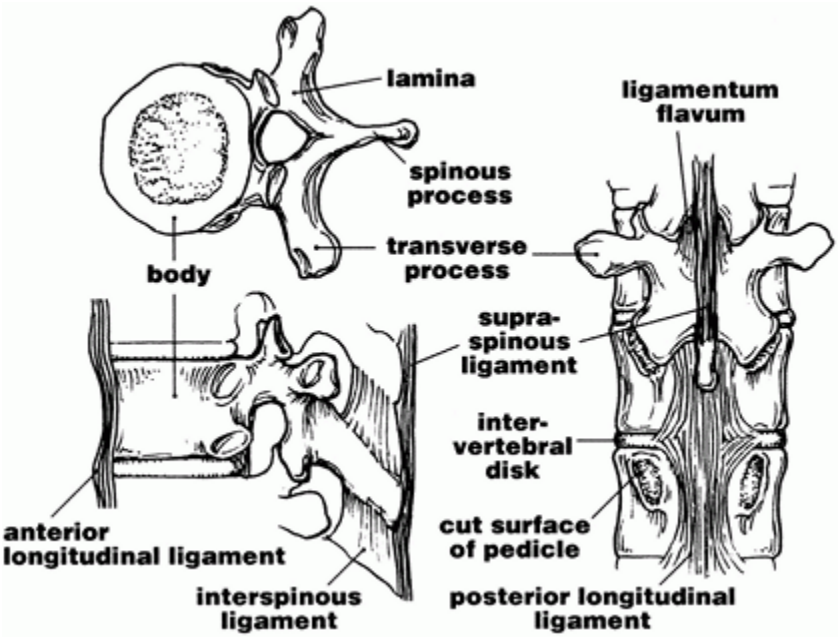


FIG. 256-1. Vertebra. Each vertebra consists of a vertebral body and posterior element. Vertebrae are stabilized by an anterior longitudinal ligament, posterior ligament, and interspinous ligament.

Spinal Stability

The determination of spinal stability is an important factor in the evaluation of the injured spine. White and Panjabi define stability as the ability of the spine to limit patterns of displacement under physiologic loads so as not to damage or irritate the spinal cord or nerve roots and to prevent incapacitating deformity or pain because of structural changes.2 Computed tomography (CT) evaluation applied to the Denis three-column system for classification of thoracolumbar injuries can be used to assess spinal stability.3 The anterior column is formed by the anterior part of the vertebral body, the anterior annulus fibrosus, and the anterior longitudinal ligament. The middle column is formed by the posterior wall of the vertebral body, the posterior annulus fibrosus, and the posterior longitudinal ligament. The posterior column includes the bony complex of the posterior vertebral arch and the

posterior ligamentous complex. For an injury to be unstable, there must be disruption of at least two columns. In evaluating stability, it is also important to include the degree of compression of the vertebral body. Vertebral body compressions of more than 50 percent are generally considered unstable.

Thoracic and Lumbar Spine

The thoracic spine is a rigid segment. The additional support provided by its articulation with the rib cage imparts a stiffness 2.5 times that of the ligamentous spine alone. Relative to other regions of the vertebral column, a large force is necessary to overcome the intrinsic stability of the thoracic spine. While injury to the thoracic spine is less common than in other regions, when it does occur it is usually significant. The spinal canal is narrower than that found in either the cervical or lumbar spine. The large spinal cord diameter relative to canal diameter increases the risk of cord injury. When cord injuries occur, most are neurologically complete (see discussion later). Of additional importance is the association between fractures of the thoracic spine and severe pulmonary injuries, including mediastinal hemorrhage. Patients with blunt chest trauma and mediastinal widening should be evaluated for both aortic and thoracic spine injuries.4

The spine is divided into alternating mobile and fixed segments. The thoracolumbar junction (T11-L2) is considered a transitional zone between the fixed thoracic and mobile lumbar regions. This distinction is important because the transitional zones sustain the greatest amount of stress during motion, and these are the areas most vulnerable to traumatic injuries. In addition to this change in bony anatomy, the thoracolumbar junction serves as the level of transition from the end of the spinal cord (about L1) to the nerve roots of the cauda equina. Relative to the thoracic spine, the width of the spinal canal is greater. Despite a large number of vertebral injuries at the thoracolumbar junction, most are associated with normal neurologic examinations or incomplete neurologic findings.

Relative to the thoracic and thoracolumbar regions, the lower lumbar spine is the most mobile. Isolated fractures of the lower lumbar spine rarely result in complete neurologic injuries. When neurologic injuries occur, they are usually complete cauda equina lesions or isolated nerve root injuries.

Sacrum and Coccyx

The sacrum supports the lumbar vertebral column and transmits loads from the trunk to the pelvic girdle and into the lower limbs. The upper border articulates with the fifth lumbar vertebra. The inferior border articulates with the coccyx. Laterally, the sacrum articulates with the iliac bones to form the sacroiliac joints. The vertebral foramina together form the sacral canal. The sacral canal contains the nerve roots of the lumbar, sacral, and coccygeal spinal nerves and the filum terminale.

The coccyx, which articulates with the sacrum, consists of four vertebrae fused together. Except for the first vertebra, the remaining coccygeal vertebrae consist of bodies only.

Injuries of the sacral spine and nerve roots are very unusual. When they occur, they are frequently associated with fractures of the pelvis. There are multiple different classification schemes of sacral fractures that help to predict neurologic deficits and establish treatment

protocols.

P.1571

In general, transverse fractures through the body are most significant in that they cause injury to part or all of the cauda equina. Longitudinal fractures may cause radiculopathy. If there is involvement of the central sacral canal, however, bowel or bladder dysfunction may also occur.5 Careful neurologic evaluation is essential. Motor and sensory evaluation of the sacral nerve roots should be performed. Rectal examination will assess anal sphincter tone and the bulbocavernosus reflex.

Coccygeal injuries are usually associated with direct falls onto the buttocks. Patients typically describe intense pain with sitting and straining. Diagnosis of fracture is made on rectal examination. Pain will be elicited with motion of the coccyx. X-rays are not needed to diagnose coccygeal fractures. Treatment is symptomatic, and includes analgesics and use of a rubber doughnut pillow.

Spinal Cord

The spinal cord is a cylindrical structure that begins at the foramen magnum, where it is continuous with the medulla oblongata of the brain. Inferiorly, it terminates in the tapered conus medullaris at the lower border of the first lumbar vertebra. The conus is continued at its apex by a prolongation of the pia mater, the filum terminale, which extends to the base of the coccyx. The spinal cord gives rise to 31 pairs of spinal nerves: 8 cervical, 12 thoracic, 5 lumbar, 5 sacral, and 1 coccygeal. Each spinal nerve emerges through the intervertebral foramen corresponding to the appropriate spinal cord level. There is disproportionate growth in the length of the spinal cord and the vertebral column. As a result of this inequality, the length of the nerve roots increases progressively from above downward. The lower nerve roots, inferior to the conus medullaris, form an array of nerves around the filum terminale; this is called the *cauda equina*.

MECHANISM OF INJURY

Motor vehicle crashes are the principal cause of traumatic injury to the spinal cord. Other etiologies, in descending order of frequency, include falls, gunshot wounds, and injuries secondary to sports or recreational activities.

Fractures of the spine can be divided into minor and major injuries. Minor injuries are those that are localized to part of a column and do not cause instability. These fractures often result from direct blunt trauma to the posterior elements of the spine. Minor injuries include isolated fractures of the transverse, spinous, and articular processes. Major spinal injuries can be classified into four categories: 1) compression (wedge) fractures; 2) burst fractures; 3) flexion-distraction (seat belt-type injuries); and 4) fracture/dislocations.

Compression fractures result from axial loading and flexion, with subsequent failure of the anterior column (Figure 256-2). The middle column remains intact. These injuries are usually stable unless they are greater than 50 percent. They are unlikely to be directly responsible for neurologic damage.

FIG. 256-2. Wedge compression fractures are often caused by axial unloading with failure of the anterior column. **A**. Schematic of a compression fracture. **B**. Lateral reconstruction CT scan demonstrates the anterior wedging. **C**. Axial CT scan demonstrates the anterior wedging and vertebral body fracture. Note the lack of retropulsion of elements into the spinal canal.

Burst fractures occur following failure of the vertebral body under axial load (Figure 256-3). In contrast to compression fractures, both the anterior and middle columns fail. There is retropulsion of bone and disk fragments into the canal. This may cause spinal cord compression.

