Degenerative and Compressive Structural Disorders (29-Jan-2003)

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In this chapter we will review a variety of neurological disorders that result from abnormalities of bones, ligaments, and other mesenchymal tissue that compress the nervous system. Most of these conditions are degenerative in nature, although some represent developmental disorders. The spinal cord and occasionally nerve roots are the nervous tissues most commonly compressed. One condition, disk disease, represents the most common cause of spinal cord compression in dogs.

An outline of this chapter is as follows:

Calcinosis Circumscripta/tumoral Calcinosis
Cervical Spondylomyelopathy
Disk Disease
Diskospondylitis
Dural Ossification
Lumbosacral Stenosis
Spinal Synovial Cysts
Spondylosis Deformans
Miscellaneous Disorders

Calcinosis Circumscripta/tumoral Calcinosis
Focal nodular aggregations of ectopic calcification occurring in the soft tissues have been referred to by several synonyms that include calcinosis circumscripta, apocrine cystic calcinosis, tumoral lipocalcinosis, lipocalcinosis, calcium gout and tumoral calcinosis. In man [1,2] the term calcinosis circumscripta is used to distinguish between those generally smaller nodular calcareous lesions occurring in the skin, subcutaneous sites and skeletal muscle and those histologically similar but often larger multinodular lesions of tumoral calcinosis occurring in the deep peri-articular sites, i.e. soft tissues located adjacent to joints. This nomenclature is beginning to be more commonly accepted to describe similar distribution patterns of nodular deposits of ectopic calcification in animals [3-5].
In the veterinary literature calcinosis circumscripta and tumoral calcinosis have been used as synonyms to describe histologically similar idiopathic conditions characterized by the formation of circumscribed single or multiple nodular masses due to deposition of calcinous material, located either in periarticular connective tissue or in cutaneous tissues over pressure points and bony prominences, in footpads, or in the mouth [6-11]. Etiology and pathogenesis remain obscure. Most reports involve dogs, but the condition has occasionally been seen in cats [12]. Concurrent illness is usually not associated with the different patterns of nodular ectopic mineralization in animals; however, some animals may have underlying renal disease [4-6,13,14]. The prevalence of the condition in young large-breed dogs, particularly German Shepherds, Great Danes and Viszlas, suggests possible hereditary predilection. Calcinosis circumscripta has occasionally been seen following surgical procedures, use of polydioxanone suture material, and progestin (medroxyprogesterone acetate) injections in dogs and cats [15-19]. Calcinosis circumscripta-like lesions have also been reported in dogs associated with the use of choke chains [20]. The pattern of deposition referred to as tumoral calcinosis usually does not directly involve bones or joints [4,8,11]. Clinical signs include one or more hard or fluctuant, spherical, well-circumscribed, non-painful subcutaneous masses [8]. When incised, the masses discharge a chalk-like material. Histopathologically, the masses are characterized by lobular areas of mineralization and degeneration in a fibrous, collagenous matrix with large foamy macrophages, giant cells, lymphocytes, and neutrophils. Some calcific foci are embedded by osseous tissue [4,8]. The mineralized material stains positively for calcium, phosphorus, carbonates and hydroxyapatite crystals. The ground substance stains positive for acid mucopolysaccharides (glycosaminoglycans). The crystalline structures can be identified using scanning electron microscopy

[21]. The heterotopic ossification seen in a 7 year old German Shepherd with fibrodysplasia ossificans may have resulted from the metaplastic change of calcinosis circumscripta lesions [22]. In this dog, several mineralized densities were seen radiographically ventrolateral to the lateral processes of the 6th cervical vertebra.

There have been several reports of calcinosis circumscripta/tumoral calcinosis (CC-TC) causing spinal cord compression in young dogs (usually less than 1 year of age) in several breeds including Bernese Mountain dog, German Shepherd, English Springer Spaniel, Rottweiler, and Great Dane [23-26]. With the exception of upper thoracic cord compression (T2 - T3) in two littermate German Shepherd puppies [25], all CC-TC masses resulting in spinal cord compression have been localized at the atlantoaxial articulation [23,24,26,27], often in the area of atlantoaxial ligament. The focal calcified masses are usually found lying over the spinal cord in the space between the caudal aspect of the dorsal arch of the atlas and the cranial edge of the spinous process of the axis, and extending into the vertebral canal. In the report involving the two young German Shepherd littermates [25], a solitary mineralized mass was found on the dorsal laminae between the dorsal spines of T2 and T3 and impinged ventrally on the intervertebral foramina. The mass projected above the articular facets between the dorsal spinous processes. Hematological and biochemical analysis of blood samples from affected animals are within normal limits, as is cerebrospinal fluid (CSF) evaluation. Clinical signs of CC-TC will reflect either a cervical syndrome or a thoracolumbar syndrome. Myelography will confirm spinal cord compression associated with these masses. Special imaging, such as computed tomography and computed tomographic myelography , may provide additional information on the extent of the mass and on the degree of spinal cord compression [26]. In the cases of CC-TC reported to date with spinal cord compression, the initiating mineralized mass lesion was the only lesion observed radiographically. There has been a report of unilateral and bilateral mineralized masses associated with the deep tendinous attachments on the lateral processes of the caudal cervical vertebrae [28]. In this report, the masses occurred in three related Great Dane puppies (around 5 to 6 months of age) but were non-clinical and only of cosmetic significance. Surgical removal of CT-TC masses in dogs with spinal cord compression is the treatment of choice and prognosis is favorable.

Reports of solitary cartilaginous exostoses in three young, large breed dogs (Rottweiler, Bernese Mountain dog, and St. Bernard), from 3.5 to 5 months of age [29], as well as in a 3.5 year old Bernese Mountain dog [30], may actually be further examples of CC-TC, or, at least variants. In each case, solitary, partially calcified lesions were localized to the atlantoaxial articulation and caused tetraparesis from spinal cord compression. The radiographic features appear identical to those described for CC-TC, including thickening of the dorsal arch of the atlas and malformation and shortening of the spinous process of the axis [23,26,29,30]. The histopathological features of the masses, however, appear to be different from those reported in CC-TC [5]. Surgical removal of the mass was successfully achieved in the older dog [30].

Cervical Spondylomyelopathy
Cervical spondylomyelopathy is a neurological disorder affecting Doberman Pinschers, Great Danes and other large and medium-sized breeds in which abnormalities of the cervical spine cause compression of the spinal cord [31]. Synonyms include wobbler syndrome, cervical malformation-malarticulation, cervical vertebral instability, cervical vertebral malformation, vertebral subluxation, cervical spondylolisthesis, cervical stenosis, caudal cervical spondylomyelopathy, and cervical spondylopathy. Approximately 80% of cases occur in Great Dane and Doberman Pinscher dogs and lesions are generally confined to the caudal cervical spine (i.e., C5 to C7). The age of onset of clinical signs is variable, ranging from 3 months to 9 years. In general, Great Danes are affected less than 2 years of age, while Doberman Pinschers more frequently manifest signs when 2 years of age or older, with a clinical incidence peak between 4 and 8 years [32-34]. Several reports suggest male dogs are more commonly affected [35,36], although no gender predilection was revealed in one study involving 170 dogs [32]. Cervical spondylomyelopathy appears to involve both bony, fibrocartilaginous, and ligamentous abnormalities of the caudal cervical spine.
These include:

a. Chronic degenerative disk disease,
b. Congenital bony malformation (stenosis of the vertebral canal and abnormal articulation of the articular facets),
c. Vertebral instability or "tipping",
d. "Hourglass" compression by the dorsal (ligamentum flavum) and ventral (dorsal longitudinal ligament and dorsal annulus fibrosus) ligaments, and
e. Hypertrophy of the ligamentum flavum. These changes, occurring alone or in combination, can result in caudal cervical spinal cord compression [32].

The cause of cervical spondylomyelopathy remains uncertain but it may be multifactorial. Some authors consider the disease

to be a developmental malformation-malarticulation disorder [35,37,274]. Hereditary factors have been suggested [38,39]. A possible familial trait was suggested for affected Dobermans in New Zealand [32]. Other reports failed to find support for a genetic basis, although recognizing the problem was more prevalent in certain lines of Dobermans [33,35]. Nutritional status has been implicated, including a hypercalorific diet and calcium excess in rapidly growing dogs [40-42], yet cervical spondylomyelopathy does occur in animals reared on a balanced diet [33]. The impact of conformation, such as longer neck and heavier head in affected dogs [42] playing a biomechanical role in this disease, was not confirmed in one study in which radiographic changes were independent of several body dimensions measured [32]. Interestingly, there was a positive correlation between longer rump length and increased risk of neurological disease in this study. Cervical trauma from use of a choker chain was not found to influence the presence of the disease [32].

Clinical signs are related to the severity of spinal cord compression and therefore are variable in nature and degree. Clinical signs usually are first noticed in pelvic limbs. Eventually, all four limbs are affected, with signs often being more pronounced in the pelvic limbs. There is ambulatory tetraparesis with the thoracic limbs moving in a short, choppy manner. The neck is often carried in flexion. There may be varying degrees of atrophy of infraspinatus and supraspinatus muscles. An affected animal can have difficulty rising from lateral recumbency or from a sitting position. The digits may knuckle when the animal walks and nails are often worn excessively as a result of scuffing and dragging. Most dogs have a conscious proprioceptive deficit and demonstrate a wide-based stance. Sometimes pain may be elicited upon neck manipulation. Clinical signs tend to be slowly progressive but can be abrupt in onset when external trauma is suspected as playing a major precipitating role. A Horner's syndrome may be present, perhaps more often in dogs with severely herniated intervertebral disks, especially at the C6 - C7 region [35].

Diagnosis is based on historical data, signalment, clinical data, and radiography. Most dogs have the following radiographic abnormalities [43]:

a. Tipping of the craniodorsal aspect of the vertebral body into the spinal canal which may be exaggerated by neck flexion;
b. Stenosis of the vertebral canal, especially at the cranial aspect of the vertebrae. Normal and abnormal values for vertebral canal dimensions have been established [32,44,275];
c. Malformations of the vertebral bodies (see below);
d. Narrowed disk spaces, often with accompanying spondylosis deformans; and
e. Degenerative changes in the articular facets. These changes may be seen radiographically, alone or in combination.

In adult dogs, the most important abnormalities seen using survey radiography are narrowed intervertebral disk space, vertebral malalignment (e.g., tilting or subluxation), ventral spondylosis deformans, and a misshapen vertebral body [31,35,45]. Vertebral tilting and coning of the vertebral canal with stenosis of the cranial orifice is primarily recognized in dogs less than 1 year of age [31,32]. Vertebral changes may be seen radiographically in puppies as early as 3 months of age [32] and the observation of normal radiographic appearance of cervical vertebrae at 6 weeks of age in one of these puppies suggested the possibility of vertebral deformity occurring between the ages of 6 and 12 weeks.
Many animals have more than one site of compression, which may not be apparent on survey radiography. In one study, plain films were inaccurate in 18 of 45 dogs (40%) [46]. Therefore, myelography is essential to establish an accurate diagnosis and prognosis, especially if surgery is to be considered. Conventional myelography is considered the technique of choice for initial evaluation of dogs with cervical spondylomyelopathy since it provides an image of the entire cervical spine [47]. Furthermore, myelographic studies may reveal various types of extradural spinal cord compression:

a. Dorsal compression associated with hypertrophied ligamentum flavum;
b. Ventral compression from a bulging/hypertrophied annulus fibrosus;
c. Lateral compression from malformation of articular facets;
d. Compression from a stenotic vertebral canal or vertebral instability associated with vertebral tipping [43].

Some clinicians are advocates of stress radiography/myelography (i.e., use of flexion, extension, or traction of the cervical vertebrae) in order to better define the nature of the compressive lesion (dynamic versus static) and better define the subsequent treatment. The measurement of "stepping" of the vertebral floor of cervical vertebrae when the neck is flexed has diagnostic significance [33]. However, complications may follow stress radiography. Hyperflexion of the neck may exacerbate cord compression caused by vertebral tipping and neck hyperextension may aggravate cord compression by closing the dorsal aspect of the intervertebral disk space which can force additional disk material (nucleus pulposus and/or

annulus fibrosus) into the spinal canal. Neck hyperextension may also worsen pre-existing compression from the ligamentum flavum [43] (it remains to be proved whether or not the apparent increase in cervical hyperextension in the Doberman Pinscher breed as a whole, between 1934 and 1972, has a role in cervical spondylomyelopathy [47]). Conversely, with soft tissue lesions, traction on the cervical spine will often reduce the degree of cord compression by stretching or flattening redundant annulus fibrosus and ligamentous structures [31,46]. While non-contrast and intravenous contrast-enhanced computed tomography appears to have little advantage over conventional myelography in cervical spondylomyelopathy [47], other specialized imaging techniques may also play an important diagnostic role, e.g., computed tomography-myelography may provide more information than conventional myelography as to the exact nature and degree of compression, particularly in cases of severe spinal cord atrophy [47].

Gross pathologic findings include stenosis of the rostral end of the involved vertebrae, unstable vertebrae associated with flattened, expanded and elongated articular facets, and hyperplasia of the dorsal annulus fibrosus and ligamentum flavum in young animals. The abnormal relationship of one vertebra to another may result in static or dynamic cord compression. Similar changes occur in older animals along with degenerative lesions affecting the articular facets including osteophyte formation, sometimes with encroachment onto the spinal cord [48]. In older dogs, the fibers of the dorsal annulus fibrosus of the intervertebral disk appear hypertrophic or hyperplastic and may be partially or totally ruptured, with disk material extruded up beneath the dorsal annulus, although herniated disk material into the vertebral canal is infrequent. The nucleus pulposus may show degenerative changes and can be mineralized [35]. Disks at C5 - 6 and C6 - 7 are most frequently affected [33,35,49]. In both young and older dogs, gross vertebral deformity may be found, often involving C6 and/or C7 in Dobermans and Great Danes [33,35,50]. The deformity can vary from rounding off of the cranioventral epiphysis to its total loss producing a triangular wedge-shape. Redundant ligamentum flavum resulting in dorsal cord compression is reported in Great Danes [46]. Similarly, the above-mentioned hourglass compression by dorsal and ventral ligaments as well as joint capsule of the facets, is seen principally in Great Danes [31]. In the spinal cord, varying degrees of compression and spinal cord atrophy may be present. Degenerative changes characterized by white and gray matter necrosis, neuronal loss, and cavitation may be seen at the site of spinal cord compression. At this level, lesions may be seen in all funiculi. Wallerian-like degeneration of white matter is seen above (e.g., ascending tracts in the dorsal funiculi and more superficial portions of dorsolateral funiculi) and below (e.g., descending tracts in ventral and deeper portions of lateral funiculi) the compressive lesion [37]. Myelin degeneration is often more predominant than axonal degeneration. Arachnoid fibrosis is not uncommon.

Prognosis for spontaneous recovery is poor. In mildly affected cases, conservative treatment may sometimes be beneficial over a 4 to 6 week period. This includes strict confinement, neck brace to immobilize the caudal cervical spine, and anti-inflammatory medication. However, long-term conservative therapy tends to be palliative [49]. Marked improvement has been reported in many cases following decompressive and/or stabilizing surgery.
A plethora of reports are available on surgical treatment that generally falls into three categories:

   a. Dorsal laminectromy for bony compression,
   b. Ventral slot decompression, which is especially useful for ventral soft tissue compressions that are not traction-responsive (such as annular protrusions or nuclear extrusions), and
   c. Distraction or ventral slot decompression for traction-responsive soft tissue compressions [34,51-58]. Distraction may be achieved with vertebral body pins/screws and bone cement, distraction rods, or intervertebral washers, often in association with bone grafts in order to encourage vertebral fusion [31,52,59].

