

# Canine Intervertebral Disk Disease

Prepared for

**The Dachshund Club of America, Inc.**

by

**Patricia J. Luttgen, DVM, MS**  
Diplomate, American College of  
Veterinary Internal Medicine,  
Specialty of Neurology  
Denver, Colorado

Copyright 1993

(This is primarily a "text only" version. A few sketches have been included to aid in understanding.)

## Comparison of Canine and Human Intervertebral Disk Disease

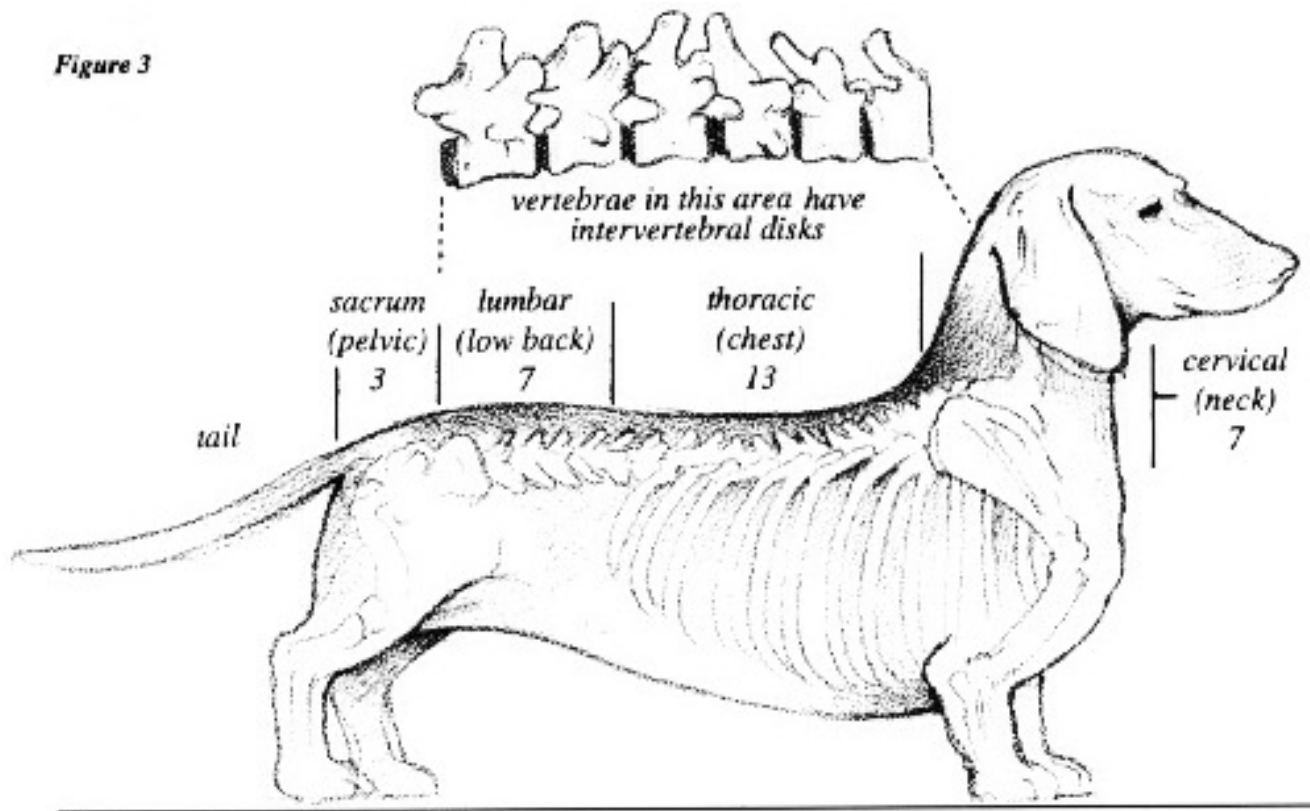
Intervertebral disk disease is a major neurologic problem affecting both canines and humans. Both species can be affected with cervical (neck) disk disease with similarities in the symptoms and outcome. However, in the thoracolumbar (mid-back) area, unique species differences alter the symptoms and outcome of canine versus human disk disease. The major neuroanatomic difference is in which vertebra the end of the spinal cord lies. In humans, the end of the spinal cord lies approximately inside the second lumbar (L2) vertebra (mid-back). Nerves exiting the spinal cord then descend inside the remaining lumbar and sacral (pelvis) vertebral segments. In comparison, the spinal cord in dogs ends at approximately the sixth lumbar (L6) vertebra (low-back area) and nerves descend through the last lumbar, sacral and coccygeal (tail) vertebral segments.

Why is this knowledge so important in determining the differences we see in canine versus human disk disease? To answer this we must first consider the postural differences between humans and dogs and the directions from which forces are applied to the vertebrae and intervertebral disks. Humans walk upright with most jarring forces being transferred straight up the spinal column from the legs. The lumbar vertebra are "first in line" to absorb and dissipate those forces. They also are not "fixed" by an attached rib cage and therefore absorb major twisting forces (axial rotation) as well. Consequently the lumbar intervertebral disks are at highest risk for injury and possible herniation in humans. When disk herniation occurs, the herniated material presses on nerve roots and not the spinal cord itself resulting in a great deal of pain but seldom profound leg paralysis. In comparison, dogs walk on all four limbs with jarring forces normally applied at a right angle to the spinal column. However, when dogs jump down onto their front limbs, the major line of force redirects down the length of the spinal column causing greater end on compression of individual disks and an increased likelihood of herniation. Additional twisting forces are greatest where the immobile thoracic spine meets the mobile lumbar spine. As a result, the most common site of disk herniation in the back of dogs is this thoracolumbar junction where the spinal cord is present and is secondarily compressed by herniated disk material. Thus the clinical presentation of thoracolumbar disk herniation in dogs can be far worse than just shooting pains down the legs. It is common for dogs to show profound paralysis of their hind limbs from the resulting spinal cord damage.

## Canine Anatomy

The spinal column is made up of four major vertebral regions: cervical (neck), thoracic (chest), lumbar (low back) and sacral (pelvic). Dogs have seven cervical, thirteen thoracic, seven lumbar and three sacral vertebrae. There are also variable numbers of coccygeal or tail vertebrae. Intervertebral disks are located between the vertebral bodies starting at the second and third cervical vertebrae (C2-3) and extending to the seventh lumbar and first sacral vertebrae (L7-S1). The three sacral vertebrae are fused and therefore do not have disks. Intervertebral disks are present between the coccygeal vertebra as well, but are of little clinical significance.

