

Canine Thoracolumbar Intervertebral Disk Disease: Pathophysiology, Neurologic Examination, and Emergency Medical Therapy*

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Abstract: Thoracolumbar intervertebral disk disease (IVDD) is a common, important cause of paraspinal hyperesthesia, pelvic limb ataxia, paraparesis, paraplegia, and urinary and fecal incontinence in dogs. Research offers insights into the pathophysiology, diagnosis, prognosis, and treatment of this disorder. The comparative efficacy of many familiar therapies remains unknown and controversial. This article reviews the pathophysiology and epidemiology of this condition and the examination and emergency medical therapy of dogs with suspected thoracolumbar IVDD. A companion article addresses diagnosis, prognosis, and treatment.

At a Glance

- ▶ Structure and Function
Page E1
- ▶ Pathophysiology
Page E4
- ▶ Acute Spinal Cord Injury
Page E6
- ▶ Myelomalacia
Page E6
- ▶ Spinal Shock
Page E6
- ▶ Chronic Spinal Cord Injury
Page E7
- ▶ Clinical Signs
Page E7
- ▶ Neurologic Findings
Page E7
- ▶ Emergency Medical Therapy
Page E8

Thoracolumbar intervertebral disk disease (IVDD) is a broad term, encompassing disk degeneration and clinical neurologic disease due to disk herniation. Canine IVDD has been reported to be the reason for presentation in 23 of every 1000 cases seen in 13 veterinary colleges in the United States and Canada, and it is the most common cause of thoracolumbar myelopathy with paraspinal hyperesthesia.^{1,2} A thorough, integrated understanding of spinal anatomy, pathophysiology, and neurologic function forms the foundation for medical decision-making and care for dogs with clinical signs of intervertebral disk herniation.

Structure and Function

The average canine vertebral column has 13 thoracic and seven lumbar vertebrae, each consisting of a body, a vertebral arch, and various processes.³ Each vertebral arch consists of right and left pedicles and a lamina. Together, the bodies and

vertebral arches form the vertebral canal, which houses the spinal cord.³ The intervertebral disks lie between the vertebral bodies, providing stability and flexibility to the vertebral column.^{4,5} Each disk is composed of three anatomic regions: the annulus fibrosus, nucleus pulposus, and cartilaginous end plate.^{4,6} Intervertebral disks account for about 16% of the vertebral column length in the thoracic and lumbar regions.⁷ The cervical and lumbar intervertebral disk spaces are wider than the caudal thoracic spaces (T9–T10 through T12–T13).⁸

The **annulus fibrosus** arises from mesenchymal cells to form a fibrous ring around the central nucleus pulposus^{3,9} (FIGURE 1A). The annulus is composed of distinct microscopic lamellae, each arising from the cartilaginous end plate and adjacent vertebrae.¹⁰ These lamellae run parallel to one another, are mostly composed of type I collagen, and have the ability to glide over one another during biomechanical

*A companion article, "Canine Thoracolumbar Intervertebral Disk Disease: Diagnosis, Prognosis, and Treatment," is also available on CompendiumVet.com.

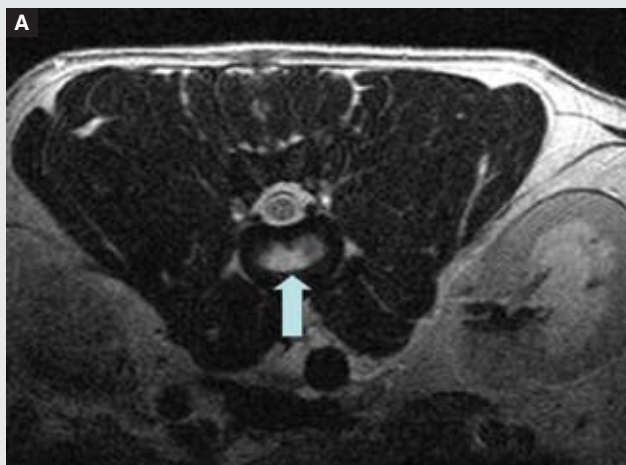
cal loading^{4,11,12} (FIGURES 1B AND 1C). The canine annulus is thickest ventrally and is sparsely innervated peripherally.^{5,13-15} This nerve supply includes nociceptive and other fibers that may be involved in sympathetic function or proprioception from the intervertebral disk.¹⁶ The nociceptive fibers probably play a role in diskogenic pain and could be a source of paraspinal hyperesthesia in some dogs with

annular tears or disk degeneration.¹⁵

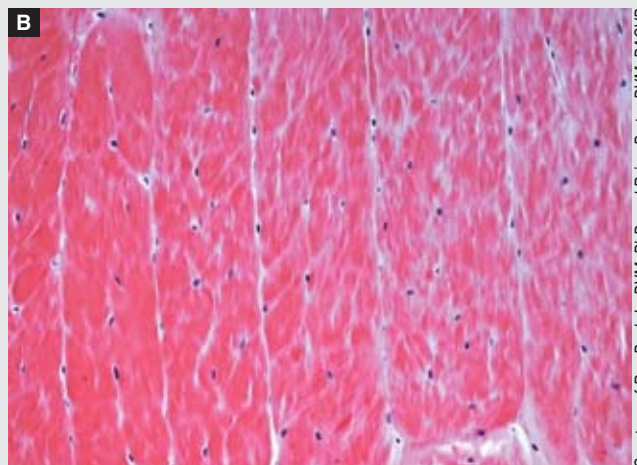
An embryologic remnant of the notochord, the **nucleus pulposus** forms the center of the intervertebral disk and comprises an extracellular matrix of water and proteoglycans^{5,13,17} (FIGURES 1D AND 2). Associated with this matrix is a sparse network of fibrous material (mostly type II collagen) and various cells (e.g., chondrocytes, fibrocytes, notochordal).^{4,5} In the healthy