The choice of surgical technique will vary according to the location of spinal cord compression (ventral, dorsal, or lateral), the nature of the compression (soft tissue or bony), and whether or not the lesion is single or multiple: up to 30% of cases in mature Dobermans may have multiple protrusions of the annulus fibrosus [34,53]. The most common lesions are ventrally located, involve soft tissue, and tend to be traction-responsive [31,46]. All surgical procedures have a high potential for morbidity and post-operative complications, which include infection, implant failure, and additional disk protrusions ("domino effect") in disks adjacent to fused or immobilized segments [49,53,60]. It has been reported that short-term success rates are high (approximately 80 per cent) after any of the surgical procedures, but there is a high rate of recurrence (around 20 per cent) [276]. Iatrogenic Horner's syndrome has been reported associated with cervical surgery, presumably due to traumatic stretching of preganglionic pathways in the thoracic vagosympathetic trunk within the carotid sheath [61]. Furthermore, neurological signs may be more pronounced the day following myelography in dogs with cervical spondylomyelopathy [62]. Non-ambulatory patients require special care and intensive nursing (see spinal trauma) for bladder control and prevention of urine scald and decubital ulcers. Physical therapy (see rehabilitation) is extremely important to combat disuse and neurogenic muscle atrophy [49,63]. One suggested prognostic guide is as follows [64]:

1. Favorable - If there is one lesion and the dog is ambulatory upon presentation;
2. Favorable to guarded - If there are two lesions and the dog is ambulatory upon presentation;
3. Guarded - If there is one lesion and the dog is non-ambulatory upon presentation;
4. Guarded to unfavorable - If there are two lesions and the dog is non-ambulatory upon presentation.

The demonstration of spinal cord atrophy and/or pooling of contrast material within the spinal cord in computed tomography-myelographic studies may also suggest a guarded to unfavorable prognosis in dogs with cervical spondylomyelopathy [47].

Control measures might include identifying Dobermans with radiographic features of cranial canal stenosis, vertebral tipping, and "stepping" of the cervical vertebrae once skeletal maturity has been reached and removing them from any breeding program since these identifiable abnormalities offer a reasonably accurate prognostication for future development of cervical spondylomyelopathy in this breed [33]. Additionally, use of balanced rations without excessive nutrition or mineral supplementation and perhaps neutering larger, faster-growing puppies might be considered [33]. Cervical vertebral ratios may have potential as a breed-specific screening tool for cervical vertebral instability [275].
There have been isolated reports of a similar wobbler syndrome in other canine breeds including Rhodesian Ridgeback, Old English Sheepdog, Weimaraner, German Shepherd, Chow Chow, Rottweiler, Pyrenean Mountain Dog, Golden Retriever, Labrador Retriever, Boxer, Irish Wolfhound, St. Bernard, Airedale Terrier, Bernese Mountain Dog, Bull Mastiff, English Setter, Irish Deerhound, and Old English Mastiff. In these breeds, the predominant sites of cord compression were C2 - C3 and/or C3 - C4 [35]. A possible hereditary malformation of C2 - C3 vertebrae occurs in Basset Hounds less than 8 months of age [65]. Involvement of the C2 - C3 articulation has been noted in Beagles [35].

The wobbler condition has also been reported in older female Borzoi dogs (from 5 to 8 years) [66]. The condition is believed to have a recessive mode of inheritance. The C6 - C7 articulation was always involved in a spectrum of abnormalities that included vertebral instability, vertebral luxation, intervertebral disk herniation, and spinal cord compression.

Disk Disease
Spinal cord compression secondary to intervertebral disk protrusion-extrusion continues to be one of the most common neurological disorders seen in clinical practice [67]. Terms used for this disorder include ruptured disk, prolapsed disk, slipped disk and herniated disk. Disk protrusion-extrusion more accurately describes this process. *Protrusion* implies that the disk is bulging into the vertebral canal as a result of dorsal shifting of central nuclear material. The outer fibrous envelope of the disk is still intact. Disk *extrusion* indicates that the outer fibrous layers have ruptured with subsequent extrusion of nuclear material into the vertebral canal. The clinical expression of disk extrusion is referred to as *disk disease*. The term *intervertebral disk displacement* is presently in vogue as another descriptor of disk disease.
There are 26 intervertebral disks in the canine and feline spinal column, excluding the coccygeal region, and they form approximately 18% of the length of the spine. Disks are widest in the cervical and lumbar regions, and narrowest in the thoracic spine. Each disk consists of two structurally different regions: (a) a central gelatinous area, the nucleus pulposus (NP), and (b) a surrounding fibrous envelope, the annulus fibrosus (AF), which contains an inner, more fibrocartilaginous matrix termed the "transitional zone" (TZ) [68-71]. The NP is oval-shaped and eccentrically positioned between the middle and dorsal thirds of the disk. It is a highly specialized tissue originating from the embryonic notochord. Throughout fetal life, the NP is the fastest growing region of the disk, and in the neonate, it occupies a considerable area of the disk. The AF is a fibrocartilaginous tissue consisting of bands of parallel fibrous bundles that run obliquely between adjacent vertebrae. The ventral annulus is about twice as wide as the dorsal annulus. Biochemically, the major macromolecular components of the canine disk include collagenous and non-collagenous protein (NCP), proteoglycan (PG) aggregates, and glycoproteins. The PG subunits consist of glycosaminoglycans (GAGs) covalently bound to a central protein core. The main GAGs in canine intervertebral disks are hyaluronic acid, chondroitin sulfate-4, chondroitin sulfate-6, and keratan sulfate. Higher orders of aggregation intimately involve hyaluronic acid. Aggregated PGs are formed by the association of many PG molecules with a single chain of hyaluronic acid, the complex being stabilized by a glycoprotein link. The GAGs are long-chained, sulfated polyanions that attach to the central protein core like the bristles of a brush.The greatest concentration of the GAGs in disk occur in NP and TZ regions of the disk.
Structures that are anatomically and physiologically closely related to disks include cartilaginous end-plates, vertebral end-plates, and conjugal and dorsal longitudinal ligaments. Conjugal ligaments, also known as transverse intercapital ligaments, are present between the second and tenth thoracic vertebral bodies in dogs, and between the second and ninth thoracic vertebral bodies in cats. Conjugal ligaments run over the dorsal part of the disk, ventral to the dorsal longitudinal ligament (a flat structure that lines the floor of the vertebral canal), and connect the heads of each set of ribs. The conjugal ligaments play an important role in the prevention of disk extrusion into the vertebral canal in the thoracic region. Dorsal longitudinal ligaments run the length of the vertebral canal, are attached to the dorsal borders of the vertebral bodies and form fan-like

coverings over the dorsal aspects of each disk. Stretch of this ligament is thought to partially account for pain associated with disk protrusion-extrusion. Cartilaginous end-plates are thin layers of hyaline cartilage that cover vertebral body epiphyses and form the rostral and caudal boundaries of each disk. Vertebrae on either side of the disk have a specialized plate of dense, smooth bone termed the vertebral end-plate. These plates are perforated by numerous small canals that are related to the underlying marrow spaces. Each plate consists of an outer peripheral zone and an inner zone that accommodates the NP region of the disk.

Intervertebral disks function as very effective shock absorbers of the vertebral column, largely due to the gel-like properties of the central NP. Specialized PGs within the nucleus bind many water molecules to form a fluid system that is virtually incompressible. This hydrophilic property allows the nucleus to deform and dissipate forces equally over the AF and cartilaginous end-plates. The transformation of an axial compressive force applied to the spine into tangential stresses on the annulus is the function of the NP, thereby reducing the compressive force on the annulus itself. Disks also provide support for the spinal column, since they represent amphiarthrodial joints in intervertebral articulations.

After birth, the canine disk undergoes structural changes that are most prominent in the NP [71-73]. The gel-like nucleus is eventually replaced by more mature fibrocartilage. This process occurs gradually in most breeds of dogs, so that by 7 to 8 years of age, the entire nucleus has changed, and the distinction between nucleus and annulus is lost. In several other breeds of dogs, however, the aging pattern is quite different. These breeds have been designated *chondrodystrophoid* due to their characteristic endochondral ossification and intervertebral disk morphology, and include Dachshunds, Beagles, Pekingese, French Bulldogs, Basset Hounds, Welsh Corgis and Cocker Spaniels [70,71]. Such breeds are characterized by varying degrees of short-limbed dwarfism. Other breeds such as Shih-tzus and Lhasa apsos probably should also be included in this group. In chondrodystrophoid breeds, replacement of notochordal cells and the gelatinous NP occurs as early as 4 months of age. This process is generally complete in all disks by 12 to 18 months of age. The central areas of the NP are usually the last to be affected, and extensive degenerative changes frequently precede the final chondrification of this area. With increasing age, degenerative changes observed in the NP include matrix disintegration, peripheral/central calcification, and localized areas of cell death. Radial fissures and clefts may appear in the AF. Commensurate with the morphologic transmutation of the NP, collagen levels approach 30 - 40% dry weight within 6 - 12 months. Extraordinary changes in all other biochemical parameters occur during the first 2 to 3 years [74-76]. In comparison with disks from non-chondrodystrophoid animals of similar age, PG levels in NP are 40 - 50% lower, glycoprotein and non-collagenous protein values are 30 - 40% lower, and chondroitin sulfate values are 30 - 50% lower. Also, during this period, keratan sulfate replaces chondroitin sulfate(s) as the major GAG. The degree of hydration of disks likely decreases with reduction in GAG content, as has been shown in people. Degenerated disks have a depressed imbibition index, which is a measure of the water-binding capacity of the disk.

The etiology of intervertebral disk protrusion-extrusion remains elusive. It is hypothesized that significant changes in morphology and biochemical parameters of the disk during the first 2 years of life result in a reduction of the disk's shock absorbing mechanisms [68,69]. While still retaining limited properties of incompressibility, the NP loses its ability to adequately deform and distribute forces in a centrifugal manner. As a result, the AF is subjected to increased loading from axial compression and lower tangential stress, which is disproportionately distributed in the disordered disk. The mechanical failure of the NP ultimately results in disruption of AF fibers and subsequent protrusion-extrusion. Results of biochemical studies suggest that the mechanical efficiency of disks is compromised in chondrodystrophoid dogs by 2 to 3 years of age [74-76]. This time-frame is consistent with the occurrence of clinical disease. Nevertheless, this theory does not explain why clinical disk disease occurs with a relatively high frequency in some non-chondrodystrophoid breeds, such as Miniature Poodles and mixed-breeds; nor does it elucidate why clinical disk disease occurs infrequently in older dogs of any breed. Studies in dogs have shown that disk metabolism in the NP is mainly anaerobic, the main route of nutrient supply into the NP is via the endplate, and that diffusion of nutrients is the main mechanism of metabolite transport [77]. There is probably an optimal, but as yet undefined, range of vertebral stress that is needed to promote and maintain nutritive requirements of disks. Half an hour of moderate exercise per day has been shown to increase nutrient flow into canine disks [78]. In contrast, spinal fusion in the dog results in significant biochemical changes in disks-metabolism is depressed in the immobilized disks but increased in the disks adjacent to the fusion mass [79]. In addition, water content and imbibition of water in NP and AF are significantly depressed in fused disks. That disk displacement occurs with some frequency in disks adjacent to totally calcified disks may also reflect an overstressed disk. Finally, it is conceivable that loss of PGs and mechanical failure of the NP profoundly influence disk nutrition. Whether disk matrix changes are the cause or the effect of nutritional diffusion impairment remains to be determined.

There is no evidence that external trauma plays a role in disk degeneration. A force of sufficient magnitude to result in spinal fractures and/or luxations rarely produces traumatic disk protrusion-extrusion. Nevertheless, trauma has been implicated in several large-breed, non-chondrodystrophoid dogs in which tearing of the dural mater secondary to intervertebral disk injury occurred during periods of vigorous running and or struggling [80]. Although trauma does not appear to play a role in the initiation of disk degeneration per se, it may be a factor in the precipitation of protrusion-extrusion once the normal mechanical efficiency of the disk is impaired. It is not unusual for dogs with clinical disk disease to be presented with a

history of spinal trauma of variable degree, such as jumping or falling. Perhaps the most logical explanation for the prevalence of disk disease in certain breeds of dogs is a genetic one. Earlier studies suggested that genetic factors are involved in the accelerated aging patterns of disks in Beagles [81]. The heightened susceptibility to disk disease in Dachshunds has been explained by a genetic model that involves the cumulative effect of several genes, with no dominance or sex linkage, subject to environmental modification [82]. In some families of Dachshunds, the prevalence of disk disease was found to be 62%, compared with the estimated breed prevalence of 19%. %. Genetic osteological factors probably play a role as well. For example, midsagittal and interpedicular diameters of the cranial and caudal aspects of cervical vertebral foramina (C3 - C7) are reportedly significantly larger in small breeds than in large breeds and Dachshunds, with seemingly potential predisposition to cervical spinal cord compression [274]. There is no evidence that autoimmune mechanisms are a factor in the pathogenesis of disk degeneration. The roles of inactivity and obesity in disk disease have not been fully evaluated, although in one study, excess body weight did not appear to be a predisposing factor in Cocker Spaniels with disk disease [83].

Neurological signs after extrusion of disk material are caused by impact injury [84], or mechanical compression of the spinal cord [85], or both. While disk protrusion usually precedes extrusion, protrusion or bulging of the disk dorsally into the vertebral canal without rupture of the AF is not usually associated with clinical signs, with the possible exception of pain. This is exemplified in dogs and cats over 7 years of age in which dorsal disk protrusion is relatively common but is subclinical. The velocity with which the disk material extrudes into the canal appears to be more important than the size of the mass. An explosive herniation results in far more severe damage than a slow extrusion. With acute impact injuries, hemorrhage and attendant inflammatory reaction may also contribute to epidural compression. Results of a quantitative radiographic study [86] suggest that the lumbar epidural space in Dachshunds is less than that in German Shepherds (a non-chondrodystrophoid breed) which implies that epidural masses of similar size would cause more spinal cord compression and more severe neurological deficits in Dachshunds. For a review of the pathophysiological events and biochemical cascade occurring with acute trauma to the spinal cord see spinal cord trauma.

Most dogs with disk disease are between 3 and 7 years of age. Eighty-five percent of disk extrusions in dogs occur in the thoracolumbar area and 15% are cervical. Approximately 80% of thoracolumbar extrusions occur between T11 and L3, with less than 2% occurring in the terminal lumbar region (L5 - S1). In one study of large-breed, non-chondrodystrophoid dogs with thoracolumbar disk disease, the mean age was approximately 7 years, and 57 dogs (92%) had Hansen type 1 disk disease, usually at the L1 - L2 site [87]. In this report, 58% of cases were acute in onset. Disk extrusion normally does not occur between T2 and T10, probably because of the presence of the conjugal ligament, although a Hansen type 1 disk extrusion has been reported at the T1 - T2 level in a 7 year old Dachshund with acute neurological deficits to the hind limbs following trauma [88]. Several studies indicate that the most common site in the cervical region is C2 - 3 [89,90]; although results of one study (105 cases) indicated no significant difference in prevalence of disk disease affecting the first four disk spaces (C2 - 3 to C5 - 6) [91] (in this study, prevalence of disk disease at C7 - T1 was significantly less than that involving the first 4 disk spaces).