Figure 3



The **vertebrae** come together at three main points: the intervertebral disk between the end plates of the vertebral bodies, and two articular facets. The ends of the vertebral bodies are covered by thin cartilaginous plates. The fibrous portion of the disk connects the vertebral bodies by attaching to these cartilaginous plates as well as directly to the bone itself. In addition, two ligaments help connect the vertebral bodies, the dorsal and ventral longitudinal ligaments. The dorsal (top) longitudinal ligament runs on the floor of the spinal canal above the disk while the ventral (bottom) longitudinal ligament runs on the bottom of the vertebral bodies. Processes of bone (articular processes) extending from two adjoining vertebrae come together to make articular facets. These are located dorsolaterally (10 and 2 o'clock positions on a clock face) on each side of the vertebral column and are true diarthrodial joints which include a joint capsule, articular cartilage, and joint synovial fluid. In addition to these three main contact points, the vertebral column is also held together by numerous muscles and their tendinous attachments, and other specialized ligaments.

**Intervertebral disks** are composed of two major anatomic zones: the annulus fibrosus and the nucleus pulposus. The annulus is composed of laminated fibrous tissue wrapped around the gelatinous nucleus pulposus. Individual annular fibers radiate outwardly at varying angles to accommodate all the angles of force that can be applied to the disk. The outer or peripheral layers of the annulus are composed of type I collagen. In comparison, the inner annular layers lying next the nucleus

pulposus are referred to as the transitional zone and are composed of fibrocartilaginous material. For the purpose of localization, the annulus is often subdivided into the dorsal (top or 12 o'clock position) annulus lying directly below the spinal cord and above the nucleus pulposus, the two lateral (side or 9 and 3 o'clock positions) annuli on the sides of the nucleus, and the ventral (bottom or 6 o'clock position) annulus below the nucleus. The dorsal annulus of the disk is approximately 1/3 the thickness of the ventral annulus; therefore the nucleus pulposus lies in the middle to the dorsal third of the disk. As a result, when disk herniation occurs, the tendency is for nuclear material to "escape" dorsally or upward into the spinal canal. The nucleus is composed of a lattice framework of collagen embedded in a highly hydrated gelatinous mass containing cells called chondrocytes and the biochemical glycosaminoglycans hyaluronic acid, chondroitin sulfate and keratin sulfate. The relative proportions for these biochemicals become important in determining those breeds of dogs predisposed to intervertebral disk herniation.

Nutrients reach the peripheral layer of the annulus fibrosus through small blood vessels adjacent to the annulus and small canals that perforate the vertebral body end plates providing direct access to the underlying vascular marrow. The transitional zone of the annulus and the nucleus pulposus receive nutrients by diffusion from the periphery and adjacent vertebral bodies. Normal body movements facilitate the removal of cellular wastes and diffusion of metabolites. Overall blood supply (vascularity) in the disk appears to decline from maturity to old age with the blood vessels remaining intact in only the outermost layers of the annulus fibrosus.

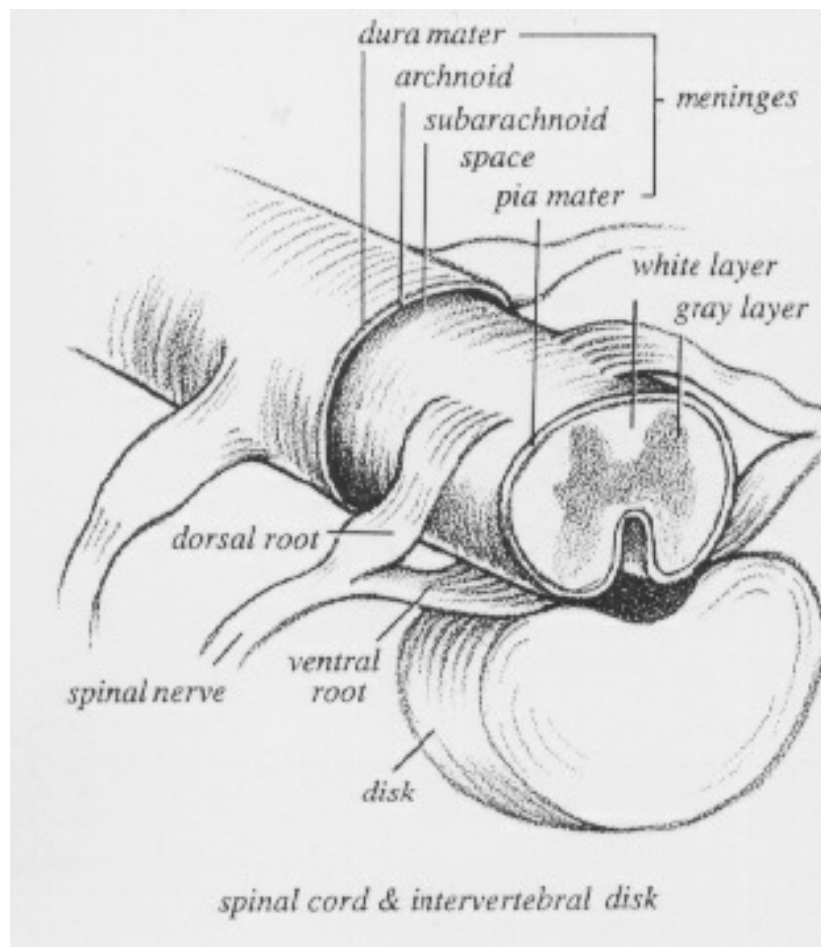
The outer layers of the annulus fibrosus and the dorsal longitudinal ligament contain sensory nerve fibers as opposed to the nucleus pulposus and transitional zone fibers which have none. So called diskogenic pain arises when there is stretching and tearing damage of the outer laminated layers of the annulus fibrosus.

Each vertebra has a central cavity which, when lined up sequentially, forms the vertebral or **spinal canal** in which the spinal cord lies. The size of this canal and the spinal cord vary in relation to each other at different levels of the spinal column. In the cervical and low lumbar areas the spinal cord that occupies it, thus allowing a great deal of space (extradural space) around the spinal cord. In comparison, the thoracolumbar spinal canal is almost entirely filled by the spinal cord; consequently there is very little extradural space at this level. These spinal canal to spinal cord relationships help explain why disk herniations in the thoracolumbar area are often far more debilitating than cervical disk herniations.