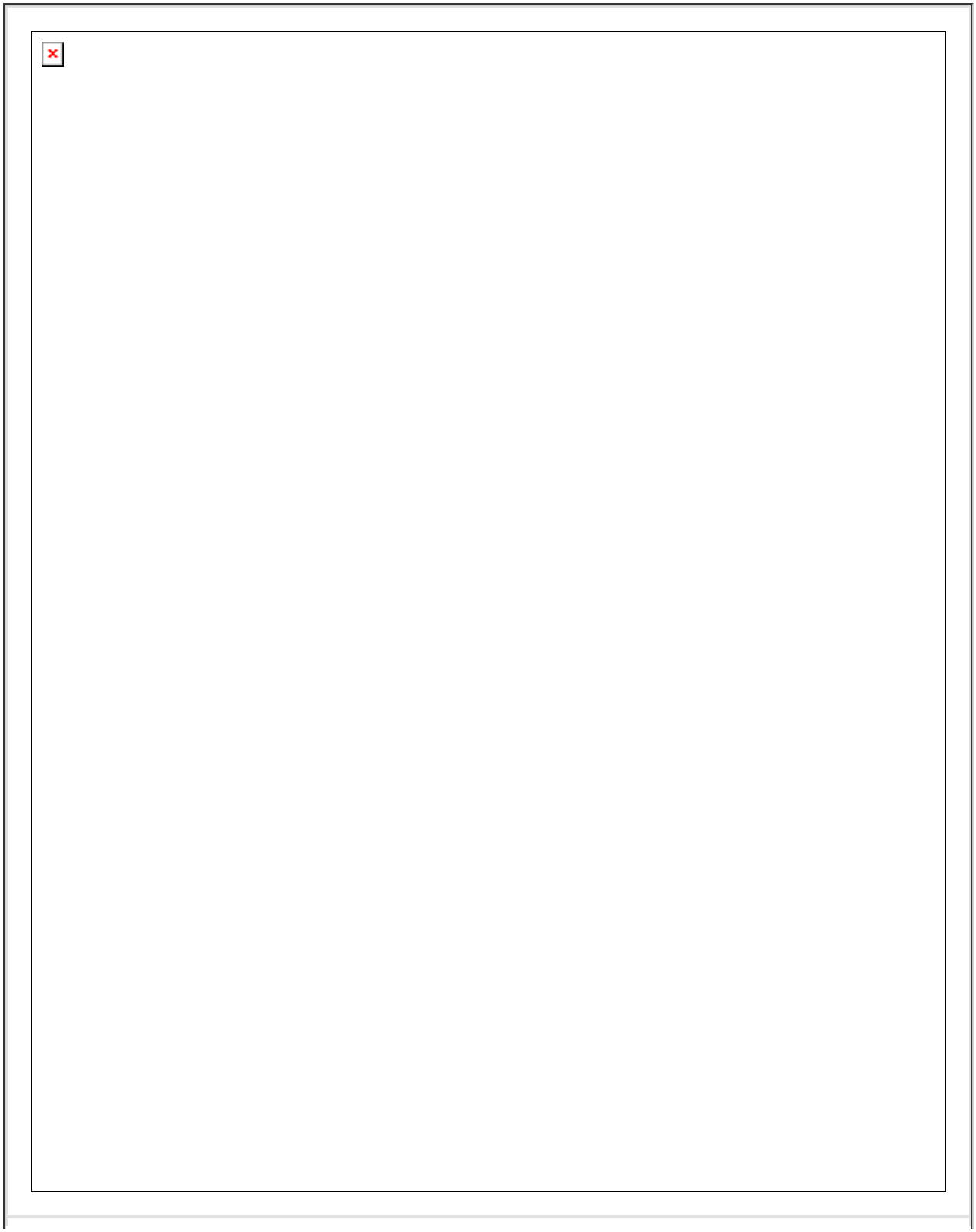


FIG. 256-3. Burst fractures are also caused by axial loading. Both anterior and middle columns have failed. **A**. Schematic of the forces transmitted. **B**. Lateral reconstruction CT scan demonstrates failure of both the anterior and middle columns. **C**. Axial CT scan demonstrates the burst vertebral body. Note the retropulsion of elements into the spinal canal.

Flexion-distraction injuries are commonly seen following seat belt-type injuries, particularly where lap belts alone are used (Figure 256-4). The seat belt serves as the axis of rotation during distraction, and there is failure of both the posterior and middle columns. The intact anterior column prevents subluxation. Typical radiographic findings reveal increased height of the posterior vertebral body, fracture of the posterior wall of the vertebral body, and posterior opening of the disk space.

FIG. 256-4. Flexion-distraction injuries involve rotation of forces. This results in failure of both the posterior and middle columns. **A**. Schematic of the forces transmitted. **B**. Lateral view of plain film demonstrating a flexion distraction injury. **C**. Lateral CT reconstruction confirming the pattern, also demonstrating posterior opening of the disk space. **D**. Axial CT scan shows loss of the middle column with fracture through the lateral elements.

Fracture-dislocations are the most damaging of injuries (Figure 256-5). Compression,

flexion, distraction, rotation, or shearing forces lead to failure of all three columns. The end result is subluxation or dislocation.

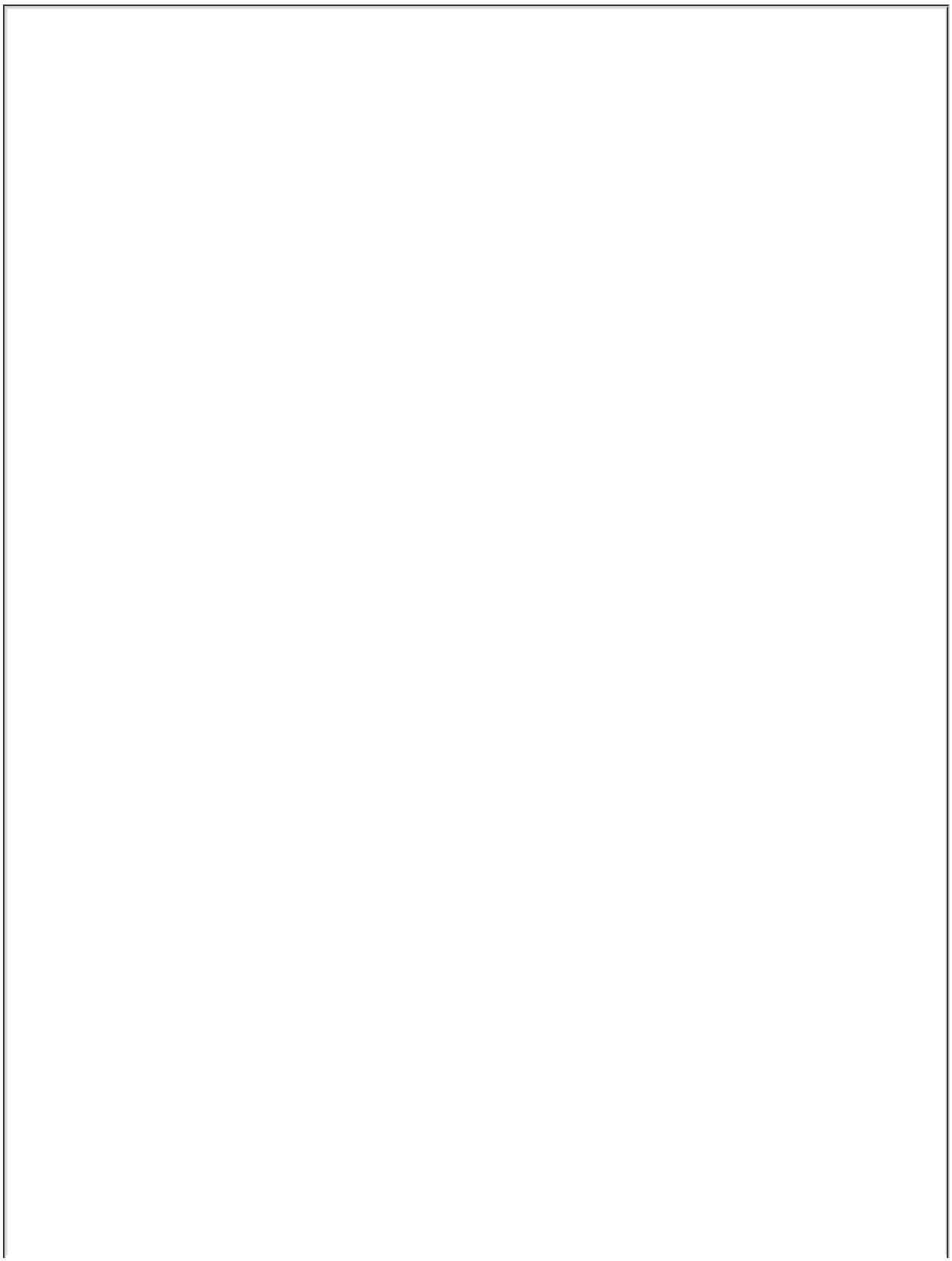


FIG. 256-5. Fracture dislocations are the most damaging of injuries leading to failure of all three columns. **A**. Demonstrates these injuries schematically. **B**. Lateral CT reconstruction demonstrates loss of all three columns. **C**. Axial CT scan demonstrates the dislocation and displacement of the vertebral body.

Blunt Injury

MOTOR VEHICLE-RELATED INJURIES

Motor vehicle crashes usually result in acceleration-deceleration injuries. The cervical spine is the most susceptible to injury by this mechanism, but the thoracic and lumbar regions are also at risk. The majority of victims are involved in low-impact crashes. Most commonly, the soft tissues are injured. Patients complain of pain in the posterior neck and back. High-speed, high-energy crashes are more likely to result in structural damage to the spine. Lap-only seat belts are associated with thoracolumbar injuries.

Pedestrians struck by vehicles and motorcyclists are at considerable risk for multiple skeletal injuries, including spinal injuries.

Obvious neurologic deficits mandate emergent treatment for an unstable injury. In the absence of deficits, the mechanism of injury with an understanding of the forces involved should guide management. When in doubt, it is best initially to overtreat and overstudy in order to avoid missing a significant injury.

Falls

Falls from a height are associated with fractures of the lower extremities, pelvis, and spine. Scalea et al. studied 161 patients who fell from a height of one or more stories, nearly one-fourth of whom suffered spine fractures.6 Of the latter, 74 percent sustained a major compression or burst fracture. The most common site of injury was the thoraco-lumbar junction. A thorough neurologic evaluation must be part of the early assessment of patients with vertical deceleration injury. All patients must be presumed to have unstable spine fractures until proven otherwise.

