Intervertebral Disk Disease (1-Jan-1985)

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Intervertebral disk (IVD) disease in the dog is a common clinical disorder manifested by pain, ataxia, paresis, motor paralysis, or sensorimotor paralysis. It occurs most frequently in the chondrodystrophic dogs but also affects the nonchondrodystrophic breeds. The clinical presentation of an animal with an IVD protrusion varies with the site of the lesion, the onset of the problem (acute versus gradual), the dynamic force at which the IVD material compresses the spinal cord, the degree of hypoxia produced in the spinal cord tissue, the mechanical displacement of the spinal cord, and the duration of clinical signs. (24)

The therapeutic measures of choice for IVD disease have long been debated. Most of the various therapeutic regimes advocated are beneficial when used properly and within the realm of their indications. Medical therapy and surgical therapy or the combination thereof seeks to alleviate the pain or neurologic deficits associated with this syndrome. It is only the patient with sensorimotor paralysis and concomitant malacia of the neural tissue that seems beyond the help of veterinary medicine.

History

Charles Bell, a British physician, has been noted as the first to describe IVD protrusion. (28) Early reports of this syndrome described the condition as enchondrosis intervertebralis-a cartilaginous proliferation (endochondroma) originating from the dorsal annulus of the IVD. (2) These “growths” were thought to produce compression of the spinal cord and nerve roots. It was not until the 1940s that the condition was recognized as a prolapse of the nucleus pulposus. (33)

IVD disease was described in the dog by Janson in 1881. (28,46) Dexter, in 1896, described chronic spinal cord compression myelitis due to IVD disease in the dog. (28,33)

From the early to middle 1900's, significant works describing the clinical and radiographic manifestations of IVD disease
were published. In the 1950s numerous reports described the predisposition for IVD disease in chondrodystrophoid dogs.(28)

In 1951, Olsson described the ventral fenestration technique for surgical management of cervical IVD protrusions.(41) The hemilaminectomy (Redding),(51) dorsal laminectomy (Green) (2l) and the pathologic manifestations of IVD protrusion (McGrath)(28,34) were also described in the literature of that year. Hoerlein published clinical data on the successful employment of the hemilaminectomy for treatment of the thoracolumbar IVD protrusions in 1952. (26,28)

Hoerlein, Olsson, Hansen, Funquist, and many others contributed significantly to the literature in the 1950s and 1960s, forming the foundations of our current medical and surgical therapies for IVD protrusion. Numerous publications concerning modification of surgical technique, radiologic diagnosis, pathology, pathophysiology, and incidence of IVD disease have appeared since the 1970s.

**Anatomy**

The IVD consists of two anatomically distinct regions: an outer layer of fibrocartilaginous material arranged in concentric layers (the anulus fibrosus) and an ovoid central region of gelatinous material (the nucleus pulposus). The anulus fibrosus is approximately two times thicker ventrally than dorsally, and its laminar structure is most prominent ventrally and least prominent dorsally (Fig. 62-1). The nucleus pulposus, which develops from the embryonic notochord, is eccentrically located in the dorsal one third of the IVD.(28,54)

The cervical, thoracic, lumbar, and sacral vertebrae collectively incorporate 26 intervertebral disks in the dog.(5) The largest IVD is located between the seventh lumbar and first sacral vertebrae (L7-S1).(5,28,68) An intercapital ligament courses dorsally over the IVDs from the second to the tenth thoracic vertebra.(35) This ligament has been proposed as a major factor in the low incidence of IVD protrusion in the T2-T10 region.(28)

The cranial and caudal borders of the IVD are formed by cartilaginous end-plates that are composed of hyaline cartilage and cover the epiphyses of the vertebral bodies.

**Pathophysiology**

**IVD DEGENERATION**

Biochemically, the IVD consists of proteoglycans, glycoproteins, and both collagenous and noncollagenous proteins. In the immature dog, the composition of the nucleus pulposus is higher in proteoglycans and glycoproteins while the anulus
fibrosus has a higher collagen content (7,68) (Figs. 62-2 through 62-4).

Degeneration of the IVD occurs with aging and results in significant changes in its biochemical structure (Figs. 62-5 and 62-6). In the nucleus pulposus, proteoglycan content decreases and collagen content increases with age. A direct relationship exists between the proteoglycan and interstitial fluid content of the nucleus pulposus. The loss of interstitial fluid content alters the gel consistency of the nucleus pulposus and decreases its ability to absorb shock and dissipate forces evenly over the IVD structure. Proteoglycan and interstitial fluid content also decreases with age in the anulus fibrosus, decreasing its ability to absorb shock. (5,7,28)

IVD degeneration occurs in all breeds of dogs; however, it is observed most frequently in the chondrodystrophoid breeds (dachshund, Pekingese, French bulldog, beagle, basset hound, American cocker spaniel). (5) In the chondrodystrophoids, a chondroid metaplasia or degeneration of the IVD begins between 2 months and 2 years of age. By one year of age, 75% to 100% of all IVDs have undergone degeneration in the chondrodystrophoid dog. Chondroid degeneration occurs rapidly and is frequently followed by mineralization of the IVD. (7,11,28,68)

A fibroid degeneration of IVD occurs in nonchondrodystrophoid dogs; it is a slow, invidious aging process most evident between 8 and 10 years of age. Fibroid degeneration is rarely accompanied by mineralization. (28,54) The influence of genetic, hormonal, autoimmune, and mechanical factors on IVD degeneration have been investigated. Their exact roles in the degenerative process are not yet fully understood. (7,28,69)

FIG. 62-2 Mesenchymal tissue taken from the lumbar area of a canine fetus 30 days postfertilization. At this stage, the notochord chord is present and intact. The spinal cord is developing at the top. The notochord, at the site of the nucleus pulposus, is looped over. The notochord cells are no longer dividing at this stage but are capable of producing matrix. Segments of the vertebral bodies, the nuclei pulposi, and the anuli fibrosi are identified. (Courtesy of Dr. W. H. Riser)

FIG. 62-3 Lumbar spine of a 35-day fetus, 5 days after Figure 62-2. The cavity of the nucleus pulposus is formed. The notochord has disappeared, the anulus fibrosus is more prominent, and the mesenchymal cells of the vertebral bodies have hypertrophied. (Courtesy of Dr. W. H. Riser)

FIG. 62-4 Lumbar spine of a l-week-old Great Dane. The vertebral epiphyses are composed of cartilage, but ossification is beginning. Primary bony trabeculae have been formed, and blood vessels are present where they entered from the periphery of the midvertebral area. The structural components of the IVD, the anulus fibrosus, the nucleus pulposus cartilage lining of the nucleus pulposus cavity, and the notochord remnant cells are all well defined. (Courtesy of Dr. W. H. Riser)

Degenerative changes in the IVD begin at the periphery of the nucleus, proceed centrally, and are usually accompanied by degeneration of the anulus. As the dorsal anulus degenerates, the nucleus pulposus follows a path of least resistance and begins to protrude dorsally (23,28) (Fig. 62-7). Other pathways of IVD protrusion (lateral, ventral) occur but may be less

FIG. 62-5 Lumbar spine of an aged beagle in which the notochord cells have been replaced entirely by chondroid cells. This nuclear material has ruptured the anulus fibrosus fibers and migrated both dorsally into the spinal canal and ventrally, causing the formation of spondolysis and osteophyte bridging of the adjoining vertebral bodies. (Courtesy of Dr. W. H. Riser)

**IVD PROTRUSION**

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Hansen described two types of dorsal IVD protrusions (Fig. 62-8). A Hansen type I IVD protrusion occurs with a total rupture of the dorsal anulus and a massive extrusion of the nucleus pulposus into the spinal canal (Fig. 62-9, A). It is observed most frequently in the chondrodystrophoid breeds and is accompanied by a severe inflammatory response. The inflammatory response often results in fibrinous adhesions between the extruded disk material and the dura. The dynamic force of compression on the spinal cord from a type I IVD protrusion and the resultant hemorrhage, vascular compromise, and inflammation are collectively responsible for the ensuing spinal cord damage and associated neurologic signs. (23,28,54)

