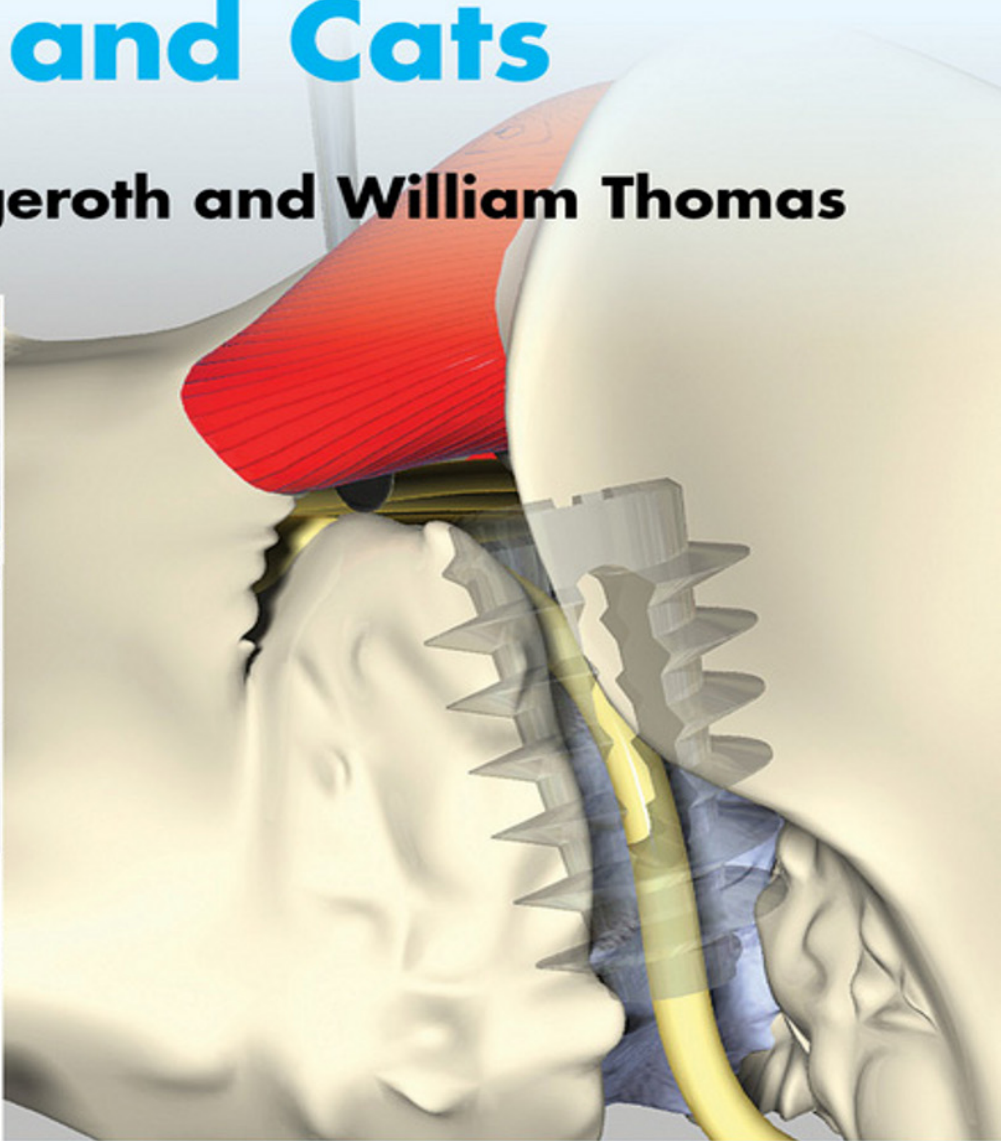


# Advances In Intervertebral Disc Disease in Dogs and Cats

**James Fingerroth and William Thomas**



**WILEY Blackwell**



Advances in  
**Intervertebral Disc  
Disease in Dogs  
and Cats**



# Advances in **Intervertebral Disc Disease in Dogs and Cats**

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# Foreword

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Hippocrates (460–370 B.C.), perhaps the most prominent physician of the distant past, is recognized as a founder of scientific medicine. The bases of the Hippocratic method were *accurate observation* and *sound reasoning*. The Hippocratic study of medicine attributed diseases to “natural causes,” thereby eradicating the concept of “luck” as a creative force. This application of scientific method to medicine was revolutionary, as the thinking of the time is epitomized in the works of the philosopher Plato (fifth to fourth century B.C.) who believed that “a divine intervention contributed to the creation of the flexible spine,” or the famous philosopher and physician Empedocles (fifth century B.C.), who suggested that at birth the spine was rigid, and that subsequently this osseous column was broken into pieces as a result of body movements.

Hippocrates considered knowledge of vertebral column anatomy essential to physicians: “One should first get a knowledge of the structure of the spine; for this is also requisite for many diseases.” Centuries later, an eminent physician of the time, Galen (second century A.D.), promoted this principle, and criticized physicians for their ignorance of the structure of the vertebral column.

What might Hippocrates have accomplished with modern technology such as magnetic resonance imaging, computed tomography, balanced

anesthesia regimens, advanced surgical instrumentation, and force plate analysis? Throughout history, great minds have been limited only by the technology of the time.

Perhaps, it is the lack of technology available at the time he made his observations that makes Hippocrates’ findings so significant. Hippocrates was forced to substitute *thought* and *reasoning* for technology. He actually had to think through problems, and to reason solutions. Hippocrates had to observe what was in front of him and describe things for the first time. Because of his thorough study of spinal diseases and their management, which was the first such study in orthopedics in the history of medicine, Hippocrates should be regarded as the *father of spine surgery*.