Sports Injuries

Spinal injuries occur in both contact and noncontact sports. The specific injury is related to the mechanism, the force involved, and the point of application of the force, rather than to the specific sport. The majority of injuries are self-limiting soft tissue injuries. Injuries at the level of the disk result in disk herniation or degeneration. Those that occur at the level of the bone can range from minimal avulsion-type fractures to compressions or fracture dislocations. Most bony injuries are not associated with neurologic sequelae. Rarely, however, sports injuries do result in significant neurologic compromise. When neurologic impairment occurs, it is usually secondary to direct axial forces. Catastrophic injuries have been associated with football, water sports (especially diving), gymnastics, rugby, and ice hockey.7

Penetrating Injury

The majority of penetrating spinal cord lesions are caused by gunshot wounds. These wounds may be localized to the spine or may involve transperitoneal trajectories. The spinal cord may be injured by direct contact with the bullet, by bony fragments, or from concussive forces.8 Most gunshot wounds result in stable vertebral injuries, although cord lesions are often complete. Stabbing injuries are much less common. These may be inflicted by a variety of implements including knives, axes, ice picks, screwdrivers, and glass fragments. The majority of stab wounds involve incomplete Brown–Séquard lesions of the thoracic cord. Among incomplete spinal injuries, these have the best prognosis. The prognosis for patients with stab injuries to the spine and incomplete paralysis is significantly better than that for patients with gunshot wounds to the spine and a similar extent of paralysis.

CLINICAL FEATURES

Damage to the spinal cord is the result of two types of injury. First is the direct mechanical injury from traumatic impact. This insult sets

P.1572

into motion a series of vascular and chemical processes that lead to secondary injury. The initial phase is characterized by hemorrhage into the cord and formation of edema at the injured site and surrounding region. Local spinal cord blood flow is diminished owing to vasospasm and thrombosis of the small arterioles within the gray and white matter. Extension of edema may further compromise blood flow and increase ischemia. A secondary tissue degeneration phase begins within hours of injury. This is associated with the release of membrane-destabilizing enzymes, mediators of inflammation, and disturbance of electrophysiologic coupling by disruption of calcium channel pathways. Lipid peroxidation and hydrolysis appear to play a major role in this secondary phase of spinal cord injury.

Spinal Cord Lesions

It is important to distinguish between complete and incomplete spinal cord injuries. The severity of injury determines the prognosis for recovery of function. The American Spinal Injury Association defines a

P.1573

complete neurologic lesion as the absence of sensory and motor function below the level of injury. This includes loss of function to the level of the lowest sacral segment. In contrast, a lesion is incomplete if sensory, motor, or both functions are partially present below the neurologic level of injury. This may consist only of sacral sensation at the anal mucocutaneous junction or voluntary contraction of the external anal sphincter upon digital examination.3 In assessing neurologic function, spinal shock must be considered. Patients in spinal shock lose all reflex activities. This generally resolves over 24 to 48 h, with the return of the bulbocavernosus reflex occurring first. Lesions

P.1574

P.1575

cannot be deemed complete until spinal shock has resolved. Complete lesions have a minimal chance of functional motor recovery. Patients with incomplete lesions are expected to have at least some degree of recovery.

Clinical syndromes classify incomplete spinal cord lesions. Damage to specific sections of the spinal cord results in predictable physical findings (Table 256-1). A discussion of the anatomy may be helpful.

TABLE 256-1 Spinal Cord Syndromes

Syndrome	Etiology	Symptoms	Prognosis
Anterior cord	Direct anterior cord compression	Complete paralysis below the lesion with loss of pain and temperature sensation	Poor
	Flexion of cervical spine	Preservation of proprioception and vibratory function	
	Thrombosis of anterior spinal artery		
Central cord	Hyperextension injuries	Quadriparesis—greater in the upper extremities than the lower extremities. Some loss of pain and temperature sensation, also greater in the upper extremities	Good
	Disruption of blood flow to the spinal cord		
	Cervical spinal stenosis		

Brown-Séquard	Transverse hemisection of the spinal cord	Ipsilateral spastic paresis, loss of proprioception and vibratory sensation and contralateral loss of pain and temperature sensation	Good
	Unilateral cord compression		
Cauda equina	Peripheral nerve injury	Variable motor and sensory loss in the lower extremities, sciatica, bowel/bladder dysfunction and “saddle anesthesia”	Good
Spinal shock	Partial or complete injury usually at the T6 level and above	Areflexia, loss of sensation, and flaccid paralysis below the level of the lesion; a flaccid bladder and loss of rectal tone; bradycarda and hypotension	Complete lesions have a poor prognosis Incomplete lesions have some degree of recovery

A large number of descending and ascending tracts have been identified in the spinal cord. The three most important of these in terms of neuroanatomic localization of cord lesions are the corticospinal tracts, spinothalamic tracts, and dorsal (posterior) columns.

The corticospinal tract is a descending motor pathway. Its fibers descend from the cerebral cortex through the internal capsule and the middle of the crus cerebri. The tract then breaks up into bundles in the pons and finally collects into a discrete bundle, forming the pyramid of the medulla. In the lower medulla, approximately 90 percent of the fibers cross (decussate) to the side opposite that of their origin and descend through the spinal cord as the lateral corticospinal tract. These fibers synapse on lower motor neurons in the spinal cord. The 10 percent of corticospinal fibers that do not decussate in the medulla descend in the anterior funiculus of the cervical and upper thoracic cord levels as the ventral corticospinal tract. Damage to the corticospinal tract neurons (upper motor neurons) in the

spinal cord results in ipsilateral clinical findings such as muscle weakness, spasticity, increased deep tendon reflexes, and a Babinski sign.

The two major ascending pathways that transmit sensory information are the spinothalamic tracts and the dorsal columns. The first neurons of both of these afferent systems begin as sensory receptors situated in the skin and stretch receptors of muscles. Their cell bodies are located in the dorsal root ganglia of the spinal nerves. The spinothalamic tract transmits pain and temperature sensation. As the axons of the first neurons enter the spinal cord, most rise one or two levels before entering the dorsal gray of the spinal cord, where they synapse with the second neuron of the spinothalamic tract. The second neuron immediately crosses the midline in the anterior commissure of the spinal cord and ascends in the anterolateral funiculus as the lateral spinothalamic tract. When the spinothalamic tract is damaged in the spinal cord, the patient experiences loss of pain and temperature sensation in the contralateral half of the body. The (pain and temperature) sensory loss begins one or two segments below the level of the lesion. The dorsal columns transmit vibration and proprioceptive information. Neurons enter the spinal cord proximal to pain and temperature neurons. They differ from pain and temperature neurons in that they do not immediately synapse. Instead, these axons enter the ipsilateral dorsal column and do not synapse until they reach the gracile or cuneate nuclei of the medulla. From these nuclei, fibers cross the midline and ascend in the medial lemniscus to the thalamus. Injury to one side of the dorsal columns will result in ipsilateral loss of vibration and position sense. The sensory loss begins at the level of the lesion. Light touch is transmitted through both the spinothalamic tracts and the dorsal columns. Therefore, light touch is not completely lost unless there is damage to both the spinothalamic tracts and the dorsal columns.

Concerning the spinal nerves and their relationship to the vertebrae, each spinal nerve is named for its adjacent vertebral body. Because there is an additional pair of spinal nerve roots compared to the number of vertebral bodies, the first seven spinal nerves are named for the first seven cervical vertebrae, each exiting through the intervertebral foramen above its corresponding vertebral body. The spinal nerve exiting below C7, however, is referred to as the C8 spinal nerve, although no eighth cervical vertebra exists. All subsequent nerve roots, beginning with T1, exit below the vertebral body for which they are named.

During fetal development, the downward growth of the vertebral column is greater than that of the spinal cord. Because the adult spinal cord ends as the conus medullaris at the level of the lower border of the first lumbar vertebra, the lumbar and sacral nerve roots must continue inferiorly below the termination of the spinal cord to exit from their respective intervertebral foramina. These nerve roots form the cauda equina. A potential consequence of this arrangement is that injury to a single lower vertebra can involve multiple nerve roots in the cauda equina. For example, an injury at the L3 vertebra can involve the L3 nerve root as well as the lower nerve roots that are progressing to a level caudal to the L3 vertebra.

