

Intervertebral Disc Disease (IVDD) – Anatomical Factors Contributing to Disc Herniation in Dogs

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Introduction

The spinal column is designed to provide both stability and flexibility. At its core, the intervertebral disc acts as a resilient cushion, allowing movement while protecting the delicate spinal cord. Each disc comprises two primary anatomical components: the central **nucleus pulposus**, a gelatinous core, and the surrounding **annulus fibrosus**, a tough, fibrous outer ring. This intricate design is inherently susceptible to degeneration and subsequent herniation, particularly under specific anatomical and genetic predispositions.

Intervertebral Disc Disease (IVDD) is one of the most common spinal disorders in companion animal practice, especially affecting dogs. It occurs due to the degeneration or displacement of the intervertebral discs. This condition can lead to varying degrees of pain, neurological dysfunction and in severe cases, paralysis.

This disease is especially seen in chondrodystrophic breeds- those with short legs and long backs, such as Dachshunds, Beagles, and Cocker Spaniels. However, it may also affect non-chondrodystrophic and large breed dogs, particularly as they age. IVDD is of significant clinical concern due to its high prevalence in certain breeds, potential for rapid progression and serious impact on the animal's mobility and quality of life.

Classification of IVDD

IVDD is primarily classified into two main types based on the anatomical changes and pathogenesis:

- **Hansen Type I IVDD:** This involves the herniation of the nucleus pulposus through torn annular fibers, resulting in the extrusion of nuclear material into the spinal canal. This condition is typically associated with chondroid disc degeneration.
- **Hansen Type II IVDD** involves the annulus fibrosus (outer disc ring) slowly protruding into the spinal canal, often due to fibroid degeneration of the disc material.

Anatomical Factors Contributing to Disc Herniation

Understanding the anatomy of the intervertebral disc and its surrounding structures is paramount to appreciating the mechanisms of disc herniation in dogs.

1. **Intervertebral Disc Structure and Degeneration:**
 - **Nucleus Pulposus:** In young, healthy dogs, it is rich in water and acts as a hydraulic shock absorber. Over time, particularly in predisposed breeds, it undergoes **chondroid degeneration**, meaning it loses water content, becomes drier, and can calcify or mineralize. This makes it less resilient and more prone to rupture. In non-chondrodystrophic breeds, the nucleus may undergo **fibroid degeneration**, where it becomes more fibrous and less likely to extrude acutely, often leading to more gradual protrusions.
 - **Annulus Fibrosus:** This outer ring, composed of concentric layers of

collagen fibers, encases the nucleus pulposus, providing structural integrity. Its fibers are strategically arranged to withstand various spinal movements. However, with age or repeated stress, the annulus can weaken, tear, or bulge. The upper part of it is often thinner, making it a common site for disc material to extrude towards the spinal canal.

2. **Spinal Canal Confinement:** The spinal cord is housed within the vertebral canal, a bony tunnel formed by the stacked vertebrae. This canal offers protection but also presents a confined space. When disc material (from the nucleus or annulus) herniates into this limited space, it directly compresses the spinal cord, leading to the neurological signs observed in IVDD. The degree of compression and the velocity of onset are critical factors in the severity of clinical signs.
3. **Breed Predispositions:**
 - **Chondrodystrophic Breeds:** These breeds (e.g., French Bulldogs, Dachshunds, Basset Hounds) are genetically predisposed to premature chondroid degeneration of their intervertebral discs, often starting as early as one year of age. This accelerated degeneration means their discs lose their elasticity and become calcified much earlier than in other breeds. This anatomical change makes them highly susceptible to **Hansen Type I IVDD**, characterized by acute extrusion of mineralized nuclear material into the spinal canal. Their elongated thoracolumbar spine also creates increased biomechanical stress on these already compromised discs.
 - **Non-Chondrodystrophic Breeds:** In breeds like, Doberman Pinschers, Labrador Retrievers and German Shepherds IVDD typically occurs later in life and is more commonly associated with **Hansen Type II IVDD**. This type involves a more gradual **annular protrusion** due to fibroid degeneration, where the annulus fibrosus slowly bulges into the spinal canal, causing chronic compression.