Where vertebrae come together, a "window" is formed on each side of the spinal column where the **spinal nerves** and the blood vessels exit and enter the spinal canal. These "windows" are called **intervertebral foramina**. One nerve exits through each intervertebral foramen. Consequently, the spinal cord is divided into

numbered segments which correspond to the number of the vertebra where the paired nerves exit. Although there are only seven (7) cervical vertebrae, there are eight (8) pairs of cervical nerves because the first pair exits in front of the first cervical vertebra and the second pair exits at the junction between the first and second cervical vertebrae. For the remaining vertebrae, there is one pair of nerves per vertebra (i.e. 13 thoracic, 7 lumbar, 3 sacral and corresponding numbers of coccygeal). In the cervical and thoracic areas of dogs, the spinal cord segments and nerves lie approximately inside the correspondingly numbered vertebral segments. However, as the spinal cord enters the lumbar segments, the spinal cord segments start to lie ever-increasingly forward of their correspondingly numbered vertebrae so that nerves exit the spinal cord and run caudally (toward the tail) inside the spinal canal before exiting.

The **spinal cord** is covered by protective membranes collectively called the meninges. The innermost layer, the pia, contains the highly vascular network that delivers nutrients and removes wastes from the nervous system. Surrounding it is the arachnoid layer which, with the pia, forms the subarachnoid space where cerebrospinal fluid flows. The outermost and strongest layer is the dura mater to which the arachnoid is closely associated. The motor and sensory nerve fibers of each cord segment join inside the meninges to become the spinal nerves before exiting this protective sack as peripheral nerves. (See figure below) The meninges are innervated by numerous sensory nerve fibers called meningeal nerves. When a disk herniates into the spinal canal, the meningeal nerves become compressed and inflamed causing the animal a great deal of pain. In addition, the nerve roots themselves are often compressed, also resulting in a great deal of pain for the affected animal.



## Purpose of the Spinal Column and Intervertebral Disks

The spinal column provides a rigid support for the attachment of muscles and bones. It also protects the delicate nervous system while still allowing for spinal column movement. The intervertebral disks form elastic cushions between the vertebrae which allow movement, minimize trauma and shock, and help connect the spinal column.

The intervertebral disk is designed well to dispose of compressive forces, but not as well to combat twisting or bending forces applied to the axis ("straight" line down the length) of the spine. When a disk is compressed, both the nucleus pulposus and the annulus fibrosus share in bearing the load, neither being able to accomplish the work effectively alone. The nucleus is a relatively incompressible mass which, when compressive forces are applied, tries to deform by spreading radially (away from the axis; approximately at right angles to the axis). The annulus fibrosus with its unique layers of fibers is present to prevent that expansion. The annulus effectively "braces" from within thus preventing buckling. Consequently the vertebral end plates are

prevented from "contacting" and the compressive force is ultimately transmitted to the vertebral bodies. The disk's ability to combat compressive forces is so efficient that failure is usually by fracture of a vertebral endplate rather than nuclear herniation or annular tearing.

In contrast, twisting forces must be borne by the annular fibers alone with the nucleus serving only as a "ball bearing" in the activity. When the spinal column is rotated around its axis, only half of the annular fibers are appropriately oriented to resist the rotation. The other half are shortened or become slack and therefore can not help prevent the movement. Consequently, far more possibility for a tearing injury of the annular fibers, with associated inflammatory response, exists and usually occurs. With time, repetitive twisting forces may significantly weaken the annulus fibrosus and prevent its ability to resist compressive forces as efficiently.

Bending forces do not cause as much damage to the annulus fibrosus as twisting forces because the joint capsules of the articular facets, epaxial muscles and spinal ligaments help limit the degree of bend that any one vertebral interspace allows. However, if these supportive structures are compromised by injury or disease, the annular fibers may become separated from the sites of attachment leading to a weakened intervertebral disk space.

## Types of Disks

Disks can be divided into two histochemical types: 1) chondrodystrophoid and 2) nonchondrodystrophoid or fibroid. The word "chondrodystrophoid" literally means faulty development or nutrition of cartilage. In humans, chondrodystrophism is recognized physically (phenotypically) as dwarfism, where individuals are smaller than normal and whose parts (especially limbs) are disproportionate. Certain breeds of dogs, such as dachshunds, show their chondrodystrophism by having disproportionately short and angulated limbs. However, phenotypic characteristics alone can not be used to identify chondrodystrophoid dogs. Other breeds, such as miniature poodles and beagles, have been histochemically identified to have chondrodystrophoid disks and yet do not appear outwardly to be chondrodystrophoid.

When comparing the disks of nine month old dogs, chondrodystrophoid disks characteristically have a larger ratio of transitional versus peripheral zone in the annulus fibrosus. Also the cells of the transitional zone lack clear orientation as is typical in nonchondrodystrophoid disks. The nucleus pulposus in chondrodystrophoid is almost completely composed of dense fibrocartilage which appears to have

completed the chondrofication process. There are only isolated "islands" of notochordal cell remnants seen. In contrast the intracellular matrix of the nonchondrodystrophoid disk is loose and fibrillar and contains notochordal cells only.

The amount of pressure that builds up inside the disk when forces are applied depends on two factors: 1) the water binding properties of the nucleus (more water equals more elasticity) and 2) the degree of resistance and elasticity of the annulus and surrounding structures. These factors are highly dependent on the histochemical makeup of the disk and the changes it undergoes during aging.

### Changes In The Disk That Predispose It To Herniation

Biochemical differences between chondrodystrophoid and nonchondrodystrophoid disks are apparent shortly after birth and explain the differences in the types of degeneration that occur. The degeneration that occurs in chondrodystrophoid disks is called chondroid metaplasia because the nucleus pulposus is gradually replaced with cartilage. Degeneration takes place rapidly and begins as early as 6 months of age starting at the periphery of the nucleus pulposus and progressing centrally. A dramatic and rapid increase in collagen content, as much as 30-40% by dry weight, is seen between 6 and 12 months of age. In addition, total glucosaminoglycan content will be 30 to 50% lower than age matched nonchondrodystrophoid dogs within the first 3 years resulting in a great loss of water content in the nucleus. When this happens, the nucleus loses its elasticity and no longer acts as an efficient shock absorber. Eventually the hyaline cartilage which forms calcifies, leading to almost complete lose of elasticity intervertebral the nucleus pulposus. The overall result is that of placing more of the "workload" on the annulus fibrosus while it is simultaneously undergoing degeneration. Disruption of the annulus fibrosus eventually occurs, especially at its weakest point, the thinner dorsal area lying just below the spinal canal. This allows nuclear material to escape, usually dorsally into the spinal canal or dorsolaterally to impinge on the nerve roots exiting the intervertebral foramina.