Anterior Cord Syndrome

The anterior cord syndrome results from damage to the corticospinal and spinothalamic pathways, with preservation of posterior column function. This is manifest by loss of motor

function and pain and temperature sensation distal to the lesion. Only vibration, position, and crude touch are preserved. This syndrome may occur following direct injury to the anterior spinal cord. Flexion of the cervical spine may result in cord contusion or bone injury with secondary cord injury. Alternatively, thrombosis of the anterior spinal artery can cause ischemic injury to the anterior cord. Immediate evaluation with CT or magnetic

resonance imaging (MRI) may reveal an extrinsic mass that is amenable to surgical decompression. The overall prognosis for recovery of function historically has been poor and remains so today.9

Central Cord Syndrome

The central cord syndrome is usually seen in older patients with preexisting cervical spondylosis who sustain a hyperextension injury. The injury preferentially involves the central portion of the cord more than the peripheral. The centrally located fibers of the corticospinal and spinothalamic tracts are affected. The neural tracts providing function to the upper extremities are most medial in position. The thoracic, lower extremity, and sacral fibers have a more lateral distribution. Clinically, patients present with decreased strength, and to a lesser degree, decreased pain and temperature sensation, more in the upper than the lower extremities. Spastic paraparesis or spastic quadriparesis can also be seen. The majority will have bowel and bladder control, although this may be impaired in the more severe cases. Prognosis for recovery of function is good; however, most patients do not regain fine motor use of their upper extremities.9

Brown–Séquard Syndrome

The Brown–Séquard syndrome results from hemisection of the cord. It is manifest by ipsilateral loss of motor function, proprioception, and vibratory sensation, and contralateral loss of pain and temperature sensation. The most common cause of this syndrome is penetrating injury. It can also be caused by lateral cord compression secondary to disk protrusion, hematomas, bone injury, or tumors. Of all of the incomplete cord lesions, this has the best prognosis for recovery.9

Cauda Equina Syndrome

The cauda equina is composed entirely of lumbar, sacral, and coccygeal nerve roots. An injury in this region produces a peripheral nerve injury rather than a direct injury to the spinal cord. Symptoms may include variable motor and sensory loss in the lower extremities, sciatica, bowel and bladder dysfunction, and “saddle anesthesia” (loss of pain sensation over the perineum). Because peripheral nerves possess the ability to regenerate, the prognosis for recovery is better than that for spinal cord lesions.

GENERAL APPROACH

Prehospital Care

The prehospital treatment of patients with spinal cord injury involves recognition of patients

at risk, triage to an appropriate facility, and early care. All patients who have complaints of neck or back pain or who have tenderness on prehospital assessment must be presumed to have a spine injury until proven otherwise. Traditionally, all patients with significant injury above the clavicle are also presumed to have cervical spine injury regardless of related complaints. All patients with neurologic complaints must be presumed to have a spinal cord injury. Sometimes this is obvious, as in a patient with flaccid paraplegia. More often, symptoms are much more subtle (numbness or tingling in an extremity). Appropriate triage is imperative, as the results of the treatment for spinal cord injury are somewhat time related. Therefore, initial triage to a center that is capable of rapid diagnostics and therapeutics is essential to optimize outcome following spinal cord injury.

Triage can be difficult. Patients may be asymptomatic or may have suffered a concomitant head injury that makes them unable to describe their injuries and hence does not allow for neurologic assessment in the field. Other injuries may preclude accurate neurologic assessment. The mechanism of injury is an important criterion on which prehospital providers can rely. High-speed or rollover vehicular accidents, falls from a substantial height (injuries to the thoracolumbar junction), and diving and surfing accidents typically produce cervical spine injuries. Any patient at risk by mechanism of injury must be presumed to have a spinal cord injury. While this may result in a substantial rate of overtriage, the consequences of undertriage can be devastating.

Prehospital care for spinal injuries involves immobilization of the entire spine and initial fluid therapy as proposed by the American College of Surgeons.10 Patients should be transported completely immobilized. The entire cervical spine can be immobilized with a rigid cervical collar supplemented with sandbags and tape. The thoracic and lumbar spine can be immobilized using a long backboard. Patients are “papoosed” onto the boards to maintain spinal alignment. While there is little scientific evidence to support any single target for systolic or mean arterial blood pressure in patients with spinal cord injuries, a mean arterial pressure of 65 to 70 mm Hg seems a reasonable target. Optimal perfusion of the spinal cord is one of the therapies that can be implemented in the field to lessen the chances of secondary spinal injury.

All efforts should be made to rapidly deliver patients with symptomatic spinal injuries to the areawide spine center. Delays engendered by transport to a different site can result in morbidity. Clearly, patients who are not hemodynamically stable must be taken to the closest available hospital.

Emergency Department Stabilization

ED evaluation should not differ substantially from any patient with multiple injuries (see Chap. 251). Consideration should be given to immediate airway control in patients with cervical spine injuries no matter how apparently stable at the time of presentation. The higher the level of spinal injury, the more compelling the indication for early airway intervention. The roots of the phrenic nerve, which supply the diaphragm, emerge at the third, fourth, and fifth cervical vertebral levels. Thus, **any patient with an injury at C5 or above should be intubated**. It may be prudent to intubate parents with cervical cord lesions even below this level. Significant spinal cord edema may progress rostrally to involve the roots of the phrenic nerve. Many patients can initially support ventilatory

function utilizing intercostal muscles or abdominal breathing, but they eventually tire and then develop respiratory failure. As the evaluation process for these patients often involves transport outside of the ED, the authors feel strongly that early airway control is the safest route. Patients who develop respiratory failure in the CT scanner or the MRI suite may suffer respiratory arrest before it can be recognized and the airway secured.

AIRWAY

It is important to perform a complete neurologic assessment, if possible, before patients are intubated and sedated. The spine must be kept immobilized while the airway is managed. In general, this is accomplished using orotracheal intubation with in-line cervical stabilization (without distraction force) and cricoid pressure. Nasal intubation can be performed in patients while maintaining spine immobilization, although it is not a preferred method because nasal intubation is generally a blind technique. Virtually all patients with potential cervical spine trauma require sedation before nasal intubation can be accomplished; but if respirations become substantially depressed, nasotracheal intubation may not be possible. If patients are inadequately prepared, they may resist intubation. Motion of an unstable fracture can worsen spinal injury. If patients are oversedated, they may become hypoxic and lose the ability to protect the airway. Also, nasal intubation is performed with a smaller endotracheal tube, which can compromise respiratory therapy later during the hospitalization. Fiberoptic bronchoscopy may later be required for diagnostic purposes, particularly in those with pulmonary injuries or significant atelectasis, to facilitate removal of inspissated mucous plugs or blood. A relatively large endotracheal tube (8 mm or greater in size) is needed during fiberoptic bronchoscopy to ensure that ventilation is not compromised

P.1578

during the procedure. An 8-mm endotracheal tube is often too large for nasotracheal intubation. In addition, the limiting factor in minimizing tube size is the increasing respiratory resistance to gas flow imposing increased work for the patient with each spontaneous breath. Thus, the work of breathing is greater with tubes of 6 to 7 mm internal diameter, sometimes used for nasal intubation, than with tubes of 8 mm or greater internal diameter. The most limiting aspect of nasal intubation is the inability to move the cervical spine to attain the “sniff” position, optimal for the procedure. Finally, nasal intubation increases the risk of sinusitis.

If nasal intubation is selected, fiberoptic guidance is a useful technique. While the concerns of nasal intubation remain, with fiberoptic guidance, it is not a blind technique.

HYPOTENSION

Following airway stabilization, hemodynamic stability is the most pressing concern. Fluid therapy is generally the treatment of choice to support cardiovascular functioning. Patients with spinal cord injuries often present with hypotension, which must be differentiated as to its cause: spinal cord injury, blood loss, cardiac injury, or a combination. Injury to the spinal cord at the level of the cervical or thoracic vertebrae causes sympathetic denervation. There is a loss of α-adrenergic tone and dilatation of the arterial and venous vessels. Elimination of sympathetic arterial tone results in decreased systemic vascular

resistance. Loss of sympathetic innervation to the heart (T1 through T4 cord levels) leaves the parasympathetic cardiac innervation via the vagus nerve unopposed, resulting in bradycardia. While it is true that neurogenic shock is associated with bradycardia, it should never be assumed that a patient with hypotension and bradycardia is suffering from isolated neurogenic shock. Vital signs are often nonspecific. Patients with neurogenic shock may have concomitant hemorrhagic shock and may not be able to mount a tachycardic response. **Blood loss must be presumed to be the cause of hypotension until proven otherwise**. In general, patients with neurogenic shock are warm, peripherally vasodilated, and bradycardic. They seem to tolerate hypotension relatively well. This makes some sense, as peripheral oxygen delivery is presumably normal. They have incurred mechanical sympathectomy, thus cardiovascular function is not impaired.

The mechanism of injury is important in determining whether hypotension is from spinal cord injury or blood loss. Factors other than spinal cord injury (mostly blood loss) are responsible at least in part for the hypotension seen in patients with blunt trauma.11 More than 90 percent of hypotensive patients with penetrating spinal cord injury have blood loss to at least partly explain their hypotension.12