A Hansen type II IVD protrusion occurs as a partial rupture or bulging of the nucleus pulposus into the spinal canal. (23) The type II IVD protrusion is more typical of the fibroid degenerative process, is seen more commonly in the nonchondrodystrophoid dog, and has a slower onset than the type I protrusion. (23,28,54)

Type I and type II IVD protrusions occur in chondrodystrophoid and nonchondrodystrophoid dogs. The clinical IVD syndrome is seen more frequently in the relatively young chondrodystrophoid breeds and is accompanied by a severe inflammatory response. The inflammatory response often results in fibrinous adhesions between the extruded disk material and the dura. The dynamic force of compression on the spinal cord from a type I IVD protrusion and the resultant hemorrhage, vascular compromise, and inflammation are collectively responsible for the ensuing spinal cord damage and associated neurologic signs. (23,28,54)

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EFFECTS OF COMPRESSION ON THE SPINAL CORD

Spinal cord compression, as it relates to the IVD syndrome, is divided into two categories: acute and chronic. This division is necessary because the pathogenesis of the acute and chronic compressive processes differs. (65)

The spinal cord can adapt to certain amounts of compression and mechanical displacement. With most IVD protrusions, however, a point is reached at which the spinal cord can no longer compensate; it is at this point that clinical signs develop. (28,65) The severity of the signs is dependent on the dynamic force of the compression, the size of the protrusion, and the location of the lesion. The effects of spinal cord compression are most readily seen in the thoracolumbar area because of the relatively small ratio of spinal canal to spinal cord diameter. In the cervical spine the spinal canal diameter is larger, allowing more room for the spinal cord to compensate for mechanical displacement. Cervical IVD protrusions frequently result in pain rather than the paresis or paralysis that is often associated with thoracolumbar IVD protrusions. (2,28,54)
The pathologic changes associated with acute spinal cord compression are related to the dynamic force of compression, the mechanical displacement of the spinal cord, and the hypoxic changes resulting from mechanical and chemical damage to the spinal cord vasculature. (6,28,42,54) Spinal cord damage from acute compression can range from a slight demyelinization to a total necrosis of the gray and white matter. (6,65)

Tarlov concluded that mechanical deformation rather than spinal cord hypoxia is the primary factor in the pathologic changes associated with acute spinal cord compression. This conclusion was based on experimental acute spinal cord compression and the benefits of surgical decompression. (60) Other studies indicate that hypoxia is the major factor in the pathologic changes associated with acute spinal cord compression. (1,42,52) Severe, acute compression of the spinal cord results in vascular modifications manifested by central necrosis of the gray matter with edema, hemorrhage, and eventual demyelinization in the white matter. The gray matter is the most severely affected area of the spinal cord in the presence of hypoxia. (6,28) This may be due to several factors: (1) Because of its histologic structure, the gray matter is more easily compressed. (2) The dura is least elastic, causing increased intramedullary pressures from hemorrhage and edema to be concentrated centrally. (3) Increased metabolic demands of the gray matter in response to injury are not met by the reduced blood flow. In less severe acute compression, the pathologic changes are not as severe; however, a predisposition for the gray matter still exists. (6)

The hypoxia that results from a reduced spinal cord blood flow may be mediated by one or more factors: direct mechanical injury to the vessels and intravascular coagulation; a massive release of neurotransmitter substances (e.g., norepinephrine, serotonin) with subsequent vasospasm, petechial hemorrhage, and, if the condition progresses, hemorrhagic necrosis of the neural tissue; and progressive venous stasis that results from pressure on the spinal cord and subsequent spinal cord edema, causing further impairment of venous return. (8,42)

The alterations produced by hypoxia can be segmental or can progress to hematomyelia (progressive hemorrhagic myelomalacia)—an ascending or descending morbid softening of the spinal cord resulting from massive hemorrhage within the spinal cord tissue (Fig. 62-9, B). Hematomyelia is rapidly progressive and often terminal in 3 to 10 days. (28,54) Patients experiencing hematomyelia have an anoxic appearance and clinically manifest sensorimotor loss and depressed hindlimb reflexes. This autocatalytic process carries a very grave prognosis, with death resulting from respiratory paralysis following ascending malacia. (2,28,54)

Chronic spinal cord compression is relatively uncommon in the IVD syndrome but does occur with some Hansen type II protrusions. The dynamic force of compression is low in the chronic spinal cord compression, and most of the pathologic changes involve the white matter. (65) Certainly some local hypoxia develops; however, the autocatalytic process associated with acute compression does not occur. It has been demonstrated that leakage of plasma proteins from intramedullary vessels occurs in acute spinal cord compression but does not occur in chronic compression. (28,66)

The initial clinical manifestation of a chronic compressive lesion may consist of pain caused by pressure on the nerve roots and on the spinal cord. As the compression progresses, the pathologic alterations in the white matter can include demyelinization, axonal and myelin necrosis, or massive malacia. These alterations are followed by the infiltration of macrophages to remove the damaged debris. The larger motor fiber tracts are more sensitive to chronic compression than the smaller sensory fibers. (65)

**Diagnosis**

The diagnosis of IVD disease is based on the medical history, physical examination, neurologic examination, and radiographic examination of the animal. (11,28,54) Every effort is made by the clinician to determine the location of the lesion, the extent of the spinal cord damage, and the presence or absence of concomitant physical or neurologic disease processes. A neurologic examination form is useful to gather the necessary information.

**HISTORY**

The history includes all pertinent medical history and a neurologic history. The neurologic history should include the following information: Duration of the problem Rapidity of onset (acute versus chronic) Status of the problem (progressive, static, improved) History of trauma History of spinal problems Previous therapy and response Alterations in bladder or bowel function Vaccination history Alterations in personality Additional information may be necessary in certain cases.

**PHYSICAL EXAMINATION**
The physical examination is essential in every case and should include the vital signs (temperature, heart rate, respiratory rate, mucous membrane color) and an evaluation of all systems. The clinician should be mindful of physical conditions that can contribute to the neurologic signs or that may require further evaluation prior to anesthesia for surgical therapy or spinal radiographs. (28,54)

**NEUROLOGIC EXAMINATION**

The purpose of the neurologic examination is to determine the presence or absence of a neurologic lesion and, when present, to determine its location and the probable extent of damage to the nervous system. The neurologic signs produced by IVD protrusions vary with the location of the protrusion. The clinician must keep in mind the possibility of multiple neurologic lesions when conducting the examination. (28,67)

When spinal cord disease is suspected, at least a cursory examination of the cranial nerves should be conducted; a more detailed examination can be given if any abnormalities are found. The neurologic examination also includes evaluation of the postural reactions (wheelbarrowing, hemistanding/hemiwalking, hopping, conscious proprioception); tendon reflexes (biceps, triceps, cranial tibialis, gastrocnemius, patellar); bladder function; anal reflex; and sensory perception. (11,25,59) Table 62-1 is a summary of the spinal reflexes produced by IVD lesions at various locations. (54) Motor control of the limbs and tail is evaluated also. (11,28) Testing for the panniculus reflex and for hyperpathia is helpful in localizing the level of a spinal lesion in the thoracic and lumbar spine. (11,28)

**RADIOGRAPHY**

Radiographic evaluation of the vertebral column is performed with the animal in a surgical plane of anesthesia. (3,39) This eliminates the problem of motion and facilitates positioning of the animal. (3)

In the lateral position, foam rubber or cotton padding is used to elevate the dependent areas (neck, shoulders, hips) and position the vertebral column parallel to the x-ray table. This eliminates the distortion created when the intervertebral foramina on each side of the vertebrae are not in perfect superimposition. It is helpful to palpate the sternum and vertebral column to ensure that they are in the same horizontal plane and that the patient is not rotated. The forelimbs are pulled cranially and the hindlimbs caudally. The ventrodorsal view is made with the animal on its back in a bilaterally symmetric position without rotation of the trunk. Sand bags or similar equipment may be helpful in positioning. (3,39)

Superior quality diagnostic films using a nonscreen Bucky or detail screen Bucky technique are necessary to demonstrate most IVD lesions. (3) Routine radiographs of animals with suspected cervical IVD disease should include lateral and ventrodorsal views of the entire cervical spine and a survey lateral radiograph of the thoracolumbar region. With suspected thoracolumbar IVD disease, lateral and ventrodorsal views of the thoracolumbar and lower lumbar regions and a survey lateral radiograph of the cervical spine are routine. The thoracolumbar view in the cervical IVD patient and the cervical view of the thoracolumbar IVD patient are indicated because multiple IVD lesions are not infrequent. (39) Single radiographs of the entire spine are avoided since adequate detail cannot be obtained with this method.

