Thoracolumbar Spine Trauma: I. Evaluation and Classification

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Abstract

A timely and thorough evaluation of thoracolumbar injuries and rational treatment based on a complete understanding of the mechanism of bone, soft-tissue, and nerve injury is essential for maximizing the patient's neurologic and functional recovery and minimizing associated complications, the time to recovery, and the problems of long-term pain and deformity. The initial evaluation includes both clinical and radiologic assessment. Clinical evaluation includes the general trauma examination as well as a detailed spinal and neurologic examination to determine the level (or levels) of spinal injury. Radiologic evaluation includes both plain radiography and the appropriate use of advanced imaging modalities. A review of the evolution of thoracolumbar injury classifications is presented.

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Proper management of thoracolumbar spine injuries is predicated on a thorough understanding of the involvement of the structural and neural tissues. Initial evaluation includes a complete and thorough clinical examination. Appropriate use of radiologic modalities provides additional information, allowing classification of the spinal injury, assessment of spinal stability, and prognosis of recovery of neurologic deficits. This comprehensive initial evaluation facilitates selection among the various surgical and nonsurgical management options available.

Initial Evaluation

Clinical Examination

The specific directed examination of a patient with suspected thoracolumbar injury begins only after the typical "ABC" (airway, breathing, circulation) evaluation that is standard for all trauma patients. The spinal examination includes inspection and palpation of the spine and a careful and complete neurologic evaluation. A high degree of suspicion of other injuries frequently associated with thoracolumbar trauma, including chest injury and intra-abdominal injury, should prompt serial chest and abdominal examinations when significant spinal trauma is diagnosed.

With a cervical collar in place and extremity injuries splinted, the patient is carefully log-rolled onto his or her side as a physician stabilizes the neck in a neutral position. Abrasions and deep lacerations are suggestive of underlying spinal column injury. Open spinal injuries are infrequent but do occur, and failure to diagnose an open injury due to an incomplete examination must be avoided. Palpation of all spinous processes is done, feeling for areas of fluid collection, crepitus, and increased interspinous distance, which signal injury to the posterior elements. Areas of localized tenderness noted in the awake patient should be remembered for later scrutiny on radiographic examination.

Neurologic Evaluation

The neurologic examination for thoracolumbar trauma includes dermatomal sensory testing, assessment of lumbar- and sacral-root motor function, and a detailed reflex examination (Table 1). "Spinal shock" refers to flaccid paralysis due to a physiologic disruption of all spinal cord function. This occurs commonly below the anatomic level of an injury to the cord. Sensory and motor func-

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Table 1
Reflex Testing in Thoracolumbar Injuries

Reflex	Level Tested
Superficial abdominal (above umbilicus)	T7-T10
Superficial abdominal (below umbilicus)	T11-L1
Cremasteric reflex	T12-L1
Knee jerk	L3-L4
Ankle jerk	S1
Anal wink	S2-S4
Bulbocavernosus reflex	S3-S4
Plantar response	Brain / cord continuity

tion are absent, as are all reflexes mediated by the spinal cord levels involved (Fig. 1). An accurate assessment of the patient's neurologic status can be made only when the patient has recovered from spinal shock, which resolves within 48 hours in more than 99% of cases. The absence of spinal shock is confirmed by the presence or return of cord-mediated reflexes below the anatomic area of injury. The bulbocavernosus reflex is the lowest cord-mediated reflex and is therefore the first to return.

A "complete" neurologic injury is marked by a total absence of sensory and motor function below the anatomic level of injury in the absence of spinal shock. In an incomplete neurologic lesion, residual spinal cord and / or nerve root function exists below the anatomic level of injury. A complete cord lesion with lumbar-root sparing is the most common type of incomplete neurologic injury associated with injury at the thoracolumbar junction.

An incomplete spinal cord lesion can be confirmed only on the basis of sensory or voluntary motor function emanating from a cord segment below the anatomic level of injury; as defined by the American Spinal Injury Association, this must include the lowest sacral segments.[1] Low sacral sensation includes perianal and deep anal sensation, and motor function is tested by voluntary contraction of the external anal sphincter on digital examination. For injuries at L2 and below, sacral sensory or motor function reflects lower motor integrity, as the spinal cord terminates above that anatomic level.