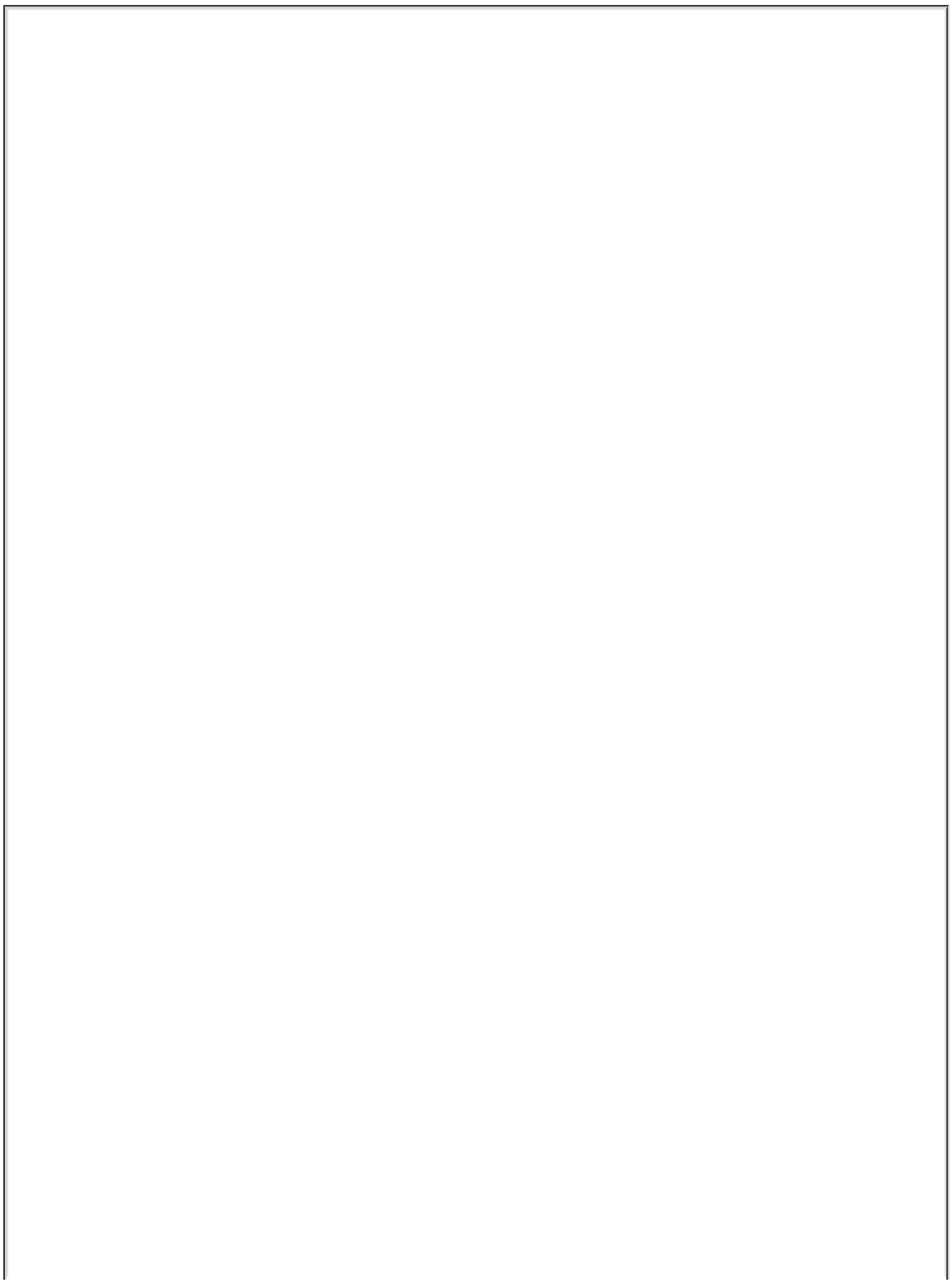
In all patients, a rapid search for potential blood loss should be undertaken. A chest x-ray will usually identify blood loss within the thorax. Retroperitoneal blood loss can come from concomitant pelvic fractures or may be secondary to lumbar arterial bleeding from spine fractures, especially in patients with substantial falls from a height. Every patient with a spinal fracture should have an abdominal diagnostic investigation. In hemodynamically unstable patients, CT scanning is often impractical; therefore ultrasonography or diagnostic peritoneal lavage should be used. Retroperitoneal bleeding should be suspected in patients without evidence of intraabdominal blood loss who develop abdominal distention or tenderness.6 Plain x-rays that demonstrate spinal fractures should help with the diagnosis. Angiography may be necessary for both diagnosis and treatment of active bleeding.

NEUROLOGIC EXAMINATION

Once patients are stabilized and other life-threatening injuries have been excluded or treated, a detailed neurologic examination should be performed. Details of history include whether the patient has had a loss of consciousness. A patient who was asymptomatic in the field and has neurologic deterioration in the ED requires emergent therapy. The presence of neck or back pain, or urinary or fecal incontinence clearly defines the patient at risk for spinal cord injury.

Physical examination should delineate the level of spinal cord injury (Figure 256-6). A complete initial neurologic examination must be documented for appropriate comparison should a patient deteriorate later. The presence or absence of neck or back tenderness should be noted. Motor function for muscle groups should be tested and recorded on a scale of 0 to 5 (Table 256-2). The level of sensory loss should be determined. Gross proprioception or vibratory function must be investigated to examine posterior column function. Deep tendon reflexes should be tested. Anogenital reflexes should also be tested, because “sacral sparing” with preservation of the reflexes denotes an incomplete spinal cord level even if the patient has complete sensory and motor loss. To test the bulbocavernosus reflex, the penis is squeezed to determine whether the anal sphincter

simultaneously contracts. Rectal tone can be assessed at the same time. Priapism implies a complete spinal cord injury. The cremasteric reflex is tested by running a pin or a blunt instrument up the medial aspect of the thigh. If the scrotum rises, there is some spinal cord integrity. The area around the anus should be tested with a pin. An “anal wink” (contraction of the anal musculature) indicates at least some sacral sparing.



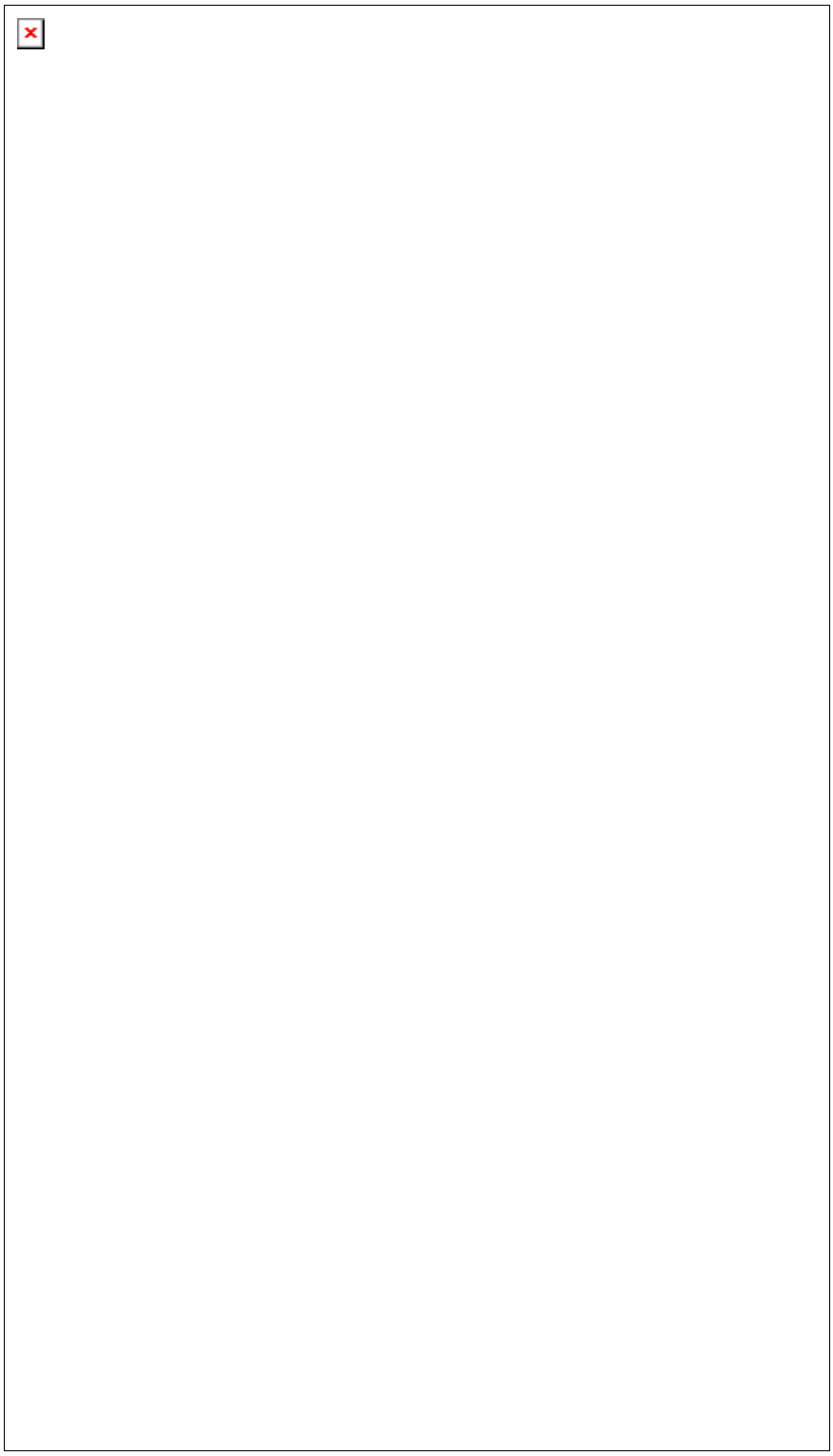


FIG. 256-6. Spinal cord level. This level can be delineated by physical examination, including a detailed neurologic examination.

TABLE 256-2 Motor Grading System

0	No active contraction
1	Trace visible or palpable contraction
2	Movement with gravity eliminated
3	Movement against gravity
4	Movement against gravity plus resistance
5	Normal power

P.1579

Diagnostic Imaging

Certainly one of the real dilemmas facing the clinician in the ED is identifying which patient requires evaluation for a potential spinal cord injury (Figure 256-7). Blind application of expensive diagnostic testing risks inappropriately increasing the cost of medical care and potentially rendering resources unavailable to other patients. Diagnostic testing must evaluate the possibility of injury to both bone and spinal cord. It is difficult to limit diagnostic testing to those who obviously require it. For example, removing a collar or allowing a patient to walk with an unstable spine risks converting a patient with no spinal cord injury to one with a complete level. Thus, it is prudent to completely image and evaluate all patients with the possibility of spinal cord pathology.