FIGURE 1

Structure of the Annulus Fibrosus and Nucleus Pulposus

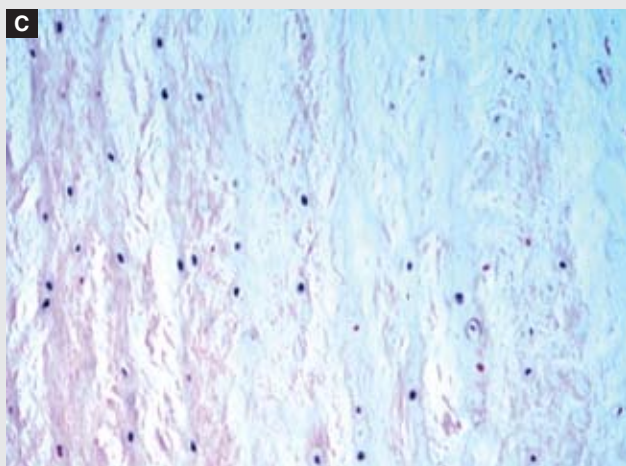


Transverse T2-weighted magnetic resonance imaging scan demonstrating the normal anatomy of the intervertebral disk. Note the clear distinction between the annulus fibrosus and nucleus pulposus. The *arrow* marks the transition between the annulus fibrosus and nucleus pulposus.



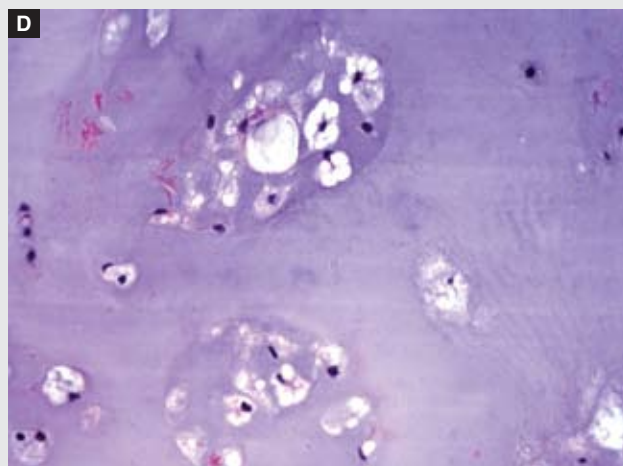
Courtesy of Roy Pool, Jr., DVM, PhD, and Brian Porter, DVM, DACVP

The annulus is composed of distinct lamellae, which are seen running in the vertical plane of this photomicrograph. These lamellae run parallel to one another and are composed mostly of type I collagen (hematoxylin and eosin stain).



Courtesy of Drs. Roy Pool and Brian Porter

The transition between annulus and nucleus is marked by decreased type I collagen content and increased proteoglycan content. The lamellae become smaller and less organized (hematoxylin and eosin stain).



Courtesy of Drs. Roy Pool and Brian Porter

The nucleus pulposus is predominantly composed of an extracellular matrix of water and proteoglycans. Associated with this matrix is a sparse network of fibrous material (mostly type II collagen) and cells (including chondrocytes, fibrocytes, and notochordal cells; hematoxylin and eosin stain).

nucleus, notochordal cells are found in large clusters connected by functional gap junctions, providing an important means of intercellular communication.¹⁸ The notochordal cells produce and assemble proteoglycans and may regulate intervertebral disk chondrocyte proteoglycan production and cell proliferation.^{19,20} Important proteoglycans include chondroitin sulfate, keratan sulfate, and hyaluronic acid.^{4,11,21} Proteoglycan content changes with aging in healthy dogs (increased keratan sulfate relative to chondroitin sulfate), and water-binding capacity decreases.^{11,21,22} It has been noted that nonchondrodystrophoid dogs maintain their intervertebral disk notochordal cells into adulthood, whereas chondrodystrophoid breeds do not, so the preserved notochordal cells may help prevent development of degenerative disk disease.¹⁹

The **cartilaginous end plate** is the site of attachment between the intervertebral disk and the vertebral body.^{6,12,13} Histologically, the end plate consists of hyaline cartilage with openings for vascular elements.⁵ Small particles diffuse across the end plate to supply the intervertebral disk with nutrients.^{23,24} It is believed that occlusion of end plate openings may lead to insufficient disk nutrition and to disk degeneration, a process that may occur with aging.²⁵⁻²⁷

Throughout the thoracolumbar vertebral column, the intervertebral disks are attached ventrally and dorsally to the **longitudinal ligaments**.¹³ The dorsal longitudinal ligament lies just dorsal to the vertebral bodies, is narrowest in the middle of the vertebra, and fans out near its attachment to each intervertebral disk.¹³ The bilateral cranial and caudal vertebral notches in the vertebral arches form the intervertebral foramina, through which spinal nerves and blood vessels pass.¹³ The lamina are bound to each other by the interarcuate ligaments.¹³ Each thoracolumbar vertebra has bilateral cranial and caudal articular processes that form synovial articulations with adjacent vertebrae.²⁸⁻³⁰ The interspinous ligaments run between adjacent spinous processes.¹³ The supraspinous ligament is a thick band of connective tissue that binds the dorsal-most aspects of the spinous processes.¹³