Now let’s jump forward to the present day of *evidence-based veterinary medicine*, where technology is available to address some of the questions previously addressed by means of thought and reasoning alone.

Evidence-based veterinary medicine may be defined as the conscientious, judicious, and explicit use of current best evidence in making decisions about individual patients. The practice of evidence-based veterinary medicine means integrating *individual clinical expertise* with the *best available external clinical evidence* obtained from systematic research.

- *Individual clinical expertise* describes the proficiency and judgment that individual clinicians acquire through clinical experience and clinical practice. This includes the essential aspects of the Hippocratic method—namely, accurate observation and sound reasoning.
- *Best available external clinical evidence* describes an aspect of evidence-based medicine that was denied to Hippocrates because of a lack of technology—namely, the results of clinically relevant research.

It should be noted that the best available external clinical evidence is not restricted to double-blinded, randomized, placebo-controlled, prospective trials with quantifiable and reliable outcome assessments. Nor is it restricted to meta-analyses. Results of such studies may be considered the “gold standard” for some types of evidence, particularly when addressing the efficacy of a particular therapeutic approach. However, results of such studies cannot take the place of the individual clinical expertise that is necessary to decide whether the external evidence applies to an individual patient, and if so, how it should be integrated into a clinical decision.

The chapters included in this book are written by authors who understand that evidence-based veterinary medicine is not “cookbook” veterinary medicine. The authors also understand that management of intervertebral disc (IVD) disease in an individual patient requires careful integration of both the best external evidence and the best individual clinical expertise. Without individual clinical expertise, the management of IVD disease risks becoming “tyrannized” by evidence, for even outstanding external evidence may be inappropriate for an individual patient or an individual situation. On the other hand, without the continual generation of best available external clinical evidence, management of IVD disease risks becoming outdated to the detriment of patients.

Several major developments have led to the present base of knowledge that permits the publication of this book. In this context, I would like to attempt an explanation of the reasons I am well qualified to write the Foreword to this extraordinary book.

In 1951, Dr Sten-Erik Olsson (1921–2000) published his doctoral thesis titled “On Disc Protrusion in the Dog.” Sten-Erik was both a human physician and a veterinarian, and an imposing figure in every sense of the word.

At a 1986 conference on IVD disease in Lund, Sweden, Sten-Erik and I were speakers on the program along with a legend in the field of vertebral column veterinary radiology, Dr Joe Morgan. After each of my presentations, Sten-Erik would correct all the “information” I had shared, not privately, but in front of the entire conference. At one stage, he had prepared his own set of slides to project after one of my lectures to debate some of the assertions I had made! I learned more during those days than anyone else at the conference. For example, I learned that data are not the plural for anecdote and that the beginning of wisdom is to call things by their correct names. In addition, I survived a focused lesson in evidence-based veterinary medicine. This experience profoundly influenced my career.

The face page of Sten-Erik’s landmark publication is something I still retain and cherish, along with Sten-Erik’s message to me written after we had been debating aspects of IVD disease for several days. The significance of this publication, as Sten-Erik stated to the entire audience, was that he had published this landmark work “in the year Rick was born.” This publication is a reminder of the importance of distinguishing data, information, knowledge, understanding, and wisdom, in the application of clinical expertise to an individual patient with clinical signs of IVD disease.

Sten-Erik and I remained good friends until he died in 2000. In my opinion, Sten-Erik Olsson is “the Hippocrates of canine IVD disease.” It was during those valuable lessons he imparted all those years ago that I learned that individual clinical expertise and best available evidence must be applied *together* to effect the best strategies for management of IVD disease in an individual patient.

It is an honor to have been asked to write the Foreword to this book, edited by two of the most experienced and talented veterinarians in the fields of surgery, neurology, and neurosurgery. I have been privileged to have known Drs Fingerhuth and Thomas for many years. Careful reading of the Preface of this book reveals much about these two clinicians, researchers, and teachers, and I highly recommend that the Preface be read before a reader proceeds to delve into the chapters within this volume. The contributions of a remarkable group of authors have been included in this most comprehensive body of information on IVD disease in dogs and cats to date. The authors have

successfully combined the Hippocratic method of *accurate observation and sound reasoning* with the *best available external clinical evidence* to produce a landmark work.

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Sackett DL et al.: Evidence based medicine: What it is and what it isn't. *BMJ* 312:71–72, 1996.

# Foreword

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The American College of Veterinary Surgeons Foundation is excited to present *Advances in Intervertebral Disc Disease in Dogs and Cats* in the book series titled *Advances in Veterinary Surgery*. The ACVS Foundation is an independently chartered philanthropic organization devoted to advancing the charitable, educational, and scientific goals of the American College of Veterinary Surgeons. Founded in 1965, the ACVS sets the standards for the specialty of veterinary surgery. The ACVS, which is approved by the American Veterinary Medical Association, administers the board certification process for diplomates in veterinary surgery and advances veterinary surgery and education. One of the principal goals of the ACVS Foundation is to foster the advancement of the art and science of veterinary surgery. The Foundation achieves these goals by supporting investigations in the diagnosis and treatment of surgical diseases; increasing educational opportunities for surgeons, surgical residents, and veterinary practitioners; improving surgical training of residents and veterinary students; and bettering animal patients' care, treatment, and welfare. This collaboration with Wiley-Blackwell will benefit all who are interested

in veterinary surgery by presenting the latest evidence-based information on a particular surgical topic.