Because conventional plain-film radiography does not always adequately demonstrate IVD protrusions, the use of myelographic techniques becomes necessary. The most popular contrast media is the water-soluble compound metrizamide. Cisternal and lumbar punctures can be employed for myelographic procedures. (3,40) Myelography is a valuable and relatively safe tool for differentiating IVD disease from other spinal cord and vertebral diseases (e.g., neoplasia); however, because of potential side-effects its use is reserved for those animals for whom surgery is planned.

**DIFFERENTIAL DIAGNOSES**

The history, physical examination, and neurologic examination are used to localize the lesion, determine its probable extent, and establish a list of differential diagnoses. Several disease entities can produce spinal cord pathology that mimics the neurologic signs associated with IVD disease (Table 62-2). After the location of a spinal cord lesion is determined, the presence or absence of an IVD protrusion can be made with plain film or contrast radiography. (54)
INCIDENCE
The diagnosis of IVD disease is common in small animal practice. In data compiled from 20 colleges of veterinary medicine in the United States and Canada by the Veterinary Medical Data Program (VMDP) during the 5-year period of 1977 to 1981, 7304 cases of IVD disease were reported in 600,630 dogs studied (1.2%). Hoerlein reported 8,117 cases of IVD disease in 356,954 dogs studied during a 10-year period by the VMDP (2.3%).(28) During the 1977 to 1981 period at Purdue University, 645 of 16,816 animals were diagnosed as having IVD disease (3.8%).

AGE AND BREED
Hoerlein reported the highest incidence of IVD disease (73.1%) in dogs 3 to 6 years of age. Dogs over 7 years of age constituted 21.2% of the reported cases. (28) Table 62-3 is a summary of the incidence of IVD disease by breed during a 5 and 1/2-year period (January 1976 June 1981) at the Purdue Small Animal Clinic.

SEX
There does not appear to be a significant difference in the incidence of IVD disease between male and female dogs. During a 5 and 1/2-year period at the Purdue University Small Animal Clinic, IVD disease was diagnosed in 136 intact female dogs, 193 spayed female dogs, 317 intact male dogs, and 37 castrated male dogs. The male/female ratio was 1.0:0.93.

Cervical IVD Syndrome

HISTORY
Patients with cervical IVD protrusions are often presented with a history of sudden crying out in pain, neck guarding, and muscle fasciculations about the head and neck. The signs may occur spontaneously or in response to exercise or may be apparent when the animal is petted about the head. The history may include a decrease in activity, since any sudden movement elicits excruciating pain, or it may indicate intermittent episodes with varying degrees of pain. (2,52)

Cervical IVD protrusion is most common in the chondrodystrophoid breeds. Nonchondrodystrophoid breeds affected with the cervical vertebral instability ("wobbler") syndrome appear to have a higher incidence of cervical IVD protrusions than other nonchondrodystrophoid breeds. (28,54,55)

CLINICAL SIGNS
The clinical signs of cervical IVD protrusion are related to the dynamic force of compression and the mechanical displacement of the spinal cord and cervical nerve roots by the extruded disk material. (41) The signs can include hyperesthesia of the neck and forelimbs, painful spasms of the neck muscles, paresis, ataxia, or quadriplegic Most cervical IVD protrusions, even when massive, are manifested by pain only (Fig. 62-10). Pain is the hallmark of cervical IVD protrusion and may be constant or intermittent. (2,28,54) Most of the pain is of radicular (nerve root) origin; some may be associated with meningeal initiation or "diskogenic" pain. (50)
Neurologic deficits are usually mild when present in cervical IVD disease (e.g., reflex alterations, proprioceptive deficits) and are often associated with progression of the disease. The acute type I protrusion can produce sudden and severe neurologic deficits (e.g., paresis, quadriplegia), can appear clinically indistinguishable from meningitis, or can be manifested by pain only. The type II protrusion has a much slower onset but may have very similar clinical signs with progression of the protrusion (2,28,54)

FIG. 62-10 Severe pain and muscle spasms associated with a cervical IVD protrusion. (Courtesy of Dr. G. Lantz)

RADIOGRAPHIC SIGNS

The radiographic signs of cervical IVD protrusion are narrowing of the IVD space, narrowing of the intervertebral foramen, increased density (“cloudiness”) in the intervertebral foramen, and the presence of a mineralized mass within the spinal canal above the IVD space (Fig. 62-11). One or more of these signs may be present. Myelography is also helpful in delineating the lesion (Fig.62-12). (3,39) Clinically, the C2-3 and C3-4 intervertebral disks have the highest incidence of protrusion. (28)

FIG. 62-11 An IVD protrusion at C4-5, demonstrating a narrowed IVD space and an opacified density within the spinal canal.

FIG. 62-12 A cervical myelogram demonstrating compression of the spinal cord by an IVD protrusion at C3-4.

MEDICAL MANAGEMENT

Neck pain and muscle spasms associated with cervical IVD protrusion are often amenable to conservative therapy (cage rest, anti-inflammatory agents, muscle relaxants). Patients exhibiting neurologic deficits (ataxia, paresis, paralysis) often have large amounts of extruded disk material within the spinal canal and are less responsive to medical management. A high incidence of recurrence exists, and those patients that respond to medical management initially may require surgery at a later date. (2,25) The objective of medical management of cervical IVD disease is to allow the spinal cord/nerve root inflammation to subside and the dorsal anulus to heal.

Dexamethasone is considered the anti-inflammatory drug of choice for the IVD syndrome. (28,54) Other frequently prescribed anti-inflammatory agents are phenylbutazone and prednisolone. (2,28,54) Greene reported that intravenous phenylbutazone was ineffective in reducing spinal cord inflammation and promoting clinical improvement following experimental spinal cord trauma. (20) Pharmacologically the anti-inflammatory effects of prednisolone are seven times less than those of dexamethasone. (22)

The optimum dose of dexamethasone has not been determined; however, some guidelines can be offered. Severe or acute spinal cord compression following a cervical IVD protrusion is treated with 2.0 mg/kg intravenous dosage. This initial therapy is followed by more conservative doses (0.2 mg to 0.4 mg/kg sid or bid) for 2 to 3 days or as needed. More subtle episodes are treated with the conservative dosage initially. The possible side-effects of corticosteroid therapy should be discussed with the client before initiating therapy.

Muscle relaxants have been recommended in cervical IVD disease manifested by severe muscle spasms. (64) Methocarbamol and chlorphenesin carbamate have been used effectively for this purpose. The recommended dosages are as follows:

- Methocarbamol: 10 mg/kg PO as a loading dose and 5 mg to 19 mg/kg PO tid thereafter (32)
- Chlorphenesin carbamate: 50 mg/kg divided into three doses for the first day, followed by 25 mg/kg PO divided into three doses per day thereafter (32)

Cage rest (no physical activity or leash exercise only) is employed in all regimens of medical management. When
corticosteroid therapy is used, cage rest is mandatory, since alleviation of pain may encourage the patient to exercise. Exercise in the presence of clinical IVD disease may result in extrusion of additional disk material and subsequent severe neurologic damage. (28,54) if the owner refuses hospitalization of the patient, confinement without corticosteroid therapy is highly recommended. (54)

**SURGICAL MANAGEMENT** of the cervical IVD syndrome is often warranted. Mild pain and muscle spasms are usually amenable to conservative therapy. The merits of medical versus surgical therapy and the incidence of recurrence should be discussed with the owner. Proper medical management may be of particular importance when there are financial considerations. Surgical candidates should be carefully evaluated through examination procedures and presurgical laboratory profiles. Thorough knowledge of the anatomy and surgical approaches to the cervical vertebrae is essential in performing the described procedures.