An incomplete spinal cord lesion may follow one of four described classic patterns, or syndromes, based on the location of neural damage within the spinal cord (Fig. 2). The central cord syndrome is the most common incomplete spinal cord injury pattern. Since the spatial orientation of the long tracts in the spinal cord maintains lumbar function more centrally and sacral function more peripherally, a central cord syndrome of the thoracolumbar spine results in greater loss in upper lumbar motor functions (hip flexion and adduction and knee extension), with relative sparing of lower sacral motor functions. Functional motor recovery can be expected in 75% of cases. In the anterior cord syndrome, only the dorsal columns remain intact, with preservation of proprioception, vibration, and light touch and complete loss of motor function and deep pain and temperature sensation. Unfortunately, in this relatively common injury pattern, functional recovery is seen in only 10% of cases. The posterior cord syndrome is very uncommon, with isolated loss of vibration, proprioception, and light-touch sensation. A unilateral hemi–spinal cord

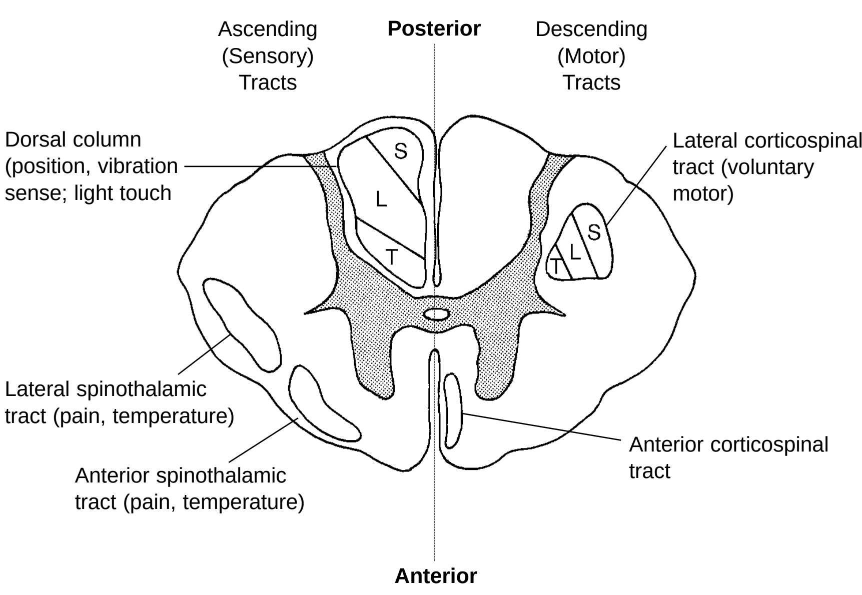


Fig. 1 Schematic drawing of a transverse section of the spinal cord at the thoracic level, showing the anatomic organization of the corticospinal tract and the posterior column (L = lumbar, S = sacral, T = thoracic).

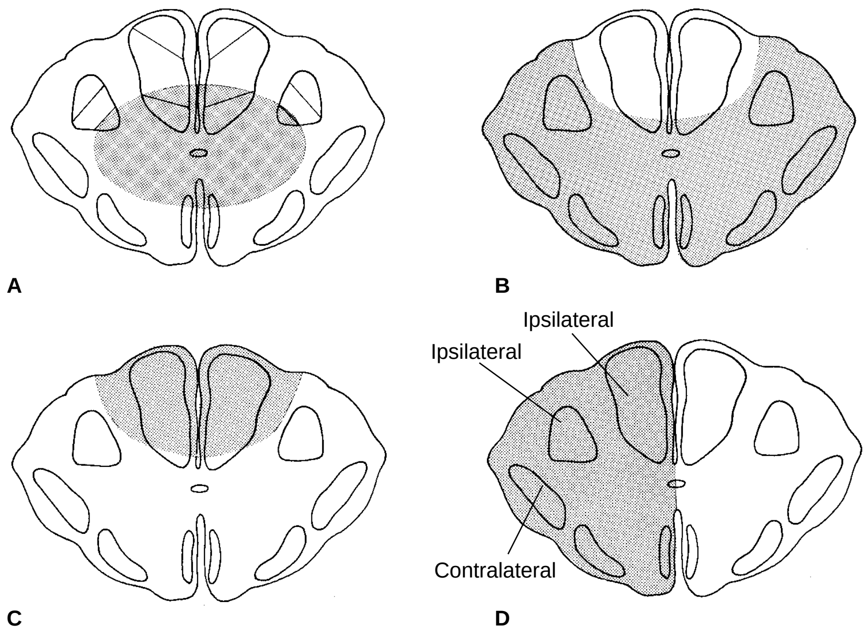


Fig. 2 Types of spinal cord injury (shaded zones) that produce the four main incomplete injury patterns seen clinically. **A,** Central cord syndrome. **B,** Anterior cord syndrome. **C,** Posterior cord syndrome. **D,** Brown-Séquard syndrome.