*Advances in Intervertebral Disc Disease in Dogs and Cats* is edited by Drs James Fingerroth and William Thomas. Dr Fingerroth is a diplomate of the American College of Veterinary Surgeons, and Dr Thomas is a diplomate of the American College of Veterinary Internal Medicine (Neurology). Both are prominent in their fields of neurosurgery and neurology, particularly as it applies to intervertebral disc disease of dogs and cats. They have assembled the leaders in this field presenting the structure and function of the intervertebral disc, the pathophysiology of disc disease in dogs and cats, the clinical features of intervertebral disc disease, and the surgical management of this important condition. They conclude this series with future directions in the field. The ACVS Foundation is proud to partner with Wiley-Blackwell in this important series and is honored to present this book in the series.

Mark D. Markel  
Chair, Board of Trustees  
ACVS Foundation



# Preface

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Clinical syndromes related to the vertebral column are among the most common and consternating problems addressed by general practitioners, emergency clinicians, diagnostic imagers, neurologists, and surgeons. Of the various causes for neck and back pain, pain referred to a limb, gait disturbance, and sphincter disturbance, intervertebral disc disease (IVDD) in its several guises is certainly the most prevalent etiology in dogs and is occasionally identified in cats. And even when the cause for such signs is something other than IVDD, there is often a *presumption* of IVDD by many clinicians who too often assume that IVDD underlies all such spinal syndromes. Yet, despite the commonness of clinical disorders related to the intervertebral disc, and decades of clinical experience in diagnosing and treating these disorders, there remains among all of the aforementioned groups of veterinarians (and their staffs and clientele) a substantial and sometimes astounding array of differing thought processes with respect to the criteria for appropriate diagnostic testing, the type and timing of interventions for treatment, and the prognostication for outcome. Moreover, while IVDD has been addressed in its various component parts in scattershot fashion in journals and previous texts, there has never been a single comprehensive source focused solely on all the aspects related to the intervertebral disc and its

diseases. While there may not be one universally agreed-upon consensus that can be derived from the extant knowledge base on how to diagnose and treat IVDD, there should be an effort to at least get everyone “on the same page,” and identify those things we know as facts, those things we speculate are true but perhaps lack sufficient evidence for, and those areas that, even as they remain controversial, ensure that we establish a common framework for arguing our theories.

To this end, the book herein is an attempt to identify areas of established fact as well as areas of established controversy, with an effort to cogently describe what we know, what we don’t know, and what we speculate is true in all the areas related to intervertebral disc disease. As with any textbook, we expect subsequent discoveries and developments to confirm some of the material presented, refute some material, and perhaps render some of it obsolete. But, even in the face of such natural expansion of knowledge, we anticipate that this book will remain a solid foundation from which present and future readers will still be able to find, in a single source, a compilation of subject areas, topics, and expression of opinion that will continue to be germane to the understanding and treatment of IVDD.

As experienced clinicians working in academia and referral centers, we have been exposed to the

wide array of thought processes, dogma, and extreme variations in knowledge that guides our veterinary students, interns, residents, general practitioners, emergency clinicians, and fellow specialists when considering the issue of “IVDD.” Each of these groups of veterinary professionals is our target audience in presenting this book. For the specialists, our goal is to compile, in a single place, all the currently existing reference material that touches upon issues related to the intervertebral disc in dogs and cats. For the nonspecialists, our goal is to provide a core of material that will enhance their understanding of IVDD, help them in dispensing with dogma, and provide a sound scientific and clinical basis for decision making as they confront patients suspected of having some form of IVDD. We may not settle every area of controversy, and we expect some of what is

presented to perhaps stir up new controversy. But regardless of our ability to “settle, once and for all” any of the disagreements that exist in our understanding or management of IVDD, we hope this book at least establishes a common playing field, where each of these controversies are acknowledged and discussed, and where each of the reader groups mentioned can more fully understand the basis for any disagreements or controversies that persist. If we are successful, we anticipate this book becoming a valuable reference to the clinician on the front lines, perhaps drawing readers to a more centralized and common understanding of IVDD and its management, and ultimately improving the quality of care offered to our patients.

JMF  
WBT

# Acknowledgments

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This book is part of a series sponsored by the ACVS Foundation. To the Foundation trustees I express my deep appreciation for approving this project initially, and then waiting patiently for it to be completed. It is my sincere hope that the final product has matched their expectations, and will sit proudly next to the other excellent texts in the series *Advances in Veterinary Surgery*.

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James M. Fingerroth

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William Thomas

# Section I

## Intervertebral Disc Structure and Function

The causes and consequences of intervertebral disc (IVD) degeneration and clinical disease are best understood in the context of the normal anatomy, physiology, and biomechanics of the IVD itself and its relationship with spinal function. In the following chapters, these fundamental principles are elucidated. And because IVD disease (IVDD) afflicts both domestic animals and

humans, we include a chapter that compares and contrasts IVDD in dogs and people. The last has esoteric value, but also can be clinically useful in that many clients have or know someone who has “back problems” or a “slipped disc,” and may make erroneous assumptions and extrapolations when trying to understand IVDD that has been diagnosed in their pet.



# 1

# Embryology, Innervation, Morphology, Structure, and Function of the Canine Intervertebral Disc

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John F. Innes and James Melrose

## Introduction

The intervertebral disc (IVD) is composed of a disparate collection of connective tissues of differing structure and function, and it is the dynamic interplay of these components in the composite IVD which endows it with its unique ability to withstand tensional stresses, to act as a visco-elastic hydrodynamic weight-bearing cushion, and to provide spinal flexibility [1]. While the cross-sectional area and angulation of IVDs vary with spinal level, all share common structural features. The outer region of the IVD, the annulus fibrosus (AF), is a collagen-rich tissue, while the central region of the IVD, the nucleus pulposus (NP), is rich in proteoglycans. The intervening region between the AF and NP is called the transitional zone (TZ). The areas of the IVD that interface with the adjacent vertebral bodies are called the cartilaginous end plates (CEPs); these are hyaline-like cartilaginous tissues containing cells of a rounded chondrocyte-like morphology.