The indications for surgical management of the cervical IVD syndrome are persistent pain, muscle spasms, or paresis after prolonged conservative therapy (1 to 2 weeks); marked neurologic deficits (proprioceptive deficits, ataxia, paresis, paralysis); and pain with radiographic evidence of extruded disk material in the cervical spinal canal. Surgical management can consist of fenestration of the cervical disks or decompression of the cervical spinal cord. Fenestration is performed to prevent additional nucleus pulposus from extruding into the spinal canal or as a prophylactic procedure. Decompression is performed to remove IVD material from the spinal canal and to relieve pressure on the spinal cord. (28,54,55)

The dorsal approach procedures are sometimes necessary; however, excessive muscle hemorrhage, increased surgery time, the difficulty of removing disk material from the ventral spinal canal, and prolonged postoperative care make this approach undesirable as a routine procedure. The ventral approach is less traumatic and requires less surgery time. The ventral-slot technique allows direct access to the extruded disk material and can be combined with prophylactic fenestration. The major disadvantage of the ventral-slot technique is the hemorrhage associated with laceration of the venous sinuses. Ventral fenestration is the most commonly employed cervical IVD procedure, but it is not a decompressive procedure. (28)

**VENTRAL FENESTRATION**

Indications for ventral fenestration are mild paresis or pain that is unresponsive to medical therapy. Ventral fenestration does not remove extruded material from the spinal canal. It is of therapeutic value when the clinical signs are mild and limited amounts of disk material are in the spinal canal. (55) it is frequently employed as a prophylactic procedure in conjunction with decompression or fenestration for therapeutic purposes. (28) The owner should be made aware that the animal may continue to experience some pain for 2 to 4 weeks following surgery because of radicular inflammation, meningeal irritation, and mechanical compression of the spinal cord. (55) In some cases severe pain unresponsive to medical therapy will persist for weeks, and a second surgery to decompress the spinal cord is necessary. (28,55) When used for the proper indications, however, ventral fenestration is most often therapeutically effective. (55)

The patient is anesthetized and positioned in perfect dorsal recumbency with the neck hyperextended over a sandbag. The area prepared for surgery extends from the middle of the ventral surface of the mandible to a point at least 2 cm caudal to the manubrium and laterally on each side to an imaginary line drawn from the wings of the atlas to the point of the shoulder. The head is secured to the table with 1-inch adhesive tape, and the forelimbs are pulled caudally to facilitate exposure of the cervical disks. (55) Poor positioning of the patient often results in dissection off the ventral midline and considerable hemorrhage. (10,16,28,29,47,55,66)

A ventral midline incision is made from the base of the larynx to the sternum. (47) The paired sternocleidomastoideus and sternothyroidus muscles are separated with blunt dissection, exposing the trachea. The tracheal blood supply and the recurrent laryngeal nerve are identified and preserved. (28,47,55) Frazier laminectomy retractors are positioned to retract the nearest carotid sheath toward the surgery and the trachea, esophagus, and the opposite carotid sheath away from the surgeon. This exposes the paired longus colli muscles, which lie on the ventral surface of the cervical vertebrae. These muscles attach diagonally to the caudal ventral processes of the cervical vertebrae. (12,28,47,55,66)

Two landmarks for identification of the cervical IVD spaces are located. The cranial landmark is found by palpating the caudal borders of the wings of the atlas and following them to the ventral midline. At this point, a sharp ventral prominence is palpated, which represents the C 1-2 interspace. The first cervical IVD (C2-3) is located by palpating caudally along the ventral midline until the next ventral prominence is encountered. The caudal landmark is the large transverse processes of the sixth cervical vertebra. These processes extend in a ventrolateral direction from the vertebral body and are lateral to the ventral midline. The surgeon should not mistake these transverse processes for the midline processes, an error that can lead to excessive hemorrhage. (12,28,55)

The ventral anuli of the cervical disks are located just caudal to the caudal ventral-cervical processes. The involved disk is located and small curved hemostats are used to separate the longus colli muscle overlying the ventral anulus. The forceps are
first positioned over the center of the ventral anulus and gently pushed downward to the anulus, separating the musculature.

The hemostats are then opened to spread the musculature and expose the white ventral anulus. With the hemostats held in place for retraction of the longus colli, a No. 15 or No. 11 scalpel blade is used to cut a window in the ventral anulus (Fig. 62-13, A). The excised anulus is removed to expose the nucleus pulposus. A small tartar scraper or a 3-0 to 4-0 bone curette is positioned in the disk space to remove the nucleus pulposus. The nucleus is "scooped out" and should be removed as far dorsally as possible (Fig. 62-13, B). The approximate depth of the IVD can be determined from measurements on the lateral cervical radiographs. (28,54) The fenestration device should enter the disk space in a craniodorsal direction to facilitate removal of the maximum amount of nucleus pulposus. (55)

Other cervical disks are often fenestrated for prophylaxis. Routinely, cervical disks C2-3 through C5-6 are fenestrated during the operation. (29,55) The incidence of C6-7 IVD protrusions is low and exposure of this space is sometimes difficult. Therefore, prophylactic fenestration of the C6-7 IVD is unwarranted in most cases. (28)

At the completion of the fenestration, the Frazier laminectomy retractors are removed and the sternohyoideus muscles are apposed with 2-0 or 3-0 absorbable suture material. The subcutaneous tissue and skin are closed in routine manner (26,28,29,41,45,55).

Postoperative care is minimal, consisting of cage rest. Corticosteroids or muscle relaxants are administered postoperatively only if severe pain and muscle guarding persist. (55)

**DECOMPRESSION OF THE CERVICAL SPINAL CORD**

The techniques described for decompression of the cervical spinal cord are the dorsal laminectomy, hemilaminectomy, and the ventral-slot. Indications for decompression are the presence of motor deficits in one or more limbs or severe pain unresponsive to proper medical management and associated with extruded disk material within the spinal canal. (28,55,57)

**VENTRAL-SLOT TECHNIQUE.** Patient positioning and surgical approach for the ventral-slot technique are identical to those described for ventral fenestration. The longus colli muscles are elevated periosteally and retracted laterally along the entire length of one vertebra cranial and one vertebra caudal to the involved IVD. (28,48,57) Gelpi retractors are positioned to retract the dissected musculature. All hemorrhage from the musculature is controlled before continuing the procedure.

![FIG. 62-13 Ventral cervical IVD fenestration.](image)

(A) Curved Kelly forceps are used to dissect and retract the longus colli muscle over the ventral anulus and a No. 11 scalpel blade is used to cut a window in the ventral anulus. (B) The nucleus pulposus is removed using a 3-0 or 4-0 bone curette.

![FIG. 62-14 Ventral cervical slot](image)

A surgical drill is used to perform a ventral cervical decompression. The slot is one half the ventral vertebral body width and one third the length of each bordering vertebra. (A, IVD anulus; B. Spinal cord) (Redrawn alter Shores A: Intervertebral disk syndrome in the dog: Part 11 Cervical disk surgery The Compendium on Continuing Education for the Practicing veterinarian 3, No. 9:805-813, 1981)

After the exposure is completed, the caudal ventral-cervical vertebral process adjacent to the disk is removed with rongeurs. Drilling is begun using a surgical drill equipped with an oblong bur. The slot should be parallel with the long axis of the vertebrae and extend from the caudal one-third of the cranial vertebra to the cranial one-third of the caudal vertebra. Its widths should not exceed one-half of the vertebral body width. (28,55,57) The surgeon begins drilling the white outer cortical bone, encounters cancellous bone next, and finally encounters the white inner cortical bone. (58) The drilling is frequently interrupted throughout the procedure to allow irrigation of the surgical site and evaluation of the slot's depth. (58) The inner cortical bone is very thin and is drilled with caution to avoid damage to the spinal cord. (58) A thin shelf of cortical bone, the endosteum, and the dorsal anulus are elevated and removed from the site with a tartar scraper or similar instrument. This opens the spinal canal, exposing the dorsal longitudinal ligament, the spinal cord, and the extruded disk material (Fig. 62-14). Hemorrhage from the cancellous bone is controlled by packing the area with bone wax. (55)

The extruded disk material is removed from the spinal canal with a small tartar scraper (28) or blunt neurosurgical probe. (55)
Care is taken not to lacerate the large venous sinuses located just lateral to the slotted area. The extruded disk material may adhere to the sinuses, and removal of the material may result in hemorrhage. If hemorrhage occurs, an absorbable gelatin sponge is gently packed into the slot for a short time to aid in hemostasis. If the hemorrhage continues, the patient's head is elevated to relieve any pressure on the jugular veins that might contribute to the hemorrhage. When hemostasis is achieved, a free fat graft or an absorbable gelatin sponge is placed over the slot. The longus colli muscle is apposed over the vertebrae. Prophylactic fenestration of other cervical disks concludes the procedure. (28,48,55,57)

Closure of the surgical site is performed as described for the ventral fenestration technique. Postoperative care consists of cage rest for 3 to 7 days. (28) Corticosteroids are administered during the surgical procedure and may be necessary for 1 or 2 days postoperatively if severe pain and muscle spasms continue. (55)

**DORSAL APPROACH TO THE CERVICAL SPINE (DORSAL LAMINECTOMY AND HEMILAMINECTOMY).**

A dorsal approach to the cervical spine is used in performing a dorsal laminectomy or a hemilaminectomy. This approach requires the dissection of the large mass of dorsal cervical musculature and therefore is more difficult than the ventral approach (14,28,47).