injury is responsible for the Brown-Séquard syndrome, an uncommon injury pattern characterized by ipsilateral loss of motor function, light touch, proprioception, and vibration sense and contralateral loss of deep pain and temperature sense. Functional motor recovery is seen in over 90% of patients with this injury pattern.

Chest and Abdominal Examination

The final aspect of the clinical evaluation of the thoracolumbar trauma patient involves the examination for and recognition of associated internal injuries in the chest and abdomen. Significant intrathoracic trauma, including hemopneumothorax, major vessel injury, and diaphragmatic rupture, may be seen in more than a third of patients with thoracic spine fractures and a neurologic deficit.[2] Intra-abdominal trauma, including bowel rupture, major vessel injury, injuries to the upper urinary tract, and hepatic, splenic, and pancreatic lacerations, is frequently associated with flexion-distraction injuries and fracture-dislocations of the thoracolumbar junction and lumbar spine.

Vital Signs

Hypotension in patients with multiple trauma and thoracolumbar spinal injuries is commonly due to "neurogenic shock," a state of relative hypovolemia due to a sudden increase in the available circulatory space caused by a loss of sympathetic tone in the thoracolumbar region with unopposed vagal parasympathetic vasodilatation. Neurogenic shock is characterized by bradycardia despite the hypotension, which is also the result of unopposed vagal output. In patients with hypotension and tachycardia, the physical examination should seek another cause for the circulatory compromise, such as cardiogenic shock or hypovolemia due to chest, abdominal, or extremity injury with local hemorrhage. In one series,[3] neurogenic shock was the cause of hypotension in 69% of patients with cervical spine injury. The percentage is probably similar in patients with upper thoracic injuries. In patients with thoracolumbar junction and lumbar injuries, however, sympathetic tone is maintained to a large extent, and neurogenic shock is less common.

Radiologic Evaluation

Radiography

The radiologic evaluation of the patient with suspected thoracolumbar spine trauma begins with plain-radiographic examination of each vertebral level (both anteroposterior and lateral views). This complete evaluation is particularly important in patients who cannot communicate about areas of pain or who are uncooperative because of head trauma, cervical trauma with neurologic deficit, or alcohol or drug intoxication.

The chest radiograph, part of the standard trauma series, must be carefully examined for evidence of mediastinal widening. Suspicion of fullness in the mediastinum must be evaluated with computed tomography (CT). An aortogram may be indicated to rule out a traumatic aortic dissection, even if thoracic spinal trauma and paraspinal hematoma are clearly visible.

Plain Tomography and CT

Computed tomography has proved invaluable in the evaluation of thoracolumbar injuries, especially in assessing the integrity of the posterior aspect of the vertebral body and posterior osseous elements (Fig. 3). Computed tomography is indicated in all cases of suspected injury to the posterior elements and posterior vertebral body. Retropulsion of

Fig. 3 Transverse CT section shows bone retropulsion from the posterior vertebral body of L5 into the spinal canal and a fracture of the lamina on the right.

bone fragments into the spinal canal can be clearly seen on transverse sections. Sagittal and coronal reconstructions can be very helpful in evaluating the alignment of the spinal canal. Both CT and plain tomography are useful for imaging areas of suspected injury seen on plain films and areas not well visualized by plain radiography, such as the cervicothoracic junction. Tomography offers the benefit of direct imaging in the sagittal and coronal planes, but is becoming less and less available as an imaging modality.