The vertebral column and associated structures must be able to provide flexibility and stability in response to compression, bending, shear, torsion, and tension.^{4,31} In physiologic

movement, several of these forces occur simultaneously.⁴ **Compression** occurs during loading of the disk on its neutral axis when adjacent vertebral bodies press together.^{4,32} Compression increases pressure within the nucleus pulposus that is circumferentially absorbed by the annulus fibrosus.^{4,31} **Bending** occurs with flexion or extension of the vertebral column, and **torsion** occurs with rotation about the long axis of the vertebral column.^{4,32} **Shear** refers to forces oriented in a plane perpendicular to the long axis of the vertebral column.³¹ **Tension** occurs when adjacent vertebral bodies are pulled apart along the long axis of the vertebral column.³¹

Resistance to bending, shear, and torsion is provided by the **articular facets** and **supporting ligaments** of the vertebral column in addition to the annulus fibrosus.^{4,28} The relative importance of these structures may vary with the type and direction of specific forces involved. For example, the annulus seems to be more important than the articular facets in opposing lateral bending forces in the lumbar vertebrae; however, the articular facets probably play a larger role in opposing lateral than dorsoventral bending forces.^{28,29} The supraspinous and interspinous ligaments impart stiffness to the vertebral column in response to ventral flexion.³³

QuickNotes

Disk degeneration is an aberrant cellular process that alters extracellular elements of the intervertebral disk to produce abnormal vertebral column biomechanics.

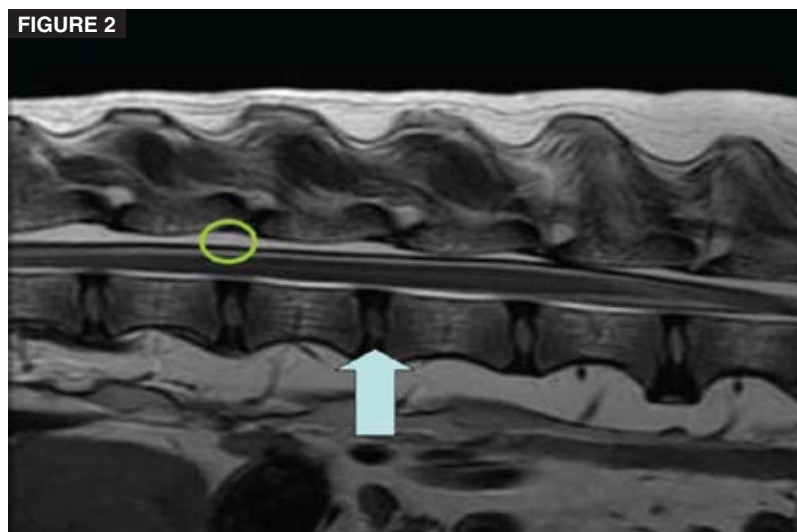


FIGURE 2
Sagittal T2-weighted magnetic resonance imaging scan demonstrating the anatomy of the lumbar vertebral column. Note that the nucleus pulposus of each intervertebral disk is hyperintense to the annulus. The arrow indicates the nucleus pulposus of the L2-L3 intervertebral disk. The circle marks epidural fat and cerebrospinal fluid. The signal intensity of each nucleus pulposus is lower than is typically expected, consistent with intervertebral disk degeneration. This dog has spondylosis deformans at T13-L1 and L4-L5.

Each thoracic vertebra articulates bilaterally with the capitulum (head) of the associated rib, and **intercapital ligaments** bind each rib to its contralateral mate.¹³ Throughout most of the thoracic vertebral column, the costovertebral joints lie in the same craniocaudal plane as the intervertebral disk.^{5,13,34,35} Hence, most of the intercapital ligaments lie immediately dorsal to the annulus.^{5,13,34,35} It has been speculated that the intercapital ligaments aid in strengthening the vertebral column, reducing the risk of disk herniation cranial to the 10th thoracic vertebra.^{5,13,34,35} Caudal to the 10th thoracic vertebra, the intercapital ligaments may lie caudal to the intervertebral disks and may be smaller or missing.^{5,13}

Pathophysiology

Thoracolumbar IVDD encompasses disk degeneration and clinical neurologic disease due to disk herniation (disk prolapse). Two patterns of disk degeneration are commonly recognized in dogs: chondroid and fibroid. Disk herniation manifests as three syndromes: disk extrusion, disk protrusion, and disk bulge. Disk herniation can result in acute or chronic spinal cord injury, with a broad spectrum of associated clinical signs.

Epidemiology

Predisposition to disk herniation likely reflects both biomechanical forces associated with body type and genetic factors associated with disk degeneration.^{5,36,37} Dachshunds, poodles, Pekingese, cocker spaniels, shih tzus, Lhasa apsos, and beagles are the most commonly affected small-breed dogs.^{1,38–40} Basset hounds, German shepherds, Labrador retrievers, and Doberman pinschers are the most commonly affected large-breed dogs.^{39,41} A study of 8117 cases of disk disease found that dachshunds were 9.9 times more likely to be affected than all breeds combined, while shih tzus, Pekingese, and Lhasa apsos were 3.9, 3.5, and 3 times more likely to be affected, respectively.¹ Dachshunds made up 48% of this study population and 72% of another large study population (654 dogs with thoracolumbar IVDD).^{1,42} The second study found that poodles and Pekingese accounted for 10.6% and 5.4% of affected dogs, respectively.⁴² Another report found that dachshunds, cocker spaniels, Pekingese, and beagles accounted for 60.3%,