## Embryology of the IVD

During gastrulation, three somatic germ cell layers are initially laid down in the developing embryo: outer ectodermal, middle mesodermal, and inner endodermal layers [2–4]. A midline longitudinal rod-shaped column of the mesoderm, the notochord, subsequently develops from cell aggregates located between the ectoderm and endoderm and establishes cranial/caudal and ventral/dorsal axes in the developing embryo [2]. Ectoderm dorsal to the notochord gives rise to the neuroectoderm from which the neural tube develops. Adjacent mesodermal tissue develops into discrete tissue units, termed as the somites [5]. The somites consist of three tissue types: (1) the dermatome which gives rise to the dermis, (2) the myotome which gives rise to the axial musculature, and (3) the sclerotome from which vertebral structures arise. Cells of the sclerotome migrate medially and ventrally to form a continuous tube of mesenchymal cells (the perichordal sheath)

which surround the notochord. Increased proliferation of cells at regular lengths along the perichordal tube creates areas of low and high cell density from which the vertebrae and AF, TZ, and spinal ligaments develop [5]. Formation of the vertebral bodies results in segmentation of the notochord. Each notochordal segment persists in the central region of the developing IVD to give rise to the NP [3]. Thus, during embryonic disc development, cells of the AF are derived from the sclerotome, whereas the NP originates from the notochord [3]. In nonchondrodystrophoid breeds, notochordal cells persist into adulthood, whereas in chondrodystrophoid breeds they disappear within 2 years of birth. This correlates with an earlier onset of IVD degeneration in chondrodystrophoid breeds.

### Innervation of the IVD

There are major neuroanatomical differences between the human and canine spines in terms of how far the spinal cord extends along the vertebral canal. In humans, the spinal cord extends as far as the second lumbar vertebra with nerves exiting the spinal cord descending inside the remaining lumbar and sacral vertebral segments to exit through their respective foramina. The spinal cord in dogs ends at approximately L6 with nerves that serve the IVDs descending through the last lumbar, sacral and coccygeal vertebral segments. The canine cervical IVDs are served by 8 pairs of nerves, the thoracic IVDs have 13 pairs, the lumbar IVDs have 7 pairs, and the coccygeal region contains 2 nerves per IVD.

The human lumbar IVD is innervated by several nerves. The sinuvertebral nerve (meningeal rami) innervates the posterior (i.e., dorsal) aspect of the disc and the posterior (dorsal) longitudinal ligament. Branches from the rami communicantes innervate the lateral aspects of the disc and the anterior (ventral) longitudinal ligament [6]. A structure similar to the sinuvertebral nerve is not apparent in the canine thoracolumbar spine and in contrast to the human IVD, sensory nerves are sparse in the outermost annular lamellae. However, the dorsal longitudinal ligament is innervated profusely [7]. The nerves in the outer AF communicate with caudal and cranial spinal levels two positions removed from the actual site of annular innervation,

which explains the referred pain reported at sites distant from damaged annular nerves.

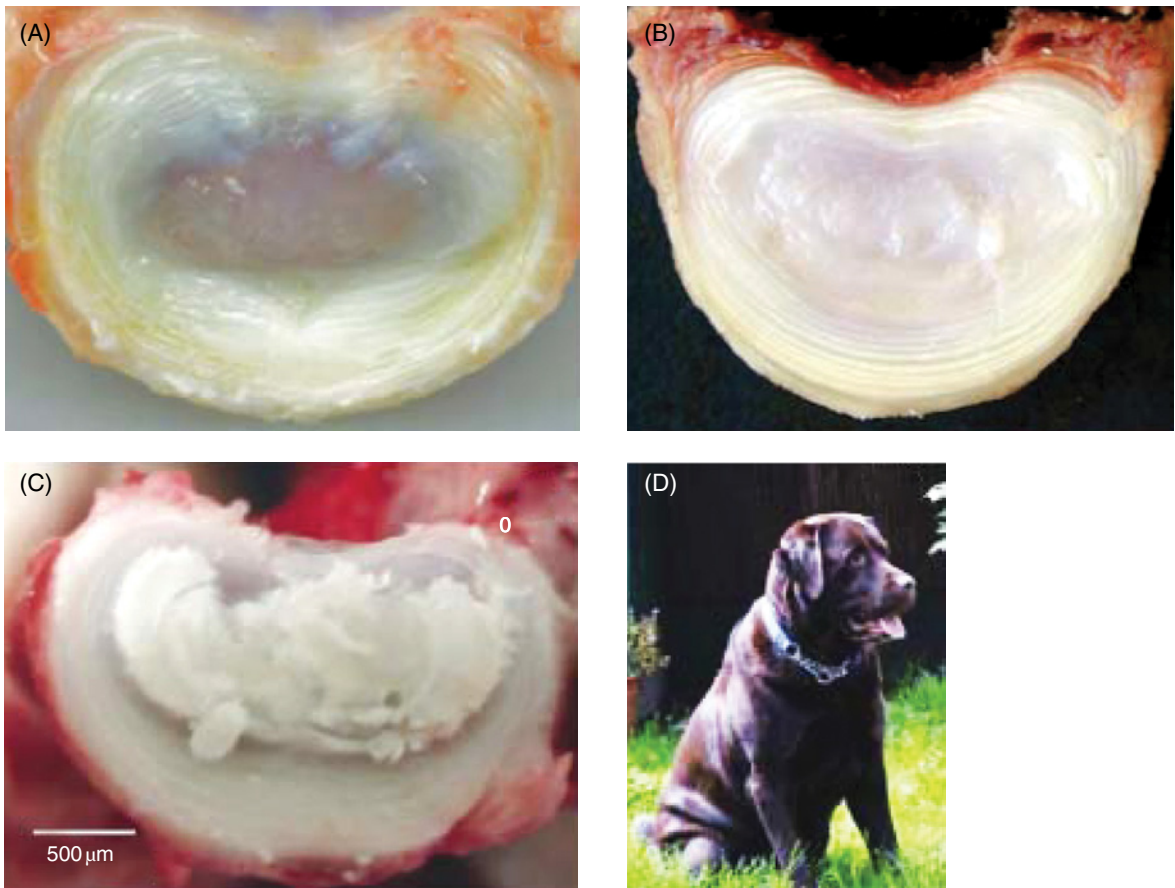
Obvious postural differences in man and dogs and effects on IVD loading contribute to differences in the resolution of forces along the spine and the incidence and distribution of spinal neurological deficits of clinical relevance [8, 9]. The upright stance of humans results in axial spinal forces being transferred down the spinal column to the lumbar region and it is this region that has the highest incidence of IVD degeneration. Posterior lumbar IVD prolapse in man can lead to significant generation of sciatic pain and impairment in mobility; however, paralysis is rarely encountered. In the canine spine, the juncture of the immobile thoracic and mobile lumbar spine is the region that has the highest incidence of disc herniation. Furthermore, since the spinal cord extends to this level in dogs, compression of the spinal cord by extruded disc material can have a significant neurological impact [10–12]. IVD degenerative diseases are generally more common in the chondrodystrophoid breeds than nonchondrodystrophoid breeds and more prevalent in older than younger dogs [13, 14] (Figure 1.1). The clinical presentation of thoracolumbar disc herniation in dogs can be severe with profound paralysis of their pelvic limbs from the resulting spinal cord damage [15]. The thoracolumbar vertebral canal is almost entirely filled by the spinal cord, and there is very little extradural space, which explains why herniations in canine thoracolumbar IVDs are so debilitating [16].