Magnetic Resonance Imaging

Magnetic resonance (MR) imaging provides direct visualization of the spinal cord and allows evaluation of intervertebral disk trauma. It is indicated in all cases with neurologic deficit to assess for both intrinsic and extrinsic cord injuries. We also use MR imaging for a more thorough preoperative evaluation of the spinal canal in all cases in which surgical treatment is planned (even those in which there is no neurologic deficit). Magnetic resonance imaging can differentiate among the various types of intrinsic cord injuries, such as edema, hematoma, and the much less frequent transection. Edema is seen as fusiform enlargement of the spinal cord with increased signal intensity on T2-weighted images. Hematoma is characterized by decreased signal intensity on T2 images acutely, and is often surrounded by a halo of T2 enhancement from adjacent edema. Edema extending more than two vertebral levels and the presence of hematoma within the spinal cord are considered poor prognostic signs for functional motor recovery. Extrinsic cord compression secondary to spinal canal compromise by osseous elements, soft tissue, or fluid collection is readily identified with MR imaging (Fig. 4).

Magnetic resonance imaging is also useful in the evaluation of "spinal cord injury without radiographic abnormalities," which is more commonly seen in children. In patients with this entity, as well as in patients with significant bone injury, MR imaging can be used to evaluate the extent of intradiskal injury and posterior ligamentous injury. In our experience, true acute disk herniations are much less common in thoracolumbar injuries than in cervical injuries, but are occasionally found. The use of MR imaging for diagnosis of acute soft-tissue injury may have a significant impact on the decision to perform early surgical stabilization. Magnetic resonance imaging may also be useful in the postinjury period in cases of late development or worsening of a preexisting neurologic injury. In these clinical situations, a treatable posttraumatic cyst or syrinx can often be diagnosed.

Myelography

Myelography may be used for many of the same indications as MR imaging, but it is invasive and does not depict the intrinsic anatomy of the spinal cord. It is indicated in cases of progressive neurologic deficit when MR imaging is not available. Myelography with postmyelogram CT scanning is also useful in the postoperative evaluation of possible persistent spinal cord compression, especially when ferromagnetic instrumentation has been used posteriorly for the indirect reduction of intracanal bone fragments.

Noncontiguous Spinal Injuries

It must be reemphasized that the entire spine must be imaged in any patient with blunt trauma to rule out additional spinal injuries that might be unstable and that, if missed, could lead to the development or progression of neurologic injury. Multilevel noncontiguous spinal injuries have been reported in up to 16.7% of cases.[4-6]

In the series of Calenoff et al,[4] noncontiguous multilevel injuries were found in 4.5% of cases. These injuries were originally missed in 50% of those cases, with the delay in

Fig. 4 T2-weighted MR image demonstrates a vertical compression injury at T8 with bone retropulsion resulting in spinal canal compromise and spinal cord edema and compression.

diagnosis averaging more than 52 days. Three common injury patterns and one subpattern were described, which accounted for 77% of the cases. Upper thoracic injuries were found to be common among patients with multilevel noncontiguous injuries, occurring in 46.7% of cases.

In a more recent series,[6] noncontiguous injuries were found in 10% of cases of spinal column injury, with only 25.6% falling into the injury patterns described by Calenoff et al.[4] This series also documented a continuing problem with failure of diagnosis of noncontiguous injuries; in 31% of patients, the secondary injury was missed initially, with an average delay in diagnosis of 7.1 days. In 25% of these missed injuries, a neurologic deficit developed or progressed due to improper initial immobilization.

Classification Schemes

Most patients with thoracolumbar injuries are still treated nonoperatively with cast or brace immobilization and early ambulation. More aggressive treatment is guided by the use of classification systems that detail the mechanisms of injury, the effects on compromised spinal structures, and the potential for late mechanical instability or neural injury.

The Spine as Two Columns

In 1963, Holdsworth[7] described a mechanistic classification of thoracolumbar fractures, including flexion, flexion-rotation, extension, and compression injuries. He felt that stability was based on the intactness of the “posterior ligament complex” (the supraspinous and interspinous ligaments, the facet joint capsule, and the ligamentum flavum). Wedge compression fractures and compression burst fractures were considered stable injuries. Extension injuries and rotational fracture-dislocations were considered unstable until healed and to be in need of temporary external stabilization. Dislocations were also considered unstable, but the ligamentous disruption was thought to require operative fusion for stabilization.

In 1968, Kelly and Whitesides[8] put forth a two-column theory of spinal stability, believing that the anterior vertebral column provided a primary weight-bearing function, while the posterior neural arch column primarily resisted tension (Fig. 5). Stability was considered to be based on the intactness of the posterior column, which they felt was strong enough to bear weight if the anterior column was compromised.