Disk disease also occurs in cats but mainly as a subclinical event [89]. One study on clinically normal cats showed that degenerative changes in disks increased with age, with dorsal protrusions found in 30% of cats 6 to 10 years of age, in 50% of cats 11 to 14 years of age, and in all cats 15 years of age and older [92]. In another report, type I disk protrusion in cats, again as a subclincial condition, was encountered most commonly in upper cervical and L4 - L5 areas [93]. Nevertheless, clinical signs of disk disease have been reported sporadically in cats [94-96]. In a recent study of disk disease in 10 cats, there was no breed or sex predilection and clinical signs included back pain, difficulty ambulating, and incontinence [96]. All herniations occurred in the thoracolumbar spine, with a peak incidence at the L4 - L5 disk space. Eight cats had a Hansen type I protrusion.

The onset of clinical signs in dogs may be acute (minutes), subacute (hours), or chronic (several days or weeks). These signs may be rapidly progressive, slowly progressive, or may remain static. Clinical signs also may undergo remission, only to recur at a later date. Clinical signs in dogs with recurrent attacks frequently are more severe than those seen at the initial episode. Recurrences have often been considered to be the result of multiple extrusions at the same disk level [97,98]. However, in a recent study, 22 of 25 dogs had a second operation (> 4 weeks after the initial surgery) at a site distinct from the initial lesion [99]. In this study, Dachshunds were at higher risk for recurrences than other breeds.

The two most common neurological syndromes associated with disk disease are thoracolumbar and cervical syndromes. With cervical disk disease, the majority of affected animals will have a history of pain, with or without paresis [90], and frequently, spasms of cervical musculature. Animals may assume a posture with the nose held close to the ground and the back arched. In some dogs, one thoracic limb may be held in partial flexion, with reluctance to support weight or walk on this limb. These animals frequently show considerable pain on manipulation of the head and neck. This combination of signs is termed *root signature*, since it is believed to be associated with nerve root entrapment near the intervertebral foramen as a result of lateral

disk extrusion [100]. A **lumbosacral syndrome** is uncommonly associated with disk disease. In some animals with lumbosacral disk extrusion, one pelvic limb may be held in partial flexion or a repetitive "stamping" motion may be observed. These animals frequently show considerable pain on manipulation of the limb and lumbosacral spine. This combination of signs has also been termed *root signature* and is believed to be associated with nerve root compression or entrapment by a fragment of extruded disk material. In a small percentage of dogs, a multifocal syndrome may develop as a result of an acute, explosive extrusion of disk material from a thoracolumbar disk that produces hemorrhagic myelomalacia. With this irreversible disorder, an initial thoracolumbar syndrome may be followed by a lumbosacral syndrome as the lesion descends the cord. As the lesion also frequently ascends the cord, signs of thoracic limb rigidity give way to flaccidity and areflexia followed by death due to respiratory paralysis.

A definitive diagnosis of disk disease requires radiographic confirmation of presence of a mass lesion or, in absence of a mass lesion, evidence of characteristic changes in the disk-vertebral articulations. Typical radiographic features of disk disease include narrowing of the disk space, intervertebral foramen and articular facet at the site of the herniated disk, wedging of contiguous vertebral bodies so that the dorsal part of the disk space appears narrower than the ventral part, and presence of an opacified mass in the vertebral canal. *In situ* calcified disks, in the absence of any other abnormality, are a common finding in chondrodystrophoid breeds of dogs and are of little significance-it has been estimated that dystrophic calcification occurs in 20 to 77% of disks in some chondrodystrophoid breeds within the first year or two of life [71,101-103], especially in Dachshunds in whom calcification appears to be inherited [104,272]. Recent studies suggest that exercise has a modulating effect on rate of occurrence of disk calcification in Dachshunds (moderate exercise reduced the rate of occurrence of disk calcification) [105]. In some cases, particularly in acute extrusions, plain radiographic findings may be minimal or equivocal and myelographic studies will be necessary to define the extent and location of spinal cord compression. In one study, accuracy for determining sites of intervertebral disk protrusion using survey radiography was only in the 51 - 61% range [280]. The importance of accurate localization of lesions is demonstrated by the presence of asymmetrical neurological signs contralateral to the myelographic and surgical lesion in some dogs, especially those with Hansen type 1 extrusion [106]. Contrast studies also are indicated when there is evidence of more than one disk lesion. The most common myelographic change is narrowing and dorsal deviation of the ventral contrast column at the level of disk protrusion/extrusion. If the disk extrusion is acute, spinal cord swelling may result in complete blockage of contrast material at, or immediately rostral to the level of the disk extrusion. Note that dogs with thoracolumbar or cervical disk disease that have clinical signs of back or neck pain alone, without neurologic deficits, may have substantial compression of the spinal cord [90,107]. Results of experimental studies suggest that high-dose contrast enhancement (e.g., 0.3 mmol/kg of gadoteridol) might facilitate the detection of recurrent herniated disk fragments [108]. While plain radiography and myelography have long been the methods of choice for the diagnosis of disk disease, other non-invasive neuroimaging procedures such as magnetic resonance imaging (MRI) [109] and computed tomography (CT) [110-112] may be more accurate, technically easier, and safer (myelography may exacerbate clinical signs and induce seizures). In one report, preoperative CT confirmation of the relationship between the spinal cord and the protruded disk was used in planning the surgical approach in dogs with cervical disk disease [113]. MRI is considered to give better information about the condition of the intervertebral disk (e.g., the hydration status of the nucleus pulposus) than radiography [114]. In fact, classification of degenerating intervertebral disks and identification of MR imaging characteristics of each type have been reported in experimental studies in dogs [115]. Hemorrhage may also be identified using MRI [278]. Analysis of CSF, especially if sampled from the lumbar subarachnoid space, may reveal markedly elevated protein levels and increased numbers of mononuclear white blood cells [116]. These changes are more likely to be found in dogs with severe and acute neurological signs. Recent studies have shown a significant increase in lumbar CSF glutamate concentrations in both acute and chronic cord compression injuries secondary to disk herniation in dogs [117].

Gross pathological findings occurring subsequent to disk disease usually depend on whether disk protrusion-extrusion is partial or complete and whether it occurs acutely or gradually. While many disks in older animals of any breed may protrude, it is uncommon to find more than one extruded disk, even in animals that have had a history of multiple episodes. This suggests that many recurrences are due to multiple extrusions from single disks (see below). In disk protrusion, the AF may bulge dorsally into the vertebral canal, without rupturing. This is known as a Hansen type 2 disk [71], and it appears as a small, round to dome-shaped bulging of the dorsal surface of the disk. A **Hansen type 1 disk** [71] is characterized by rupture of the dorsal annulus, with extrusion of degenerate NP into the vertebral canal around the spinal cord. In some instances, the extruded nuclear material will be contained by the dorsal longitudinal ligament. Typically, disks extrude in a dorsomedian, paramedian, or dorsolateral plane. In the cervical region, where the vertebral canal/spinal cord ratio is larger than that of the thoracolumbar region, lateral and intraforaminal extrusions may be more common than in other spinal regions, producing spinal root rather than spinal cord compression. Rarely, disk material may herniate through the cartilaginous end-plate into the vertebral body (resulting in an intravertebral herniation or Schmorl's node) [118], or into the spinal cord itself

(intramedullary extrusion) [279].

The spinal cord may be swollen, indented, flattened, or atrophic. In chronic cases, a fibrous adhesion may be evident between the extruded mass and the dura mater. In many instances of Hansen type 1 disk extrusion, hemorrhage will be associated with the extruded disk material, producing a soft, granular, salt and pepper consistency. In some cases, the volume of epidural hemorrhage may exceed that of the extruded disk material. The extruded material may form a circumscribed mass or may lie flattened around the sides of the dura mater. The extruded material may have migrated one or two vertebral levels away from the site of the affected disk. This form of extrusion is usually present in dogs with thoracolumbar disk disease. Since extruded disks are not completely absorbed, single disks that may have had multiple extrusions are recognized by their stratified appearance. The oldest component may be dark gray, hard, and adherent to the dura. Subsequent laminations are lighter in color and more friable [97]. In chronic disk disease with slow, progressive extrusion, the degenerate material frequently has a gritty consistency and an opaque and cheesy appearance. This type of extrusion is more often observed in dogs with cervical disk disease.

Microscopic changes in the spinal cord are dependent on the rate of disk extrusion and duration of cord compression. Gradual or mild compression produces varying degrees of demyelination and axonal degeneration. Sudden, massive extrusions often result in focal or multifocal hemorrhage and necrosis in gray and white matter. Localized edema may result in pronounced cord swelling and collapse of the subarachnoid space. Rarely, disk material will be present within the cord parenchyma. In necrotic areas of the spinal cord, vessels and mesenchymal (connective tissue) elements are usually preserved. Lipid macrophages are observed in those cases of a few days duration. In more chronic cases, marked proliferation of astrocytes and microglial cells may be a feature, especially in areas that border the necrotic zone, together with trabeculae of blood vessels and connective tissue that cross the necrotic areas [84]. In longer standing lesions, the gray matter often has a fenestrated appearance due to loss of neurons and fibers. Astrocytic gliosis may result in marked sclerosis of the gray matter. An epidural inflammatory reaction composed of neutrophils, red blood cells, fibroblasts, large mononuclear cells, occasional multinucleate giant cells, chondrocytic-like cells, and fibrocartilaginous debris may be present.

Medical management usually is directed at animals with their first signs of disk disease. Mild clinical signs often resolve after at least three weeks of confinement with outside activity limited to leash exercise. Recurrences of clinical signs are common in this group of animals. Severe, unremitting pain may be managed with prednisolone, 0.5 mg/kg, PO, bid, for 72 hours. Muscle spasms may respond to muscle relaxants, e.g., methocarbamol (Robaxin), 20 mg/kg, PO, tid, for 7 to 10 days, or diazepam, 2 - 5 mg, PO, tid, for several days. High dose methylprednisolone succinate should be considered in paraplegic/tetraplegic animals with acute spinal cord injury (see spinal trauma). Acupuncture is considered another form of conservative treatment [119-122]. The analgesic response to acupuncture is reportedly most effective in dogs showing pain with or without mild paresis. Animals receiving this treatment should have restricted activity.

Surgical treatment is indicated in animal with clinical signs unresponsive to medical management, recurrent and/or progressive clinical signs, or in animals that are paralyzed. The approaches most widely used are dorsolateral hemilaminectomy / pediculectomy or dorsal laminectomy for thoracolumbar diskdisease and ventral slot-decompression for cervical disk extrusions, although a thoracolumbar lateral approach has its proponents [89,123-128]. In a recent study, significant improvement in clinical results was seen in caudal cervical disk protrusions when additional surgical distraction and stabilization were provided following ventral slot decompression [129]. Dorsal laminectomy has also been successfully performed in dogs (especially those < 15 kg) with cervical disk disease [130].While some studies of thoracolumbar disk disease indicate that removal of disk material using these techniques significantly improves the degree of completeness of recovery [131], successful results have been reported using fenestration alone [98,132-134]. Prophylactic fenestration [89] in addition to decompression remains somewhat controversial [135] but is still performed by many surgeons in order to reduce the chance of subsequent herniation involving other disks [136-138]. A variety of other surgical procedures have been described, including percutaneous diskectomy [139], but their effectiveness await large clinical trials. Although still not commonly employed for the treatment of disk disease, chemonucleolysis (e.g., using collagenase or chymopapain injected directly into the disk) has its exponents [140-143] and may be more effective than fenestration at removing nuclear material from the disk [144]. Experimental autographic disk transplantation for potential use in humans with chronic disk disease is in its infancy but initial surgical studies in dogs showed promise [145]. Potential treatment complications include cardiac dysfunction from manipulation of the vagosympathetic trunk during cervical surgery, and vertebral luxation as a complication of the ventral slot procedure, especially in mid to lower cervical vertebrae [146]. Furthermore, cervical vertebral fusion may predispose adjacent disks to herniation [147]. Corticosteroid therapy (usually associated with use of dexamethasone) may lead to gastrointestinal hemorrhage, ulceration, colonic perforation and pancreatitis [148-150]. Complications may be kept to a minimum by administering corticosteroids for as short a time as possible. Prophylactic use of intestinal protectants, e.g., bismuth subsalicylate (Pepto-Bismol®) in conjunction with frequent administration (at least four times daily) of antacids, e.g., magnesium or aluminum hydroxide, or H2 antagonists such as cimetidine (Tagamet®, at 20

mg/kg, PO, tid) also may reduce the prevalence of gastrointestinal hemorrhage. Corticosteroids should be stopped immediately, when gastrointestinal complications are noted. In a recent study in dogs with acute degenerative disk disease treated by surgery and corticosteroid administration, both omeprazole (a gastric acid pump inhibitor) and misoprostol (a synthetic prostaglandin E1 analog) were ineffective in treating or preventing the further development of gastric mucosal lesions [150].

Paralyzed patients need to be maintained in a sanitary environment, with twice daily bladder catheterization, frequent removal of soiled bedding, and use of foam rubber pads or water beds to prevent development of decubital ulcers. In addition, active physiotherapy (see also spinal trauma and chapter on rehabilitation) that includes assisted standing and walking exercises, and supervised swimming for 15 minutes twice daily, is an integral part of the nursing care since it will delay disuse muscle atrophy.

The following statements may be used as a general guide to assess prognosis:

1. Animals that are paretic or paralyzed but have normal pain sensation have a good prognosis following medical and/or surgical management. Results of a recent surgical study (using hemilaminectomy and fenestration) with an 86% success rate indicated that the rate of onset of clinical signs significantly influenced the clinical outcome but not the length of recovery time, while the duration of clinical signs did not seem to significantly affect the outcome, but did affect the length of recovery time [281]. The presence of postoperative voluntary motor function is also reported to be a favorable prognostic indicator for early return to ambulation [282].
2. Animals that are paralyzed with loss of bladder control and with reduced pain sensation have a guarded-to-favorable prognosis following surgical intervention (decompression and/or fenestration).
3. Animals that are paralyzed with loss of bladder control and loss of pain sensation have a guarded-to-unfavorable prognosis.

Dogs with absent deep pain perception that undergo surgery within 12 to 36 hours have a better chance of recovery (more complete and over a shorter time-period) than those in which surgery is delayed [100]. Evaluation of the degree of myelographic spinal cord swelling might also assist in establishing a prognosis in severely affected animals [151]. As a caveat to prognostication, several studies have shown that severity of spinal cord dysfunction, based on clinical signs, does not necessarily predict outcome. In one recent report, 50% of dogs with loss of bladder control and loss of deep pain sensation recovered completely or partially [152].

A functional scoring system for pelvic limb gait of dogs with acute thoracolumbar spinal cord trauma (from spontaneously-occurring disk disease) has been developed to allow quantification of recovery to be assessed and potentially facilitate evaluation of pharmacotherapeutic clinical trials [153]. Spinal cord evoked potentials and somatosensory potentials may be useful in localizing spinal cord lesions and assessing lesion severity [154,155]. Other evoked potentials such as magnetically elicited transcranial motor evoked potentials may be sensitive indices of severity of spinal cord lesions in dogs with disk disease but do not appear to be reliable predictors of neurologic recovery [156]. In one report involving 10 cats with disk disease, prognosis was adjudged to be most favorable in cats following surgical decompression [96].