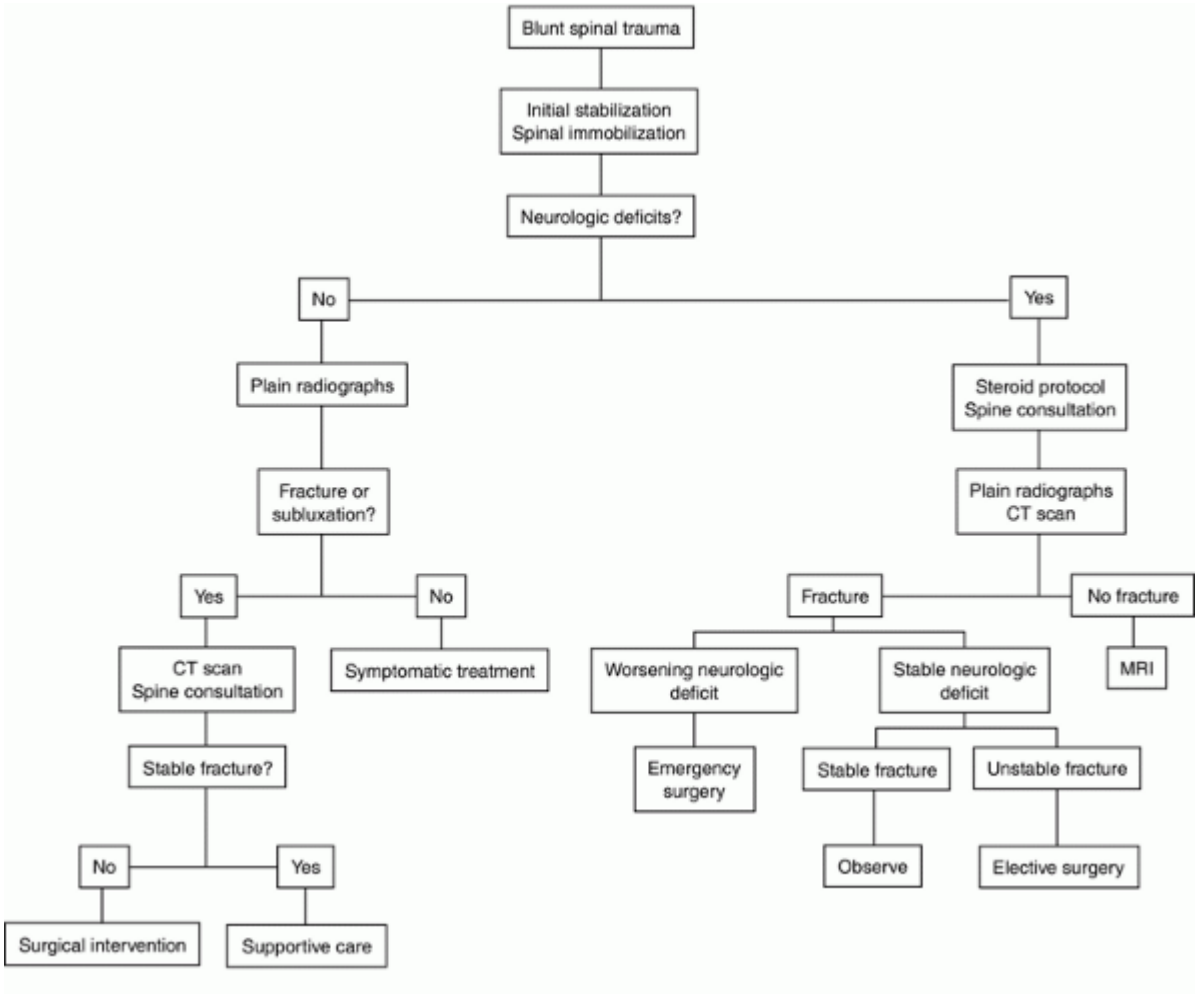


FIG. 256-7. Algorithm for blunt spinal trauma.

C-SPINE

In general, an x-ray of the cervical spine is part of the standard triage for blunt trauma. Patients with possible head or neck trauma who are not fully alert [Glasgow Coma Scale (GCS) <15] should have imaging of their cervical spines. The frequency of cervical spine injury in association with blunt head trauma is approximately 2 to 5 percent. However, it increases to almost 9 percent in patients with significant head injury, defined as a GCS score <10.13

The utility of plain films of the cervical spine in patients who are alert, oriented, and have no neck or back pain or tenderness is questionable. The National Emergency X-Radiography Utilization Study (NEXUS) group identified five clinical criteria (absence of midline cervical tenderness; normal level of alertness; no evidence of intoxication; absence of focal neurologic deficit; absence of painful distracting injury) as factors that should be

present to eliminate the performance of cervical radiographs.14 The NEXUS criteria were 99 percent sensitive for detecting clinically significant cervical spine (C-spine) injuries. The specificity of the criteria, however, was only 12.9 percent, raising concerns that use of the NEXUS criteria would actually increase the use of radiography. The “Canadian C-Spine Rule for Radiography” was subsequently developed for alert, stable trauma patients to further reduce practice variation and inefficiency in ED use of C-spine radiography.15 The Canadian Rule consists of three questions: 1) Are there any high-risk factors that mandate radiography? 2) Are there any low-risk factors that would allow a safe assessment of range of motion? and 3) Is the patient able to actively rotate the neck 45 degrees to the left and to the right? High-risk factors include age 65 years or older, a dangerous mechanism of injury (fall from a height of ≥1 m; an axial loading injury; high-speed motor vehicle crash, rollover, or ejection; motorized recreational vehicle or bicycle collision), or the presence of paresthesias in the extremities. Low-risk factors include simple rear-end motor vehicle crashes, patient able to sit up in the ED, patient ambulatory at any time, delayed onset of neck pain, or the absence of midline cervical tenderness. The Canadian C-Spine Rule had 100 percent sensitivity and 42.5 percent specificity for identifying patients with “clinically important” cervical spine injuries. Stiell et al. suggest that their rule could significantly reduce the use of C-spine radiographs ordered for alert, stable trauma patients.

The “gold standard” for the identification of bony cervical injury includes three views of the cervical spine: anteroposterior (AP), lateral, and odontoid. They allow for imaging of the entire cervical spine. It is important that all seven cervical vertebrae be imaged, including the junction between the seventh cervical and the first thoracic vertebrae. A single lateral cervical spine film will identify 90 percent of injuries to bone and ligaments.16 The film must be examined for the presence or absence of prevertebral swelling. The prevertebral space anterior to C3 should be less than 5 mm. The predental space should be less than 3 mm. The open-mouth odontoid view will identify many of the remaining abnormalities. Cervical immobilization must be maintained until the patient also has an AP and open-mouth odontoid view.

P.1580

If the initial lateral view is normal and the patient is neurologically intact, the AP and open-mouth views can be delayed until other injuries are adequately stabilized. The combination of lateral, AP, and odontoid views is generally adequate to identify or at least raise the suspicion of cervical spine injury.

Alternatively, CT scanning can be used to visualize the entire cervical spine. Patients can have a cervical spine injury even with normal plain films. The current trend in many trauma centers is to CT the entire C-spine. Plain films are poor for imaging C_1 and C_2. The only possible advantage to plain films is for visualization of prevertebral swelling and spinous process. Further, visualization of the entire cervical spine via plain films often can be problematic. Patients' body habitus or the presence of upper-extremity injuries may limit the clinician's ability to pull on the arms, a maneuver necessary to visualize all seven vertebral bodies. An alternative is a swimmer's view, which is aimed through the axilla in an attempt to image the lower cervical spine. Oblique views (45 degrees) can also be obtained. These views have the added advantage of showing the neural foramina well. They demonstrate the pedicles as well as the laminae, which should stack like shingles on a roof. Plain and even CT films of the neck may not identify patients with pure ligamentous

injuries. In these patients, ligaments are disrupted but the spine spontaneously reduces to a normal position. Motion, however, risks neurologic injury.

Flexion and extension views demonstrate the degree of spinal column stability. In general, these views are obtained when patients have pain or tenderness but normal plain films. They can only be obtained in a fully awake, unsedated, cooperative patient. The patient carefully and slowly flexes and extends the neck. Motion should be limited by increasing pain or the appearance of any neurologic symptomatology. A stepoff of 3.7 mm or an angulation of greater than 11 degrees denotes cervical spine instability.17 It is possible to have ligamentous injury even with normal flexion/extension films, because muscle tone can splint the bones in a stable configuration. Most patients in this latter category note pain improvement with analgesics after a few days.

Reliable patients without a substantial mechanism of injury with persistent pain but normal radiographs, including flexion/extension views, can be discharged in a hard collar with outpatient follow-up in 3 to 5 days. Most patients' symptoms will resolve over a few days. A patient with persistent symptoms will require additional outpatient workup. Unreliable patients or those with a significant mechanism of injury or risk factors, such as advanced age, should undergo MRI although this is rarely indicated as part of the initial investigation.