The Spine as Three Columns

In 1983, Denis[9,10] devised a new classification of thoracolumbar injuries based on a three-column theory of spinal stability (Fig. 5). The anatomic spine was divided into three sections, or columns, with radiographs and CT scans being used to assess the integrity of each. The anterior column consists of the anterior longitudinal ligament and the anterior two thirds of the annulus and vertebral body. The middle column consists of the posterior third of the vertebral body and annulus and the posterior longitudinal ligament. The posterior column consists of the osseous neural arch, the interspinous and supraspinous ligaments, and the ligamentum flavum.

On the basis of a radiographic review, 412 thoracolumbar injuries were divided into minor and major injuries. Minor injuries, which accounted for over 15% of fractures, included fractures of the spinous and transverse processes, the pars interarticularis, and the facet articulations. Major spinal injuries were divided into compression fractures, burst fractures, seat-belt injuries, and fracture-dislocations.

Compression fractures represented 47.8% of all thoracolumbar spinal injuries in Denis’ series. They are defined as a compression failure of the anterior column with an intact middle column and a posterior column that is intact or disrupted in tension. Failure of the posterior column occurs when there is more than 40% to 50% loss of anterior vertebral-body height. In burst fractures, compression failure occurs in the anterior and middle columns, often with retropulsion of middle-column bone into the spinal canal. Seat-belt injuries are flexion injuries about an axis near the anterior longitudinal ligament, with tension failure of the posterior and middle columns through bone or soft tissue. The anterior longitudinal ligament remains intact, but there may be compression failure of the anterior column. Fracture-dislocations include flexion-rotation, shear, and flexion-distraction subtypes. In each type, all three columns fail in compression, tension, rotation, or shear. Vertebral translation causes narrowing of the spinal canal at the site of injury and a high incidence of neurologic deficits.

Also in 1983, McAfee et al[11] described a classification of six fracture types based on the failure mode of the middle column. Compression fractures have only anterior column compression failure, while stable burst fractures have both anterior- and middle-column compression failure. In the unstable burst fracture, anterior- and middle-column compression failure occurs along with failure of the posterior column, either in compression, lateral flexion, or rotation. Factors such as a progressive neurologic deficit, progression of kyphosis by more than 20 degrees, more than 50% loss of vertebral body height, and free bone fragments within the spinal canal are indicative of instability in compression burst fractures. The Chance fractures described in this system resemble the flexion-distraction injuries of Denis, and the flexion-distraction injuries

