SPINAL CORD TRAUMA – HOW TO MANAGE INTERVERTEBRAL DISC DISEASE

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Acute spinal cord injuries of dogs or cats result most commonly from direct physical trauma such as missile injury or vertebral fracture or luxation. Also, spinal cord trauma is the underlying cause of neurologic signs in numerous myelopathies (e.g., intervertebral disk disease). Chronic spinal cord compression usually is seen in association with chronic progressive diseases such as neoplasia or type II disk protrusion.

Following injury, the spinal cord may undergo sustained compression, distraction, or both. The severity of a spinal cord injury, as determined by the eventual degree and quality of recovery, is related to three factors: the *velocity* with which the compressive force is applied, the *degree* of compression (transverse deformation), and the *duration* of the compression.

An understanding of differences between acute and chronic spinal cord injury is essential for effective management and determination of prognosis in cats or dogs with spinal trauma.

Acute Spinal Cord Injury

Blunt traumatic injury to the spinal cord causes neurologic deficits through both *direct* and *indirect* mechanisms. The direct effects are due to *immediate mechanical disruption* of neural pathways, and have been considered by most investigators not to be amenable to therapy. Indirect effects develop during the first few hours following injury, and result in *delayed secondary injury to the spinal cord*. This is mediated by a cascade of pathological changes including hemorrhagic necrosis, lipid peroxidation, lipid hydroxylation with subsequent prostaglandin and leukotriene (eicosanoid) formation, loss of calcium ions from the extracellular space and loss of potassium ions from the intracellular space, ischemia with consequent decline in tissue oxygen tension and energy metabolites and development of lactic acidosis, and inflammation and neuronophagia by PMN leukocytes. This secondary damage has been considered potentially reversible through the use of either physical (e.g., hypothermia) or pharmacologic interventions.
A special feature of acute spinal cord injury is progressive hemorrhagic myelomalacia.

Chronic Spinal Cord Compression

Chronic spinal cord compression results either from a slowly developing lesion (e.g., neoplasia), or from an acute compression that is sustained. In contrast to acute spinal cord injury, chronic compression injury affects white matter more severely than it affects gray matter. Hemorrhage and edema, the major findings of acute trauma, are not significant in chronic compression. Characteristic lesions are degeneration of myelin, focal areas of malacia, vacuolization, and loss of white matter axons. Mechanical deformation is likely to be the major factor in pathogenesis of these lesions; however, ischemia and venous obstruction also may be important consi

INTERVERTEBRAL DISK DISEASE

Etiology and Pathogenesis

Degeneration of intervertebral disks may result in protrusion of anulus fibrosus or extrusion of nucleus pulposus into the vertebral canal, causing spinal cord compression and clinical signs ranging from apparent pain to complete transverse myelopathy. Degenerative changes may occur in any of the intervertebral disks. However, it occurs most commonly in the cervical, caudal thoracic, and lumbar

spine. The intervertebral disks between T1 and T11 are stabilized dorsally by the intercapital ligaments and as a result disk protrusion or extrusion is less likely in this region.

Two types of disk degeneration (type I and type II) have been reported to occur in dogs. Type I disk extrusion occurs with degeneration and rupture of the dorsal anulus fibrosus and extrusion of nucleus pulposus into the vertebral canal. Type I disk extrusion is most commonly associated with chondroid disk degeneration . Although it occurs most commonly in chondrodystrophoid breeds (Dachshund, Beagle, Pekingese, Lhasa Apso, Shih Tzu) and breeds with chondrodystrophoid tendencies (miniature Poodle and Cocker spaniel) chondroid disk degeneration and type I disk extrusion may occur in any breed, including large breeds of dog. Type II disk protrusion is characterized by bulging of the intervertebral disk without complete rupture of the anulus fibrosus. Type II disk protrusion is most commonly associated with fibroid disk degeneration .

Recent studies have demonstrated differences between the vertebral canal and spinal cord mensuration in Dachshunds and German shepherd dogs. The spinal cord of Dachshunds was found to terminate further caudally than that of German shepherd dogs. Further, the ratio of spinal cord to vertebral canal heights in the lumbar region was notably greater in Dachshunds than in German shepherd dogs. The smaller lumbar epidural space in Dachshunds may explain the occurrence of severe clinical signs seen in this breed in association with apparently small amounts of extruded disk material. It is also possible that the larger epidural space present in large breeds of dog may account for the fact that small amounts of extruded disk material within the spinal canal in these breeds may not cause spinal cord compression and associated clinical signs.