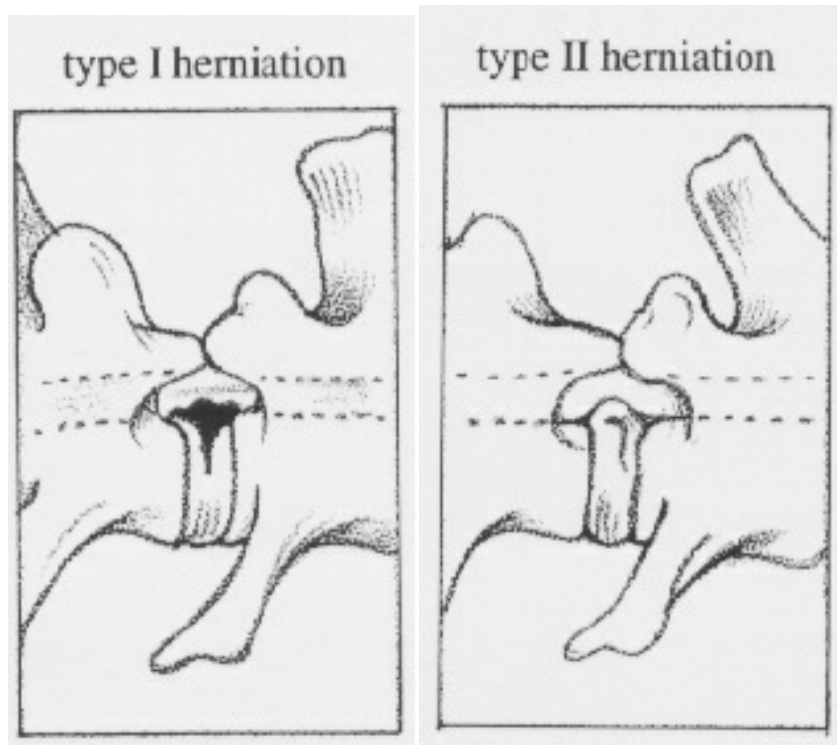
In comparison, nonchondrodystrophoid disks degenerate by fibroid metaplasia with the process becoming clinically significant at 8 to 10 years of age. Fibroid degeneration involves a gradual process of dehydration, and therefore loss of elasticity, of the nucleus pulposus with the incorporation of increasing amounts of collagen and polysaccharides (chondroitin sulfate and keratin sulfate). This causes a gradual diminishing of the border between the annulus fibrosus and the nucleus pulposus, and thus a weakening of the disk's overall biomechanical abilities. Partial



rupture of the annulus fibrosus may result allowing the nucleus pulposus to bulge into the annulus and possibly the spinal canal.

### Pathology of Intervertebral Disk Herniation

Disk herniations have been classified according to the way in which the herniation occurs. **Type I herniation** (see figure below) refers to a large tear in the annulus allowing a large quantity of nucleus pulposus to escape, usually in an acute and profound way. The majority of this type of herniation occurs in the chondrodystrophic breeds but can be caused in any breed by extreme physical activity or trauma. **Type II herniation** (see figure below) refers to small partial tears in the annulus fibrosus which allow nuclear material to escape into the annular area resulting in bulges with only occasional actual "escapes" of nuclear material outside the annulus fibrosus. Type II herniations are the most common type seen in fibroid degenerating disks but can occur in chondroid degenerating disks as well. Typically these herniations are more insidious than the type I herniation.



The spinal cord is made up of an outer "white" layer composed of nerve fiber tracts and an inner "gray" layer composed of the nerve cell bodies, interconnecting nerve fibers and a rich vascular supply. When injury occurs, a chain of metabolic events is set into motion which, if allowed to go unchecked, can result in irreversible damage to the spinal cord. The spinal cord is extremely sensitive to a lack of oxygen and glucose, its only energy sources, which can only be delivered by a viable vascular supply. Consequently, if vascularity is compromised by disruption or occlusion, the spinal cord starts to die. The degree and reversibility of this damage depend on the length of time the spinal cord goes without proper oxygen and glucose delivery and the degree of actual physical derangement of neurologic tissue that occurs. When the spinal cord dies it liquifies. This liquified state is called malacia. Once that occurs, nothing can reverse the process. Spinal cord trauma must therefore be dealt with as quickly as possible if the viability of nervous tissue is to be maintained. The aggressiveness of the chosen therapy will correspond to the degree of clinical dysfunction the injury is causing. Minor trauma with minimal clinical dysfunction may respond well to limited amounts of medical (drug) therapy alone. More damaging trauma may respond well to aggressive medical therapy, or may require surgical intervention as well.

When a disk herniates, it causes damage to the nervous system in several ways. The spinal cord and/or nerve roots can be compressed causing physical as well as physiologic derangement of nervous tissue. Vascular supply can be compromised by tearing, compression or secondary release of vasoactive substances which cause vasoconstriction resulting in a lack of oxygen (anoxia or hypoxia) and glucose delivery. Vascular compromise also leads to the release of destructive chemicals from the blood and from the by-products of nervous tissue breakdown which cause further damage. If this metabolic cascade of destruction can not be stopped in time, nervous tissue will liquify, an irreversible state referred to as myelomalacia. Acute type I disk herniations that apply great forces in a brief period of time cause more damage than chronic progressive type II herniations that apply pressure gradually allowing time for the spinal cord to compensate. Therefore, type I acute disk herniations have a more guarded prognosis in general than type II chronic progressive disk herniations.

In addition to the direct damage caused to the nervous system herniated disk material can cause secondary damage when the animal's body mounts an immune response against the presence of disk material in an abnormal site. Normally the nucleus pulposus is totally isolated from the rest of the body due to a lack of blood supply and the animal's immune system never "sees" this material to recognize it as its own. As a result, the body mounts an immune reaction against "foreign" material when nucleus pulposus escapes into the spinal canal and the inflammatory reaction that results further damages the already compromised nervous system.