THORACOLUMBAR SPINE

Plain films of the thoracic and lumbar spine are the initial examinations generally used to image these spinal levels. Many of the same principles used for cervical spine imaging are important for thoracic and lumbar imaging. All patients with a mechanism of injury, those with complaints of back pain, and those who have tenderness on physical examination must be assumed to have a fracture of the thoracic or lumbar spine. They must be kept immobilized. AP and lateral films are generally obtained and examined for abnormality. In general, the lateral x-rays are much more easily obtained with patients still on a backboard. Skin breakdown and pressure sores can develop very quickly, particularly in obese patients. Our goal is to remove patients from the backboard in less than 2 h. Patients can then be nursed supine in a bed as long as they are logrolled. **A standard hospital mattress provides adequate spinal support**. However, patients must be carefully moved and care must be taken to keep spinal immobilization complete in transfers from bed to stretcher. It may be helpful to place patients back on a backboard for the transportation phases of their care. Alternatively, a scoop stretcher may be used for transport.

It can be difficult to image the upper thoracic spine adequately, even if maximal power of the x-ray beam is used. One alternative is to clear the reliable patient by physical examination. Unreliable or unexaminable patients with a concerning mechanism should be logrolled until the thoracic spine can be appropriately evaluated. Patients with point tenderness and normal films are a special subset. CT scanning can be useful in this subset, although the yield is low. The thoracic spine has inherent stability from the rib cage. Few fractures in these patients will be unstable. Alternatively, patients can be treated with analgesics and investigated selectively if symptoms persist.

More recently, CT has assumed a much more important role in the imaging of spine injuries. Plain films can be imperfect and may miss a number of such patients. Newer-

generation helical scanning is rapid, and CT allows for a complete three-dimensional imaging of bony structures. CT scanning is indicated in almost all patients with proven bony spinal injury, subluxations, for patients with neurologic deficits but no apparent abnormalities on plain films, those with severe neck or back pain and normal plain films, and those in whom the thoracic and lumbar spine should be examined to define the anatomy of a fracture and the extent of impingement on the spinal canal. CT can reveal the exact anatomy of an osseous injury, the extent of spinal canal impingement by bone fragments, and the stability of an injury. CT scans are indicated for all patients with fractures that can be seen on plain films. CT is especially useful when the lower cervical spine cannot be adequately visualized on plain films because of overlying soft tissues.

MRI

MRI is not as sensitive as CT for detecting or delineating bone injuries. MRI is superb at defining neurologic, muscular, and soft tissue injury. It is the diagnostic test of choice for describing the anatomy of nerve injury. Entities such as herniated disks or spinal cord contusions are easily seen on MRI. Many of these require only supportive therapy. However, some require acute surgical intervention. Early identification helps plan therapy. MRI may also be used to identify ligamentous injury. MRI is indicated in patients with neurologic findings with no clear explanation following plain films and CT scanning. CT myelography is an alternative when MRI is unavailable and immediate diagnosis of a neurologic lesion is required. If the patient is neurologically stable and MRI is unavailable, delayed MRI and/or transfer to a tertiary care facility may be appropriate.

The determination of a spinal column injury at one level should prompt imaging of the remainder of the spine. Approximately 10 percent of patients with spine fracture in one segment will have a second fracture in another. Often plain films will suffice, but multilevel CT scanning and/or MRI may be necessary to investigate such patients completely.

TREATMENT

The goals of treatment are to prevent secondary injury, alleviate cord compression, and establish spinal stability. Spinal immobilization must be maintained, and movement kept to a minimum.

Once the patient is stabilized, it should be determined whether the patient has a neurologic deficit and/or the spinal column is unstable. If either of those conditions exists, subspecialty consultation should be requested emergently. The consultant, be it a neurosurgeon or orthopedic surgeon, must have the opportunity to perform a detailed neurologic examination early in the patient's course, so as to optimize outcome. In a few cases, this will be impossible if the patient requires intubation. Patients with progressive neurologic deterioration require urgent surgical intervention. The method of stabilization (collar or traction) must be determined, as must the need for further CT or MRI imaging.

Corticosteroids

Although controversial, the use of high-dose methylprednisolone presently remains the standard of care for the treatment of blunt spinal cord injury in the US. The National Acute

Spinal Cord Injury Study (NASCIS) group conducted a series of multi-institutional studies to evaluate the efficacy of methylprednisolone in spinal trauma.18

Methylprednisolone infusion resulted in improvement of both motor and sensory function in patients with complete and incomplete neurologic lesions. This positive outcome was dependent upon dosage of steroids and time of administration. The current recommended steroid protocol for victims of spinal injury with neurologic deficits is as follows:

1. Treatment must be started within 8 h of injury.
2. Methylprednisolone (30 mg/kg) bolus administered IV over 15 min.
3. This is followed by a 45-min pause.
4. A maintenance infusion of methylprednisolone (5.4 mg/kg per h) is continued for 23 h.

NASCIS evaluated only blunt spinal cord injury. Patients with penetrating injuries were excluded from the study. **Massive steroid therapy has not been shown to be effective in penetrating spinal cord injury**, and in fact may impair recovery of neurologic function.19

The major neuroprotective mechanism by which high-dose methylprednisolone is believed to work is its inhibition of free radical-induced lipid peroxidation. Other proposed beneficial actions include its ability to increase levels of spinal cord blood flow, increase extracellular calcium, and prevent loss of potassium from injured cord tissue. Methylprednisolone is advocated in preference to other steroids because it crosses cell membranes more rapidly and completely.

Recent studies question the validity of the NASCIS trials and the effectiveness of high-dose steroid therapy in patients with spinal cord injury.20,21 Potential complications associated with prolonged, high-dose steroids such as pneumonia, sepsis, wound infection, thromboembolism, gastrointestinal bleeding, and delayed healing are a frequently cited concern; however whether any of these concerns relate to a 23-h protocol has not been investigated to date.

Penetrating Injury

Treatment goals are the same for penetrating and blunt spinal injury. There are, however, additional considerations in penetrating trauma. Figure 256-8 shows an approach for gunshot wounds. Optimal treatment of these injuries has been the subject of debate. One concern is that of infectious complications related to the presence of foreign bodies. Additional contamination is associated with transperitoneal and transintestinal trajectories of gunshot wounds to the spine. Intravenous antibiotics should be given in the ED. Surgical débridement with laminectomy has not proven effective in reducing the incidence of infectious complications, as most are managed nonoperatively. If the patient requires laparotomy for abdominal trauma, irrigation and débridement of the spinal injury through the missile tract may be appropriate.8

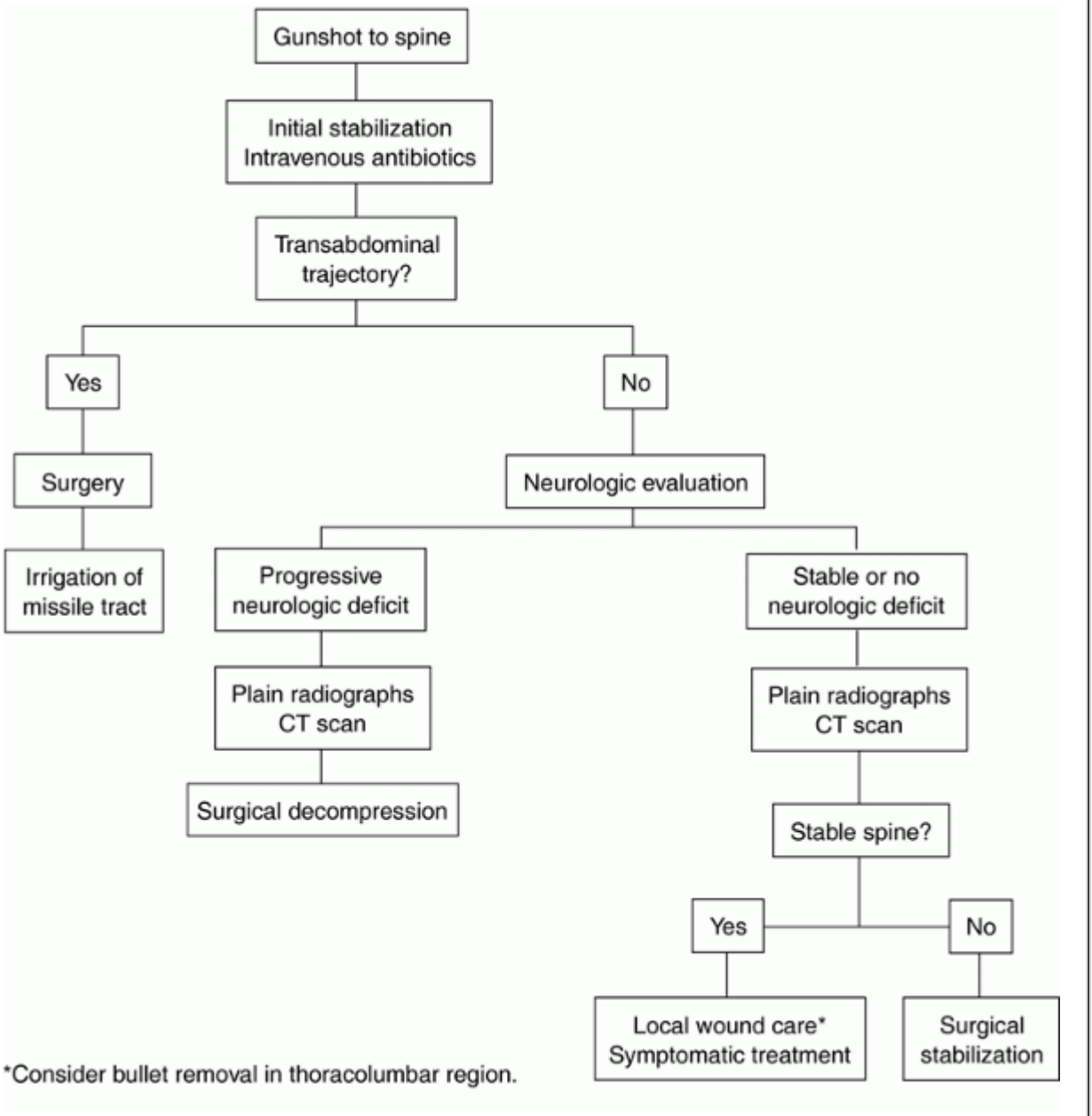


FIG. 256-8. Algorithm for gunshot to spine.