### IVD morphology, structure, and function

The immature nonchondrodystrophoid canine IVD has an extremely gelatinous NP that with age becomes progressively more fibrous and less hydrated with the decline in proteoglycan levels (Figure 1.1 A). IVDs of chondrodystrophoid canine breeds have a relatively fibrous NP (Figure 1.1 B). The NP is surrounded by well-defined collagenous annular lamellae (Figure 1.1 B). Calcification of the NP occurs in the chondrodystrophoid canine breeds but infrequently in nonchondrodystrophoid dogs (Figure 1.1 C).

The annular lamellae contain collagenous fibers of type I and II collagen, which comprise 40–60%

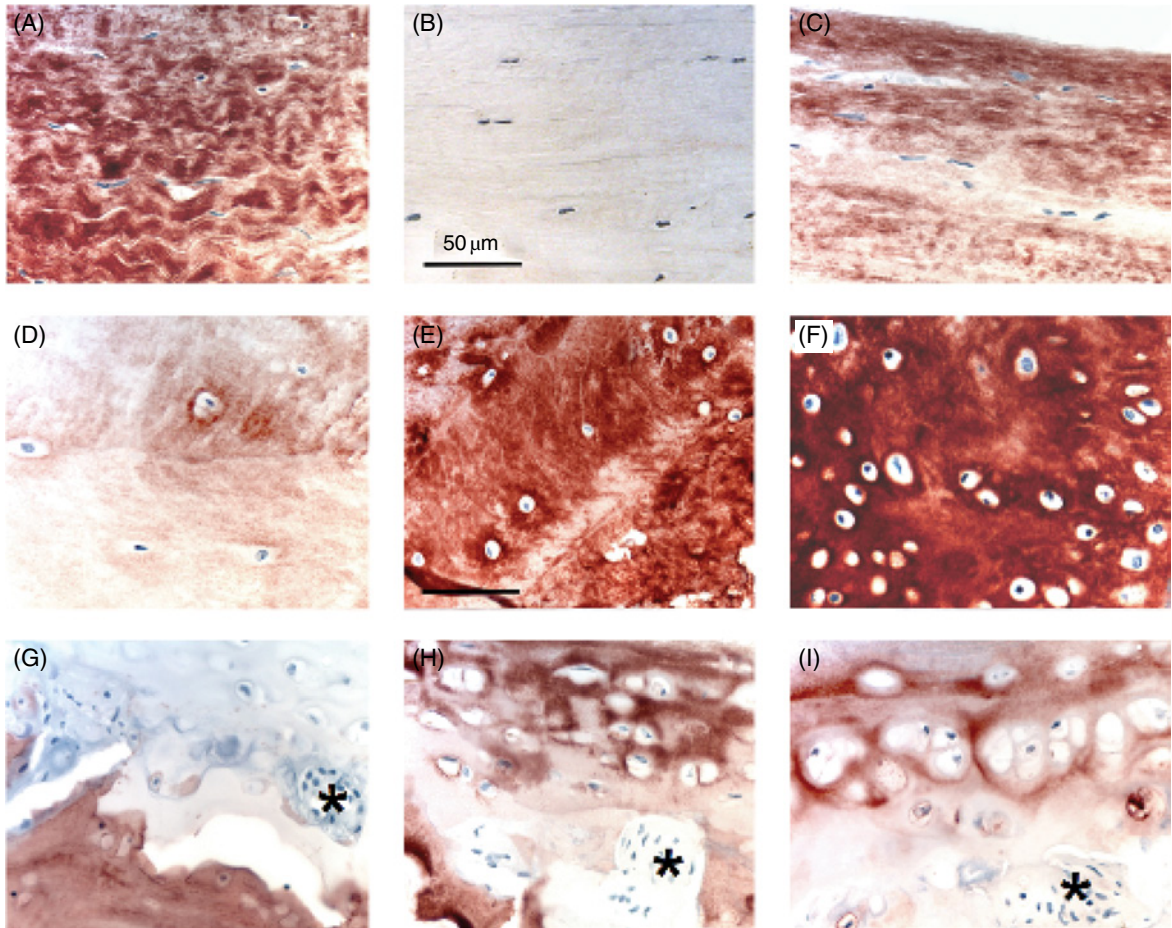




**Figure 1.1** Composite figure depicting macroviews of horizontally bisected lumbar intervertebral discs demonstrating their characteristic morphology with the peripheral annular lamellae clearly evident and central nucleus pulposus. Discs typical of nonchondrodystrophic (A) and chondrodystrophic (B, C) canine breeds are shown. In plate (A), the nucleus pulposus is gelatinous, while in (B) the nucleus pulposus has a fibrous consistency typical of a chondrodystrophic breed (beagle). In plate (C), the entire nucleus pulposus has undergone calcification (Hansen type I). Plate (D) depicts an example of typical nonchondrodystrophic canine breed, labrador retriever. Labradors typically present with disc degeneration at ages of 5–12 years. Figures (A) and (C) supplied by courtesy of Dr PN. Bergknut, Faculty of Veterinary Medicine, Utrecht University.

of the dry weight of the outer annulus and 25–40% of the inner annulus. Type I and II collagens are radially distributed in opposing gradients from the disc periphery to the NP with the concentration of type I collagen greatest in the outer AF, while type II collagen predominates in the NP (Figure 1.2 A, B, D, and E). The tension-bearing properties of the AF are principally conveyed by type I collagen fiber bundles; however, the resistance to compression provided by the NP is provided by proteoglycans (aggrecan) and their associated hydration entrapped within a type II collagen network

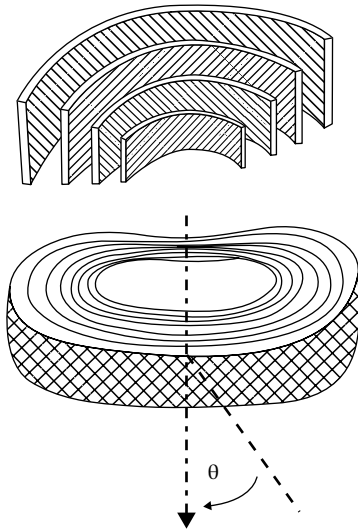
(Figure 1.2C and F). Collagen fibers are virtually inextensible and their major role is in the provision of tensile strength. Elastin fibers located in intralamellar margins interconnect adjacent lamellae and return the fully extended collagen fibers to their preloaded dimensions. The elastin content of the IVD is small (1–2%) but nevertheless essential in the provision of elastic material properties [17]. Type I collagen fiber bundles insert firmly but imperceptibly with the CEPs and underlying vertebral bone to form anchorage points for the IVD to adjacent bony structures (Figures 1.2G–I).