Chondroid degeneration of disks is characterized by an increase in collagen content of the disk, alteration of specific glycosaminoglycan concentration of the nucleus pulposus, and a decrease in water content of the disk. The normally gelatinous nucleus pulposus becomes progressively more cartilaginous and granular and eventually may mineralize (calcify). Extrusion of degenerative nucleus pulposus occurs through fissures in, or rupture of, the anulus fibrosus. Hansen has reported that in chondrodystrophoid breeds of dog, 75 to 100 per cent of all disks undergo chondroid metaplasia by one year of age. The radiographic pattern of degenerative changes that occur with aging in the vertebral column and intervertebral disks of beagles has been described. This study aids in the differentiation of clinically insignificant degenerative changes and pathologic changes that may produce clinical signs.

Fibroid disk degeneration occurs in older dogs of all breeds but is most often recognized as a clinical problem in older, large-breed, nonchondrodystrophoid dogs and is characterized by fibrous metaplasia of the nucleus pulposus. An increase in the noncollagenous glycoprotein content of intervertebral disks occurs in nonchondrodystrophoid breeds of dog with aging. Calcification of the disk may occur, but is rare. Protrusion of the disk occurs with a bulging of the anulus fibrosus due to partial rupture of the anular bands. Rupture of the anulus fibrosus and extrusion of nucleus pulposus (characteristic of type I disk extrusion) uncommonly is seen in association with type II disk protrusion.

Intervertebral disk protrusion or extrusion may occur in a ventral, dorsal, or lateral direction. In most instances, only dorsal protrusions or extrusions are of clinical signficance as meningeal irritation and nerve root and/or spinal cord compression may occur. Occasionally a lateral disk protrusion or extrusion may result in nerve root or spinal nerve compression with associated clinical signs.

The cause of intervertebral disk degeneration is unknown. Trauma does not appear to play a major role in chondroid degeneration but may be a factor in acute disk extrusion. Mechanical and anatomic factors are probably important, as disk extrusions are most common in the cervical and T11 to L3 regions of the vertebral column. Genetic factors probably have a role in the accelerated degeneration of disks in chondrodystrophoid breeds, but the exact influence of these factors is not known. Hypothyroidism and autoimmune disease have also been proposed as contributing factors.

Type I disk extrusion often results in more severe clinical signs than type II protrusion although the mechanical distortion and compression of the spinal cord caused by type II protrusion may be greater. Nucleus pulposus is most often extruded into the spinal canal acutely (minutes to hours) or subacutely (days) from disks undergoing chondroid degeneration, whereas slowly progressive spinal cord compression most often accompanies protrusion of disks undergoing fibroid degeneration as the bulging fibrous mass increasingly enlarges within the spinal canal. The spinal cord changes seen in acute versus chronic spinal cord compression differ, and are reflected in the difference in clinical signs and response to treatment seen in these different types of intervertebral disk disease. The severity of spinal cord injury depends on the velocity at which the compressive force is applied, the degree of compression, and the duration of the compression. Vascular factors, as well as mechanical distortion of the spinal cord as a result of herniated disk material, are important in the pathogenesis of resulting spinal cord lesions. Severe spinal cord lesions may be found in spinal cord that does not have evidence of compression, presumably as a result of vascular changes.

Hemorrhage, edema, and necrosis of spinal cord gray and white matter are characteristic of acute spinal cord injury associated with acute type I disk extrusion. Hemorrhage and edema are not a major feature of chronic spinal cord compression in which white matter changes such as demyelination, focal malacia, vacuolization, and loss of axons are seen. Type I disk extrusions often are associated with rupture of vertebral venous sinuses, and hemorrhage into the epidural space may increase the degree of spinal cord compression. Pulmonary emboli arising from the nucleus pulposus have been described in three chondrodystrophoid dogs with acute thoracolumbar transverse myelopathies as a result of type I disk extrusions, presumably as a result of disk material entering the vertebral venous sinuses. Nucleus pulposus may also penetrate the aura mater. Traumatic rupture of the anulus fibrosus and extrusion of normal nucleus pulposus may occur, resulting in spinal cord compression and an acute onset of clinical signs indicative of a transverse myelopathy.