Diskospondylitis
Diskospondylitis is intervertebral disk infection with concurrent osteomyelitis occurring in contiguous vertebral bodies [157-169]. This disorder occurs in young to middle-aged adult dogs (typically non-chondrodystrophoid) usually of the larger breeds. Male dogs outnumber females by approximately 2:1. Diskospondylitis has also been reported in cats, albeit infrequently [170-174]. Diskospondylitis may occur following iatrogenic trauma of the vertebral column (e.g., disk curettage), foreign body migration, paravertebral injection, extension from a body organ abscess, or more commonly from blood-borne septic emboli that reach the avascular intervertebral disk via the capillary networks in the vertebral end-plates [158-161,175,176]. The source of infection is not established in most cases. Possible initiating sites include the genitourinary tract, skin, gingiva, and infected heart valves. In one dog, epidural abscess and diskospondylitis developed after administration of a lumbosacral epidural analgesic [177]. Diskospondylitis has also been found in a Bernese Mountain dog with immune-mediated polyarthritis [178]. Bacterial infection is the most common cause of diskospondylitis and coagulase positive *Staphylococci* (*S. aureus* or *S. intermedius*) are the most frequent isolates. Other organisms identified include *Brucella canis*, *Nocardia*, *Streptococcus canis*, *Escherichia coli*, *Acaligenes* sp, *Micrococcus* spp, *Proteus* sp, *Corynebacterium diphtheroides*, *Mycobacterium avium*, *Erysipelothrix tonsillarum* and *Actinomyces viscosus*. In a recent study, novel organisms incriminated in canine diskospondylitis included *Pseudomonas aeruginosa*, *Enterococcus faecalis* and *Staphylococcus epidermidis* [179]. Fungal organisms including *Aspergillus terreus*, *Paecilomyces* sp (e.g.,*Paecilomyces varioti*), *Penicillium* sp, *Chrysosporium* sp, *Pseudallescheria boydii*, and *Coccidioides immitis* have also been cultured [163,164,166,180,181]. In one retrospective study involving 135 dogs with diskospondylitis, the prevalence of dogs with

Brucella canis was approximately 10% and sexually intact male dogs were at risk as were dogs from the southeastern United States [182]. Immunosuppression may predispose some breeds, such as German Shepherds, Airedale Terriers, and Basset Hounds to bacterial or fungal infection and subsequent diskospondylitis [181,183-185]. Respiratory or gastrointestinal portals of entry are suggested for animals with aspergillosis. Curiously, the majority of reports of disseminated aspergillosis in dogs have involved German Shepherds [181,186-188] with organisms localizing most frequently in kidneys, spleen, and vertebrae. In one dog with diskospondylitis due to *Aspergillus terreus*, multiple granulomas with fungal elements were also found in the subarachnoid space associated with the nerve roots of the cauda equina [183]. Hypothyroidism does not appear to be a predisposing factor in the development of diskospondylitis.

Clinical signs are variable according to vertebral involvement, ranging from subtle spinal hyperesthesia and stiffness, to severe paresis/paralysis. In more than 80% of affected dogs, spinal pain is observed [189]. Affected animals may manifest depression, anorexia, and pyrexia. Often they are reluctant to exercise or jump. Heart murmurs can be detected on auscultation in some animals. Pleural effusion associated with paecilomycosis was reported in one dog [284]. Spinal cord and/or nerve root compression may result from proliferation of inflammatory tissue and exostosis, subarachnoid or epidural abscessation, vertebral pathological fractures, intervertebral disk protrusion/herniation, or excessive vertebral instability [177,187,190,191]. Spinal cord myelitis may also occur by extension of infection through the meninges.

Radiographic abnormalities include a concentric area of lysis of adjacent vertebral end-plates early in the disease process. More chronic lesions are characterized by varying degrees of bone lysis and proliferation, vertebral sclerosis, shortening of vertebral bodies, narrowed intervertebral disk spaces, and ventral osseous proliferation that may bridge the affected disk space. Extensive destruction of a vertebra may result in its collapse. Diskospondylitis may be present in more than one disk space and commonly occurs in one or more adjacent disk spaces. Common sites of diskospondylitis are the caudal cervical area, midthoracic and thoracolumbar regions, and the lumbosacral joint. In dogs with grass awn migration, reactive bony changes may be seen on ventral and lateral surfaces of vertebrae L2 through L4 [189]. The nature and location of the changes along the spine may help differentiate diskospondylitis from malignant bone disease [192]. The severity of the radiographic changes do not necessarily correlate with the degree of clinical involvement. Results of a recent multicenter, retrospective study evaluating contrast radiographic findings (myelograms or epidurograms) in canine bacterial diskospondylitis revealed that 15 of 27 cases (56%) showed some degree of spinal cord compression, although in the majority (approximately 73%) soft tissue was the compressive mass and the median compression for all cases was only 5% of the vertebral canal [193]. Vertebral subluxation was evident in 20% of these dogs. Stress radiography has been recommended for further evaluating dogs with vertebral instability [193]. Radiographic signs of the disease may not appear for several weeks after the onset of clinical signs. Hence, a radiographically normal spine does not preclude the diagnosis of diskospondylosis. Magnetic resonance imaging can be diagnostic prior to development of definitive radiographic abnormalities [194]. MRI findings in affected dogs have revealed increased T2 and decreased T1 signal intensity of the soft tissues ventral to vertebral bodies, the end plates of the same vertebral bodies and the intervertebral disk [195].

Blood and urine cultures should be obtained before starting antibiotic therapy. Reports of positive blood cultures range from 45% to 75% of affected dogs, while urine cultures can be positive in up to 50% of dogs [189,196]. In one report, fungal hyphae were identified in urine sediment from 6 dogs [181]. While serologic *Brucella* titers should be checked because of the public health significance, positive blood cultures are reportedly lower in dogs with *Brucella canis*-induced diskospondylitis [182]. Percutaneous aspiration of the infected vertebrae using fluoroscopy is a very useful diagnostic aid. In one study, positive bacterial cultures were obtained from 9 of 12 aspirated disk spaces including 2 dogs in which blood and urine cultures were negative [197].

Prognosis is usually favorable with aggressive long-term antibiotic therapy (e.g., from 2 to 4 months) if neurological signs are mild and the vertebrae are stable [198,199]. As a rule of thumb, until culture results are available, the organism should be assumed to be a *Staphylococcus*. The cephalosporins have been effective in the majority of small animal cases, e.g., cephalexin, at 22 mg/kg, PO, tid or cefazolin 20 mg/kg IV, qid for up to 5 days initially, if animals have fever or progressive neurological signs, followed by oral antibiotics. The following drugs have been recommended for treating diskospondylitis caused by other organisms [189,200,201]:

Microorganism	Antibiotic	Dose
beta-hemolytic *Streptococcus* sp.	Amoxicillin	20 mg/kg, PO sid
Brucella canis	Enrofloxacin Doxycycline Gentamycin	10 - 20 mg/kg PO sid 25 mg/kg PO sid 5 mg/kg IM or SQ sid
Actinomyces sp.	Penicillin G	100,000 U/kg IV, IM, or SQ qid
Coccidioides immitis	Ketoconazole Fluconazole	10 mg/kg PO bid (dog); 50 mg (total) PO bid or sid (cat) 5 mg/kg PO bid (dog); 25 - 50 mg(total) PO bid or sid (cat)
Aspergillus sp.	Amphotericin B (deoxycholate) Itraconazole	0.25 mg/kg IV every 48 hours until a cumulative dose of 8 - 12 mg/kg (dog) or 4 - 8 mg/kg (cat) is reached 5 mg/kg PO bid (dog and cat)

Prognosis for dogs with fungal diskospondylitis is guarded. In one report of 10 cases, 8 dogs were euthanized because of severe neurological signs, although one dog was alive after 4 years of continuous treatment with itraconazole [181]. Prognosis may also remain guarded in dogs with *Brucella canis* infection since serologic testing and radiographically active lesions may remain positive long after resolution of clinical signs [182]. Dogs with brucellosis should be neutered and clients advised of potential zoonotic infection. Vertebral curettage may expedite clinical resolution in cases refractory to medical treatment. In animals with severe neurological signs, spinal cord decompression and/or vertebral immobilization are indicated. In one report, surgical treatment involving distraction and stabilization to obtain intervertebral fusion was effective in treating lumbosacral instability caused by diskospondylitis [191]. Prognosis for surgically treated animals with severe neurological signs is often favorable [179]. Analgesics may also be required in some dogs because of pain. Recurrences may be common, especially in dogs with *Brucella canis* and in those with fungal infections, thereby necessitating re-treatment. The resolving lesion is characterized radiographically by cessation of the lytic process and by gradual replacement with new bone, sometimes causing fusion of the adjacent vertebrae. Radionuclide bone imaging, especially using gallium scans, may be a sensitive technique for confirming successful treatment [189]. Note that there is no apparent correlation between the ambulatory status and the ultimate outcome of dogs with diskospondylitis [193].

Dural Ossification

Dural ossification is a degenerative disorder of dogs characterized by deposition of bone plaques on the inner surface of the dura mater [202-204]. Synonyms are osseous metaplasia of the dura mater and, incorrectly [205,206], ossifying pachymeningitis. These plaques occur in more than 60% of large and small breeds, of either gender, over 2 years of age and occur most often in the cervical region (e.g., C3 - T1) and lumbar (L1 - L6) areas of the spine [202,207]. Over 40% of 2 year old dogs had lesions in one report [202]. The etiology of this condition is unknown but seems unrelated to mechanical stress due to distribution of the changes [202]. Dural ossification is a common, incidental necropsy finding in dogs [203]. In extreme cases, the dura may be transformed into a solid bony tube[205]. The plaques often contain marrow cavities. The majority of plaques in one study were located ventrally while the remainder were found on dorsal and lateral aspects of the dural tube [202]. In general, dural ossification rarely causes clinical disease, but spinal cord compression with secondary degenerative changes in white and gray matter including edema, loss of neuronal cells, gliosis, and rarely, marked spinal cord compression with malacia have occasionally been reported in the dog [203,205]. Degenerative changes also may occur in nerve roots closely associated with the bony plaques [205]. Affected animals may be presented with a history of chronic paresis over several months, or tetraparesis, according to the location of the bony plaques, sometimes with atrophy of limb musculature and pain. Dural ossification is characterized radiographically (often as an incidental finding) by thin radiopaque linear shadows in cervical and lumbar areas, especially at the site of intervertebral foramina. The plaques sometimes may be confused with calcified herniated intervertebral disk material, vertebral osteophytes, or accessory processes on thoracic and lumbar segments [202]. They may be further defined using advanced imaging techniques, such as computed tomography [112].

If a definitive diagnosis is made in a dog with neurological signs, decompressive surgery may be attempted [202]. More common disorders should be given priority in the differential diagnosis of chronic spinal cord compression.

Lumbosacral Stenosis
Stenosis (narrowing) of the vertebral canal and/or the intervertebral foramina in the lumbosacral area with compression of the nerve roots that form the cauda equina (L6 - 7 + S1 - 3 + coccygeal segments) and/or their related vasculature is an entity reported in dogs [208-211] and rarely, in cats [171]. This condition has been termed cauda equina syndrome, spondylolisthesis, lumbosacral instability, lumbar spinal stenosis, degenerative lumbosacral stenosis, and lumbosacral malarticulation and malformation. In most dogs, the spinal cord usually ends within the sixth or cranial half of the seventh lumbar vertebra, although in chondrodystrophoid and small-breed dogs the cord extends to the L7 - S1 level [86,212]. Most cases of acquired lumbosacral stenosis appear to be related to intervertebral disk degeneration at L7 - S1, especially Hansen type 2 protrusion (see disk disease) [211] with subsequent development of osteophytes at L7 - S1 endplates and articular facets, narrowing of the disk space at L7 - S1, subluxation of articular facets, thickening and in-folding of the normally taut interarcuate ligament, and thickened lamina and pedicles. The end result is degenerative stenosis with compression of the cauda equina. Lumbosacral instability, including dorsal dislocation of L7, has also been reported [213]. Morphometric studies suggest that multilevel congenital or developmental stenosis of the lumbosacral canal may contribute to acquired lumbosacral stenosis in large-breed dogs [214]. Other causes of acquired stenosis include diskospondylitis, neoplasia, and traumatic fracture/luxation of L7 - S1, sacrum, or the sacrococcygeal junction. Also, lumbosacral osteochondrosis, a developmental disturbance of the end plate of either the sacrum or L7 vertebra, with subsequent separation of an osteochondral flap, has been reported as a cause of lumbosacral stenosis in mature dogs [269-271]. This condition is often associated with disk disease, consequently, compressive lesions result from the flaps alone or in combination with disk material. An infrequently reported form of congenital ("idiopathic") stenosis in dogs occurs unassociated with disk disease. It is characterized by shortening of the pedicles, thickened and sclerotic apposition of the lamina and articular processes, in-folding and hypertrophy of the ligamentum flavum, and sclerotic and bulbous articular facets that bulge into the dorsal half of the canal, sometimes accompanied by malformations (e.g., hemivertebra, block vertebra, and transitional vertebra, such as lumbarization of S1 [215]. This congenital condition is thought to be associated with a developmental defect in the neural arch.

Acquired degenerative stenosis occurs most commonly in large breed dogs, many of which are highly trained or working dogs, including Border Collies [216-218]. German Shepherds are especially at risk for this degenerative disorder [219], possibly because of the presence of destabilizing transitional lumbosacral vertebral anomalies that predispose to premature disk degeneration [220]. The vertebral anomalies in the German Shepherd are considered to be inherited [221]. Smaller breed dogs appear to be more often affected by the congenital form of lumbosacral stenosis. In both forms, clinical signs are noted usually when dogs are mature to middle-aged (e.g., 5 to 8 years), possibly associated with age-related soft tissue and bony changes, along with altered spinal mechanics, resulting in cauda equina compression [222]. Males appear to be at higher risk than females in the acquired disease. In dogs with lumbosacral osteochondrosis, the mean age was 6.3 years, German Shepherds (56%), Boxers (11%) and Rottweilers (9%) were overrepresented, and the male:female ratio was 4:1 [271]. Irrespective of etiology, dogs with lumbosacral stenosis usually show varying signs of a lumbosacral syndrome depending on the level and extent of the lesion. Owners often note that affected dogs have difficulty rising or climbing stairs, and show signs of pain or stiffness during extensive physical activity [218]. Clinical signs may include pain (the most commonly reported sign) during direct palpation (especially downward pressure) of the lumbosacral area or during lumbosacral hyperextension, unilateral or bilateral pelvic limb paresis or lameness, proprioceptive deficits, tail paresis, hypotonia of anal sphincter with fecal incontinence, and urinary incontinence [211,216,222]. In some animals self-mutilation of pelvic limbs, tail, perineum, anal area, and genitalia may be noted. The occurrence of exercise-induced pain in some affected dogs, termed *neurogenic intermittent claudication* [222], may be related to dilatation of radicular vessels and subsequent compression of adjacent nerve roots in a stenotic region, e.g., intervertebral foramen or lateral recess of the caudal L7 vertebral foramen [223] narrowed by a degenerative process.