## Symptoms of Intervertebral Disk Disease

Any breed of dog can have a disk herniation, especially when external trauma is involved. However, because of the unique metabolic differences of their disks, the chondrodystrophoid breeds are far more likely to develop significant intervertebral disk disease in their lifetimes. Analysis of the frequency of occurrence of disk disease within particular breeds shows that standard and miniature dachshunds are at the highest risk of all dog breeds followed by Pekingese. Approximately one in every four dachshunds will have some degree of disk related problems in their lifetimes. The age of incidence for chondrodystrophoid breeds is highest between three and seven years, as opposed to eight to ten years for nonchondrodystrophoid breeds. No sex predilection has been identified. Clinical signs seen in dogs with intervertebral disk disease vary highly depending on which disk herniates, the amount and speed at which disk material actually protrudes, and which nervous system structures become involved and to what extent.

When increasing pressure is applied to the spinal cord, as occurs with a type II herniation, clinical symptoms develop in a very predictable manner. This is because the different nerve fibers in the spinal cord vary in sensitivity to pressure based on their size. The largest nerve fibers are the most sensitive to pressure and the smallest the least sensitive. When a neurologic examination is performed, the knowledge gained is used to make accurate assessments (prognosis) of the degree of spinal cord and/or nerve root compression present, the possible reversibility of the damage, and how aggressively to instigate therapy.

Proprioceptive (position sense) nerve fibers are the largest and therefore the most susceptible to pressure. Proprioceptive losses can be seen with only slight amounts of pressure applied to the spinal cord. Clinically proprioceptive deficits are recognized as incoordination (ataxia). If proprioceptive losses are the only ones identified on the neurologic examination, the prognosis is usually good and less aggressive therapy is usually adequate.

Motor fibers are the next smallest size of nerve fiber and therefore require more pressure to become dysfunctional than do proprioceptive fibers. Moderate damage to these fibers causes decreased muscle strength and function resulting in clinical signs called paresis. In general, paresis holds a guarded to good prognosis and indicates the need for more aggressive medical, and often surgical, therapy if normal function is to return. More severe damage to motor fibers can result in an absence of muscle strength and function called paralysis. The presence of paralysis is accompanied by a fair to guarded prognosis and indicates the need for fairly rapid and aggressive medical and surgical therapy if the spinal cord is to regain reasonable function.

Superficially or cutaneous sensory nerve fibers are small and therefore require a great deal of pressure to become dysfunctional. Unfortunately this neurologic modality can be hard to evaluate because dogs can not talk to tell what they can or can not feel on their skin. If a lack of conscious perception of pain sensation in corresponding cutaneous sensory zones is identified, the prognosis is guarded to poor. Aggressive medical and surgical therapy must be initiated immediately if the animal is ever to function reasonably normally again.

The smallest nerve fibers transmit deep pain sensation and are the most resistant to pressure. Deep pain is pain perceived when hard pressure is applied to bones or joints. Lack of deep pain perception is the gravest of clinical signs but must be considered in light of how long it has been lost before deciding if the spinal cord is irreversibly damaged.

In acute type I disk herniations, spinal cord nerve fibers are sensitive to externally applied pressure in the same manner as in type II disk herniation. However, type II disk herniations do not cause as much of the acute internal damage to the spinal cord described above. Even so, the neurologic examination findings should be interpreted in basically the same way following an acute spinal cord trauma: only ataxia indicates a relatively good prognosis, additional motor deficits indicate the need for more aggressive therapy and loss of deep pain sensation is a grave sign.

Type I disk herniations tend to occur explosively with large amounts of nucleus pulposus escaping into the spinal canal at one time. The spinal cord is damaged first by impactive forces when nuclear material strikes it, and then by compressive forces when nuclear material occupies the extradural space. When this occurs, major physiologic disruptions of nerve transmission occur which are not always permanent if aggressive medical therapy is instigated immediately. It is not unusual for dogs presented shortly after severe spinal cord trauma to show no signs of conscious perception of deep painful stimuli. The veterinarian is left with the task of trying to differentiate if the dog's failure to show that it feels the clamp on its toe indicates 1) a true lack of deep pain sensation from irreversible destruction of the ascending pain pathways which can not be altered by any amount of therapy, 2) severe physiologic interference with signal transmission in an otherwise viable nerve fiber which could return to normal function if immediately and aggressively treated, or 3) if so many painful stimuli are bombarding the dog's brain that it simply doesn't show behavioral acknowledgment of this one additional painful stimulus and deep pain sensation is actually intact. Unfortunately, there is no way to clinically differentiate these different states of "no deep pain" perception. With the goal of preserving the maximum amount of neurologic tissue and regaining the maximum amount of clinical function possible, the best course it always to treat these dogs as aggressively as possible (medical and surgical therapy) and wait to see if deep pain perception returns. In

most cases it will and these dogs will be saved. However, sometimes owners are unable or unwilling to spend money for surgery on a gamble that deep pain function will return. In that case, the best course of action is aggressive medical therapy and reevaluation of the neurologic examination in a few hours to see if deep pain perception has returned. If it does, surgery should then be performed immediately. In this case, the owner understand that this delayed surgical intervention probably won't be as effective as if surgery had been performed earlier. In general the longer the state of no deep pain perception exists, the poorer the prognosis. In most case of acute spinal cord trauma, well documented lack of deep pain perception for 24 or more hours is most often indicative of functional transection (transverse malacia) of the spinal cord. However, due to the subjective nature of the interpretation of the presence or absence of deep pain perception, it is probably wise to extend the "window" of time in which aggressive medical and surgical therapy is attempted to 48 hours.

The pain suffered with intervertebral disk disease arises from several sources. The spinal cord has no sensory nerve fibers. However, the peripheral layers of the annulus fibrosus do. So called diskogenic pain arises from abnormal stresses being placed on the annulus fibrosus or physical tearing of fibers. Herniation of the nucleus pulposus is not necessary for diskogenic pain to occur. Another source of pain arises from the sensory nerves in the the meningeal coverings. When the spinal cord, and therefore the meningeal coverings, are compressed or when nuclear material contacts the meninges resulting in a "foreign body" inflammatory process, these sensory nerves are activated and meningeal pain occurs. A third source of pain from intervertebral disk disease arises when nerve roots are entrapped by herniated disk material. The resulting pain is referred to as radicular pain and the clinical signs called a "root signature."