**Figure 1.2** Composite figure depicting the immunolocalization of type I (A, D, G) and type II collagen (B, E, H) and the major space-filling and water-imbibing disc proteoglycan aggrecan (C, F, I) in the outer annulus fibrosus (A–C), inner annulus fibrosus/nucleus pulposus (D–F), and cartilaginous end plate (G–I). Plate (A) depicts strong localization of type I collagen displaying a crimp pattern in the outer annulus fibrosus. This is consistent with the hoop stresses generated within and tensional forces carried by this tissue. The outer annulus fibrosus is devoid of type II collagen (B) while it contains a sparse distribution of aggrecan (C). The characteristic elongated fibroblastic morphology of the annular cells is also evident (A–C). The inner annulus fibrosus/nucleus pulposus contains a little type I collagen (D) but is rich in type II collagen (E) and aggrecan (F). The cells in this region of the intervertebral disc display a characteristic rounded morphology (E–F). The cartilaginous end plate is a hyaline cartilage-like tissue that forms the interface of the intervertebral disc with the vertebral bodies (G–I). This tissue also contains cells of a rounded chondrocytic morphology surrounded by type II collagen (H) and aggrecan (I) but does not contain type I collagen (G), while the underlying vertebral bone is stained positively for type I collagen (G). The cartilaginous end plate has important roles to play in the nutrition of the disc cells with small blood vessels (\*) clearly in evidence in the underlying vertebral vascular bed (G–I). The intervertebral discs shown are vertical midsagittal sections from an L1–L2 disc of a 2-year-old French bulldog, a typical chondrodystrophic canine breed.

The NP acts as a viscoelastic hydrodynamic cushion that counters compressive loading of the spine. Upon axial loading of the spine, compression of the NP results in load transference to the AF which is arranged in collagenous lamellar layers

with collagen fiber bundles arranged at a 50–60° angle relative to one another in adjacent lamellae (Figure 1.3). This results in bulging of the annular lamellae with the generation of hoop stresses that dissipate axial compressive forces.



**Figure 1.3** Schematic depiction of the lamellar structure of the annulus fibrosus in a partially exploded view and surrounding the central nucleus pulposus with the parallel arrays of collagen fiber bundles indicated oriented at 50–60° (q) relative to collagen fiber bundles in adjacent lamellae in the transverse plane.

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# 2 Biomechanics of the Intervertebral Disc and Why Do Discs Displace?