On plain films, indirect evidence of degenerative lumbosacral stenosis includes spondylosis deformans, disk space narrowing, and end-plate sclerosis. There may be evidence of lumbosacral fracture/luxation, osseous neoplasia, intradiskal osteomyelitis associated with diskospondylitis, or congenital lumbosacral stenosis. In dogs with lumbosacral osteochondrosis, a radiolucent defect occurs in the dorsal aspect of the affected end-plate along with one or more bone fragments in the vertebral canal and lipping, angling, and sclerosis of the dorsal part of the end-plate [271]. Stress radiography, such as dynamic flexion/extension studies, may accentuate the lumbosacral instability. Epidurography and diskography may provide useful information. In one study, combined survey radiography and discography-epidurography were correctly positive in 16 of 18 dogs (89%) [224]. Myelography has limited value in the evaluation of the cauda equina because the dural sac is elevated from the vertebral canal floor and often ends before the lumbosacral junction [225]. Computed tomography and MRI are probably the diagnostic procedures of choice [217,222,223,225-227], although findings of similar CT changes (but not vertebral subluxation) in the lumbosacral spine of older dogs without clinical disease may complicate diagnosis [112]. MRI can clearly reveal soft tissue, such as cauda equina, epidural fat, and intervertebral disk, at

the lumbosacral region without use of contrast medium [228]. MRI is also considered to give better information about the condition of the intervertebral disk (e.g., the hydration status of the nucleus pulposus) in dogs with degenerative lumbar spine diseases, than radiography [114]. CT scans also have an important diagnostic role. In a study evaluating canine lumbosacral stenosis using intravenous contrast- enhanced CT, the positive predictive values for compressive soft tissues involving the dorsal canal, ventral canal and lateral recesses were 83%, 100%, and 81% respectively [229]. However, no correlation was found between severity of the clinical signs and the severity of cauda equina compression as assessed by MRI in another study [277]. A gas-filled lumbosacral disk space (*vacuum disk* phenomenon) along with smaller gas bubbles in between the degenerated L5 - L6 dorsal articular facets (*vacuum facet* phenomenon) has also been revealed by CT in a 7 year old Rottweiler with cauda equina syndrome [230]. A diagnostic role for CT densitometry awaits further studies [283]. Electromyographic studies can demonstrate fibrillation potentials in lumbosacral paraspinal muscles, pelvic limbs, coccygeal muscles, and anal sphincter. In those cases where results of ancillary aids are equivocal, exploratory surgery may be the only means available for definitive diagnosis and treatment [222].

Grossly, marked compression and indentation of nerve roots may be seen, associated with stenotic lesions, bone fragments, disk material, inflammatory lesions, neoplasia, etc. Histological sections of samples removed from dogs with lumbosacral osteochondrosis revealed the osteochondral flaps consisted of a core of bone with or without a hyaline cartilage cap covering its margin, with the cartilage present consisting of a mixture of cartilaginous overgrowth and cartilage separation-necrosis [271]. I have commonly found extensive axonal degeneration characterized by linear rows of ovoids and balls, along with variable demyelination and remyelination in teased nerve fiber studies of biopsied nerve roots. In semithin sections, nerve fiber loss can be pronounced. Evidence of nerve regeneration may be seen in chronic lesions. In experimental studies in dogs, the involvement of intrinsic spinal cord neurons in the compression-induced cauda equina syndrome includes anterograde, retrograde and transneuronal degeneration in the lumbosacral segments [231] as well as marked changes in NADPH (nicotinamide adenine dinucleotide phosphate, reduced form) diaphorase-exhibiting and Fos-like immunoreactive neurons and heat-shock protein 72 (a cytoprotective protein whose expression is induced by a variety of harmful stimuli) [232].

Prognosis will depend on the underlying cause and the degree of damage to the nerve roots of the cauda equina. For congenital lumbosacral stenosis, prognosis is usually favorable with surgical decompression alone. Animals with acquired lumbosacral stenosis presenting with severe neurological deficits that include urinary or fecal incontinence have a guarded prognosis [222]. In dogs with less severe clinical signs, prognosis may be favorable when decompression by dorsal laminectomy is combined with foraminotomy and/or mass removal (e.g., disk, facets, ligaments, joint capsule, or osteophytes) [211,216,218,222,233]. In one report, dogs with lumbosacral osteochondrosis and instability treated with distraction-fusion along with dorsal laminectomy had a better prognosis than dogs without instability that were treated with dorsal laminectomy alone [271]. Medical treatment, such as rest, weight loss, anti-inflammatory or analgesic drugs, has usually been disappointing for congenital or acquired degenerative lumbosacral stenosis [222]. As for other acquired causes of lumbosacral stenosis, diskospondylitis can be successfully managed with antimicrobial therapy (see diskospondylitis), traumatic lumbosacral stenosis has a guarded prognosis (see spinal trauma), while lumbosacral neoplasia (see neoplasia) has a poor prognosis.

Spinal Synovial Cysts
Extradural spinal synovial cysts originating from articular facet joint capsules and causing spinal cord compression have recently been described in dogs [109,234-237]. These cysts are characterized by a lining of single or multiple layers of flattened or cuboidal synovial cells with a wall composed of hypercellular synovium or fibrocollagenous tissue that may have cellular infiltrates (e.g., lymphocytes, plasma cells and macrophages) and focal areas of mineralisation or mucoid material. Another histological intraspinal articular facet joint cyst, called a ganglion cyst, is very similar to the synovial cysts but has no synovial lining. This form of cyst has also been recently reported involving L6 - L7 and L7 - S1 articular process joints in a 6 year old German Shepherd [238]. Adipose tissue and numerous capillaries were present on the outer surface of the cyst and foci of metaplastic cartilage were noted within the cyst wall. In humans, there is no clinical distinction between synovial and ganglion cysts of the spine that collectively have been termed "juxta-facet" cysts [239-244]. The pathophysiology of these cysts is uncertain, although they appear to develop secondary to osteoarthritis of the facet joints, e.g., synovial cysts arising from the synovial outpouchings through areas of weakened or destroyed capsular tissue; or ganglion cysts developing from mucinous degeneration of periarticular connective tissue [239,240]. In the canine cases reported to date, there has been no antecedent history of trauma or signs of vertebral instability. Furthermore, the cysts do not appear to be associated with concurrent degenerative structural disorders such as cervical spondylomyelopathy.
Preliminary data indicate that juxta-facet cysts in dogs occur most commonly in the cervical vertebrae, followed by thoracic vertebra, and least commonly in lumbar vertebrae. Dickinson and colleagues identified two groups [237]:
a) young (e.g., 12 - 36 months), giant breed dogs (e.g., Mastiffs and Great Danes) with multiple cysts involving one or more

levels of the cervical spinal cord usually affecting several vertebrae from C4 - C5 to C6 - C7; and
b) older (e.g., 7 - 9 years), large breed dogs (including German Shorthaired Pointer and German Shepherds) with solitary cysts involving the thoracolumbar spinal cord and affecting vertebrae T13 - L1 or L1 - L2.

This classification seems appropriate since the 4 cases reported by Levitski and colleagues included 3 Mastiffs and a Great Dane aged between 15 and 18 months with variable cervical lesions from C3 - C4 to C6 - C7 associated with single or multiple cystic lesions [234]. The case reported by Flegel and colleagues also involved an 18 month old Great Dane with multiple cervical synovial cysts [235]. Consistent with the above-mentioned classification, the case report of an 8 year old Siberian Husky involved a single cyst located at the level of T13 - L1 vertebral level [236].

Clinical signs reflect location of spinal cord compression, e.g., cervical, cervicothoracic, thoracolumbar, or lumbosacral. Many dogs with cervical lesions show evidence of cervical pain [234]. The clinical course may be slowly progressive over several months. In one Great Dane, progressive ataxia and tetraparesis was noted over a 1 year period [237]. In the single case report to date on lumbar ganglion cysts in a 6 year old German Shepherd, the dog had a 6 month history of intermittent hind limb lameness, especially after exercise, difficulty handling stairs, and evidence of lumbosacral pain on hip extension and tail dorsiflexion [245]. Radiographic studies in dogs are usually unremarkable except for presence of degenerative arthritis of the facet joints and/or degenerative changes in intervertebral disks [234,237,238]. Lumbarization of S1 vertebra and fusion of the first caudal vertebra to the sacrum were present in the dog with multiple lumbar ganglion cysts [238]. Spinal cord compression has been demonstrated using myelography, usually with areas of axial deviation at sites of articular degeneration, and typically medial to the articulations [236,237]. In one report, a 1 by 2 cm cyst was found in the ventral epidural space associated with a pedicle attachment of the right T13 - L1 vertebral articular processes [236]. In a magnetic resonance imaging study, cysts appeared as well-defined circular defects, slightly hyperintense on post-contrast T1-weighted axial images and hyperintense on T2-weighted scans [234]. MRI revealed presence of multiple cysts arising from the L6 - L7 and L7 - S1 articular processes in one dog with lumbar lesions [245]. In addition to the presence of extradural transparent, fluid-filled cysts (typically 0.2 - 1 cm in size) usually adjacent to articular facets, surgical findings may include hypertrophic interarcuate ligaments, articular facets, and joint capsules [234].

Cerebrospinal fluid may be normal or characterized by mild to moderate protein increase ranging from 40 to 500 mg/dL, with normal cellularity or mild pleocytosis (usually mononuclear but sometimes with occasional neutrophils) [234,236,237]. Treatment by surgical decompression (e.g., hemilaminectomy or dorsal laminectomy) and removal of the cysts has usually resulted in satisfactory resolution of clinical signs and improvement in function postoperatively [234,236,237]. Vertebral stabilization using arthrodesis (lumbosacral) or a dynamic compression plate (thoracolumbar) has been performed in some dogs in addition to spinal cord decompression. Long-term prognosis appears to be good [234,236,237], although in one case, clinical recovery was incomplete following decompressive surgery [235]. To date, recurrence has been reported in one dog with a cervical lesion that improved with conservative treatment [237].

Spondylosis Deformans
Spondylosis deformans is a degenerative, proliferative disease of the vertebral column characterized by the presence of vertebral osteophytes at intervertebral spaces, resulting in the formation of spurs or complete bony bridges [246,247]. Synonyms include spondylitis ossificans deformans, ankylosing spondylitis, spondylitis deformans, deformative ossifying spondylitis, spondyloarthritis, and spondylitis [246,247], all of which incorrectly imply the presence of inflammation [248]. Spondylosis deformans has been reported in dogs and cats, usually middle-aged, but some as early as 2 years of age. The incidence increases with age. Spondylosis occurs in about 50% of dogs by 6 years of age and 75% by 9 years [247]. It reportedly occurs in about 70% of asymptomatic domestic cats. A high incidence has been noted in the Boxer breed in which the condition is considered to be inherited [249]. In Boxers, dogs as young as one year of age may be affected, females are more often affected than males, and a positive correlation between hip dysplasia and spondylosis deformans has been noted [250,251]. In several comprehensive canine studies, Flat-coated Retrievers, Irish Setters, Bloodhounds, Rhodesian Ridgebacks, German Shepherds, Airedale Terriers, and Cocker Spaniels were identified as having a medium-high risk of spondylosis deformans, and females had a significantly higher incidence than males [246,252]. In dogs, vertebral sites most often affected were T9 - T10 and L7 - S1 in one study based on radiography of vertebral columns removed after death [246]. In some animals, the entire spine may be extensively involved [253]. Thoracic vertebrae are more commonly affected in cats [247].

While heritability appears to be a factor in some (perhaps all ?) of the high-susceptibility breeds [249], the underlying mechanisms responsible for spondylosis deformans remain unclear. It may be associated with degenerative changes in the annulus fibrosus of the intervertebral disks [246,254], particularly where the peripheral fibers of the annulus fibrosus (Sharpey's fibers) attach to the vertebral rim [247]. Interestingly, in an experimental collagenase chemonucleosis study in cervical disks of normal dogs, spondylosis deformans developed at the sites of cervical enzyme injections [143], suggesting that trauma may be a predisposing factor in some instances (curiously, osteophytes did not develop in injected thoracic or

lumbar disks). Spondylosis deformans has been observed 1 - 4 years after intervertebral disk fenestration [255]. While spondylosis may occur secondary to disk degeneration/herniation (see cervical spondylomyelopathy), some consider the condition to be simply a manifestation of chronic degenerative disk disease, with bony spurs forming around diseased disks in an attempt to re-establish stability to the weakened disk spaces [246,254,256]. This view is not universally held (at least by me), especially since spondylosis deformans is uncommonly observed in chondrodystrophoid breeds predisposed to disk disease, such as Dachshunds and Poodles [246]. In one study involving 30 healthy Beagles [257], histological evidence of disk degeneration and changes in the mechanical properties of the intervertebral disk joint preceded radiographic changes of spondylosis. Karkkainen and colleagues, using magnetic resonance imaging, noted the presence of marked spondylosis deformans adjacent to intervertebral disks without evidence of disk degeneration [114]. Also, it is difficult to see a relationship between radiographic spondylosis deformans and disk degeneration in dogs one to two years of age [246,250,251]. While a radiographic study of a closed colony of Beagles (a chondrodystrophoid breed) did reveal the presence of spondylosis deformans that was age-related and located principally at low cervical, mid thoracic and cranial lumbar vertebral sites, these sites were somewhat different from the usual reports, and it is noteworthy that the lumbosacral site was minimally involved [258]. The role of spinal stress in spondylosis remains enigmatic. Some workers favor the idea that the sites most frequently involved represent those regions subjected to greatest mechanical stresses [256], while Morgan and colleagues state that normal or abnormal spinal motion, trauma, or areas of ligamentous attachment do not satisfactorily explain the variable frequency of lesions [246]. Lumbosacral osteophyte formation secondary to lumbosacral joint instability was considered as an unlikely event, based on quantitative lumbosacral angulation measurements [259]. Conversely, results of a recent radiographic study examining position and shape of osteophyte formations at canine vertebral endplates favored a role for mechanical factors in the pathogenesis of spondylosis deformans [260]. Anatomical dissection of the lumbosacral spinal columns from German Shepherds with spondylosis deformans revealed that diseased vertebrae have more flexibility than healthy vertebrae in the sagittal and frontal planes than but less so for dorsal flexion [261]. An imperfect L7 facet geometry may also predispose dogs, especially German Shepherds, to spondylosis deformans and may be influenced by congenital factors as well as body weight and locomotion in immature dogs [262]. Immunogenetic studies of Boxers failed to show any significant correlation between spondylosis deformans and leukocyte antigens and complement C4 allotypes [263]. Osteophytes tend to develop on ventral, lateral, or dorsolateral aspects of vertebral margins. Dorsally projecting osteophytes are rare. Classification based on extent of osteophytic proliferation has been proposed [246], ranging from small spurs projecting vertically from the vertebral body (grade 1), larger osteophytes with parrot's beak shape on both sides of the joint space (grade 2), osteophytic projection beneath the intervertebral space (grade 3), to extensive bridging of the intervertebral space resulting in bony fusion (grade 4). Osteophytic projections into the spinal canal, with compression of the spinal cord, is rare [264]. Similarly, osteophyic compression of spinal nerves at the level of the intervertebral foramina is infrequently encountered. In animals with spondylosis deformans, the disk space is usually of normal width [247,264]. Osteophytes seen after disk disease, with narrowing of the intervertebral spaces, tend to develop perpendicular to the vertebral body rather than bridge the disk space, and sclerosis may be seen in the vertebral end-plates [264].