4. **Location of Herniation:** While disc herniation can occur at any level of the spine, certain regions are more commonly affected due to their inherent biomechanics and mobility:

- **Thoracolumbar Region (T11-L2):** This is the most common site for disc herniation, particularly Hansen Type I, in chondrodystrophic breeds.
- **Cervical Region (C2-C7):** The neck region is another common site, especially for Hansen Type II IVDD or for Type I in some breeds. The high degree of mobility in the neck makes these discs vulnerable to stress and degeneration.
- **Lumbosacral Region (L7-S1):** Less common for acute IVDD but can be involved in chronic low back pain, particularly in larger breeds, often referred to as lumbosacral stenosis.

Causes

The primary cause of IVDD is the age-related degeneration of the intervertebral discs, significantly influenced by breed-specific anatomical predispositions. For **Hansen Type I IVDD**, the degenerated, often mineralised, nucleus pulposus forcefully extrudes through a weakened dorsal annulus. This acute extrusion leads to direct ventral, ventrolateral, or circumferential compression of the spinal cord. As mentioned, chondrodystrophic breeds like Dachshunds, Pekingese, Welsh Corgi, Beagle, Lhasa Apso, and Miniature Poodle have a genetic predisposition to this type, with Dachshunds showing the highest incidence. The thoracolumbar region is most commonly affected in these breeds due to the biomechanical forces at play.

Hansen Type II IVDD involves a slow, progressive protrusion of the annulus fibrosus into the spinal canal, leading to chronic spinal cord compression. This sustained pressure can result in focal ischemia and microvascular derangements of the spinal cord. Type II IVDD usually occurs at the more mobile points of the spinal column and is more common in older, non-chondrodystrophic breeds like Dobermanns and Labrador Retrievers.

Symptoms

The symptoms of IVDD can vary greatly, primarily depending on where the spinal cord is compressed, how severely it's compressed, and how quickly the issue developed. Neurological signs can

appear very suddenly (within an hour), acutely (within 24 hours), or more gradually (over more than 24 hours). Dogs experiencing a sudden or acute disc herniation in their mid-to-lower back might show signs of spinal shock or Schiff-Sherrington postures. While these indicate a severe, sudden injury to the spinal cord, they don't necessarily determine the long-term outlook. The extent of neurological impairment can range from simple back pain (spinal hyperesthesia) to uncoordinated walking (ataxia), weakness (paresis), or even complete paralysis (paraplegia), with or without the ability to feel deep pain.

Dogs that are only experiencing pain often seem reluctant to move and might have a hunch in their back (kyphosis). It's important to note that even dogs showing only pain can still have significant spinal cord compression, which advanced imaging can reveal. The clinical signs of Hansen type II IVDD include slowly progressive hindlimb ataxia, weakness, reluctance to rise or jump on furniture and difficulty climbing stairs. Onset of clinical signs is considered chronic and progressive, although acute exacerbations of signs are not uncommon. Localization is focal with asymmetrical or symmetrical weakness. Paraspinal hyperesthesia may or may not be present.

Diagnosis

Accurate diagnosis of IVDD requires a comprehensive approach integrating clinical evaluation, neurological assessment, and advanced imaging.

Clinical Signs: Animals typically present with neurological and pain-related signs such as neck or back pain, ataxia, paresis/paralysis, reluctance to move, and muscle tension.

Neurological Examination: Crucial for localizing the lesion (cervical, thoracolumbar, or lumbosacral). It assesses proprioception, spinal reflexes, and pain perception, which has significant prognostic implications.

Diagnostic Imaging:

- **Radiography (X-rays):** Can show narrowed disc spaces or mineralized discs, but limited for soft tissue visualization or definitive spinal cord compression.
- **Myelography:** Involves injecting a contrast agent to outline the spinal cord, helping identify the location and severity of compression. Invasive with risks.

- **Computed Tomography (CT):** Excellent for detecting mineralized disc material and often used with myelography. Useful for visualizing bone and soft tissue structures, aiding surgical planning.
- **Magnetic Resonance Imaging (MRI):** Considered the gold standard. Provides high-resolution images of the spinal cord, intervertebral discs, nerve roots, and surrounding soft tissues. Effective in identifying both Hansen Type I and II herniations, as well as secondary changes like spinal cord edema or hemorrhage.