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Lucas A. Smolders and Franck Forterre

## Biomechanical function of the healthy intervertebral disc

From a biomechanical viewpoint, the intervertebral disc (IVD) can be regarded as a water-filled cushion that mediates and transmits compressive forces between vertebral bodies and provides mobility as well as stability to the spinal segment [1–4]. The IVD functions in relation to the ligamentous apparatus of the spine, which consists of the interspinal, interarcuate, dorsal longitudinal, and ventral longitudinal ligaments and the annulus fibrosus of the IVD. The healthy IVD exerts a high swelling pressure, which accounts for the separation of contiguous vertebrae. The separation of adjacent vertebrae creates constant tension on the spinal ligaments, preventing uncontrolled displacements that would cause stress peaks of the vertebrae. Therefore, the IVD creates the necessary tension for optimal functionality and stability of the ligamentous apparatus of the spine [4].

The healthy IVD is composed of three distinct components: the nucleus pulposus (NP), annulus fibrosus (AF), and cartilaginous end plates (EPs).

Each component exhibits specialized physical-mechanical properties and specific biomechanical functions. The collaboration of these individual structures results in optimal biomechanical function of the IVD.

The healthy NP is composed of approximately 80% water. This high water content results in a high intradiscal swelling pressure that allows the NP to serve as a hydraulic cushion transmitting compressive forces while providing spinal mobility and stability [1–3, 5–9]. The NP is surrounded ventrally, dorsally, and laterally by the AF, with the ventral part of the AF being 2–3 times thicker than the dorsal part [10–12]. The fibers of the AF provide reinforcement when the IVD is twisted (axial rotation), bent (flexion/extension), and/or compressed (axial compression), with the inner and outer AF mainly resisting compressive and tensile forces, respectively [5, 6, 8]. The AF contains the NP, preserving its internal swelling pressure and protecting it against shearing [5, 6, 8, 13].

The cranial and caudal borders of the IVD are formed by the cartilaginous EPs, situated between the disc and the epiphyses of the respective cranial



and caudal vertebral bodies [5, 11, 14]. The EPs are partially deformable due to their high water content (50–80%) and serve to contain the NP during loading of the spine [15].

All in all, this IVD can be viewed as an inflated tire, with the NP providing intradiscal pressure and resistance to compressive loads, the AF coping with tensile forces, and the partially deformable EP containing the NP. Owing to the specialized conformation of these structurally and functionally divergent entities, the IVD concurrently provides mobility and stability to compressive, tensile, and shear stresses applied to the spine [2, 5, 6, 8].

## Biomechanical failure of the IVD

Degeneration of the IVD is the fundamental process that lies at the root of most IVD displacements. Due to this degeneration, the NP loses the ability to absorb and maintain water and thereby to function as a hydraulic cushion [16–20]. Consequently, more of the compressive load bearing, which is normally resisted by the hydrated NP, is transmitted to the AF [21–23]. This results in a compensatory increase in functional size of the AF [21, 24–26]. However, the AF is not built to resist compressive forces, and the increase in functional size consists of biomechanically inferior matrix [17–19, 25]. As a result, the AF becomes stiffer and weaker leading to structural failure that impedes the ability of the AF to resist tensile forces and to contain the NP. Eventually, these degenerative changes result in outward bulging of the IVD when subjected to physiological loading [16]. In addition, structural failure of the AF can result in annular defects or tears, through which degenerated NP material can extrude and which further compromise the function of the IVD [12, 16]. In essence, the degenerated IVD functions as a flat tire, being unable to cope with physiological loading, with consequent displacement of the IVD. Since the dorsal AF is 2–3 times thinner than the ventral AF, the dorsal side is usually where the AF shows structural failure and IVD displacement. In addition to structural failure of the NP and AF with consequent disc displacement, degeneration of the NP and AF results in an uneven distribution of load onto the EP, making the EP more susceptible to damage [27]. Although the EPs are deformable when axially loaded, they are a weak link within the IVD [13].

Degeneration of the IVD can cause cracks in the EP [28–30]. The degenerated NP can displace through these EP defects, which is referred to as a Schmorl's node [31].

Although displacement of the IVD is commonly the result of IVD degeneration, displacement can also occur as a result of strenuous exercise or trauma. This type of IVD displacement involves abrupt extrusion of nondegenerated NP material through the dorsal or dorsolateral AF and is referred to as acute noncompressive nucleus pulposus extrusion [12, 32–34] (see Chapter 13).

The biomechanical alterations involved in degeneration of the IVD can occur in any dog. However, when speaking of actual herniation of the IVD, a clear distinction can be made between chondrodystrophoid (CD) and nonchondrodystrophoid (NCD) dogs. Chondrodystrophoid and NCD dogs display significant differences in the character, age of onset, prevalence, and spinal location of IVD displacement. Due to these distinct differences, it is conceivable that the etiological factors for IVD displacement are different between these two groups of dogs.

## Displacement of the IVD in CD dogs

Chondrodystrophoid dog breeds are characterized by an accelerated form of IVD degeneration [12]. Consequently, the NP abruptly loses its hydraulic function, with consequent degenerative changes in the AF [12]. This predisposes to explosive herniations of the IVD, with complete rupture of the AF and dorsal longitudinal ligament, and extrusion of the NP into the vertebral canal [12]. These disc displacements are referred to as Hansen type I IVD herniations [12, 35–37].