Degenerative disk disease also occurs in cats, although the incidence of clinical signs associated with disk protrusion is low. Degenerative changes and distribution of disk protrusions are similar to type II disk protrusions in nonchondrodystrophoid dogs. Clinical signs seen usually are indicative of a slowly progressive transverse cervical or thoracolumbar myelopathy. Type I disk extrusion associated with calcification of intervertebral disks and an acute onset of neurologic deficits also have been reported in cats. Diagnosis and treatment are similar to that described for dogs.

Clinical Findings

Chondroid degeneration and type I disk extrusion most commonly occur in dogs three years of age and older, but may occur in younger animals. Fibroid degeneration and type II disk protrusion most commonly occur in dogs older than 5 years of age. There does not seem to be a sex predilection for intervertebral disk disease.

Clinical signs seen in association with type I disk extrusion include apparent pain and/or motor and/or sensory deficits. These clinical signs usually develop rapidly, within minutes or hours of disk extrusion. However, clinical signs may progress slowly over several days or manifest periods of improvement and subsequent worsening over weeks or months. These findings are probably associated with extrusion of small amounts of disk material into the spinal canal over a period of time.

Clinical signs associated with type I disk extrusion in the cervical spine usually are less severe than those associated with extrusions in the thoracolumbar region as the vertebral canal in this region is larger in diameter in relation to the spinal cord than is the case in the thoracolumbar region. Apparent neck pain is the most common clinical finding in dogs with cervical disk extrusion. Affected dogs often hold the head and neck rigidly and cry out when moved, and may show spasms of cervical musculature.

Neurologic deficits indicative of a cervical myelopathy such as proprioceptive deficits, tetraparesis, or tetraplegia are seen less commonly.

Ipsilateral Homer's syndrome and hyperthermia have been described in cases of acute, severe, dorsolateral cervical disk extrusions. Lower motor neuron deficits in the thoracic limbs may be seen in caudal cervical disk extrusions. Thoracic limb lameness may also be seen in caudal cervical disk extrusions as a result of nerve root compression, particularly from lateral disk extrusions where disk material enters an intervertebral foremen.

Clinical findings in animals with thoracolumbar type I disk extrusion depend on the severity of spinal cord injury, and range from apparent back or abdominal pain to complete paraplegia and loss of deep pain perception. Neurologic deficits usually are indicative of a transverse myelopathy between T3 and L3, as most disk extrusions in this region occur between T 11 and L3. Lower motor neuron signs may be seen in the pelvic limbs if disk extrusion occurs caudal to L3 as a result of compression of the lumbosacral spinal cord or nerves of the cauda equina. Lower motor neuron signs also may be seen in paraplegic animals with progressive hemorrhagic myelomalacia (PHM). The clinical signs and diagnosis of PHM are discussed later.

The panniculus reflex may be depressed or absent caudal to the site of disk extrusion. The site of a lesion is usually one or two vertebral spaces cranial to the loss or depression of panniculus reflex. The Schiff-Sherrington sign may be seen in animals with acute type I disk extrusion caudal to T2.

Clinical signs seen in both cervical and thoracolumbar type I disk extrusion may be asymmetric, especially if extrusion occurs dorsolaterally within the spinal canal.[172,173] Apparent pain associated with disk extrusions results from inflammation and/or ischemia caused by compression of meninges and/or spinal nerve roots. Extruded disk material initiates an extradural inflammatory reaction that results in fibrous adhesions between the aura mater and extruded disk material. Pain may also arise from stimulation of sensory nerve endings in the anulus fibrosus and dorsal longitudinal ligament. The nucleus pulposus of each disk does not contain nerve fiber endings.

Clinical signs associated with type II disk protrusion generally are slowly progressive over a period of months. Clinical signs, however, may develop acutely over days in some animals. Neurologic deficits usually are indicative of a cervical or thoracolumbar myelopathy. Paraparesis or tetraparesis, depending on the site of the lesion, is the most common clinical finding, and deficits may be asymmetric. In the cervical spine, type II protrusions most commonly occur in caudal cervical disks. In some cases, caudal cervical type II disk protrusion may be part of the spectrum of abnormalities associated with cervical spondylomyelopathy. Apparent neck or back pain may or may not be a feature of type II disk protrusion.