The patient is positioned in a sternal recumbency with a sandbag under the neck for dorsiflexion of the cervical spine. (28) The skin is prepared for aseptic surgery from midcranium to the fourth thoracic vertebra and several inches laterally on each side of the dorsal midline.(55)

The skin incision extends from the external occipital protuberance to the dorsal spinous process of T1. The subcutaneous tissue is incised to expose the platysma muscle and a thin median raphe. The incision continues on the midline through the dorsal cervical musculature until the nuchal ligament is exposed. Several vessels are encountered during the dissection. (47,55) Electrocautery is very helpful in controlling the hemorrhage.

Frazier laminectomy retractors are positioned in the incision to retract the musculature and further expose the nuchal ligament. The cervical dorsal spinal processes are palpated under the nuchal ligament to identify the laminectomy site. (47,55)

The dorsolateral musculature is incised on the near side of the nuchal ligament and periosteal elevation used to bluntly dissect it from the dorsal vertebral arches. Lateral elevation of the muscles is limited by the large vertebral vessels that course through the muscle near the articular processes. The dorsolateral musculature is elevated and retracted to one side for the hemilaminectomy and to both sides for the dorsal laminectomy. The nuchal ligament is retracted to one side with the dorsolateral musculature when performing the dorsal laminectomy (28,46,55).

The hemilaminectomy is begun with a surgical drill, creating an opening in the lateral laminae just above the lesion. The hemilaminectomy is enlarged cranially and caudally with the drill or with rongeurs until adequate decompression is accomplished (usually one to two vertebral lengths). The dorsal limit of the hemilaminectomy is near the dorsal midline. The ventral limit is near the floor of the spinal canal, preserving a small shelf of the laminae above the ventral border of the spinal cord (Fig. 62-15). (28 29 45 55) Disk material cannot be removed from the floor of the spinal canal without some degree of difficulty. Excessive manipulation of the spinal cord is avoided. (45)

The dorsal laminectomy is begun by removal of the dorsal spinous processes over the decompression site with rongeurs. The dorsal laminae are removed with the surgical drill or rongeurs. The laminectomy defect should extend to the most medial

![FIG. 62-15 Hemilaminectomy of cervical vertebrae C3-5. A small shelf of the laminae above the ventral border of the spinal cord is preserved. (A, dorsal laminae; B. caudal articular process of C3; C, vertebral vessels; D, spinal nerve root)](image)

![FIG. 62-16 Dorsal laminectomy of the cervical vertebrae. (A, laminectomy site; B. dura; C, spinal cord; D, articular process; E, spinal nerve root; F. vertebral vessels; G. extruded nucleus pulposus; H. anulus fibrosus) (Redrawn after Shores A: Intervertebral disk syndrome in the dog: Part II. Cervical disk surgery The Compendium on Continuing Education for the Practicing Veterinarian 3:805-813, 1981)](image)

The dorsal laminectomy is begun by removal of the dorsal spinous processes over the decompression site with rongeurs. The dorsal laminae are removed with the surgical drill or rongeurs. The laminectomy defect should extend to the most medial
aspect of the dorsal articular processes and for a distance of two to three vertebral body lengths (Fig. 62-16). Removal of disk material from the ventral spinal canal is difficult without excessive spinal cord manipulation and is generally not attempted. (14,28,55)

The incision is closed with 1-0 or 2-0 absorbable suture material. The external fascia of the deep dorsal cervical muscles is sutured to the nuchal ligament, and the medium raphe is closed, followed by the subcutaneous and skin layers. (47) Patients that are ambulatory prior to a dorsal decompressive procedure may be recumbent for several days postoperatively. (55)

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**Thoracolumbar IVD Syndrome**

**HISTORY**

The history described for a dog with thoracolumbar IVD protrusion often varies with the severity of the lesion. Any history of trauma should be noted. Dogs with acute type I protrusions may experience an episode of severe pain prior to sudden hindlimb paresis or paralysis. Less severe lesions may be described as a gradual onset. (2,28,54)

Type II protrusions may be described as a painful episode that has responded to analgesics (i.e., aspirin) or rest. The owners will frequently correlate an event such as climbing stairs, jumping onto furniture, or standing on the hindlimbs with the problem. A reluctance to move is also reported. (28)

**Clinical Signs**

An early sign of a thoracolumbar IVD protrusion is severe pain. The pain may be manifested by vocalization, reluctance to move, or aggressive behavior. In the type I thoracolumbar IVD protrusion, the acute pain is often quickly followed by motor deficits (paresis, paralysis) and analgesia distal to the level of spinal cord trauma. (2,28,54) The neurologic deficits are assessed by examination and are influenced by the site of the lesion and its severity (dynamic force of compression, vascular compromise, mechanical displacement of the spinal cord). (5,28,54)

The initial sign of the type II thoracolumbar IVD protrusion is pain. If the protrusion and its pathophysiological effects progress, varying degrees of ataxia, paresis, or paralysis and alterations in bladder function can occur. Type I and Type II thoracolumbar protrusions can progress to sensory and motor paralysis of the hindlimbs. (54) Figure 62-17 is representative of the progression of signs with thoracolumbar IVD protrusions.

Severe spinal cord damage can progress to hematomyelia. Patients exhibiting hematomyelia have an anxious appearance and dilated pupils; clinically they manifest sensorimotor paralysis with depressed hindlimb reflexes. A grave prognosis accompanies this syndrome (28,54)

The presence or absence of sensory function ("deep pain") in the hindlimbs is the most important prognostic sign. Sensory paralysis for over 24 hours warrants a very poor prognosis. (2,19,28)

The common site of thoracolumbar disk protrusion in the dog is at T11-12. (28) Gage reported a 71.3% incidence in T11-12 to L2-3 area. (17)

**RADIOGRAPHIC SIGNS**

Evaluation of the radiographic signs associated with IVD disease is an important step in the diagnostic process. The clinician
should coordinate clinical signs with the radiographs. Radiographic evidence of a thoracolumbar IVD protrusion (Fig. 62-18) may include the following: a narrowing of the IVD space, a narrowing or cloudiness of the intervertebral foramen, abnormal spacing of the articular processes, or the presence of a mineralized mass above the IVD space (a highly significant finding). (3,28,39)

![FIG. 62-18 An IVD protrusion at L3-4. The mineralized disks at L2-3 and L5-6 were not involved clinically.]

FIG. 62-18 An IVD protrusion at L3-4. The mineralized disks at L2-3 and L5-6 were not involved clinically.

![FIG. 62-19 A myelogram demonstrating an IVD protrusion at L3-4.]

FIG. 62-19 A myelogram demonstrating an IVD protrusion at L3-4.