Other Diagnostic Tests: Cerebrospinal fluid (CSF) analysis to rule out inflammatory/infectious conditions and electrodiagnostic testing (EMG, nerve conduction studies) for ambiguous or chronic cases.

Treatment

Treatment for IVDD is divided into conservative (medical) management and surgical intervention.

1. Conservative (Medical) Management: Recommended for mild cases (Grades I–II) or when surgery is not feasible.

- **Strict Cage Rest:** 4 to 6 weeks of minimal movement to reduce inflammation and swelling, alleviating pressure on nerves.
- **Medications:** NSAIDs (carprofen, meloxicam) for pain/inflammation, Gabapentin for nerve pain, Methocarbamol for muscle spasms, and Opioids (tramadol) for severe pain. *Never give steroids and NSAIDs concurrently.*
- **Bladder & Bowel Care:** Manual bladder expression if needed to prevent complications.
- **Supportive Measures:** Soft bedding, using a harness instead of a collar, strict avoidance of stairs and jumping.
- **Physical Rehabilitation (Post-Rest):** Passive range-of-motion, laser therapy, acupuncture, and hydrotherapy to rebuild strength and mobility.
- **Monitoring:** Regular assessment; worsening signs or paralysis/loss of deep pain sensation warrant surgical reconsideration.

2. Surgical Treatment: Reserved for severe cases (Grades III–V), significant neurological deficits, or lack of improvement with medical management.

- **Surgical Options:**
 - **Hemilaminectomy:** Most common for thoracolumbar herniations; removes part of the vertebra to relieve spinal cord compression.
 - **Ventral Slot:** Primarily for cervical herniations; creates an opening in the vertebra to remove disc material.
 - **Fenestration:** Performed to prevent future herniations by creating openings in the disc.
- **Postoperative Care:** Pain management, bladder care, early physical rehabilitation, and meticulous wound care are crucial for optimal recovery.

Prognosis

The prognosis depends on disease severity and treatment:

- **Mild signs (Grade I–II):** Excellent prognosis with conservative treatment, often leading to full recovery.
- **Moderate signs (Grade III):** Good recovery with surgical intervention.
- **Severe signs (Grade IV–V):** More guarded prognosis, especially with loss of deep pain perception for >48 hours. Full recovery may not occur, impacting quality of life.

Recovery

The recovery process involves four key phases:

1. **Immediate Phase (0–7 Days):** Strict crate rest, pain management, bladder expression (if needed), and post-surgery monitoring.
2. **Early Recovery Phase (1–4 Weeks):** Focus on mobility and nerve recovery with controlled leash walks, passive range-of-motion exercises, and hydrotherapy.
3. **Intermediate Phase (4–8 Weeks):** Regaining motor function and coordination; gradual increase in activity, introduction of active rehabilitation exercises.
4. **Long-Term Recovery (2–6 Months):** Focus on functional independence; many dogs regain walking ability. Lifelong adjustments may be needed to prevent re-injury.

With proper care and rehabilitation, most dogs can recover well, though some may require ongoing lifestyle changes.

Conclusion

Intervertebral Disc Disease (IVDD) represents a significant neurological condition in veterinary practice, particularly among predisposed canine breeds. The impact of the disease ranges from mild discomfort to severe neurological deficits and paralysis, making early diagnosis and appropriate intervention essential. By understanding the distinct types of IVDD—Hansen Type I and II—veterinarians can tailor treatment plans that include either conservative management or surgical correction, depending on the severity and progression of clinical signs. Advanced diagnostic tools such as MRI and CT imaging enhance accuracy and aid in effective therapeutic decision-making. Post-treatment care, including physical rehabilitation and long-term management strategies, plays a vital role in recovery and relapse prevention. With a comprehensive, informed approach, many affected animals can regain function and enjoy a good quality of life, reinforcing the importance of early intervention, breed-specific awareness and client education in managing IVDD successfully.

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