Diagnosis

A tentative diagnosis of type I disk protrusion or extrusion may be made on the basis of age, breed, history, and clinical signs; however, other causes of transverse myelopathy or apparent pain should be considered in the differential diagnosis. It must be remembered that apparent spinal pain is seen in animals with meningitis. Dogs with thoracolumbar disk extrusions may show apparent abdominal pain, and causes of pain such as pancreatitis and peritonitis must be considered in the differential diagnosis.

The differential diagnosis in animals with type II disk protrusion includes other causes of progressive transverse myelopathy, the most likely being neoplasia or degenerative myelopathy.

Vertebral column radiographs and, in almost all cases, CSF analysis and myelography are necessary to confirm a diagnosis of disk extrusion or protrusion. General anesthesia is required to achieve the

precise positioning needed to obtain radiographs of diagnostic value. Foam wedges or sandbags are usually needed to align the vertebral column parallel to the table top for lateral projections. Care must be taken, however, in anesthetizing and positioning animals that have acute type I disk extrusions, as further extrusion of disk material and further spinal cord compression may occur with manipulation and movement of the spine.

Calcification of the nucleus pulposus is best seen on lateral radiographic views and usually is seen in one or more disks of most chondrodystrophoid dogs more than one year of age. Calcified disks also may be seen in older nonchondrodystrophoid breeds of dog. Calcified material within the nucleus pulposus is indicative of disk degeneration, but alone is not of clinical significance.

The disk space of an extruded disk may be narrower than adjacent disk spaces and may be wedge-shaped with a decrease in the width of the disk space dorsally. However, positioning is important as some disk spaces (C7-T1, T9-10 or T10-11, and L7-SI) are normally narrower than adjacent spaces, and cervical and lumbosacral disks are normally wedge-shaped on hyperextension and flexion of the spine. "Spikes" of calcified material suggestive of disk extrusion may extend dorsally from a disk. Calcified material may be present within the vertebral canal but often is difficult to visualize due to overlying vertebral articular processes or ribs. Intervertebral foramina are larger in the lumbar spine, and calcified material often is easily visualized in the spinal canal in this region. Disk material within the spinal canal may appear as a hazy, indistinct shadow or as a dense mass with distinct margins. In many cases of disk extrusion calcified material is not visualized within the spinal canal, as disk material is probably not sufficiently mineralized to be visible on radiographs. Ventrodorsal views, and in some cases oblique views, are important in determining laterality of any visible mineralized material within the spinal canal. Vertebral osteophytes and vertebral end-plate sclerosis may be seen associated with chronic disk degeneration and extrusion or in cases of chronic disk degeneration without disk extrusion or protrusion.

Type II disk protrusion may be associated with narrowing of the disk space, osteophyte production, and end-plate sclerosis. Calcification of disk material rarely is seen in association with type II disk protrusion. In some animals with type I or type II disk herniation obvious abnormalities are not seen on noncontrast vertebral radiographs.

Myelography is almost always necessary to confirm that disk material has herniated into the spinal canal resulting in spinal cord compression. Myelography is most important in determining the site (or sites) of disk herniation and in lateralization of disk material within the spinal canal prior to surgical decompression. Myelography, is necessary as a means of distinguishing disk protrusion from other causes of slowly progressive transverse myelopathy such as spinal neoplasia and degenerative myelopathy.

Cerebrospinal fluid should be collected and analyzed prior to myelography to rule out inflammatory or infectious disease of the spinal cord and/or meninges. Clinical signs in animals with GME, distemper myelitis, FIP, spinal lymphoma, and other disorders may mimic those of cervical or thoracolumbar disk disease.

The characteristic myelographic findings in both type I and type II disk herniation into the spinal canal are extradural compression of the spinal cord with displacement of the spinal cord and narrowing of the subarachnoid space on lateral and/or ventrodorsal views, depending on the location of the compressive mass. Type II, and most type I, disk herniations result in a ventral or ventrolateral epidural mass that causes dorsal displacement of the spinal cord. Disk material may extend over more than one vertebral segment in type I extrusions and may result in deviation or narrowing of contrast columns over more than one vertebral length. Disk material may completely encircle the spinal cord. Acute type I disk extrusions often are accompanied by spinal cord edema and swelling, and occasionally dural laceration.

The spinal cord may be widened over several spinal cord segments and the myelographic appearance is similar to that of an intramedullary mass, making precise determination of the site of disk extrusion difficult. In some animals disk material is scattered along the spinal canal without obvious mechanical distortion of the spinal cord.