Another remarkable feature of CD dogs is that IVD degeneration occurs throughout the entire vertebral column [12, 38, 39]. Therefore, biomechanical factors inherent to individual spinal levels seem to be of less importance to IVD degeneration in CD dogs. At first impression, a logical biomechanical factor in CD dogs may be the disproportion between the length of the spine and the length of the legs. However, there is no correlation between a relatively long spine and IVD degeneration in these dogs [12, 28]. Moreover, CD dogs with a relatively shorter spine, a larger height at the withers, and a large pelvic circumference are at

higher risk for IVD herniation [29]. In CD dogs, all IVDs show signs of degeneration at an early age; therefore, a genetic component linked to the CD trait causing aberrant synthesis of the NP extracellular matrix appears to be the main etiological factor [12, 30, 38, 40]. However, causative factors for the high susceptibility of IVD herniation at certain spinal levels (cervical and thoracolumbar spine) in CD dogs are still unclear. It has been proposed that the transition from the rigid, thoracic spine to the more flexible, lumbar spine is a causative biomechanical factor for herniation of thoracolumbar IVDs in CD dogs; however, definitive evidence to support this theory is still lacking [41]. In contrast, it is well known that IVD displacement is seldom seen in the midthoracic spine (T1–T9). This may be because of the intercapital ligaments present ventral to the dorsal longitudinal ligament at each level from T1–T2 to T9–T10 [12, 42, 43]. These ligaments may prevent dorsal and dorsolateral displacement of the IVD at these levels [12, 36].

## Displacement of the IVD in NCD dogs

In NCD dogs, degeneration of the IVD occurs more gradually [17, 18]. Degeneration of the AF can occur independently of NP degeneration, and mainly consists of partial ruptures of the AF fibers [12]. Due to the more gradual character of IVD degeneration in NCD dogs, lesions in the IVD are less dramatic, generally characterized by partial herniation of the NP through a partial rupture of the AF, and protrusion of the IVD and dorsal longitudinal ligament. This type of disc displacement is referred to as Hansen type II IVD herniation [12].

In NCD dogs, IVDs at specific spinal levels, such as C5–C7 and L7–S1, have the highest rate of degeneration and displacement [12, 44–47]. Therefore, it seems that in NCD dogs biomechanical factors related to individual spinal levels play a key role in degeneration and consequent displacement of the IVD.

In the cervical spine of large-breed NCD dogs, the caudal cervical spine is at the highest risk for developing IVD degeneration and displacement [44, 45, 48]. This may be related to the conformation of the facet joints of the caudal cervical spine. Due to their shape and conformation, the facet joints of the caudal cervical spine allow considerably more axial rotation and can induce significantly more spinal

instability compared to more cranial spinal segments [49, 50]. Therefore, the workloads and stresses on the IVDs of the caudal cervical spine may be relatively high, thereby promoting IVD degeneration and displacement at these locations [51].

The L7–S1 disc of NCD large-breed dogs is also frequently affected by IVD displacement [46, 52–54]. This may be related to the conformation and mobility of the L7–S1 junction in these breeds. The conformation of the IVD and facet joints of the L7–S1 spinal segment permits considerable mobility in flexion/extension, axial rotation, and ventrodorsal translation [51, 55–58]. Also, the L7–S1 junction in large-breed dogs is subject to proportionally higher loads due to an imbalance between body weight and the dimensions of the lumbosacral contact area (IVD and facet joints) as compared to smaller dogs [57]. These factors indicate that the L7–S1 IVD of large-breed dogs is subject to relatively high workloads and stress (wear and tear), predisposing the disc to degeneration and structural failure, with consequent displacement of the IVD.

In addition to these factors for NCD dogs in general, specific biomechanical factors apply to the German shepherd dog, which is highly predisposed to degeneration and displacement of the L7–S1 IVD [53]. In this breed, the orientation of the L7–S1 facet joint causes a disproportionately high workload in the L7–S1 IVD [46, 47, 51]. Also, lumbosacral transitional vertebrae are relatively common in the German shepherd dog. This developmental abnormality is associated with abnormal mobility and distribution of force on the lumbosacral IVD [59–61]. Last, the German shepherd dog is predisposed to sacral osteochondrosis, which involves degeneration and fragmentation of the sacral end plate [62–64]. Besides aberrant nutritional supply to the IVD, the abnormal shape of the sacral end plate may cause aberrant mechanical loading of the IVD [62–64]. These specific factors in the German shepherd dog may cause aberrant loading of the IVD with consequent IVD degeneration and displacement.

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# 3 Comparisons between Biped (Human) and Quadruped (Canine/Feline) Intervertebral Disc Disease

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There are many similarities between the canine and human spines and their vertebral components. The vertebral units comprising two adjacent vertebrae—an intervertebral disc and facet joints—are comparable in both species. The human spine averages 33 vertebrae, whereas the canine spine generally averages 50 [1]. Humans generally have 12 thoracic and 5 lumbar vertebrae, whereas dogs generally have 13 thoracic and 7 lumbar vertebrae.

Intervertebral disc (IVD) degeneration and herniation are considered the main causes of acute and chronic low back pain in humans [2–6], with a lifetime prevalence of over 70% in the global population [7]. Also, dogs commonly suffer from back pain caused by degenerated IVDs subsequently herniating; it is in fact the most common cause of neurological deficits in dogs [8] and is one of the most common causes for euthanasia in dogs less than 10 years of age [9]. Moreover, the clinical presentation, macroscopic and microscopic appearance, diagnostics, and treatment of IVD degeneration and herniation are similar in humans and dogs [6, 10–12].

Decompressive surgery and spinal fusion are common treatments for IVD disease (IVDD) in both humans and dogs, with fusion less often used in the latter species.

The many similarities between canine and human spines and their diseases have led to dogs being frequently used for translational studies of surgical procedures and biomechanical studies of the spine [13–18]; and recently, the dog has been proposed as a translational model for acute spinal cord injury (SCI) [19] as well as for IVD degeneration [20].

## Anatomy and gross pathology

The occurrence of asymptomatic degenerated IVDs is common in both dogs and humans [21, 22]; and in general, similar pathological changes have been reported in degenerated IVDs from humans and dogs [23]. A notable difference between human and canine vertebrae is the absence of growth plates in growing human vertebrae and the thicker cartilaginous end plates in humans.