It is an absolute necessity that the neurologic findings be coordinated with the radiographic findings. Mineralization of the nucleus pulposus with or without anular involvement is a common finding in the asymptomatic chondrodystrophoid dog. Lesions of this type are often not considered clinically significant unless there is other radiographic, myelographic, or clinical evidence of spinal cord compression at that site. (28)

Myelography is occasionally necessary to demonstrate spinal cord compression (Fig. 62-19). The lumbar technique is most often used to demonstrate thoracolumbar IVD protrusions with myelography. (3,28,39)

**MEDICAL MANAGEMENT**

Indications for medical management of the thoracolumbar IVD syndrome are clinical signs of ataxia, paresis with pain, or pain only and no previous history of disk disease. Recurrent episodes of thoracolumbar disk disease may warrant strong consideration of surgical therapy. (28,54) Radiographic confirmation of a disk protrusion should be made whenever possible. When surgery is prohibited by financial considerations or severe systemic disease (e.g., congestive heart failure), medical management is indicated. (54)

Medical management has been shown to be as ineffective as surgical therapy in the majority of patients with sensorimotor paralysis for more than 24 hours with signs of hematomyelia. (28) However, some clients do not consider euthanasia as an immediate alternative in these cases and may request some form of therapy.

Proper medical therapy for the thoracolumbar IVD patient includes cage rest, corticosteroid therapy, muscle relaxants if indicated, and frequent observation for deterioration of neurologic signs. (28,54,55) Hospitalization for 1 to 2 weeks is often required and is mandatory if corticosteroid therapy is used. The bladder is expressed two or three times daily in the incontinent patient. Frequent evaluation for cystitis is made. (54) Clean soft padding (fleece pads or foam rubber) is maintained in the cage to minimize the potential for decubital ulcer formation. (28,54)

Client education is an important component of the medical management regime. The client should be informed of the severity of the disease and of the fact that the signs may suddenly become progressively worse in which case surgical therapy is indicated. (54) Recurrent episodes are frequent and are commonly more severe than the previous one (28,54)

**SURGICAL MANAGEMENT**

Indications for surgical management are pain or paresis unresponsive to medical therapy, recurrent or progressive signs of thoracolumbar IVD disease, paraplegia without sensory paralysis, and sensory and motor paralysis for less than 24 hours. (54) Every surgical candidate must have radiographic evidence of an IVD protrusion that coincides with the neurologic findings. (28,54)

The surgical procedure of choice in thoracolumbar IVD disease is based on the condition of the patient and the surgeon's experience, ability, and preference. (49,54) Surgical procedures described for thoracolumbar IVD disease are the lateral, (13,53) dorsolateral, (28) and ventral fenestrations; (4) the dorsal laminectomy, (15,21,63) and the hemilaminectomy. (18,28,51)

Fenestration is performed as a prophylaxis in combination with a decompressive procedure or following successful medical management of an IVD protrusion. Therapeutically, fenestrating is performed on dogs exhibiting only back pain or mild
paresis associated with a Hansen type II IVD protrusion.

Decompression of the thoracolumbar spine is indicated when extrusion of IVD material into the spinal canal produces severe ataxia, paresis, or paralysis. The severely paretic or paralyzed dog must be properly managed medically and surgically within the first 24 hours to afford optimal results. Delay in proper management may result in a prolonged recovery period or permanent sensorimotor paralysis.

The hemilaminectomy procedure provides good decompression, allows access to the ventral spinal canal for removal of the extruded IVD material, and is easily combined with prophylactic fenestration. The dorsal laminectomy provides no additional advantages and requires more muscle dissection.

FENESTRATION

IVD fenestration is defined as the removal of the nucleus pulposus by perforation and curettage of the IVD space. (28,56) Fenestration is employed in two ways: as a method of surgical therapy and prophylaxis in animals with minimal neurologic deficits (mild ataxia, loss of conscious proprioception) and/or pain associated with a type II thoracolumbar IVD protrusion; and as a prophylactic procedure in conjunction with a decompressive procedure in animals with severe neurologic deficits (severe paresis, paralysis) exhibiting radiographic evidence of several degenerated IVDs in the thoracolumbar spine. IVDs T11-12 through L3-4 are routinely fenestrated in these procedures. (56) The T10-11 IVD is not a common site of disk protrusion because of the intercapital ligament and is not routinely fenestrated. (28,35)

VENTRAL FENESTRATION

The patient is positioned in right lateral recumbency and a paracostal incision is made. The abdominal cavity is entered, and the left kidney and peritoneum are reflected ventrally. Frazier laminectomy retractors are positioned in the incision.

Exposed abdominal viscera are packed off with moistened laparotomy pads. The iliopsoas muscle is elevated, and the short transverse process of L1 is identified for orientation. The L1-2 through L5-6 IVDs are fenestrated through this incision (Fig. 62-20). Exposure of the IVD space is made by digitally depressing the aorta and sympathetic nerve trunk. The ventral anulus is incised with a No. 11 or No. 15 scalpel blade and the nucleus is removed with a small tartar scraper. (4,33)

The abdominal musculature is closed with 2-0 absorbable sutures, and the skin incision is moved cranially to the tenth intercostal space. A thoracotomy incision is made and the retractors positioned. Ventilatory assistance is required during this phase of the operation. IVDs T9-10 through T13-L1 are located and dissected free of pleura, avoiding the sympathetic trunk, aorta, and intercostal vessels (Fig. 62-21). Fenestration is performed in the manner described for the lumbar disks. The thoracotomy is closed in a routine fashion. (4)

This procedure offers the advantages of minimal hemorrhage and avoidance of the spinal nerve roots in the dissection. The disadvantages are the necessity of a thoracotomy and ventilatory assistance; dissection is required near major vessels, nerve trunks, and organs; and decompression of the spinal cord cannot be accomplished with this approach. (27,56)

LATERAL FENESTRATION

Lateral fenestration of the thoracolumbar IVDs is performed with the dog in a lateral recumbency; (13,51) a left lateral recumbency for the left-handed surgeon, a right lateral recumbency for the right-handed surgeon. (13) An area is prepared for
aseptic surgery from the seventh thoracic vertebra to the greater trochanter and from the dorsal spinous processes to a few inches below the level of the lumbar transverse processes (53,56)

A skin incision is made along a line from a dorsal spinous process of T9 to the ventral aspect of the wing of the ilium. The incision is continued through the subcutaneous fat, the lumbar fascia, and through a second layer of fat overlying the longissimus dorsi and iliocostalis muscles. The transverse processes of the lumbar vertebrae are palpated through these muscles, locating the short transverse process of L 1 for orientation. The L1 process is located medial to the dorsal curvature of the thirteenth rib. (13,53)

The transverse process of the fourth lumbar vertebra is identified and palpated with the index finger. The musculature attaching to the dorsal aspect of the L4 transverse process is separated with curved Kelley hemostatic forceps. An Adson periosteal elevator is positioned on the lateral edge of the dorsal aspect of the process and the musculature is elevated to the level of the vertebral body. These muscles are retracted dorsally with a deep wide-blade retractor while a second retractor is used to reflect the musculature ventral to the transverse process (Fig. 62-22). (53,56)

The lumbar IVDs are located just cranial and slightly ventral to the junction of the transverse process and the vertebral body. The loose fascia over the lateral anulus is elevated and reflected cranially, preserving the blood vessels and nerves in the immediate area (Fig. 62-23). Once the IVD is positively identified, a rectangular window is cut in the lateral anulus with a No. 11 scalpel blade. The dorsal border of the window does not extend above the transverse process-vertebral body junction. Incising dorsal to this area may invade the spinal canal and damage the spinal cord. (53,56)

The excised portion of the lateral anulus is removed, and the nucleus is fenestrated with a tartar scraper or a 14- or 16-gauge spinal needle. The fenestration device is never angled dorsally to avoid entering the spinal canal.(53)

The IVDs at L2-3, L1-2, and T13-L1 are fenestrated in the same manner. Thoracic IVDs (T10-11, T11-12, T12-13) are fenestrated in a similar manner; however, the approach is more difficult. The iliocostalis lumborum muscle is elevated from the craniodorsal aspect of the proximal one-third of the rib and is then retracted craniodorsally. The remaining musculature on the cranial surface of the proximal rib is reflected ventrally, exposing the fibrous tissue overlying the lateral anulus. The thoracic pleura is very near the ventral aspect of the dissection. Rupture of this pleura will result in a pneumothorax. (53,56)

The dorsal limit for excision of the lateral anulus and fenestration of the thoracic IVDs is the arch of the rib. Dissection or fenestration above this limit will enter the spinal canal. Invasion of the thoracic disks is limited with this approach. (53,56)

The incision is closed with continuous 2-0 absorbable sutures through the lumbar fascial layer and routine subcutaneous tissue and skin closure. (53) Postoperative care should include 2 days of cage rest.