Rarely, in the cervical region, type I disk extrusion may occur laterally or intraforaminally, resulting in neck pain or thoracic limb pain due to nerve root compression. In such cases myelograms may be normal; however, increased density associated with calcified disk material may be visualized intraforaminally on ventral oblique radiographs of the cervical spine. Traumatic disk protrusion is usually associated with narrowing of the intervertebral disk space on radiographs. Other abnormalities such as vertebral fracture, luxation, or instability also may be seen. Myelography is useful in determining the presence or absence of spinal cord compression in such cases, and therefore whether surgical decompression is indicated.

The use of advanced imaging techniques, such as CT or MRI, may aid in the exact localization of intervertebral disc extrusions, particularly in cases of intraforaminal disc extrusion.

Treatment

Type I Disk Extrusion. The appropriate treatment for animals with type I disk extrusion depends on an individual animal's neurological status. Medical treatment directed at decreasing spinal cord edema by means of corticosteroids is indicated only in those animals with an acute onset of neurological deficits, that are examined within eight hours of the injury. The recommended agents and dosages are as described for spinal cord trauma. The use of corticosteroids in dogs with type I disk extrusion has been associated with pancreatitis, gastrointestinal bleeding, or colonic perforations.

Nonsurgical (medical or conservative) treatment is recommended for animals with apparent pain only or animals that have mild neurologic deficits but are ambulatory and have not had previous clinical signs associated with disk disease. These animals should be strictly confined to a small area such as a hospital cage or a quiet place away from other pets for at least 2 weeks, and walked (on a leash or harness) only to urinate and defecate. The objective of confinement is to allow fissures in the anulus fibrosus to heal, thus preventing further extrusion of disk material, and allowing resolution of the inflammatory reaction caused by small amounts of extruded disk material.

Use of analgesics, muscle relaxants, or non-steroidal anti-inflammatory agents, is not recommended in most cases as it is believed that their use encourages animals to exercise and risk further disk extrusion. Very cautious use of analgesics or non-steroidal anti-inflammatory agents occasionally may be indicated. However, strict confinement followed by a period of restricted exercise is imperative. Owners should also be warned that an animal's neurological status may deteriorate owing to extrusion of further disk material despite this treatment and to observe the animal very carefully. If the neurological status worsens, an animal's treatment should be reevaluated immediately. Owners should also be warned that a recurrence of clinical signs is common due to further disk extrusion at the same or a different site, and subsequent episodes may be more severe, especially in the thoracolumbar spine.

Animals with severe cervical pain frequently do not respond to cage rest. These dogs often have large amounts of disk material within the spinal canal, and dogs that do not show improvement after 7 to 10 days of confinement should be evaluated further by means of radiographs and myelography, and ventral cervical decompression should be considered.

Surgical disk fenestration has been recommended as a prophylactic measure to prevent further extrusion of disk material into the spinal canal. Fenestration of the disks most likely to herniate (C2-3 through C6-7 in the cervical spine and T11-12 through L3-4 in the thoracolumbar spine) is recommended

in animals that have had one or more episodes of apparent neck or back pain and have evidence of intervertebral disk disease on radiographs. Various surgical techniques have been described. Fenestration of disks does not remove disk material from the spinal canal and therefore is not recommended as the sole surgical procedure in dogs that have evidence of disk material within the spinal canal and spinal cord compression on radiographs and myelography.

The role of disk fenestration in the management of intervertebral disk disease is controversial. Disk fenestration in the thoracolumbar region is not easily done, and complications such as scoliosis, pneumothorax, and hemorrhage may occur. Disk fenestration in the cervical region is achieved more easily and rarely is associated with such complications. Fenestration does not prevent recurrence of disk extrusion in all animals. The effectiveness of fenestration depends largely on the amount of nucleus pulposus removed. Completion of disk fenestration is recommended at the time of spinal cord decompression.