In dogs, cervical and thoracolumbar disks are the most commonly herniated, but the degree of clinical dysfunction seen can vary remarkably. As mentioned previously, there is a great amount of extradural space in the cervical area. Large quantities of disk material may herniate without causing significant impact on or compression of the spinal cord leaving little to moderate clinical dysfunction. In comparison, the thoracolumbar area has almost no extradural space around the spinal cord. Consequently, a comparable amount of herniated disk material at this level may significantly impact and/or compress the thoracolumbar spinal cord resulting in major clinical dysfunction. The spinal cord has a great ability to compensate if compression is applied slowly over a long period of time, such as occurs with type II disk herniation. In comparison, it has little ability to compensate for the acutely applied compression that occurs in type I disk herniation. Unfortunately, thoracolumbar disks herniate more frequently than cervical disks.

One of the cardinal signs of **cervical disk disease** is neck pain. Dogs display this

pain by tightened neck muscles, reluctance to move the neck, inability to lower the head to eat or drink, and painful cries when the neck is manipulated or touched. Their posture reminds you of a turtle with its head partially pulled into its shell. They may also walk with the back in an arched position to try to straighten and lower the neck to avoid pain. Unfortunately this posture is often misinterpreted as a sign of back pain. Probably all three types of pain (diskogenic, meningeal and radicular) are involved. Nagging neck pain that responds to appropriate medications and then recurs when medications are withdrawn is a very typical history. Large quantities of herniated disk material can lie on the floor of the spinal canal without proprioceptive or motor deficits ever being observed because there simply isn't enough pressure on the spinal cord. However the irritation causing the pain remains and surgical removal of the herniated material is the only solution. If enough disk material herniates, ataxia and perhaps motor deficits may be seen. Classically all four limbs will be affected but this can be highly variable. If disk material herniates mainly to one side, only the limbs on that side of the body may be affected. Low cervical disk herniations may cause only forelimb or only hind limb involvement. Luckily there is seldom enough pressure on the spinal cord in cervical disk herniation to cause a loss of deep pain sensation.

Clinical signs of **thoracolumbar disk herniation** unfortunately are usually more profound than in the cervical area. With almost no extradural space for herniated disk material to occupy, the spinal cord can be severely compressed. These dogs have normal forelimb function with one exception. If the disk is just starting to protrude but has not actually herniated, dogs will walk with an arched stiff back due to pain. They often cry out when picked up or their back muscles are squeezed. They are usually reluctant to move around as usual. If more pressure is present on the spinal cord, ataxia and paresis of the hindlimbs is usually evidenced by the dog dragging its toes and walking with a wobbly gait. At this point immediate surgical intervention can provide excellent results. Further pressure causes hindlimb paralysis and eventually loss of deep pain sensation if therapy is not instigated. The greatest value of surgery is in the earliest stages of disk herniation. The longer clinical signs exist without surgical intervention, the less value the surgery can provide and the more permanent damage the spinal cord sustains.

Occasionally injury to the thoracolumbar spinal cord can cause clinical signs in the forelimbs. Almost invariably the injury is extremely acute and severe, such as a spinal fracture or profound type I disk herniation. There are motor fibers in the upper lumbar spinal cord segments that run forward to the cervicothoracic area to influence the nerves exiting to the muscles of the forelimbs and neck. When these motor fibers are damaged, the forelimbs become rigidly extended and the dog holds its head back. Also, deep pain perception is often not recognizable. This is called Schiff-Sherrington syndrome and, in the past, was said to always be an indication of irreversible spinal cord malacia. Unfortunately that is not true because many dogs

that display this syndrome can recover if immediate aggressive medical and surgical therapy is instigated. Unfortunately, many dogs have been needlessly euthanized due to failure to understand that this syndrome can be reversible. However, if signs of Schiff-Sherrington syndrome persist after aggressive therapy has been administered, or persist when therapy has been delayed for whatever reason, then the prognosis is grave.

## Diagnosics

Intervertebral disk herniation is usually suspected based on the signalment (breed, age, sex), history of appropriate clinical dysfunction, and a neurologic localization of the cervical or thoracolumbar areas. To make a positive diagnosis, spinal radiographs or Xrays will need to be taken. It is absolutely imperative that no movement occur and that the muscles along the spinal column be relaxed if an accurate assessment of vertebral relationships and disk spaces is to be made. Consequently, the only way to get good quality spinal radiographs is by administering a general anesthetic to the affected dog. The only exception would be if some other physical abnormality, such as severe heart disease, precludes this being done safely. In that event, surgical intervention is probably also not an option and specifically locating the offending disk is not necessary to carry out good medical therapy.

Once the dog is anesthetized, a series of plain radiographs will be taken with the dog lying on its side (lateral view). The veterinarian will be looking for changes in the normal relationships and density of the vertebrae and the disks. Those changes include narrowed or wedged disk spaces, displaced calcified disk material in the intervertebral disk space or intervertebral foramina, narrowed articular joint spaces, and/or many other suggestive signs. However, calcification of the nucleus pulposus does not necessarily mean that a disk will herniate. Unfortunately, most herniated disk material is not calcified enough to be seen on Xray. Therefore the exact position of the herniated material most often can not be determined on plain Xrays and a specialized study called a myelogram must be performed. Risks are involved and the possibility of worsening a delicately balanced situation exists. However, the manufacture of new less irritative dyes has removed a great deal of the risk and myelograms are now routinely performed without the great concern of the past.

In a myelogram, radioopaque dye is injected into the cerebrospinal fluid in the subarachnoid space. In a normal study, two lines of dye will be seen running on each side of the spinal cord more or less like railroad tracks running parallel to one another. When a disk has herniated, nuclear material and torn annular fibers can

occupy the extradural space compressing the spinal cord. The normal parallel configuration of the two dye lines will be disrupted by either pushing them closer together or farther apart depending on the particular radiographic view examined. Additional oblique views can be taken to help identify which side of the spinal cord is more affected. By examining all views carefully, the amount of herniated material, and therefore the amount of pressure on the spinal cord, can be estimated. Also the preferred side to enter surgically can be determined.

## Therapeutics

### Restricted Physical Activity and Nursing Care

Owners of chondrodystrophic dogs should be advised by their veterinarians of the potential dangers of intervertebral disk disease when those dogs are first seen as puppies. At that age, dogs can be trained to a life style that limits the possibilities of disk herniation, such as restricting excessive jumping, preventing obesity, and training the dog to be confined quietly in an airline crate. Taking these measures will help, but do not provide an absolute guarantee that disk herniation will not occur.