The lateral approach to the thoracolumbar IVDs requires minimal muscle dissection, produces minimal hemorrhage, and can be combined with a hemilaminectomy for decompression. (27,53,56) It also requires an assistant to provide retraction and the availability of a good light source that can be adjusted by the surgeon during the procedure.(51) Exposure may be difficult in heavily muscled animals and is limited for the thoracic disks. The surgical dissection is near the spinal nerve roots and vessels and for this reason should not be performed below the L3-4 IVD space. (53,56)

DORSOLATERAL FENESTRATION.

The patient is placed in sternal recumbency for the dorsolateral approach. A sandbag or rolled towel can be placed under the abdomen to arch the spine and facilitate surgical exposure. (28) The area prepared for the surgery extends from the seventh
thoracic vertebra to the lumbosacral junction and 3 to 4 inches below the dorsal spinous processes on each side.

The skin incision is made on the dorsal midline from T9 to L6. The subcutaneous tissues and fat are incised next and reflected toward the surgeon to expose the thoracolumbar fascia. (28,56) A scalloped incision is made through the fascia, beginning on the dorsal midline between the ninth and tenth thoracic vertebrae, continuing around the near side of the dorsal spinous process, and returning to the midline between each vertebra. The incision is continued to include the fifth lumbar vertebra. (56)

The thoracolumbar musculature is dissected from the lateral aspect of the vertebrae on the side nearest the surgeon. The dissection begins at L5 and continues cranially to T10. (28,56) A scalpel handle or periosteal elevator is used to reflect the musculature from the dorsal spinous processes. Tendinous attachments to the dorsal spinous processes are severed with scissors. (18,28)

A second level of muscle is elevated from the articular processes. The periosteal elevation begins at the caudal aspect of the articular process and continues around the process to the cranial aspect. The tendinous attachments are cut close to the process to minimize hemorrhage. (18) When this dissection is completed from L5 to T10, Gelpi retractors are placed at each end of the incision to increase exposure. (56)

The IVDs are exposed by elevating the spinal musculature from the dorsal aspect of the lumbar transverse processes and the cranial surface of the proximal one-third of the ribs. The spinal nerve and vessels are located within the muscle that attaches to the accessory process of each vertebra. The muscle attachments, nerve, and vessels are retracted cranially to visualize the lateral anulus. The loose fascia is dissected from the lateral surface of the anulus, and a sharp tartar scraper or a 14 to 16-gauge needle is used to penetrate the anulus. (28) The nucleus is removed with the tartar scraper using a downward and outward movement, or with the 14to 16-gauge needle or small burette. Fenestration above the level of the transverse process-vertebral body junction or the head of the rib will enter the spinal canal. Fenestration through or below the ventral anulus may result in severe hemorrhage if the aorta is punctured. A pneumothorax can result from dissection performed too far ventrally in the thoracic area. (28)

The incision is closed in three layers. The thoracolumbar fascia is closed with 1-0 or 2-0 absorbable sutures in an interrupted fashion. The thoracolumbar muscle should be completely covered with the fascia. The subcutaneous layer is closed with 3-0 absorbable suture, and the skin is closed in a routine manner.

An advantage of the dorsolateral fenestration is that it can easily be combined with a hemilaminectomy when decompression is necessary. The disadvantages of this procedure include the muscle dissection required, the hemorrhage that can result from the muscle dissection, and the necessity of dissection near the spinal nerves and vessels. (27,56)

**DECOMPRESSION**

Decompression of the thoracolumbar spinal cord is defined as the removal of the dorsal or lateral components of the vertebral arch to relieve pressure on the spinal cord. (28,56) The decompressive procedures described for the thoracolumbar spine are the hemilaminectomy and the dorsal laminectomy. Decompression of the spinal cord segment or segments involved in a thoracolumbar IVD protrusion can be combined with prophylactic fenestration of the thoracolumbar disks.

**HEMILAMINECTOMY.**

A dorsolateral (18,28) or a lateral (8) approach to the thoracolumbar spine is used for the hemilaminectomy procedure. The dorsolateral approach provides the best exposure (28,56)

After completing the muscle dissection, the short transverse process of L1 and the thirteenth rib are located for orientation (Fig. 62-24). The site of the suspected IVD protrusion is identified and the hemilaminectomy begun by removing the articular processes directly over the involved IVD with a bone rongeur or surgical drill (Fig. 62-25, A) (18,28,56)

![FIG. 62-24 Anatomy of the thoracic and lumbar vertebrae. (A, dorsal spinous process; B, articular process; C, accessory process; D, transverse (lateral) process of lumbar vertebrae; E, IVD; F, articulation of 13th rib)](image)

The spinal canal is entered by one of three methods:

- The dorsal spinous process of the vertebra just cranial to the IVD is clamped with Backhaus towel forceps and gently
elevated by an assistant. This increases the space between the vertebrae at their articulation. A 3-mm Lampert rongeur is used to widen the space and expose the spinal cord (Fig. 62-25, B and C). (28,56) This method is used most effectively in dogs weighing less than 20 pounds. (28)

- A 5/16 inch Michele trephine is placed over the articular space and used to remove a circular core of the lateral lamina, exposing the spinal cord. (28)
- A surgical drill is used to create the hemilaminectomy. (28,58) This is the preferred method in larger dogs.

The hemilaminectomy should be at least one vertebral body length cranial and caudal to the affected IVD (Fig. 62-25, D). The final length of the hemilaminectomy defect is governed by the appearance of the spinal cord and adjacent tissue within the canal. The length is extended until "normal" appearing tissue is encountered (presence of epidural fat; absence of IVD material or cord swelling). The Lampert rongeurs or the surgical drill can be used to lengthen the hemilaminectomy when necessary. (19) The opening extends ventrally to the floor of the spinal canal.

After creating the hemilaminectomy defect, a thin, tough layer of endosteum may be encountered. A dural hook or similar instrument is used to penetrate this layer, allowing visualization of the spinal canal. The site of the IVD protrusion often contains necrotic epidural fat. This necrotic material is often associated with the extruded IVD material.

The extruded IVD material is removed at the hemilaminectomy site with a small, curved blunt probe (e.g., ophthalmic strabismus hook) or tartar scraper. The probe is carefully passed under and above the spinal cord to dislodge the extruded material. (56) This portion of the surgery is performed with extreme care to avoid damage to the spinal cord. If the IVD material is hardened or adhered to the dura, it may be especially difficult to dislodge. The IVD material is often adhered to the ventral venous sinuses, and its removal results in hemorrhage from these vessels. Application of an absorbable gelatin sponge over the hemilaminectomy site for a few minutes will often control the hemorrhage. (28,56)

The hemilaminectomy exposes only one side of the spinal cord, and therefore disk material on the contralateral side may be inaccessible. A bilateral hemilaminectomy can be performed in such instances if the surgeon deems it necessary. (59)

The majority of the extruded IVD material is removed with the small probe. The remaining small amounts of material are removed through irrigation with Ringer's solution and careful suctioning. The suction tip should never contact the spinal cord. (28,56)

A durotomy can be performed to allow direct visualization of the spinal cord. This procedure is reserved for severe spinal cord edema or for diagnostic purposes when malacia of the spinal cord is suspected. (28) The durotomy does not provide significant additional decompression in the majority of cases. (44) The durotomy is performed by tenting the dura with small tissue forceps (microsurgical, ophthalmic) or a 5-0 silk suture and incising the dura with a No. 11 or No. 12 scalpel blade. The cutting edge of the scalpel blade is directed upward to protect the spinal cord. (19,28)

Several applications of irrigation solution are applied to the decompression site to remove any remaining blood clots, tissue debris, or small bone fragments. Complete hemostasis is achieved before closure. The exposed spinal cord is protected with a layer of sublumbar fat or absorbable gelatin sponge. (28,49) The incision is closed by the methods described previously.

Prophylactic fenestrations of IVDs T11-12 through L3-4 are routinely performed in association with the hemilaminectomy. (28) Neither the effects nor the benefits of prophylactic fenestration have been demonstrated. In the older, nonchondrodystrophic dog, prophylactic fenestration may be an unnecessary procedure that lengthens the total surgical time. In the chondrodystrophic dog, however, IVD degeneration is a systemic problem, and therefore prophylactic fenestration warrants strong consideration.