7.3%, 4.7%, and 3.1% of all cases of IVDD, respectively.⁴³ Chondrodystrophoid dogs are most commonly affected between the ages of 4 and 6 years, whereas nonchondrodystrophoid dogs are generally affected between 6 and 8 years of age.^{1,5,42} Some studies suggest a slight male predisposition.^{1,44}

As the most susceptible breed, dachshunds have been studied to identify predisposing factors.^{36,42,45–47} Pedigree analysis suggests that IVDD in dachshunds is inherited in an autosomal polygenic manner that may be related to haircoat type.⁴⁵ Body weight, body condition score, and various body dimensions do not seem to affect intervertebral disk calcification.⁴⁸ Shorter vertebral column length (as measured from T1 to S1) and a shorter distance from the tuber calcaneus to the midpatellar tendon do appear to predispose to acute intervertebral disk herniation.³⁶ In addition, longer vertebral column lengths, taller height at the withers, and decreased pelvic circumference were associated with more severe myelopathy.³⁶ When dachshunds were compared with German shepherds, dachshunds were found to have an increased ratio of spinal cord height to vertebral canal height in the lumbar region and a spinal cord that terminates further caudally—differences that could leave less room within the vertebral canal to accommodate disk herniation.^{36,47}

Disk Degeneration

Chondroid metaplasia is a predictable, degenerative change in the disks of chondrodystrophoid dogs younger than 2 years^{5,22,46} (**FIGURE 3**). This process begins shortly after birth and may be caused by early chondrocyte senescence within the nucleus pulposus, leading to nuclear calcification, disk dehydration, and increased keratan sulfate content relative to chondroitin sulfate content.^{21,22,48,49} The affected nucleus in turn has abnormal biomechanical properties that may allow annular tearing when torsion and compression are applied to the disk.^{5,50} In some dogs, the annulus is weakened sufficiently to permit disk herniation. Although chondroid metaplasia is most often associated with disk extrusion, protrusion and bulge are also possible.⁵

Fibroid metaplasia is a degenerative change in the disks of older dogs that may be a pathologic response to chronic torsion.^{5,51} Repetitive microtrauma likely leads to fissures in the

QuickNotes

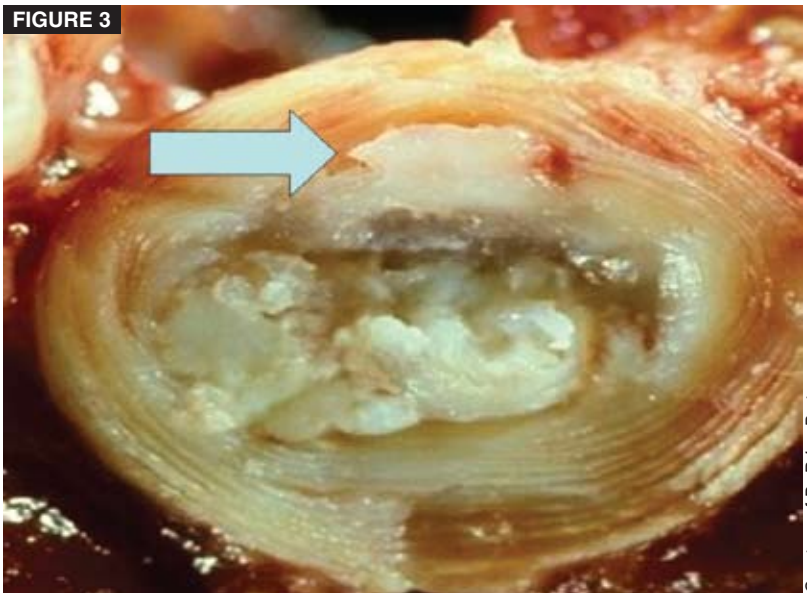
Disk herniation is the displacement of disk material into the vertebral canal, commonly manifested as disk extrusion, protrusion, or bulge.

annulus fibrosus, altered disk biomechanics, and secondary fibroid metaplasia of the nucleus pulposus.^{5,51,52} Fibroid metaplasia of the nucleus is histologically characterized by fibrous tissue deposition, disk dehydration, and increased keratan sulfate content relative to chondroitin sulfate content.^{5,22,52,53} As with disk degeneration in chondrodystrophoid dogs, fibroid degenerated disks may become calcified.^{5,52}

Disk Herniation

Disk herniation typically occurs within two disk spaces of the thoracolumbar junction (T13–L1).^{5,6,40,42,54–61} The spaces between T1–T2 and T9–T10 are seldom affected, probably due to the relative lack of mobility cranial to T10 and the presence of the intercapsular ligament.^{5,34,35,52,62} Herniation usually occurs in a dorsolateral orientation, likely influenced by the presence of the dorsal longitudinal ligament on midline and the decreased width of the annulus dorsally; the ventral aspect of the annulus fibrosus is approximately twice as thick as the dorsal aspect.^{5,63,64} Young adult chondrodystrophoid dogs are most likely to be affected by disk extrusion (**FIGURE 4**), whereas older, large-breed dogs are more susceptible to disk protrusion and bulge.^{5,6,40,42,54–61} Disk herniation in large dogs is most common at the L1–L2 disk space, whereas small dogs tend to have involvement at the T12–T13 or T13–L1 disk spaces.^{6,40–42,54–61,63} Disk extrusion is usually acute in onset, whereas disk protrusion and bulge are usually chronic.^{2,5}