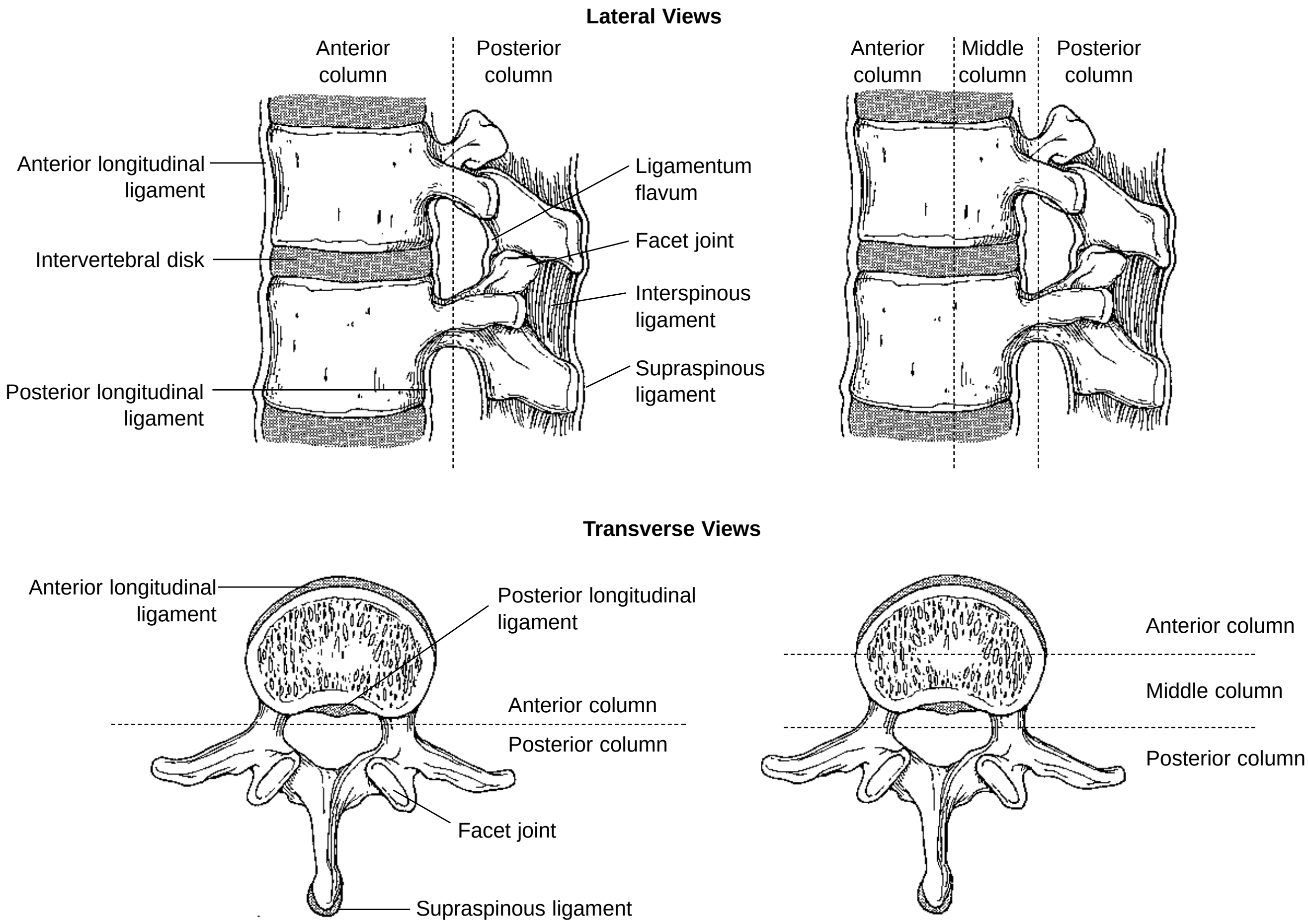


Fig. 5 **Left,** The two columns of the spine, as described by Kelly and Whitesides.[8] **Right,** The three columns of the spine, as described by Denis.[9,10]

are similar to Denis' seat-belt injuries, with compression failure of the anterior column and distraction failure of the middle and posterior columns. Translational injuries are failures in shear or rotation, which primarily occur in combination with the other injury types.