When a dog starts to show mild signs of pain indicative of intervertebral disk disease but is still ambulatory, restricted activity in a cage is indicated. Allowing nature time to heal the tear in the annulus fibrosus may be all that is necessary. If cortiosteroids are simultaneously administered, restricted activity is an absolute must.. Corticosteroids will mask the pain the body is using to tell the dog to "slow down." If the nucleus pulposus has not herniated but significant weakening of the annulus fibrosus exists, one good jump off the couch is all it may take for the dog to become hopelessly paralyzed from irreversible spinal cord injury. The absolute necessity of cage rest in these animals cannot be overstressed. Failure to do so often contributes to disk herniation. Such a dog can never be hurt by too much cage rest, but certainly devastating results can occur if too little cage rest is used.

When disk disease causes motor deficits, a great deal of damage can be done to the feet and legs if the dog is not restricted from dragging across rough or hard surfaces. When the owner can not directly supervise the dog's activity, it should be put in its well padded cage to avoid further injury. When outside, protective foot coverings are a great help. Once sores are allowed to form they are very difficult to clear up; preventing them is far better.

Disk herniation not only damages descending motor tracts to the legs, but also those that are involved in normal urination and defecation. Consequently, a number of



dogs will require special attention to these functions. It is very common to see urinary incontinence following disk herniation. Care must be taken to see that the bladder is emptied properly several times a day to avoid urinary tract infection. This may require regular (4 to 5 times per day) manual expression of urine from the bladder. Fecal incontinence is less of a problem but still must be watched and treated if necessary with stool softeners.

Especially in paralyzed patients, regular cleaning of the dog and its bedding is imperative to avoid urine scalding and infection of the skin. Ointments to provide a water barrier are very useful. Additionally, a well padded bed must be provided to avoid the development of bed sores (decubital ulcers). Once the skin lesions break open, massive systemic infection may result which can be life threatening. It is far simpler to prevent these problems from arising rather than treat them after the fact.

## Medical Therapy

Aggressive medical therapy is recommended in any form of spinal cord trauma. It becomes especially important in acute trauma, such as type I disk herniation, to interrupt the destructive metabolic consequences described previously.

**Corticosteroids** are the first line of attack in handling any acute spinal cord trauma. The currently preferred corticosteroid is methylprednisolone sodium succinate (Solu-Medrol R; Upjohn). Compared to the previously favored corticosteroid dexamethasone (AziumR; Scherring), methylprednisolone sodium succinate seems to have far better sparing effects on the spinal cord while causing fewer side effects (gastrointestinal bleeding: excessive water consumption/urination). Since **oxygen** is so critical to the survival of nervous tissue, delivery by way of a face mask or an oxygen cage can be advantageous. **Hyperosmolar agents** such as mannitol or glucose do not have as much effect in spinal cord injury as brain injury because they have no effect on the compressing mass of herniated material. However, in severe cases, they can be useful in reducing some of the spinal cord swelling. Numerous other medications have been advocated for spinal trauma, such as narcotic antagonists and calcium channel blockers. Some are of most benefit when administered within minutes of the spinal cord trauma and this can not always be accomplished. Others do little to fight the primary injury but have long term sparing effects. The use of these additional medications will vary with each individual case.

## Surgical Therapy

The spinal cord can compensate immensely if a compressive force is applied in a chronic progressive manner, as occurs in type II disk herniation. Clinical dysfunction develops in the classic stepwise fashion described previously (ataxia >> paresis >> paralysis >> lose of pain perception) at a rate proportional to the speed of the increasing compression. Because damage is occurring on a "one cell at a time" basis rather than the profound total involvement that occurs in acute spinal injury, the spinal cord has time to retrain itself and compensate clinically for a great deal of the damage occurring. Consequently, the outward clinical picture may not truly reflect the total degree of internal pathology that exists. Performing surgery in cases where spinal cord compression has existed for long periods of time and significant clinical loss has occurred can do little to improve clinical function if at all. What function is lost is most often irretrievable, so the best therapy in these cases is to arrest the slowly progressive nature of the compression before too much clinical dysfunction develops, most especially a loss of pain perception. By comparison, in cases of explosive type I disk herniation, decompressive surgery is of most value when performed immediately. Delays of even a few hours may make a major difference in the outcome of the case, especially if significant compression exists.

Medical therapy combined with restricted activity and physiotherapy is a good approach as long as an affected dog is only displaying ataxia and/or discomfort. However, once the line is crossed to motor deficits, more aggressive therapy is usually needed. It is true that many dogs that become paretic, or even paralyzed, given enough time can regain "normal" clinical function without surgical intervention. However, the degree of permanent pathologic changes inside the spinal cord and the time to functional recovery will be far greater than for the dog that receives surgical decompressive therapy early in the course of disease. If additional trauma is sustained in the future, the dog that has "recovered" without surgery will probably "decompensate" more easily because of greater preexisting spinal cord pathology, resulting in a much greater degree of dysfunction than the dog with similar dysfunction that was operated early.

There are basically three categories of surgical procedures applied to the spinal column: decompression, stabilization and preventative disk fenestration. Very seldom does disk herniation destabilize the vertebral column so stabilizing techniques are seldom necessary. Decompressive surgery basically creates a window through vertebral bone so that the spinal canal can be entered and examined for compressive masses of material which are then removed. The particular type of decompressive surgery done depends on the site, or sites, of disk herniation and the location of the herniated material within the canal.

There are two main decompressive surgical procedures performed in the **cervical area**: 1) a **dorsal laminectomy** where the surgeon approaches from the dorsal side by separating the muscles on the back of the neck to reach the laminae of the vertebrae and 2) **ventral spondylectomy or slot** where a ventral approach is made by displacing the trachea, esophagus and neurovascular trunks to the side to reach the vertebral bodies. Because most of the herniated disk material will be found on the floor of the spinal canal, the ventral slot procedure is far superior to dorsal laminectomy to reach and retrieve that material. If material has herniated up the sides of the cord, the ventral slot affords good exposure to retrieve it as well. However, the ventral slot procedure can not be safely extended over multiple disk sites in a row. In the event multiple disks herniate, the dorsal laminectomy can be extended over several disk spaces without compromising the stability of the vertebral column. Pressure on the cord will be relieved, but the floor of the canal can not be reached without causing significant trauma to the spinal cord. Therefore most of the disk material will probably have to be left under the spinal cord and can possibly cause problems later from the inflammatory reaction to its presence. Recently a lateral approach to the cervical vertebrae has been described, but probably has little advantage over the ventral slot procedure for access to the herniated disk material. Fenestration can only be performed from the ventral approach in the cervical area and therefore can be performed simultaneously with a ventral slot.