Disk extrusion (Hansen's type I IVDD) is defined as complete rupture of the annulus fibrosus with translocation of the nucleus pulposus into the vertebral canal.^{2,5,6} Complete rupture of the annulus fibrosus is probably caused by abnormal stresses associated with altered biomechanical properties of the nucleus pulposus.^{5,50,65} Disk extrusion is often associated with chondroid metaplasia.⁵ The extruded nucleus usually lies in the adjacent epidural space but may also migrate cranially, caudally, or dorsally.^{5,6} Disk extrusions can lead directly to spinal cord compression and may cause laceration of the ventral vertebral venous sinuses with epidural hemorrhage, compression of the ventral spinal artery with spinal cord ischemia, and fibrocartilaginous embolic myelopathy.^{66–68} Rarely, the nucleus



Courtesy of Dr. Brian Porter

FIGURE 3
Gross specimen demonstrating degeneration of the intervertebral disk. Note the disruption of the inner fibers of the dorsal annulus (arrow) and mild protrusion of the annulus dorsally. The nucleus pulposus appears granular and lobulated, which is common in chondroid degeneration.

extrudes into the spinal cord or causes high-velocity concussion.⁶⁹

Disk protrusion (Hansen's type II IVDD) is caused by rupture of the inner layers of the annulus fibrosus, partial displacement of the nucleus into the disrupted annulus, and annular hypertrophy.⁵ Clinical disk protrusion usually leads to chronic spinal cord compression. Spondylosis deformans, a noninflammatory osteophytic reaction associated with the cartilaginous joints of the vertebral column, may be spatially related to sites of disk protrusion, but no link has been demonstrated between spondylosis and disk extrusion.^{70,71} The putative association between spondylosis deformans and disk protrusion may reflect similar predisposing factors, such as vertebral column biomechanical abnormalities and resulting annular tears.⁷⁰

Disk bulge is poorly defined in veterinary medicine and is often equated with disk protrusion. However, although these forms of disk herniation may have a shared pathogenesis, they are distinct entities.⁶ Disk bulge is defined as symmetric hypertrophy of the annulus fibrosus, probably in response to injury of the annulus and microscopic instability.^{6,51} Such injury may be associated with nuclear degeneration leading to altered vertebral column biomechanics.^{5,65}

FIGURE 4



Courtesy of Dr. Brian Porter

Gross specimen demonstrating a typical intervertebral disk extrusion in the sagittal plane. Note the compression of the overlying spinal cord, erythema of the adjacent dura, and proximity to the nerve roots.

Intravertebral disk herniation (Schmorl's nodes) is a rare manifestation of disk herniation that may be attributable to weakening of the cartilaginous end plate or of the subchondral trabeculae of the vertebral body.⁷² Abnormal axial loading causes nuclear material to herniate through the cartilaginous end plate without annular degeneration.^{11,73} Back pain is the most consistent clinical finding in dogs, with radiography and magnetic resonance imaging demonstrating defects just beneath the cartilaginous end plate.^{72,74} In one report, three of five affected dogs were German shepherds younger than 4 years.⁷²

Acute Spinal Cord Injury

Acute spinal cord injury is usually divided into primary and secondary events. *Primary injury* refers to the initial mechanical insult to the spinal cord and associated vascular structures. Primary injury can involve compression, concussion, contusion, or laceration.^{75,76} Compression occurs when adjacent structures exert pressure on the spinal cord.³² Concussion results from abrupt acceleration and deceleration of the spinal cord in response to trauma and may involve temporary axonal impairment.⁷⁷ More severe trauma may result in contusion, which is defined as a loss of vascular integrity resulting in hemorrhage into the spinal cord parenchyma and meninges.⁷⁷ Laceration occurs

when the spinal cord is physically torn or disrupted.⁷⁷ Disk extrusion can cause primary injury by each of these mechanisms, although laceration is rare.^{6,58,69,77-79} The severity of the injury is thought to correspond to the rate of extrusion, amount of disk material extruded, and duration of compression; the amount of material extruded is not necessarily proportional to the severity of the injury.^{6,60,80-82}

Primary injury results in a cascade of events that initially affect the spinal cord gray matter.^{76,83,84} Secondary injury results in neuronal cell death by necrosis and apoptosis. Necrosis typically occurs shortly after the primary injury, whereas apoptosis can occur for weeks following the injury.⁸⁵ Secondary injury can involve many interconnected systemic, local, and cellular mechanisms.^{75,83,86} Systemic mechanisms include arterial hypotension and hypoxemia.^{83,87} Local mechanisms include loss of autoregulation of spinal cord circulation, ischemia, vasogenic edema, neurotransmitter release, oxidative injury, release of matrix-degrading enzymes, loss of neurotrophic factor support, and inflammation.^{76,83,86,88-93} Cellular mechanisms include ionic derangements, altered membrane permeability, and loss of energy metabolism.