Animals with neurological deficits such as paresis or paralysis with deep pain perception intact, animals with recurrent bouts of apparent back or neck pain, or animals with apparent back or neck pain (or mild neurological deficits) that are unresponsive to strict confinement, should be evaluated by means of spinal radiographs, CSF analysis, and myelography. Surgical decompression of the spinal cord and removal of disk material from the spinal canal should be considered. Although many dogs with moderate or severe paresis improve neurologically if treated with cage rest, neurologic recovery is often more rapid and more complete in animals following surgical decompression of the spinal cord. In addition, the neurological status of some dogs with type I disk extrusion, especially in the thoracolumbar spine, suddenly worsens over a period of hours or days despite medical treatment. Such deterioration usually results from further disk extrusion that may result in irreversible spinal cord damage and permanent paralysis. This progression of signs always is a risk with medical treatment of animals with thoracolumbar disk disease. Progression is impossible to predict on the basis of history, clinical signs, or radiography. Owners should be made aware of treatment options and offered the opportunity of referral to an appropriate surgical facility when animals are initially presented. Surgical decompression should be done as soon as possible to prevent further spinal cord damage incurred as a result of sustained compression or further extrusion of disk material. In addition, if surgery is delayed 2 to 3 weeks, disk material hardens and becomes adherent to dura mater, and becomes difficult or impossible to remove from the spinal canal.

Prognosis for neurological recovery in animals that retain deep pain perception postsurgically is fair to very good. The major factors that correlate with the degree of neurological improvement seen postsurgically are the animal's neurological status prior to surgery, the rapidity of onset of clinical signs, and the time interval between onset of clinical signs and surgical decompression. Animals that have severe neurological signs, a rapid onset of clinical signs (hours), and a long period of time before surgery generally have a prolonged recovery period and may have varying degrees of permanent neurological deficit.

The incidence of recurrence of clinical signs due to disk extrusion is greater in nonsurgically than surgically treated dogs. One author found that one-third of dogs with type I disk herniation that were treated nonsurgically had a recurrence of clinical signs, and generally showed greater severity of neurological deficits at the time of recurrence. Another author reported a recurrence rate of 40 per cent in nonsurgically treated dogs.

The advantages and disadvantages of various techniques for spinal cord decompression have been discussed. Surgical treatment is not without risks. Anesthesia is necessary, and surgery occasionally results in further spinal cord damage due to surgical manipulation. Nonsurgical treatment should be attempted in animals that are poor anesthesia or surgical candidates or if surgical treatment is not possible financially.

In animals with clinical signs of a complete transverse myelopathy, without deep pain perception for a period of more than 24 hours, the prognosis for return of spinal cord function is poor despite medical or surgical treatment. Some of these animals may improve neurologically if given sufficient time. However, it is a matter of controversy whether surgical treatment increases the probability of improvement or not. In cases in which deep pain perception has been absent for less than 24 hours, the prognosis for return of spinal cord function is poor. However, surgical treatment may increase the likelihood of neurological improvement in this group.

Regardless of whether medical or surgical treatment is instituted, animals that are paretic or paralyzed require intensive nursing care. Neurological improvement may take weeks or months and this requires owner cooperation and enthusiasm regarding care and physical therapy. Manual expression, intermittent catheterization, and /or indwelling catheterization of the bladder are often required to ensure emptying of the bladder. Weekly urinalysis, especially in animals that do not have voluntary control of micturition, is important in monitoring for urinary tract infection. It is also important to keep animals well padded, clean, and dry to prevent formation of pressure sores, and to ensure that caloric and water intake is adequate. Physical therapy does not result in neurologic improvement but helps to prevent disuse muscle atrophy associated with paraplegia or tetraplegia. Physical therapy should not be attempted in animals treated medically for at least the first 2 weeks following onset of signs, as further extrusion of disk material may occur.

Type 11 Disk Protrusion. Treatment with corticosteroids may result in neurological improvement for variable periods of time in animals with type II disk protrusion. However, corticosteroid therapy is not curative. The reason for this improvement is not clear, as intramedullary hemorrhage and edema seen in cases of acute spinal cord injury are not a feature of chronic spinal cord compression. In the thoracolumbar spine, surgical removal of protruded disk material may result in clinical improvement. However, the neurological status of some dogs is worsened permanently despite careful surgical technique. The reasons for this are not known, but increased vascular permeability has been described in the spinal cord associated with release of chronic spinal cord compression and this probably plays a role in this phenomenon. Ventral decompression in the cervical spine allows removal of protruded type II disk material and neurologic improvement may occur over several months; however, some dogs, especially those with moderate to severe neurologic deficits prior to surgery, may manifest temporary or permanent worsening of clinical signs postoperatively.