As with blunt trauma, there is general agreement that progressive neurologic deficits warrant surgical decompression. The indication for removal of bullet and bone fragments in those patients with nonprogressive neurologic deficits is less clear. Wound location may determine the need for surgical intervention. Bullet removal does not significantly improve the neurologic status of patients with stable cervical and thoracic spinal cord lesions. In contrast, bullet removal from the thoracolumbar spine may significantly improve motor recovery in both complete and incomplete injuries.22 Most gunshot wounds to the spine

following penetrating trauma are stable and require only symptomatic treatment with a supportive orthosis and analgesics.

Patients who present with stab wounds to the spinal region with no neurologic deficits should receive antibiotics and local wound care. Plain films and/or CT scan may be performed to evaluate for a retained foreign body, but the literature is controversial as to whether or

not to surgically remove the blade if there are no or stable neurologic deficits. If no metallic foreign bodies are present, neurologic deficits are best evaluated with MRI. Progressive neurologic deficits are generally treated surgically.

Chap. 258 details approaches to stab wounds to the neck. Significant spinal injuries are related to direct penetration of spinal cord neural elements, spinal infarction, or, rarely, from a spinal epidural hematoma. Vertebral instability is not an issue per se. Delayed deficits are rare. When they do occur, they are related to retained fragment of blade within the spinal canal.

Nonoperative Spinal Stabilization

The goal of stabilization is to reduce deformities and then restrict motion and maintain alignment. In the cervical spine, it is important to determine the adequacy of cervical bony reduction. Subluxations are generally reduced using Gardner–Wells tongs, which are placed into the soft tissue of the temples under local anesthesia. The location and type of injury determine the amount of weight applied. The upper cervical spine generally requires less weight for traction than the lower cervical spine. Depending upon location, initial weight should be started at 5 to 10 lb. Weight should be increased in 2.5-to 5-lb increments. Ideally, this should be done under fluoroscopic guidance. If fluoroscopy is unavailable, radiographs and neurologic examinations should be performed after each increment of weight. The radiographs should be evaluated for alignment of the spinal column and to ensure that overdistraction has not occurred. Neurologic performance can improve if reduction is achieved. Inability to achieve adequate reduction is an indication for early spinal decompression and fusion.

Spinal orthoses are used to immobilize well-reduced cervical fractures. The cervical spine is the region most effectively stabilized by external splinting devices. There is less soft tissue separating the brace from the spine at this level. In addition, some braces can be solidly secured by fixation points at the cranium and the thoracic cage. Cervical orthoses consist of cervical and cervicothoracic types. Cervical collars fit around the neck and contour to the mandible and occiput. They restrict flexion and extension in the middle and lower cervical spine. Lateral bending and rotational movements, however, are poorly controlled. Examples of cervical orthoses include the hard collar, the Philadelphia collar, and the Miami J collar. Cervicothoracic braces provide additional support. The “gold standard” is the halo cervical immobilizer, which provides the most rigid stabilization. Consisting of a halo ring pinned to the skull, a vest, and upright posts, it can be used for traction and reduction of unstable fractures as well as immobilization.

Immobilization of the upper thoracic spine by orthoses is difficult. Fortunately, an intact rib

cage and sternum provide relative stability. Although brace immobilization is not always necessary in the treatment of these fractures, braces can provide additional comfort. Thoracic corsets provide minimal control of motion and are appropriate only for minor injuries. Jewett and Taylor braces provide intermediate control of spinal motion. Maximum limitation of motion is provided by the Risser jacket and the body cast.

The thoracolumbar junction and lower lumbar regions are also difficult to immobilize externally. Splints are limited by lack of an adequate caudal fixation point. The functions of most thoracolumbosacral orthoses are the following: to create an awareness and remind the patient to restrict movements, to support the abdomen and relieve some of the load on the lumbosacral spine, to provide some restriction of motion of the upper lumbar and thoracolumbar spine by three-point fixation, and to reduce lumbar lordosis in order to provide a straighter, more comfortable lower back.

Complications of external immobilization devices include pain, pressure, muscle weakness and disuse atrophy, venous compromise, psychological dependence, ineffective stabilization, and pin-site complications (halo vest) (see Chap. 236 for ED evaluation).

Operative Management of Spine Injuries

The indication for operative stabilization is somewhat controversial and varies from institution to institution. Those favoring an aggressive approach stress the importance of early mobilization of the multiply injured patient as it helps decrease pulmonary problems, skin breakdown, deep venous thrombosis, and pulmonary embolus. Rigid fixation may also decrease time in hospital as well as long-term pain and deformities.

Those advocating a nonoperative approach point out the possibility of worsening neurologic performance by operative manipulation. In addition, the long-term results with operative intervention may not be substantially better than with nonoperative therapy.

All would agree that progressive neurologic deterioration is an indication for urgent surgery. In addition, spinal instability should most often be managed operatively even in the case of a complete spinal cord level. This helps prevent long-term deformity.

REFERENCES

1. Burney RE, Maio RF, Maynard F, et al: Incidence, characteristics, and outcome of spinal cord injury at trauma centers in North America. *Arch Surg* 128:596, 1993.

2. White AA, Panjabi MM: *Clinical Biomechanics of the Spine*, 3rd ed. Philadelphia, Lippincott, 1990.

3. Denis F: The three column spine and its significance in the classification of acute thoracolumbar spinal injuries. *Spine* 8:817, 1983.

4. Woodring JH, Lee C, Jenkins K: Spinal fractures in blunt chest trauma. *J Trauma*

28:789, 1988.

5. Denis F, Davis S, Comfort T: Sacral fractures: An important problem. Retrospective analysis of 236 cases. *Clin Orthop* 227:67, 1988.

6. Scalea T, Goldstein A, Phillips T, et al: An analysis of 161 falls from a height: The “jumper syndrome.” *J Trauma* 26:706, 1986.

7. Tall RL, De Valut W: Spinal injury in sport: Epidemiologic considerations. *Clin Sports Med* 12:441, 1993.

8. Kihtir T, Ivatury RR, Simon R, et al: Management of transperitoneal gunshot wounds of the spine. *J Trauma* 31:1579, 1991.

9. Bosch A, Stauffer ES, Nickel VL: Incomplete traumatic quadriplegia: A ten-year review. *JAMA* 216:473, 1971.

10. American College of Surgeons: *The Advanced Trauma Life Support Course*. Chicago, American College of Surgeons, 2003.

11. Soderstrom C, McArdle DQ, Ducker TB, Militello PR: The diagnosis of intra-abdominal injury in patients with cervical cord trauma. *J Trauma* 23:1061, 1983.

12. Zipnick R, Scalea TM, Trooskin SZ, et al: Hemodynamic responses to penetrating spinal cord injuries. *J Trauma* 35:578, 1993.

13. Bayless P, Ray VG: Incidence of cervical spine injuries in association with blunt head trauma. *Am J Emerg Med* 7:139, 1989.

14. Hoffman JR, Mower WR, Wolfson AB, et al: Validity of a set of clinical criteria to rule out injury to the cervical spine in patients with blunt trauma. *New Engl J Med* 343:94, 2000.

15. Stiell IG, Wells GA, Vandemheen KL, et al: The Canadian C-spine rule for radiography in alert and stable trauma patients. *JAMA* 286:1841, 2001.

16. MacDonald RL, Schwartz ML, Mirich D, et al: Diagnosis of cervical spine injury in

motor vehicle crash victims: How many x-rays are enough? *J Trauma* 30(4):392, 1990.

17. Panjabi MM, Tech D, White AA: Basic biomechanics of the spine. *Neurosurgery* 7:76, 1980.

18. Bracken MB, Shepard MJ, Collins WF, et al: A randomized, controlled trial of methylprednisolone or naloxone in the treatment of acute spinal cord injury: Results of the second national acute spinal cord injury study. *New Engl J Med* 322:1405, 1990.

19. Prendergast MR, Saxe JM, Ledgerwood AM, et al: Massive steroids do not reduce the zone of injury after penetrating spinal cord injury. *J Trauma* 37:576, 1994.

20. Nesathurai S: Steroids and spinal cord injury: Revisiting the NASCIS 2 and NASCIS 3 trials. *J Trauma* 45:1088, 1998.

21. Short DJ, El Masry WS, Jones PW: High-dose methylprednisolone in the management of acute spinal cord injury—A systematic review from a clinical perspective. *Spinal Cord* 38:273, 2000.

22. Waters RL, Adkins RH: The effects of removal of bullet fragments retained in the spinal canal. A collaborative study by the National Spinal Cord Injury Model Systems. *Spine* 16:934, 1991.