Myelomalacia

About 5% to 10% of dogs with severe spinal cord injury (absent nociception) may develop myelomalacia—gross softening of the spinal cord resulting from hemorrhagic necrosis.^{5,58,60,77} Myelomalacia may ascend and descend through the spinal cord parenchyma and is believed to result from secondary spinal cord injury.^{5,58,77,94} Dogs with a lesion initially involving the T3–L3 spinal cord segments that develop myelomalacia may have decreased pelvic limb reflexes, anal and urethral sphincter hypotonia, cranial migration of panniculus reflex, flaccid abdominal muscles, and ultimately flaccid forelimb paralysis and respiratory arrest.^{2,58} The prognosis is grave, and there is no known treatment. It is not known how many dogs with focal myelomalacia develop ascending and descending myelomalacia.^{2,5,95,96}

Spinal Shock

Spinal shock consists of temporary hypotonia and hyporeflexia caudal to a severe spinal cord injury; such decreases in reflexes are not caused

by lower motor neuron injury.⁹⁷ Spinal shock may be caused by an acute disruption of upper motor neuron facilitatory input to lower motor neurons.⁹⁷ In dogs with experimentally induced spinal cord injury, some reflexes may return in minutes (patellar), whereas others may take hours (flexor withdrawal).^{97,98} With time, adaptations such as altered excitatory neurotransmitter levels and receptor modifications often restore function in lower motor neurons caudal to the injury.^{97,98} The exact time course of spinal shock and the return of reflexes in clinical cases of disk herniation remain unknown.

Chronic Spinal Cord Injury

Disk herniation may result in chronic spinal cord compression.^{5,86,99–101} Although the increased pressure is thought to be distributed throughout the cross-sectional area of the cord, dogs with compressive lesions can have asymmetric clinical signs.^{86,102} Interestingly, the lateralization of the clinical signs does not necessarily coincide with the source or side of compression.¹⁰² Chronic spinal cord compression results in gliosis, demyelination, perivenous fibrosis, loss of cells in the gray matter, vasogenic edema, and permanent axonal loss.^{77,86,103,104} Decreased expression of neurotrophic factors in chronic spinal cord compression is probably an important cause of neuronal loss by apoptosis.¹⁰⁵ Histopathologic analysis reveals degeneration of descending upper motor neuron fibers caudal to the lesion and ascending proprioceptive fibers cranial to the compressive lesion (Wallerian degeneration).^{106,107} The oxytocin content of cerebrospinal fluid is increased in dogs with chronic spinal cord compression and may be involved in pain modulation.¹⁰⁸

In both the acute and chronic settings, the fiber diameter of the white matter dictates the progression of spinal cord injury.^{2,86,99} The variable susceptibility to injury in the white matter may be explained by the law of Laplace, which states that wall tension is directly related to pressure and radius.^{99,109} Thus, larger myelinated fibers under pressure would be expected to sustain more severe injury due to increased cell membrane tension. Conversely, small, unmyelinated fibers that carry nociceptive information to the brain are relatively resistant to injury.^{84,99,109,110} Loss of deep nociceptive fibers indicates severe spinal cord injury.^{2,58,60,63,110,111}

Clinical Signs

Paraspinal hyperesthesia is the earliest and most consistent clinical sign of thoracolumbar disk herniation.^{38,82} Progressive spinal cord dysfunction may occur and usually results in overlapping development of clinical signs. Pelvic limb proprioceptive ataxia is also seen early, followed by ambulatory paraparesis (pelvic limb weakness). Later clinical signs include nonambulatory paraparesis, urinary retention, fecal incontinence, paraplegia, and loss of nociception progressing from superficial to deep structures.^{38,40,41,58,60,61,63,82,100,110,112–114} This process can take minutes to months.²

Neurologic Findings

With few exceptions (e.g., myelomalacia, Schiff-Sherrington posture), neurologic examination reveals abnormalities limited to the pelvic limbs.² Gait analysis may show general proprioceptive ataxia, alterations in stride length (elongated with upper motor neuron involvement, shortened with lower motor neuron involvement), paraparesis, and paraplegia.¹¹⁵ The presence of motor function in a nonambulatory dog should be evaluated by supporting the dog's weight in the tail or inguinal region and walking the dog on a leash while observing for purposeful pelvic limb movement.¹¹⁰ It is important to evaluate the dog on a surface that provides good traction, such as grass or concrete. Postural reactions (knuckling, hopping, hemi-walking) are decreased or absent in dogs with paraparesis and paraplegia.^{110,115}

Pelvic limb myotatic reflexes may be normal, increased, or decreased.^{58,94,97,98,110} Lesions causing dysfunction of the T3–L3 segments are usually associated with normal to increased pelvic limb reflexes.¹¹⁰ Lesions causing dysfunction of segments L4 to S2 are usually associated with decreased pelvic limb reflexes.¹¹⁰ Spinal shock may complicate differentiation between T3–L3 and L4–S2 lesions. Depression of the patellar reflex in clinically normal geriatric dogs can also complicate neuroanatomic localization of spinal cord injury.¹¹⁶ Patellar hyporeflexia in older dogs with myelopathy must be interpreted with caution, as a subset of these patients may not have a true lesion in the L4–L6 segments.

The cutaneous trunci (panniculus) reflex may be weak or absent in some dogs with thoracolumbar disk herniation.^{110,115} This reflex

QuickNotes

Pelvic limb reflexes are typically increased with T3–L3 lesions. Decreased pelvic limb reflexes may be due to a lesion in the lumbosacral intumescence, spinal shock, or ascending/descending myelomalacia.

can be assessed by using hemostats to lightly pinch the skin of the dorsal trunk, cranial to the wings of the ilia, on each side of the midline^{110,115}; in healthy dogs, this produces ipsilateral “twitching” of the skin by the cutaneous trunci muscles.^{110,115} The afferent limb of the reflex involves the dorsal cutaneous branches of the spinal nerves. Ascending fibers course bilaterally through the fasciculus proprius to the C8–T1 cord segments, where they synapse on lower motor neurons of the lateral thoracic nerve (efferent limb), which innervates the cutaneous trunci muscle. Loss of the cutaneous trunci reflex in an animal with T3–L3 myelopathy usually implies a lesion located one or two vertebrae cranial to the cutoff point.^{110,115}

The crossed-extensor reflex occurs with upper motor neuron lesions due to decreased descending inhibitory input to lower motor neurons.^{98,117} The measurable outcome is extension of the limb ipsilateral to the lesion after flexor withdrawal is performed on the contralateral limb.^{110,115,117} This reflex must be carefully distinguished from the dog’s attempt to escape the noxious stimulus applied to initiate the withdrawal reflex.