In spite of the often dramatic radiographic changes, spondylosis deformans in dogs and cats tends to be a subclinical disorder, although stiffness, restricted motion, and pain might be attributed to spondylosis deformans in a small percentage of patients [247,265], sometimes in association with fracture of bony spurs or bridges. It is possible that subtle signs may be more easily detected in dogs required to be agile in work or sport [266]. Diagnosis is based on spinal radiography, with osteophytic presence often seen as incidental findings. Standard ventrodorsal and lateral radiographs without oblique views may miss many ventrolateral osteophytes [247,264]. Langeland and Stigen [267] found that oblique views contributed to a more exact localization of osteophytes, especially in the region of L6 - 7 and L7 - S1 where the intervertebral spaces are overlapped by the os ilium. In general, however, they considered that oblique views provided only minimal additional information for evaluating number and size of osteophytes. Advanced imaging, such as magnetic resonance imaging, may demonstrate evidence of nerve root impingement [227] and demonstrate soft tissue changes suggestive of degeneration of intervertebral disks, including loss of hydration of the nucleus pulposus [114]. Note that spondylosis deformans is commonly seen radiographically in dogs with degenerative lumbosacral stenosis [268]. Spondylosis deformans was also present in a 6 year old German Shepherd with intraspinal cysts (see spinal synovial cysts) of the L6 - L7 and L7 - S1 articular process joints along with lumbarization of the first sacral vertebra and fusion of the first caudal vertebra to the sacrum [238]. Treatment of spondylosis deformans is usually unnecessary. Nevertheless, analgesics can be given if spinal pain can be attributed to spondylosis deformans [265]. Surgery is only indicated in those rare instances in which both pain and neural deficit are present due to spinal cord or nerve root compression. Prognosis in animals with spondylosis deformans is usually very favorable.

Miscellaneous Disorders
Several structural disorders, some of which are quite uncommon, may also be associated with compressive neurological

signs. These include **Chiari malformations**, **atlantoaxial subluxation**, and scoliosis/kyphosis/hemivertebrae/block vertebrae (see vertebral anomalies). These conditions are discussed in the chapter on Developmental Disorders. Skeletal abnormalities occasionally causing spinal cord and or nerve root compression include mucopolysaccharidoses (see Mucopolysaccharidosis type VI), osteochondromatosis, hypervitaminosis A, and osteopenia-related spinal fractures and lordosis/kyphosis associated with nutritional secondary hyperparathyroidism. Compression of neural structures (spinal cord, brainstem, nerve roots) may be caused by a variety of mesenchymal and/or ectodermal cysts, including arachnoid cysts / intra-arachnoid cysts, and epidermoid and dermoid cysts (see malformation tumors. Cervical fibrotic stenosis (at C2 - C3 articulation) associated with yellow ligament proliferation, but without evidence of cervical instability, has been reported in an 18 month old neutered male Rottweiler [273]. Clinical signs were similar to those seen in dogs with cervical spondylomyelopathy. The stenosis was confirmed using myelography (no abnormalities were seen with survey radiography) and at surgery. Severe degeneration of the spinal cord affecting all funiculi and gray matter was found at the level of the yellow ligament compression. Identical myelographic and necropsy findings were observed by the authors in another young Rottweiler (15 months of age) presented for progressive ataxia. Acute and chronic spinal cord compression may occur from foreign bodies such as wood fragments secondary to oropharyngeal stick injuries [285,286].

References

1. Calcinosis circumscripta and tumoral calcinosis.In: Anderson DM, Keith J, Novak PD, et al, eds. Dorland's Illustrated Medical Dictionary. 28th ed. Philadelphia: WB Saunders Co, 1988; 246.
2. Spjut HJ, Dorfman HD, Fechner RE, et al. Tumoral calcinosis. Tumors of bone and cartilage. Washington: AFIP, 2nd series, Fascicle 5, 1970; 423-428.
3. Scott DW, Miller WH, Griffin CE. Muller & Kirk's Small Animal dermatology. 6th ed. Philadelphia: WB Saunders Co, 2001; 1400-1401.
4. Liu S-K. Tumors of bone and cartilage. In: Bojrab MJ, Smeak DD and Bloomberg MS, eds. Disease mechanisms in small animal surgery. 2nd ed. Philadelphia: Lea & Febiger, 1993; 923-925.
5. Pool RR. Tumoral calcinosis. In: Moulton JE, ed. Tumors in domestic animals. 3rd ed. Berkeley: University of California Press, 1990; 123-125.
6. Kowalewich N, Hawkins EC. Calcinosis circumscripta involving the metatarsal region in a dog with chronic renal failure. Can Vet J 1992; 33:465-466.
7. Scott DW, Buerger RG. Idiopathic calcinosis circumscripta in the dog: a retrospective analysis of 130 cases. J Am Anim Hosp Assoc 1988; 24:651-658.
8. Roudebush P, Maslin WR, Cooper RC. Canine tumoral calcinosis. Compend Contin Educ Pract Vet 1988; 10:1162-1163.
9. Stampley A, Bellah JR. Calcinosis circumscripta of the metacarpal pad in a dog. J Am Vet Med Assoc 1990; 196:113-114.
10. Joffe DJ. Calcinosis circumscripta in the footpad of a dog. Can Vet J 1996; 37:161-162.
11. Morgan PW, Cockshutt J. What is your diagnosis? [Tumoral calcinosis in dog]. J Am Vet Med Assoc 1993; 203:969-970.
12. Berrocal A, Tjalsma EJ, Koeman JP. Calcinosis circumscripta in two cats. Feline Pract 1992; 20:9-12.
13. Croom AL, Houston DM. Hyperphosphatemic tumoral calcinosis in a young dog with renal failure. Can Vet J 1994; 35:438-440.
14. Komori S, Washizu M. Metastatic calcinosis circumscripta treated with an oral charcoal absorbent in a dog. J Vet Med Sci 2001; 63:913-916.
15. Kirby BM, Knoll JS, Manley PA, et al. Calcinosis circumscripta associated with polydioxanone suture in two young dogs. Vet Surg 1989; 18:216-220.
16. Ginel P, Perez J, Rivas R, et al. Calcinosis circumscripta associated with medroxyprogesterone in two Poodle bitches. J Am Anim Hosp Assoc 1992; 28:391-394.
17. Ginel PJ, Lopez R, Rivas R, et al. A further case of medroxyprogesterone acetate associated with calcinosis circumscripta in the dog. Vet Rec 1995; 136:44-45.
18. Davidson EB, Schulz KS, Wisner ER, et al. Calcinosis circumscripta of the thoracic wall in a German shepherd dog. J Am Anim Hosp Assoc 1998; 34:153-156.
19. O'Brien CR, Wilkie JS. Calcinosis circumscripta following an injection of proligestone in a Burmese cat. Aust Vet J 2001; 79:187-189.
20. Gardner DE, Alley MR, Wyburn RS, et al. Calcinosis circumscripta-like lesions in dogs associated with the use of choke chains. N Z Vet J 1975; 23:95-97.
21. Jacobson LS, Kirberger RM. Canine multiple cartilaginous exostoses: unusual manifestations and a review of the literature. J Am Anim Hosp Assoc 1996; 32:45-51.
22. Guilliard MJ. Fibrodysplasia ossificans in a German shepherd dog. J Small Anim Pract 2001; 42:550-553.

23. Lewis DG, Kelly DF. Calcinosis circumscripta in dogs as a cause of spinal ataxia. J Small Anim Pract 1990; 31:35-37.

24. Marks SL, Bellah JR, Wells M. Resolution of quadriparesis caused by cervical tumoral calcinosis in a dog. J Am Anim Hosp Assoc 1991; 27:72-76.

25. McEwan JD, Thomson C, Sullivan M, et al. Thoracic spinal calcinosis circumscripta causing cord compression in two German shepherd dog littermates. Vet Rec 1992; 130:575-578.

26. van Ham LM, Bree HJv, Tshamala M, et al. Use of computed tomography and computed tomographic myelography for assessment of spinal tumoral calcinosis in a dog. Vet Radiol Ultrasound 1995; 36:115-118.

27. Corlazzoli DS, Marinucci AMT, Degna MT. Tumoral calcinosis at the atlantoaxial joint causing a neurological impairment: a case report in a dog. [Italian]. Veterinaria (Cremona) 1997; 11:101-104.

28. Flo GL, Tvedten H. Cervical calcinosis circumscripta in three related Great Dane dogs. J Am Anim Hosp Assoc 1975; 11:507-510.

29. Bichsel P, Lang J, Vandevelde M, et al. Solitary cartilaginous exostoses associated with spinal cord compression in three large-breed dogs. J Am Anim Hosp Assoc 1985; 21:619-622.

30. Bhatti S, van Ham L, Putcuyps I, et al. Atlantoaxial exostosis causing spinal cord compression in a mature Bernese mountain dog. J Small Anim Pract 2001; 42:79-81.

31. McKee WM, Lavelle RB, Richardson JL, et al. Vertebral distraction-fusion for cervical spondylopathy using a screw and double washer technique. J Small Anim Pract 1990; 31:21-26.

32. Burbidge HM, Pfeiffer DU, Blair HT. Canine wobbler syndrome: a study of the Dobermann Pinscher in New Zealand. N Z Vet J 1994; 42:221-228.

33. Lewis DG. Radiological assessment of the cervical spine of the Dobermann with reference to cervical spondylomyelopathy. J Small Anim Pract 1991; 32:75-82.

34. McKee WM, SJ B, HW S. Management of cervical spondylopathy-associated intervertebral disc protrusions using metal washers in 78 dogs. J Small Anim Pract 1999; 40:465-472.

35. Lewis DG. Cervical spondylomyelopathy ("wobbler" syndrome) in the dog: a study based on 224 cases. J Small Anim Pract 1989; 30:657-665.

36. Read RA, Robins GM, Carlisle CH. Caudal cervical spondylo-myelopathy (wobbler syndrome) in the dog: a review of thirty cases. J Small Anim Pract 1983; 24:605-621.

37. de Lahunta A. Veterinary Neuroanatomy and Clinical Neurology. 2nd ed. Philadelphia: WB Saunders Co, 1983; 199-204.

38. Selcer RR, Oliver JE, Jr. Cervical spondylopathy-wobbler syndrome in dogs. J Am Anim Hosp Assoc 1975; 11:175-179.

39. Mason TA. Cervical vertebral instability (wobbler syndrome) in the dog. Vet Rec 1979; 104:142-145.

40. Hazewinkel HAW. Nutrition in relation to skeletal growth deformities. J Small Anim Pract 1989; 30:625-630.

41. Teare JA, Hintz HF, Krook L. Rapid growth and skeletal disease in dogs. In: Proceedings of the Cornell Nutritional Conf (Nutrition Abstracts and Reviews, B 52, 1508) 1980; 126-130.

42. Hedhammer A, Wu F, Krook L, et al. Over-nutrition and skeletal disease. An experimental study in growing Great Dane dogs. Cornell Vet 1974; 64:58-64.

43. VanGundy T. Canine wobbler syndrome. Part 1. Pathophysiology and diagnosis. Compend Contin Educ Pract Vet 1989; 11:144-157.

44. Wright J. The use of sagittal diameter measurements in the diagnosis of cervical spinal stenosis. J Small Anim Pract 1979; 20:331-344.

45. McKee WM, Lavelle RB, Mason TA. Vertebral stabilisation for cervical spondylopathy using a screw and washer technique. J Small Anim Pract 1989; 30:337-342.

46. Seim H, Withrow S. Pathophysiology and diagnosis of caudal cervical spondylo-myelopathy with emphasis on the Doberman Pinscher. J Am Anim Hosp Assoc 1982; 18:241-251.

47. Sharp NJH, Cofone M, Robertson ID, et al. Computed tomography in the evaluation of caudal cervical spondylomyelopathy of the Doberman Pinscher. Vet Radiol Ultrasound 1995; 36:100-108.

48. Wright F, Rest JR, Palmer AC. Ataxia of the Great Dane caused by stenosis of the cervical vertebral canal: comparison with similar conditions in the Basset Hound, Doberman Pinscher, Ridgeback and the Thoroughbred horse. Veterinary Record 1973; 92:1-6.

49. VanGundy TE. Disc-associated wobbler syndrome in the Doberman pinscher. Vet Clin North Am Small Anim Pract 1988; 18:667-696.

50. Olsson SE, Stavenborn M, Hoppe F. Dynamic compression of the cervical spinal cord. A myelographic and pathologic investigation in Great Dane dogs. Acta Vet Scand 1982; 23:65-78.

51. Bruecker KA, Seim HB, Blass CE. Ventral decompression and stabilization using Steinmann pins and polymethyl methacrylate for the treatment of caudal cervical spondylomyelopathy: results of 39 cases. Vet Surg 1988; 17:1,31.

52. Bruecker KA, Seim HB, Blass CE. Caudal cervical spondylomyelopathy: decompression by linear traction and

stabilization with Steinmann pins and polymethyl methacrylate. J Am Anim Hosp Assoc 1989; 25:677-683.

53. Bruecker KA, Seim HB, Withrow SJ. Clinical evaluation of three surgical methods for treatment of caudal cervical spondylomyelopathy of dogs. Vet Surg 1989; 18:197-203.

54. Bruecker KA, Seim HB, Withrow SJ. Ventral decompression and lubra-plate stabilization for the treatment of caudal cervical spondylomyelopathy: results of 37 cases. Vet Surg 1987; 16:84-85.

55. Dixon BC, Tomlinson JL, Kraus KH. Modified distraction-stabilization technique using an interbody polymethyl methacrylate plug in dogs with caudal cervical spondylomyelopathy. J Am Vet Med Assoc 1996; 208:61-68.

56. Ellison GW, Seim HB, Clemmons RM. Distracted cervical spinal fusion for management of caudal cervical spondylomyelopathy in large-breed dogs. J Am Vet Med Assoc 1988; 193:447-453.

57. Goring RL, Beale BS, Faulkner RF. The inverted cone decompression technique: a surgical treatment for cervical vertebral instability "Wobbler syndrome" in Doberman Pinschers. Part 1. J Am Anim Hosp Assoc 1991; 27:403-409.

58. Rusbridge C, Wheeler SJ, Torrington AM, et al. Comparison of two surgical techniques for the management of cervical spondylomyelopathy in dobermanns. J Small Anim Pract 1998; 39:425-431.

59. Adamo PF, Cherubini GB, Mocavero A, et al. Short and long term results in eight cases of caudal cervical spondylomyelopathy by using an interbody methylmethacrylate plug. In: Proceedings of the 14th Annu Symposium, ESVN 2000; 38.

60. Wilson ER, Aron DN, Roberts RE. Observation of a secondary compressive lesion after treatment of caudal cervical spondylomyelopathy in a dog. J Am Vet Med Assoc 1994; 205:1297-1299.

61. Boydell P. Horner's syndrome following cervical spinal surgery in the dog. J Small Anim Pract 1995; 36:510-512.

62. Lewis DD, Hosgood G. Complications associated with the use of iohexol for myelography of the cervical vertebral column in dogs: 66 cases (1988-1990). J Am Vet Med Assoc 1992; 200:1381-1384.

63. VanGundy T. Canine wobbler syndrome. Part II. Treatment. Compend Contin Educ Pract Vet 1989; 11:269...284.

64. Seim HB. Wobbler syndrome in the Doberman pinscher. Canine Practice 1994; 19:23-26.

65. Palmer A, Wallace M. Deformation of cervical vertebrae in bassett hounds. Vet Rec 1967; 80:430.

66. Jaggy A, Gaillard C, Lang J, et al. Hereditary cervical spondylopathy (wobbler syndrome) in the Borzoi dog. J Am Anim Hosp Assoc 1988; 24:453-460.

67. Bray JP, Burbidge HM. The canine intervertebral disk: part one: structure and function. J Am Anim Hosp Assoc 1998; 34:55-63.

68. Braund KG. Canine intervertebral disk disease. In: Bojrab MJ, ed. Pathophysiology in Small Animal Surgery. Philadelphia: Lea and Febiger, 1981; 739-746.

69. Braund KG. Intervertebral disk disease. In: Kornegay JN, ed. Neurologic Disorders: Contemporary Issues in Small Animal Practice. New York: Churchill Livingstone, 1986; 21-39.

70. Braund KG, Ghosh P, Taylor TK, et al. Morphological studies of the canine intervertebral disc. The assignment of the beagle to the achondroplastic classification. Res Vet Sci 1975; 19:167-172.