The hemilaminectomy provides good decompression and visualization of the spinal cord and nerve roots, it allows removal of IVD material from the spinal canal without excessive spinal cord manipulation, and, when necessary, a durotomy can be
performed with the exposure provided. This procedure is easily combined with prophylactic fenestration from a dorsolateral or lateral approach. (27,56)

Disadvantages of the hemilaminectomy are the muscle dissection necessary with the dorsolateral approach, the difficulty in exposing the thoracic vertebrae, and the required dissection near the spinal nerve roots and vessels. (27, 56)

**DORSAL LAMINECTOMY.** Dorsal laminectomies are most often performed using the Funquist B method or the modified dorsal lateral laminectomy method. The two procedures differ in the width of the dorsal laminae removed (28,63)

The dog is positioned in a sternal recumbency. The area is prepared for surgery in the same manner as that described for the hemilaminectomy. The skin incision is made on the dorsal midline for a distance of two vertebrae cranial and caudal to the involved IVD space. The thoracolumbar muscles are elevated from both sides of the vertebra to the level of the accessory processes in a manner similar to that described for the hemilaminectomy. Gelpi retractors are used to improve the exposures

The dorsal spinous processes of one vertebra cranial and one vertebra caudal to the suspected IVD protrusion are removed with bone rongeurs. The processes are removed with the cutting action of the rongeurs in a manner that does not exert torsional forces on the vertebral bodies. A surgical drill is used to create the laminectomy. The dorsal laminae are very thin in comparison with the lateral laminae, and are easily penetrated with the drill bur. (63)

The modified dorsal laminectomy is created by removing the dorsal laminae to the junction of the cranial and caudal articular processes, leaving the cranial articular process intact (Fig. 62-26, A). (25,28,63) The Funquist B dorsal laminectomy is completed in a similar fashion, but its width stops short of the articular processes (Fig. 62-26, B). (28) The decompression is continued cranially and caudally until normal-appearing epidural fat is encountered. (63) Extension of the laminectomy is performed with the surgical drill or rongeurs.

In most instances, Funquist B dorsal laminectomy is not wide enough to allow removal of the extruded IVD material. A small blunt probe, irrigation, and suction are used to remove IVD material from the modified dorsal laminectomy site. It may be necessary to place 5-0 silk sutures transversely to roll the spinal cord to one side for removal of IVD material from the ventral spinal canal. (63)

Cicatricial compression of the spinal cord and cosmetic defects along the dorsal midline can occur following a dorsal laminectomy. To prevent these complications, 20- to 22-gauge orthopaedic wire can be used to span the defect. The wire is looped around the dorsal spinous processes cranial and caudal to the defect in a figure-eight fashion. The wire is moderately tightened and the thoracolumbar fascia is sutured over the wire. (49) The remainder of the thoracolumbar fascia is closed over the dorsal processes with 1-0 to 2-0 absorbable suture. The subcutaneous tissue and skin are closed in routine fashion. (63)

Some descriptions of the dorsal laminectomy have advocated a durotomy as a routine part of the procedure. (15,63) Studies of acute spinal cord trauma have not shown any significant additional decompression to be obtained with a durotomy. (44)

Selective regional spinal cord hypothermia has also been advocated as a component of the dorsal laminectomy procedure. A lactated Ringer's slush is applied directly to the spinal cord tissue to produce the hypothermia. (63) This procedure has been shown to be effective only in acute spinal cord trauma and when it is continued for a minimum of 3 hours. (51) The time factor alone makes this procedure prohibitive. (56)

The dorsal laminectomy provides good visualization and good decompression of the spinal cord. The exposure necessary for performing a durotomy or myelotomy is also provided. It can be combined with prophylactic fenestration of the disks; however, additional muscle dissection is required. The dorsal laminectomy does require considerable muscle dissection and may result in a cosmetic defect or cicatricial compression unless additional measures are taken. The extruded IVD material cannot be removed from the floor of the spinal canal with the Funquist B procedure, and removal of the material requires manipulation of the spinal cord when using the modified procedure. (27,56)
IVD Disease in Cats

The incidence of clinical IVD protrusions in the cat is low. (28) I have seen one such case (Fig. 62-27). Hoerlein reports two cases. (28) Butler, King, and Smith have published the most significant amount of data on feline IVD disease. (9,30,31) Their studies have shown that IVD protrusions occur frequently in the adult cat; however, the incidence of clinical signs associated with the protrusions is extremely low. (28,31) Most of the clinical IVD protrusions occurred as a result of direct trauma, and the protrusions are similar to those associated with nonchondrodystrophoid dogs. (9,28,30)

The incidence of IVD protrusions is higher in the older cat. Butler, King, and Smith report the highest incidence of partial ruptures at 6 to 14 years of age and of complete ruptures at 15 to 20 years of age. (31) The cervical spine has the highest incidence of IVD protrusions in the cat, followed by the midlumbar spine. (39) The reason for the extremely low incidence of clinical signs associated with IVD protrusion in the cat is unknown and warrants further research.

Clinical signs of spinal cord compression in the cat are more frequently associated with epidural lymphosarcoma than with IVD protrusions. (28) An exploration of the spinal canal may be necessary to make an accurate diagnosis. A histologic examination should be performed on any material removed from the spinal canal during surgery for an IVD protrusion in the cat.

Postoperative Management/Physical Therapy for the Recumbent Animal

The goal of postoperative therapy is overall patient comfort and speedy recovery. The major postoperative considerations are diet, exercise, fecal and urinary elimination, and surgical wound management. Most of these concerns are also valid for the medically managed IVD patient. (2,28,54)

The recumbent animal or the animal experiencing pain should be handled carefully and affectionately while in the hospital. All personnel should show interest and compassion for the patient. Frequently one member of the hospital staff can establish a good rapport with the patient, promoting a good attitude in the animal that encourages the desire to recover. (28,37,54)

It is important that the animal remain clean. Dermatitis, orchitis, and decubital ulcers develop rapidly if the patient is allowed to sit in feces or urine. The cage should be padded with clean synthetic fleece blankets or foam rubber pads. (28,54)

The occurrence of corticosteroid-induced enteropathies has been reported as a serious postoperative complication. This syndrome is encountered most frequently in the thoracolumbar surgical patient receiving corticosteroid therapy for more than 3 days. Severe diarrhea, melena, gastric perforations, and colonic perforations have all been described to be associated with this syndrome. (62) Oral antacid or type 2 histamine antagonists have been advocated as prophylactic agents. (28,62) As a guideline for postoperative corticosteroid therapy, 0.4 mg dexamethasone is administered bid on the first preoperative day and 0.2 mg bid on the second postoperative day. Corticosteroid therapy is discontinued as of the third postoperative day.

Aggressive therapy is necessary when urinary incontinence is present. Total evacuation of the bladder either by catheterization or manual expression is performed as often as necessary, usually two to three times daily. The development of urinary tract infections and a delay in the return of urinary function are enhanced by allowing the bladder to become greatly distended. Prophylactic or therapeutic antibiotic therapy is indicated in the incontinent patient. (2,28,43,54) Serial urinalyses are performed to monitor the patient's progress. Once the patient regains full control of urinary function, hospitalization is usually not required. Additional physical therapy can be performed at home.

Physical therapy is begun on the third postoperative day. (54) The objective of physical therapy in the recumbent animal is to promote return of function by strengthening the limb muscles and encouraging their use. Supervised exercises on grass or non-stick indoor surfaces are conducted twice daily and may include standing with support, massage of the limbs, extension and fixation of the limbs, and walking while supported by a towel under the abdomen. (28)
Swimming and whirlpooling are excellent methods of physical therapy. The warm water encourages circulation to the limbs and contributes to removal of any feces or urine present on the skin. (28) These exercises encourage walking movements of the limbs without the added weight required to support the body. (36) Strict supervision of the animal is necessary during hydrotherapy. (28,36) Hydrotherapy sessions are strenuous for the patient and should not extend past the animal's capabilities (usually 10-20 minutes twice daily). (36) Iodinated whirlpool concentrates can be added to the water to promote overall skin care and to combat any contamination of the tub by the patient.

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