A common mistake in the assessment of spinal cord injury is to confuse the withdrawal reflex with nociception. The withdrawal reflex can localize a lesion.^{2,110,115} A decreased withdrawal reflex can localize the lesion to either central (L4–S2) or peripheral lower motor neuron disease but provides no specific prognostic information.^{2,63,110,115} This reflex may remain intact in dogs with a spinal cord that is com-

pletely severed between the T3 and L3 spinal cord segments.^{98,110}

Nociception can be confirmed only by observing a brain-mediated response to a painful stimulus.^{2,110,115} This may be behavioral (biting, vocalizing, panting) or physiologic (increased heart rate, mydriasis). In dogs with spinal cord disease, nociception can help to evaluate lesion severity.¹¹⁰ Many clinicians differentiate between deep and superficial nociception.¹¹⁰ Superficial nociception is tested by pinching the skin with fingers or forceps. Deep nociception is tested by applying heavy pressure with forceps to bones of the digits or tail.^{58,60} Deep nociception depends on a network of small-diameter, bilateral, multisynaptic fibers that are relatively resistant to injury.^{3,118,119}

Dogs with severe T3–L3 myelopathy may adopt the Schiff-Sherrington posture: increased thoracic limb extensor tone with normal thoracic limb postural reactions. Damage to border cells or their ascending projections within the fasciculus proprius results in disinhibition of thoracic limb extensor motor neurons.¹¹⁰ Pelvic limb reflexes are classically decreased (which may relate to spinal shock) but may be normal or increased.^{75,110}

Traditionally, modified Frankel spinal cord injury scores (**BOX 1**) have been used in veterinary medicine to assess the extent of myelopathy.¹²⁰ A number of different schemes have been employed.^{36,54,59,61,74,92,110,111,121,122} A more specific functional scoring system was proposed to facilitate clinical outcome trials in ambulatory dogs with pelvic limb dysfunction caused by acute spinal cord injuries.^{123,124} This 14-point scale compared favorably with a visual analog scale in terms of intraobserver and interobserver variability in gait evaluation. These scores can be used to characterize therapeutic outcomes and compare studies in the future.¹²³

Emergency Medical Therapy

The goal of emergency therapy is to improve the likelihood of recovery by subsequent surgical or nonsurgical means. Severely affected (nonambulatory) or rapidly deteriorating dogs should be regarded as surgical emergencies because their prognosis worsens as clinical signs progress.^{2,41,55,55,58,60,63,74,113,125} Because ischemia and hypoxia are important pathophysiologic mediators of spinal cord injury, intravenous (IV) fluid replacement should be implemented.^{83,87} Physical examina-

QuickNotes

Assessment of deep nociception has important prognostic ramifications, and the presence of a withdrawal reflex does not confirm that nociception is present.

BOX 1

Modified Numerical Frankel Scale for Dogs with Thoracolumbar Spinal Cord Disease^{36,92}

- ▶ **Grade 0:** Paraplegia with absent deep nociception
- ▶ **Grade 1:** Paraplegia with absent superficial nociception
- ▶ **Grade 2:** Paraplegia with intact nociception
- ▶ **Grade 3a:** Nonambulatory paraparesis; inability to bear weight on the pelvic limbs without support
- ▶ **Grade 3b:** Nonambulatory paraparesis; ability to bear weight on the pelvic limbs without support
- ▶ **Grade 4:** Ambulatory paraparesis
- ▶ **Grade 5:** Normal gait with paraspinal hyperesthesia

tion, packed cell volume, and total protein levels are unreliable indicators of hydration, so it is prudent to apply 1.5 to 2 times maintenance fluid rates.¹²⁶ Appropriate analgesia should be administered as well.¹²⁷ Catheterization or expression can be used to relieve urine retention and bladder distention.^{128,129} Surgical candidates should be closely monitored for progression of clinical signs.

In practices that do not perform spinal surgery, it is crucial to determine whether the case should be referred to a surgical facility. Surgery is advisable in dogs with progressive, nonresponsive, or severe clinical signs such as nonambulatory paraparesis. Conservative management is usually reserved for cases with recent-onset mild myelopathy or paraspinal hyperesthesia.^{94,130-134} When the clinician is in doubt, the dog should be reevaluated and monitored at the surgical facility. The sooner a nonambulatory dog is admitted to a surgical facility, the better.^{2,41,55,56,58,60,63,74,113,125}

High-Dose Methylprednisolone

Therapy with high-dose methylprednisolone sodium succinate (MPSS) is widely used to treat acute spinal cord injury. MPSS is thought to be integrated into cell membranes, decreasing lipid peroxidation through a nongenomic mechanism of action.¹³⁵⁻¹⁴⁰ Its use in humans is controversial, appearing to yield little benefit and sometimes producing serious side effects.^{135,141-150} Evidence supporting its use in dogs with intervertebral disk herniation is likewise lacking. Several retrospective studies found no significant benefit from MPSS in this setting.^{63,151,152} Spinal cord injury models in dogs, cats, and rats have produced inconsistent and inconclusive histopathologic and functional results.^{99,153,155}