71. Hansen H-J. A pathologic-anatomical study on disc degeneration in the dog. Acta Orthop Scand 1952; suppl 11:1-117.

72. Ghosh P, Taylor TK, Braund KG, et al. A comparative chemical and histochemical study of the chondrodystrophoid and nonchondrodystrophoid canine intervertebral disc. Vet Pathol 1976; 13:414-427.

73. Braund KG, Ghosh P, Taylor TK, et al. The qualitative assessment of glycosaminoglycans in the canine intervertebral disc using a critical electrolyte concentration staining technique. Res Vet Sci 1976; 21:314-317.

74. Ghosh P, Taylor TK, Braund KG. The variation of the glycosaminoglycans of the canine intervertebral disc with ageing. I. Chondrodystrophoid breed. Gerontology 1977; 23:87-98.

75. Ghosh P, Taylor TK, Braund KG. Variation of the glycosaminoglycans of the intervertebral disc with ageing. II. Non-chondrodystrophoid breed. Gerontology 1977; 23:99-109.

76. Ghosh P, Taylor TK, Braund KG, et al. The collagenous and non-collagenous protein of the canine intervertebral disc and their variation with age, spinal level and breed. Gerontology 1976; 22:124-134.

77. Holm S, Maroudas A, Urban JP, et al. Nutrition of the intervertebral disc: solute transport and metabolism. Connect Tissue Res 1981; 8:101-119.

78. Holm S, Nachemson A. Variations in the nutrition of the canine intervertebral disc induced by motion. Spine 1983; 8:866-874.

79. Taylor TK, Ghosh P, Braund KG, et al. The effect of spinal fusion on intervertebral disc composition: an experimental study. J Surg Res 1976; 21:91-104.

80. Hay CW, Muir P. Tearing of the dura mater in three dogs. Vet Rec 2000; 146:279-282.

81. Ghosh P, Taylor TK, Yarroll JM. Genetic factors in the maturation of the canine intervertebral disc. Res Vet Sci 1975; 19:304-311.

82. Ball MU, McGuire JA, Swaim SF, et al. Patterns of occurrence of disk disease among registered dachshunds. J Am Vet Med Assoc 1982; 180:519-522.

83. Brown DC, Conzemius MG, Shofer FS. Body weight as a predisposing factor for humeral condylar fractures, cranial cruciate rupture and intervertebral disc disease in Cocker Spaniels. Veterinary & Comparative Orthopaedics & Traumatology 1996; 9:38-41.

84. Griffiths IR. Some aspects of the pathology and pathogenesis of the myelopathy caused by disc protrusions in the dog. J Neurol Neurosurg Psychiatry 1972; 35:403-413.

85. Wright F, Palmer AC. Morphological changes caused by pressure on the spinal cord. Pathol Vet 1969; 6:355-368.

86. Morgan JP, Atilola M, Bailey CS. Vertebral canal and spinal cord mensuration: a comparative study of its effect on lumbosacral myelography in the dachshund and German shepherd dog. J Am Vet Med Assoc 1987; 191:951-957.

87. Cudia SP, Duval JM. Thoracolumbar intervertebral disk disease in large, nonchondrodystrophic dogs: a retrospective study. J Am Anim Hosp Assoc 1997; 33:456-460.

88. Liptak JM, Watt PR, Thomson MJ, et al. Hansen type I disk disease at T1 - 2 in a dachshund. Aust Vet J 1999; 77:156-159.

89. Hoerlein BF. Canine Neurology: Diagnosis and Treatment. 3rd ed. Philadelphia: WB Saunders Co, 1978; 470-560.

90. Morgan PW, Parent J, Holmberg DL. Cervical pain secondary to intervertebral disc disease in dogs; radiographic findings and surgical implications. Prog Vet Neurol 1993; 4:76-80.

91. Dallman MJ, Palettas P, Bojrab MJ. Characteristics of dogs admitted for treatment of cervical intervertebral disk disease: 105 cases (1972-1982). J Am Vet Med Assoc 1992; 200:2009-2011.

92. King AS, Smith RN. Disc protrusion in the cat: incidence of dorsal protrusions. Vet Rec 1960; 72:381-383.

93. King AS, Smith RN. Disc protrusion in the cat: distribution of dorsal protrusions along the vertebral column. Vet Rec 1960; 72:335-337.

94. Sparkes AH, Skerry TM. Successful management of a prolapsed intervertebral disc in a Siamese cat. Feline Pract 1990; 18:7-9.

95. Salisbury SK, Cook JR, Jr. Recovery of neurological function following focal myelomalacia in a cat. J Am Anim Hosp Assoc 1988; 24:227-230.

96. Munana KR, Olby NJ, Sharp NJH, et al. Intervertebral disk disease in 10 cats. J Am Anim Hosp Assoc 2001; 37:384-389.

97. Prata RG. Neurosurgical treatment of thoracolumbar disks: the rationale and value of laminectomy with concomitant disk removal. J Am Anim Hosp Assoc 1981; 17:17-26.

98. Knapp DW, Pope ER, Hewett JE, et al. A retrospective study of thoracolumbar disk fenestration in dogs using a ventral approach: 160 cases (1976 to 1986). J Am Anim Hosp Assoc 1990; 26:543-549.

99. Sarit D, Glickman N, Waters DJ. Reoperative neurosurgery in dogs with thoracolumbar disc disease. Vet Surg 1999; 28:421-428.

100. Coates JR. Intervertebral disk disease. Vet Clin North Am Small Anim Pract 2000; 30:77-110.

101. Stigen O. Calcification of intervertebral discs in the dachshund: a radiographic study of 21 stud-dogs. Acta Vet Scand 1995; 36:329-334.

102. Stigen O. Calcification of intervertebral discs in the dachshund. A radiographic study of 327 young dogs. Acta Vet Scand 1991; 32:197-203.

103. Stigen O. Calcification of intervertebral discs in the dachshund: a radiographic study of 115 dogs at 1 and 5 years of age. Acta Vet Scand 1996; 37:229-237.

104. Stigen O, Christensen K. Calcification of intervertebral discs in the dachshund: an estimation of heritability. Acta Vet Scand 1993; 34:357-361.

105. Jensen VF, Ersboll AK. Mechanical factors affecting the occurrence of intervertebral disc calcification in the Dachshund - a population study. J Vet Med A Physiol Pathol Clin Med 2000; 47:283-296.

106. Smith JD, Newell SM, Budsberg SC, et al. Incidence of contralateral versus ipsilateral neurological signs associated with lateralised Hansen type I disc extrusion. J Small Anim Pract 1997; 38:495-497.

107. Sukhiani HR, Parent JM, Atilola MA, et al. Intervertebral disk disease in dogs with signs of back pain alone: 25 cases (1986-1993). J Am Vet Med Assoc 1996; 209:1275-1279.

108. Nguyen C, An H, Ho KC, et al. Utility of high-dose contrast enhancement for detecting recurrent herniated intervertebral disks. AJNR Am J Neuroradiol 1994; 15:1291-1297.

109. Levitski RE, Lipsitz D, Chauvet AE. Magnetic resonance imaging of the cervical spine in 27 dogs. Vet Radiol Ultrasound 1999; 40:332-341.

110. Olby NJ, Munana KR, Sharp NJ, et al. A comparison of computed tomography and myelography in the diagnosis of acute intervertebral disc disease in dogs. J Vet Intern Med 1999; 13:239.

111. Olby NJ, Munana KR, Sharp NJ, et al. The computed tomographic appearance of acute thoracolumbar intervertebral disc herniations in dogs. Vet Radiol Ultrasound 2000; 41:396-402.

112. Jones JC, Inzana KD. Subclinical CT abnormalities in the lumbosacral spine of older large- breed dogs. Vet Radiol

Ultrasound 2000; 41:19-26.

113. Hara Y, Tagawa M, Ejima H, et al. Usefulness of computed tomography after myelography for surgery on dogs with cervical intervertebral disc protrusion. J Vet Med Sci 1994; 56:791-794.

114. Karkkainen M, Punto LU, Tulamo RM. Magnetic resonance imaging of canine degenerative lumbar spine diseases. Vet Radiol Ultrasound 1993; 34:399-404.

115. Sether LA, Nguyen C, Yu SN, et al. Canine intervertebral disks: correlation of anatomy and MR imaging. Radiology 1990; 175:207-211.

116. Thomson CE, Kornegay JN, Stevens JB. Canine intervertebral disc disease: changes in the cerebrospinal fluid. J Small Anim Pract 1989; 30:685-688.

117. Olby N, Sharp NJ, Munana KR, et al. Chronic and acute compressive spinal cord lesions in dogs are associated with increased lumbar CSF glutamate levels. J Vet Intern Med 1999; 13:241.

118. Gaschen L, Lang J, Haeni H. Intravertebral disc herniation (Schmorl's node) in five dogs. Vet Radiol Ultrasound 1995; 36:509-516.

119. Janssens LA. Acupuncture for the treatment of thoracolumbar and cervical disc disease in the dog. Probl Vet Med 1992; 4:107-116.

120. Still J. Auriculotherapy and canine thoracolumbar disc disease. J S Afr Vet Assoc 1991; 62:3.

121. Janssens LA, Rogers PA. Acupuncture versus surgery in canine thoracolumbar disc disease. Vet Rec 1989; 124:283.

122. Janssens L. Acupuncture in thoracolumbar disc disease. J S Afr Vet Assoc 1991; 62:2.

123. Braund KG, Taylor TKF, Ghosh P, et al. Lateral spinal decompression in the dog. J Small Anim Pract 1976; 17:583-592.

124. Lubbe AM, Kirberger RM, Verstraete FJM. Pediculectomy for thoracolumbar spinal decompression in the Dachshund. J Am Anim Hosp Assoc 1994; 30:233-238.

125. Shores A. Neurosurgical techniques. In: Braund KG, ed. Clinical Syndromes in Veterinary Neurology. 2nd ed. St Louis: Mosby, 1994.

126. Scott HW, McKee WM. Laminectomy for 34 dogs with thoracolumbar intervertebral disc disease and loss of deep pain perception. J Small Anim Pract 1999; 40:417-422.

127. McKee M. Intervertebral disc disease in the dog: 2. Management options. In Pract 2000; 22:458-471.

128. Seim HB, III. Dorsal decompressive laminectomy for T-L disk disease. Canine Practice 1995; 20:6-10.

129. Fitch RB, Kerwin SC, Hosgood G. Caudal cervical intervertebral disk disease in the small dog: role of distraction and stabilization in ventral slot decompression. J Am Anim Hosp Assoc 2000; 36:68-74.

130. Gill PJ, Lippincott CL, Anderson SM. Dorsal laminectomy in the treatment of cervical intervertebral disk disease in small dogs: a retrospective study of 30 cases. J Am Anim Hosp Assoc 1996; 32:77-80.

131. McKee WM. A comparison of hemilaminectomy (with concomitant disc fenestration) and dorsal laminectomy for the treatment of thoracolumbar disc protrusion in dogs. Vet Rec 1992; 130:296-300.

132. Denny HR. The lateral fenestration of thoracolumbar disc protrusions in the dog. Veterinary Annual 1982; 22:169-174.

133. Butterworth SJ, Denny HR. Follow-up study of 100 cases with thoracolumbar disc protrusions treated by lateral fenestration. J Small Anim Pract 1991; 32:443-447.

134. Davies JV, Sharp NJH. A comparison of conservative treatment and fenestration for thoracolumbar intervertebral disc disease in the dog. J Small Anim Pract 1983; 24:721-729.

135. Fingeroth JM. Fenestration. Pros and cons. Probl Vet Med 1989; 1:445-466.

136. Muir P, Johnson KA, Manley PA, et al. Comparison of hemilaminectomy and dorsal laminectomy for thoracolumbar intervertebral disc extrusion in Dachshunds. J Small Anim Pract 1995; 36:360-367.

137. Holmberg DL, Palmer NC, Vanpelt D, et al. A comparison of manual and power-assisted thoracolumbar disc fenestration in dogs. Vet Surg 1990; 19:323-327.

138. Scott HW. Hemilaminectomy for the treatment of thoracolumbar disc disease in the dog: a follow-up study of 40 cases. J Small Anim Pract 1997; 38:488-494.

139. Simonet M, Cazenave A. Surgical management of a intervertebral disc herniation by percutaneous discectomy in a dog. [French]. Le Point Vétérinaire 1997; 24:1399-1404.

140. Atilola MA, Bailey CS, Morgan JP. Cervical chemonucleolysis in the dog. A surgical technique. Vet Surg 1988; 17:135-140.

141. Fry TR, Johnson AL. Chemonucleolysis for treatment of intervertebral disk disease. J Am Vet Med Assoc 1991; 199:622-627.

142. Miyabayashi T, Lord PF, Dubielzig RR, et al. Chemonucleolysis with collagenase. A radiographic and pathologic study in dogs. Vet Surg 1992; 21:189-194.

143. Atilola MAO, Cockshutt JR, McLaughlin R, et al. Collagenase chemonucleolysis - a long term radiographic study in normal dogs. Vet Radiol Ultrasound 1993; 34:321-324.

144. Bray JP, Burbidge HM, Thompson KG. A comparative study of chemonucleolysis with collagenase and fenestration on the canine intervertebral disc. Prog Vet Neurol 1996; 7:117-123.

145. Frick SL, Hanley EN, Jr., Meyer RA, Jr., et al. Lumbar intervertebral disc transfer. A canine study. Spine 1994; 19:1826-1834; discussion 1834-1825.

146. Lemarie RJ, Kerwin SC, Partington BP, et al. Vertebral subluxation following ventral cervical decompression in the dog. J Am Anim Hosp Assoc 2000; 36:348-358.

147. Bagley RS, Forrest LJ, Cauzinille L, et al. Cervical vertebral fusion and concurrent intervertebral disc extrusion in four dogs. Vet Radiol Ultrasound 1993; 34:336-339.

148. Moore RW, Withrow SJ. Gastrointestinal hemorrhage and pancreatitis associated with intervertebral disk diseases in the dog. J Am Vet Med Assoc 1982; 180:1443-1447.

149. Hoerlein BF, Spano JS. Non-neurological complications following decompressive spinal cord surgery. Arch Am Coll Vet Surg 1975; 4:11-16.

150. Neiger R, Gaschen F, Jaggy A. Gastric mucosal lesions in dogs with acute intervertebral disc disease: characterization and effects of omeprazole or misoprostol. J Vet Intern Med 2000; 14:33-36.

151. Duval J, Dewey C, Roberts R, et al. Spinal cord swelling as a myelographic indicator of prognosis: a retrospective study in dogs with intervertebral disc disease and loss of deep pain perception. Vet Surg 1996; 25:6-12.

152. Kazakos G, Polizopoulou ZS, Patsikas M, et al. Correlation of clinical, radiographic and surgical localization of thoracolumbar intervertebral disc extrusion in dogs: a report and follow up of 30 cases. In: Proceedings of the 14th Annu Symposium, ESVN 2000; 11-12.

153. Olby NJ, De Risio L, Munana KR, et al. Development of a functional scoring system in dogs with acute spinal cord injuries. Am J Vet Res 2001; 62:1624-1628.

154. Poncelet L, Michaux C, Balligand M. Somatosensory potentials in dogs with naturally acquired thoracolumbar spinal cord disease. Am J Vet Res 1993; 54:1935-1941.

155. Poncelet L, Michaux C, Balligand M. Study of spinal cord evoked injury potential by use of computer modeling and in dogs with naturally acquired thoracolumbar spinal cord compression. Am J Vet Res 1998; 59:300-306.