In the **thoracolumbar area**, decompression can be performed via **dorsal laminectomy** as in the neck. This approach has the same disadvantages of not being able to reach the floor of the spinal canal to retrieve herniated disk material. Also, additional extensive retraction of muscle would have to be done to perform preventative fenestration in combination with dorsal laminectomy. The more commonly preferred approach is dorsolaterally or laterally on one side of the dorsal spines to reach the articular facet and side (pedicle) of the vertebrae of the affected disk. A **hemilaminectomy** (removal of half the lamina and the pedicle) can then be performed by removing the articular facet and entering the spinal canal from the side. From this position, herniated disk material is easily removed from the floor of the canal and the side from which entry was made. Unfortunately, if material has herniated up the opposite side, it probably can not be retrieved without significant damage to the spinal cord. This is why it is so critically important to know to which side the disk material has herniated. The neurologic examination and the myelogram should be used to decide from which side the hemilaminectomy should be performed. Preventative fenestration can be easily accomplished in combination with a hemilaminectomy. A ventral slot procedure is not feasible in the thoracolumbar area. The abdomen and chest would both have to be entered, and the aorta courses just below the vertebral bodies. Using a bone drill in these areas would be too dangerous with no added advantages over hemilaminectomy. For the same reasons the formerly advocated ventral approach to fenestrate these disks have been abandoned.

**Fenestration** is the surgical procedure that creates an opening through the ventral (cervical area or lateral (thoracolumbar area) disk annulus fibrosus through which nucleus pulposus can be removed, hopefully preventing disk herniation at the site in the future. It is usually performed in the cervical area from C2-3 to C5-6 or C6-7 and in the thoracolumbar area from T11-12 to L2-3 or L3-4. The use of preventative fenestration as part of the treatment of intervertebral disk disease remains controversial. Certainly, if done poorly, numerous complications can arise, the worst being laceration of the spinal cord or aorta. Infection can be introduced into the disk space causing diskospondylitis. Trauma to the vertebral end plates can result in degenerative bony proliferation called spondylosis which may entrap nerve roots. It is also very difficult to get every bit of the nucleus pulposus out of the disk space leaving the possibility that the disk may still herniate sometime in the future. The overall biomechanics of the fenestrated area is altered such that the remaining disks on either end of the fenestrated chain must absorb greater forces, creating the risk of future disk herniation in atypical sites. Also, studies have shown a very low overall incidence of a second herniation disk in an individual dog's lifetime. The argument can then be effectively made "is the potential danger of preventative fenestration worth it?". Proponents of disk fenestration point out that, even if most chondrodystrophic dogs will only herniate one or perhaps no additional disks in their lifetime, they are frequently plagued with chronic pain from degenerating disks. It has been repeatedly shown that preventative fenestration in these dogs is of great value in relieving that discomfort, even if no herniation has actually occurred. In dogs undergoing thoracolumbar hemilaminectomy or cervical ventral spondylectomy, access to the disks does not require any additional surgical exposure. If done carefully and atraumatically, fenestration can prevent future disk herniation, additional surgery, and future pain. I personally advocate preventative fenestration and believe it is a valuable service if rendered atraumatically.

## Physiotherapy

In dogs who have suffered motor losses, physiotherapy is extremely useful to speed their recovery to more normal function. **Swimming** is the best exercise because it allows the limbs to move freely in the water without having to bear weight. It also provides a bath to clean the dog and prevent skin infections. The lukewarm water also serves to increase circulation in the limbs and massage the muscles. Most chondrodystrophic dogs will fit nicely into a bath tub filled with just enough water to allow the dog to float with its toes off the bottom. Care should be taken not to leave a dog unattended to avoid drowning. Swimming exercises may be begun 4 to 5 days post-operatively. The surgical incision may be "waterproofed" with a thin film of

Vaseline. The dog should be exercised until tired and then removed and thoroughly dried. Each day the sessions will lengthen as the dog's endurance builds up. Swimming once daily is good but twice daily is even better. This will vary with the time available to the owners.

Additional exercises include "**towel walking**" paraplegic dogs where the owner holds onto a cloth sling placed under the dog's abdomen allowing the dog to "walk" alongside the owner. Allowing the hindlimbs to support some weight will stimulate motor responses and strength. Exercises which manipulate the joints and massage the muscles will help prevent muscle atrophy and joint contracture. These may be done by lying the dog on its back and "**bicycling**" the hindlimbs and forelimbs, moving the limbs through a pattern of full range of motion excursions to keep the joints limber, and pushing on the limbs to encourage muscular resistance.

**Massaging** the muscles of the affected limbs will also help improve circulation and will feel good to the dog.

**Carts** are made commercially or can be made at home to provide "mobility" for paralyzed dogs. Carts allow dogs to get up and participate in more household activities, but do not provide any form of physiotherapy. Owners who "abandon" their dogs to carts will probably never have a walking dog again. Carts have a place in the overall management of paralyzed dogs but can not be substituted for good nursing care and physiotherapy.

Occasionally a dog that has transverse malacia of its thoracolumbar spinal cord (no deep pain sensation) can learn to "walk" again using the crossed extensor reflex that may still exist in the hindlimbs. Without motor connections to the brain, this reflex is "released" and causes the involuntary motor movements that are frequently observed in the limbs. Through extensive physiotherapy, many of these dogs learn to swing their bodies to get their hindlimbs under them and allow the reflex walking movements to be effectual. This form of walking is called **spinal walking** and looks a bit "motorized" but serves the function well. Unfortunately many of these dogs are also permanently incontinent. Many owners lifestyles do not allow the time necessary to properly care for incontinent dogs. As a result many of these dogs are euthanized rather than allowed the time to see if they can develop spinal walking abilities.

## Summary

Intervertebral disk disease is a major clinical problem in the dachshund breed. However, by recognizing the causes and consequences of the problem, owners may

prevent a great deal of difficulties by restricting the amount of excessive jumping and managing the weight of their dogs. In the event disk herniation does occur, veterinary attention should be sought immediately since the earlier medical and surgical intervention are begun, the better the therapeutic outcome.

---

Further information and support may be found at:

<[www.dodgerslist.com](http://www.dodgerslist.com)>