Dexamethasone

Dexamethasone is also widely used in the treatment of canine thoracolumbar intervertebral disk herniation despite a similar lack of supporting data.^{59,61,63,83,113,152,156,157} MPSS was reported to be superior to dexamethasone in promoting functional and histopathologic out-

It is unclear whether the benefits of high-dose methylprednisolone therapy outweigh the risks. High-dose dexamethasone therapy is not recommended.

comes in one rat model of spinal cord injury.¹⁵⁵ A rodent model of gradual spinal cord compression over 7 days demonstrated improved motor function in rats treated with high- and low-dose dexamethasone compared with nontreated rats; mortality was higher in the high-dose group.¹⁵⁷

Complications

The complications of high-dose corticosteroid therapy are well known. Studies have shown that 33% of dogs treated with high-dose prednisolone had gastrointestinal (GI) side effects and that nine of 10 healthy dogs treated with high-dose MPSS had severe gastric hemorrhage.^{158,159} Dachshunds treated with MPSS were more likely to have GI side effects, required more GI protectant drugs, and had an increased cost of hospitalization.¹⁵¹ Most GI side effects are not life threatening, but increased rates of sepsis and pneumonia have been linked to MPSS.^{147,151} Colonic perforation is a life-threatening side effect reported in a few dogs treated with dexamethasone.^{160,161}

Other Therapies

21-Aminosteroid compounds such as tirilazad inhibit lipid peroxidation and may be beneficial in minimizing secondary spinal cord injury. An advantage of these compounds is that they do not have many of the side effects of high-dose corticosteroids.¹³⁵ Nonetheless, a clear demonstration of therapeutic benefit is lacking.^{99,135} Other pharmacologic options include IV surfactants, which may seal cell membrane defects and thereby repair spinal axons.¹⁶² Dogs with acute disk herniation treated with two surfactants, polyethylene glycol and poloxamer 188, exhibited no adverse drug effects and recovered spinal cord function faster than historical controls. Other medications have been tried (e.g., dimethyl sulfoxide, solcoseryl, mannitol, naloxone, crocetin, thyrotropin-releasing hormone), but none has shown clinical efficacy in dogs; mannitol has had harmful effects in a feline model of acute spinal cord injury.^{154,156} **C**

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3 CE
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1. The normal nucleus pulposus is predominantly composed of

- a. keratan sulfate.
- b. water and proteoglycans.
- c. a sparse network of type I collagen fibers.
- d. type I collagen fibers arranged in lamellae.

2. Which statement is true regarding the cartilaginous end plates?

- a. They represent the site of attachment between the intervertebral disk and the interarcuate ligaments.
- b. Histologically, the end plate consists of hyaline cartilage with openings for cortical bone.
- c. Small particles diffuse across the end plate to supply the intervertebral disk with nutrients.
- d. Occlusion of end plate openings may be associated with in utero migration of neuroendocrine precursor cells.

3. Which statement is false regarding compression of the vertebral column?

- a. Compression occurs during loading of the disk on its neutral axis when adjacent vertebral bodies press together.
- b. A cause-and-effect relationship has been demonstrated between repeated compressive forces and disk degeneration in chondrodystrophoid dogs.
- c. Compression results in increased pressure within the nucleus pulposus.
- d. none of the above

4. Which statement characterizes the intercapital ligaments?

- a. They bind each rib to adjacent, ipsilateral ribs.

- b. They are present throughout the length of the thoracic vertebral column.

- c. They lie immediately ventral to the annulus or vertebral body.

- d. They may serve as a protective force against disk herniation.

5. Which statement regarding disk degeneration is true?

- a. Chondroid metaplasia is a predictable, degenerative change of the disks of chondrodystrophoid dogs younger than 2 years.

- b. Disk degeneration involves cellular swelling and increased water content of the disk.

- c. Disk calcification is specific for chondroid disk degeneration.

- d. Disks undergoing chondroid and fibroid degeneration typically have increased chondroitin sulfate content relative to keratan sulfate content.

6. Which statement regarding disk herniation is true?

- a. Disk herniation occurs most commonly within two disk spaces of L3-L4.

- b. Herniation usually occurs in a ventral orientation.

- c. Disk extrusion is usually chronic in onset, whereas disk protrusion and bulge are usually acute.

- d. Young chondrodystrophic dogs are more likely to be affected by disk extrusion than older, large-breed dogs.

7. Which statement regarding types of spinal cord injury is true?

- a. Primary injury can involve compression, concussion, contusion, or laceration.

- b. Concussion involves focal disruption of vascular elements and hemorrhage into the parenchyma and meninges.

- c. Laceration occurs commonly with disk extrusion.

- d. Delayed neuronal cell death occurs exclusively by necrosis.

8. Spinal shock involves

- a. increased extensor tone of the thoracic limbs with normal postural reactions.

- b. decreased reflexes that are not caused by lower motor neuron injury.

- c. myelomalacia.

- d. chronic, progressive myelopathy due to disk protrusion.

9. An acutely nonambulatory dog with decreased pelvic limb reflexes and normal mentation that regains normal reflexes in a few hours is most likely exhibiting clinical signs of

- a. a synovial cyst.

- b. spinal shock.

- c. an intervertebral disk protrusion.

- d. diskospondylitis.

10. High-dose corticosteroid therapy

- a. has been shown to significantly improve outcome in dogs treated within 8 hours of spinal cord injury.

- b. is associated with sepsis, pneumonia, and gastric hemorrhage.

- c. causes pulmonary thromboembolism in most dogs.

- d. a and b.