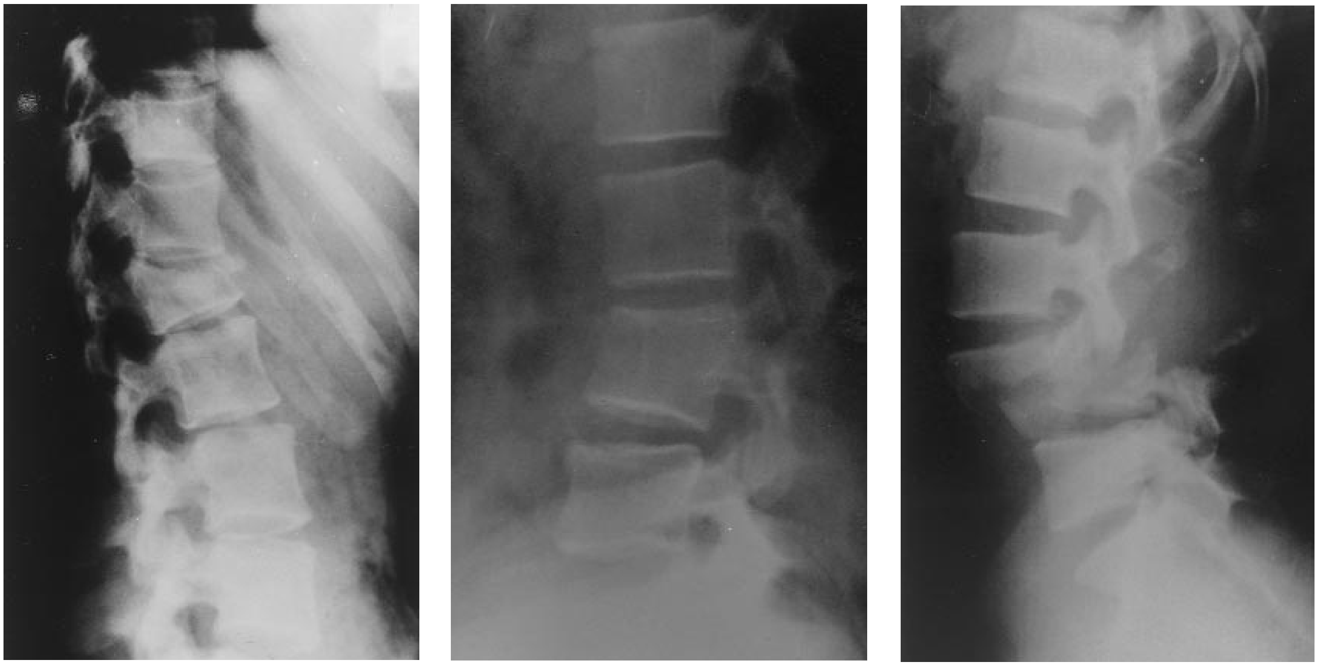
Mechanistic Classification

In 1984, Ferguson and Allen[12] presented a mechanistic classification of thoracolumbar injuries, describing seven injury patterns. This system categorizes injuries by the forces that create them and is therefore useful in guiding nonoperative and operative stabilization.

In compressive flexion injuries, three types are described, all having in common compression failure of the anterior column. Type I injuries involve only anterior column failure in compression. Type II compressive flexion injuries involve compression failure of the anterior column combined with tension failure of the posterior column, with the axis of rotation being within the middle column. In type III injuries, the middle column fails as well, rotating back into the spinal canal due to either tension or "hydraulic blowout" (Fig. 6, A). With this mechanism, the middle-column height is maintained or increased. In one series of surgically treated thoracolumbar fractures, this injury mechanism was most common, seen in 48% of cases.[13]

Distractive flexion injuries represent tension failure of all three columns due to flexion about an axis at or anterior to the anterior longitudinal ligament. This category includes flexion-distraction fracture-dislocations and the seat-belt injuries described by Denis (Fig. 6, B).

Lateral flexion injuries are seen in two patterns, the first being unilateral compression failure of the anterior and middle elements. In the second pattern, the posterior ele-



A B C

Fig. 6 Lateral injury radiographs. **A,** Ferguson-Allen type III compressive flexion injury. **B,** Distractive flexion injury. **C,** Translational injury (primarily anteroposterior translation in combination with

ments are disrupted as well, with ipsilateral compression failure and contralateral tension failure, including unilateral facet dislocations.

Translational injuries are the result of anterior-posterior or lateral shear forces. They are uncommon as isolated injuries but are often associated with other injury mechanisms (Fig. 6, C). In torsional flexion injuries, the anterior column fails in compression and rotation, and the posterior elements fail in tension and rotation, commonly with middle-column involvement as well. These and translational injuries are the most unstable thoracolumbar injuries and have the highest propensity to cause paraplegia.

Vertical compression injuries are characterized by anterior- and middle-column compression failure with vertebral-body shortening. Middle-column failure is osseous, not ligamentous, and the posterior bony elements may also fail in compression.

Distractive extension, the final injury mechanism, is rare in the thoracolumbar spine. This mechanism is characterized by tension failure of the anterior column and compression failure of the posterior elements.

Neurologic Injury and Recovery

In 1988, Dall and Stauffer[14] classified burst fractures of the thoracolumbar junction by the degree of regional kyphosis and the location of maximal canal compromise in order to correlate fracture pattern with neurologic injury and recovery. The severity of neurologic injury did not correlate with the fracture pattern or the amount of spinal canal compromise. Neurologic recovery was greatest in patients with more than 15 degrees of initial kyphosis and least in patients with less than 15 degrees of initial kyphosis and maximal canal compromise at the level of the bony posterior neural arch.

Summary

Successful management of thoracolumbar spine injuries depends on a thorough knowledge of spinal anatomy and an understanding of the injury mechanism and the resultant compromise of bone and soft-tissue structures. A complete classification scheme for thoracolumbar fractures that incorporates the mechanism of injury, a description of osseous and ligamentous destruction, and the degree of neurologic damage is still lacking. Such a classification system would enable the treating physician to identify an unstable thoracolumbar injury, make a prognosis as to neurologic recovery, and direct the choice of nonoperative or operative management.

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