156. Sylvestre AM, Cockshutt JR, Parent JM, et al. Magnetic motor evoked potentials for assessing spinal cord integrity in dogs with intervertebral disc disease. Vet Surg 1993; 22:5-10.

157. Norsworthy GD. Discospondylitis as a cause of posterior paresis. Feline Pract 1979; 9:39-40.

158. Henderson RA, Hoerlein BF, Kramer TT, et al. Discospondylitis in three dogs infected with Brucella canis. J Am Vet Med Assoc 1974; 165:451-455.

159. Johnson DE, Summers BA. Osteomyelitis of the lumbar vertebrae in dogs caused by grass-seed foreign bodies. Aust Vet J 1971; 47:289-294.

160. Kornegay JN, Barber DL. Diskospondylitis in dogs. J Am Vet Med Assoc 1980; 177:337-341.

161. LaCroix JA. Vertebral body osteomyelitis: a case report. Am Vet Radiol J 1973; 14:17-21.

162. Bennett D, Carmichael S, Griffiths IR. Discospondylitis in the dog. J Small Anim Pract 1981; 22:539-547.

163. Patnaik AK, Liu S-K, Wilkins RJ, et al. Paecilomycosis in a dog. J Am Vet Med Assoc 1972; 161:806-813.

164. Wood GL, Hirsh DC, Selcer RR, et al. Disseminated aspergillosis in a dog. J Am Vet Med Assoc 1978; 172:704-707.

165. Kornegay JN, Barber DL, Earley TD. Cranial thoracic diskospondylitis in two dogs. J Am Vet Med Assoc 1979; 174:192-194.

166. Johnson RG, Prata RG. Intradiskal osteomyelitis: a conservative approach. J Am Anim Hosp Assoc 1983; 19:743-750.

167. Gilmore DR. Diskospondylitis and multifocal osteomyelitis in two dogs. J Am Vet Med Assoc 1983; 182:64-66.

168. Hurov L, Troy G, Turnwald G. Diskospondylitis in the dog: 27 cases. J Am Vet Med Assoc 1978; 173:275-281.

169. Smith KR, Kerlin RM, Mitchell G. Diskospondylitis attributable to gram-positive filamentous bacteria in a dog. J Am Vet Med Assoc 1994; 205:428-430; discussion 430-422.

170. Moore MP. Discospondylitis. Vet Clin North Am Small Anim Pract 1992; 22:1027-1034.

171. Hurov L. Laminectomy for treatment of cauda equina syndrome in a cat. J Am Vet Med Assoc 1985; 186:504-505.

172. Malik R, Latter M, Love DN. Bacterial discospondylitis in a cat. J Small Anim Pract 1990; 31:404-406.

173. Watson E, Roberts RE. Discospondylitis in a cat. Vet Radiol Ultrasound 1993; 34:397-398.

174. Aroch I, Shamir M, Harmelin A. Lumbar diskospondylitis and meningomyelitis caused by Escherichia coli in a cat. Feline Pract 1999; 27:20-22.

175. Siems JS, Jakovljevic S, Adams LG, et al. Discospondylitis in association with an intra-abdominal abscess in a dog. J Small Anim Pract 1999; 40:123-126.

176. Jacob F, Bagley RS, Moore MP, et al. Cervical intervertebral disk protrusion, discospondylitis, and porcupine quill foreign body in a dog. Prog Vet Neurol 1996; 7:53-55.

177. Remedios AM, Wagner R, Caulkett NA, et al. Epidural abscess and discospondylitis in a dog after administration of a lumbosacral epidural analgesic. Can Vet J 1996; 37:106-107.

178. van der Wel TJ, Meyer HP. Discospondylitis and immune-mediated polyarthritis in a Bernese mountain-dog. Tijdschr Diergeneeskd 1995; 120:75-77.

179. Adamo PF, Cherubini GB. Discospondylitis associated with three unreported bacteria in the dog. J Small Anim Pract 2001; 42:352-355.

180. Booth MJ, van der Lugt JJ, van Heerden A, et al. Temporary remission of disseminated paecilomycosis in a German shepherd dog treated with ketoconazole. J S Afr Vet Assoc 2001; 72:99-104.

181. Watt PR, Robins GM, Galloway AM, et al. Disseminated opportunistic fungal disease in dogs: 10 cases (1982-1990). J Am Vet Med Assoc 1995; 207:67-70.

182. Kerwin SC, Lewis DD, Hribernik TN, et al. Diskospondylitis associated with Brucella canis infection in dogs: 14 cases (1980-1991). J Am Vet Med Assoc 1992; 201:1253-1257.

183. Dallman MJ, Dew TL, Tobias L, et al. Disseminated aspergillosis in a dog with diskospondylitis and neurologic deficits. J Am Vet Med Assoc 1992; 200:511-513.

184. Carpenter JL, Myers AM, Conner MW, et al. Tuberculosis in five basset hounds. J Am Vet Med Assoc 1988; 192:1563-1568.

185. Turnwald GH, Shires PK, Turk MA, et al. Diskospondylitis in a kennel of dogs: clinicopathologic findings. J Am Vet Med Assoc 1986; 188:178-183.

186. Berry WL, Leisewitz AL. Multifocal Aspergillus terreus discospondylitis in two German shepherd dogs. J S Afr Vet Assoc 1996; 67:222-228.

187. Butterworth SJ, Barr FJ, Pearson GR, et al. Multiple discospondylitis associated with Aspergillus species infection in a dog. Vet Rec 1995; 136:38-41.

188. Kaufman AC, Greene CE, Selcer BA, et al. Systemic aspergillosis in a dog and treatment with hamycin. J Am Anim Hosp Assoc 1994; 30:132-136.

189. Thomas WB. Diskospondylitis and other vertebral infections. Vet Clin North Am Small Anim Pract 2000; 30:169-182, vii.

190. Lobetti RG. Subarachnoid abscess as a complication of discospondylitis in a dog. J Small Anim Pract 1994; 35:480-483.

191. Auger J, Dupuis J, Quesnel A, et al. Surgical treatment of lumbosacral instability caused by discospondylitis in four dogs. Vet Surg 2000; 29:70-80.

192. Wrigley RH. Malignant versus nonmalignant bone disease. Vet Clin North Am Small Anim Pract 2000; 30:315-347, vi-vii.

193. Davis MJ, Dewey CW, Walker MA, et al. Contrast radiographic findings in canine bacterial discospondylitis: a multicenter, retrospective study of 27 cases. J Am Anim Hosp Assoc 2000; 36:81-85.

194. Kraft SL, Mussman JM, Smith T, et al. Magnetic resonance imaging of presumptive lumbosacral discospondylitis in a dog. Vet Radiol Ultrasound 1998; 39:9-13.

195. Gonzalo-Orden JM, Altonaga JR, Orden MA, et al. Magnetic resonance, computed tomographic and radiologic findings in a dog with discospondylitis. Vet Radiol Ultrasound 2000; 41:142-144.

196. Kornegay JN. Diskospondylitis revisited. In: Proceedings of the 9th Annu Meet Vet Med Forum, ACVIM 1991; 291-293.

197. Fischer A, Mahaffey MB, Oliver JE. Fluoroscopically guided percutaneous disk aspiration in 10 dogs with diskospondylitis. J Vet Intern Med 1997; 11:284-287.

198. Gilmore DR. Lumbosacral diskospondylitis in 21 dogs. J Am Anim Hosp Assoc 1987; 23:57-61.

199. van Bree H, de Rick A, Verschooten F, et al. Successful conservative treatment of cervical discospondylitis in a dog. J Small Anim Pract 1981; 22:59-65.

200. Greene RT. Coccidioidomycosis. In: Greene C, ed. Infectious diseases of the dog and cat. 2nd ed. Philadelphia: WB Saunders Co, 1998; 391-398.

201. Day MJ. Canine disseminated aspergillosis. In: Greene C, ed. Infectious diseases of the dog and cat. 2nd ed. Philadelphia: WB Saunders Co, 1999; 409-413.

202. Morgan JP. Spinal dural ossification in the dog: incidence and distribution based on a radiographic study. J Am Vet Radiol Soc 1969; 10:43-48.

203. Wilson JW, Greene HJ, Leipold HW. Osseous metaplasia of the spinal dura mater in a Great Dane. J Am Vet Med Assoc 1975; 167:75-77.

204. McGrath JT. Letter: Spinal ossifying pachymeningitis. J Am Vet Med Assoc 1975; 167:1045, 1048.

205. McGrath JT. Neurologic examination of the dog. 2nd ed. Philadelphia: Lea & Febiger, 1960; 245-248.

206. de Lahunta A. Veterinary Neuroanatomy and Clinical Neurology. 2nd ed. Philadelphia: WB Saunders Co, 1983; 175-214.

207. von Sandersleben J, el Sergany MA. A study of so-called ossifying spinal pachymeningitis in the dog with special

reference to its pathogenesis and etiology. Zentralbl Veterinarmed A 1966; 13:526-540.

208. Lenehan TM. Canine cauda equina syndrome. Compend Contin Educ Pract Vet 1983; 5:941-951.

209. Palmer RH, Chambers JN. Canine lumbosacral diseases. Part I. Anatomy, pathophysiology, and clinical presentation. Compend Contin Educ Pract Vet 1991; 13:61-68.

210. Palmer RH, Chambers JN. Canine lumbosacral diseases. Part II: Definitive diagnosis, treatment, and prognosis. Compend Contin Educ Pract Vet 1991; 13:213-221.

211. Watt PR. Degenerative lumbosacral stenosis in 18 dogs. J Small Anim Pract 1991; 32:125-134.

212. Evans HE, Christensen GC. Miller's Anatomy of the Dog. 2nd ed. Philadelphia: WB Saunders Co, 1979; 947.

213. Koppel E, Rein D. [Lumbosacral instability. The cauda equina compression syndrome in dogs]. Tierarztl Prax 1992; 20:637-645.

214. Jones JC, Wright JC, Bartels JE. Computed tomographic morphometry of the lumbosacral spine of dogs. Am J Vet Res 1995; 56:1125-1132.

215. Tarvin G, Prata RG. Lumbosacral stenosis in dogs. J Am Vet Med Assoc 1980; 177:154-159.

216. Ness MG. Degenerative lumbosacral stenosis in the dog: a review of 30 cases. J Small Anim Pract 1994; 35:185-190.

217. Jones JC, Banfield CM, Ward DL. Association between postoperative outcome and results of magnetic resonance imaging and computed tomography in working dogs with degenerative lumbosacral stenosis. J Am Vet Med Assoc 2000; 216:1769-1774.

218. Danielsson F, Sjostrom L. Surgical treatment of degenerative lumbosacral stenosis in dogs. Vet Surg 1999; 28:91-98.

219. Jaggy A, Lang J, Schawalder P. Cauda equina syndrome in the dog. Schweiz Arch Tierheilkd 1987; 129:171-192.

220. Morgan JP. Transitional lumbosacral vertebral anomaly in the dog: a radiographic study. J Small Anim Pract 1999; 40:167-172.

221. Morgan JP, Bahr A, Franti CE, et al. Lumbosacral transitional vertebrae as a predisposing cause of cauda equina syndrome in German shepherd dogs: 161 cases (1987-1990). J Am Vet Med Assoc 1993; 202:1877-1882.

222. De Risio L, Thomas WB, Sharp NJ. Degenerative lumbosacral stenosis. Vet Clin North Am Small Anim Pract 2000; 30:111-132, vi.

223. Jones JC, Cartee RE, Bartels JE. Computed tomographic anatomy of the canine lumbosacral spine. Vet Radiol Ultrasound 1995; 36:91-99.

224. Barthez PY, Morgan JP, Lipsitz D. Discography and epidurography for evaluation of the lumbosacral junction in dogs with cauda equina syndrome. Vet Radiol Ultrasound 1994; 35.

225. Ramirez O, 3rd, Thrall DE. A review of imaging techniques for canine cauda equina syndrome. Vet Radiol Ultrasound 1998; 39:283-296.

226. Jones JC, Sorjonen DC, Simpson ST, et al. Comparison between computed tomographic and surgical findings in nine large-breed dogs with lumbosacral stenosis. Vet Radiol Ultrasound 1996; 37:247-256.

227. Adams WH, Daniel GB, Pardo AD, et al. Magnetic resonance imaging of the caudal lumbar and lumbosacral spine in 13 dogs (1990-1993). Vet Radiol Ultrasound 1995; 36:3-13.

228. Taga A, Taura Y, Nishimoto T, et al. The advantage of magnetic resonance imaging in diagnosis of cauda equina syndrome in dogs. J Vet Med Sci 1998; 60:1345-1348.

229. Jones JC, Shires PK, Inzana KD, et al. Evaluation of canine lumbosacral stenosis using intravenous contrast-enhanced computed tomography. Vet Radiol Ultrasound 1999; 40:108-114.

230. Schwarz T, Owen MR, Long S, et al. Vacuum disk and facet phenomenon in a dog with cauda equina syndrome. J Am Vet Med Assoc 2000; 217:862-864, 844.

231. Marsala J, Sulla I, Jalc P, et al. Multiple protracted cauda equina constrictions cause deep derangement in the lumbosacral spinal cord circuitry in the dog. Neurosci Lett 1995; 193:97-100.

232. Orendacova J, Cizkova D, Kafka J, et al. Cauda equina syndrome. Prog Neurobiol 2001; 64:613-637.

233. Oliver JE, Jr., Selcer RR, Simpson S. Cauda equina compression from lumbosacral malarticulation and malformation in the dog. J Am Vet Med Assoc 1978; 173:207-214.

234. Levitski RE, Chauvet AE, Lipsitz D. Cervical myelopathy associated with extradural synovial cysts in 4 dogs. J Vet Intern Med 1999; 13:181-186.

235. Flegel T, Kagan K, Munana K. Spinal cord compression due to synovial cysts in a Great Dane. [German]. Kleintierpraxis 2000; 45:787-792.

236. Perez B, Rollan E, Ramiro F, et al. Intraspinal synovial cyst in a dog. J Am Anim Hosp Assoc 2000; 36:235-238.

237. Dickinson PJ, Sturges BK, Berry WL, et al. Extradural spinal synovial cysts in nine dogs. J Small Anim Pract 2001; 42:502-509.

238. Webb AA, Pharr JW, Lew LJ, et al. MR imaging findings in a dog with lumbar ganglion cysts. Vet Radiol Ultrasound 2001; 42:9-13.

239. Kao CC, Winkler SS, Turner JH. Synovial cyst of spinal facet. Case report. J Neurosurg 1974; 41:372-376.

240. Deinsberger W, Schindler C, Boker DK. [Juxta-facet cysts. Pathogenesis, clinical symptoms and therapy]. Nervenarzt 1997; 68:825-830.

241. Antoniadis G, Richter HP, Kast E, et al. [Juxta-facet cysts as space-occupying intraspinal processes]. Nervenarzt 1997; 68:515-520.

242. Prestar FJ. Juxta facet cysts of the lumbar spine. Minim Invasive Neurosurg 1996; 39:45-49.

243. Finkelstein SD, Sayegh R, Watson P, et al. Juxta-facet cysts. Report of two cases and review of clinicopathologic features. Spine 1993; 18:779-782.

244. Weyns F, Van Calenbergh F, Goffin J, et al. Intraspinal juxta-facet cysts: a case of bilateral ganglion cysts. Clin Neurol Neurosurg 1992; 94:55-59.

245. Webb AA. Intradural spinal arachnoid cyst in a dog. Can Vet J 1999; 40:588-589.

246. Morgan JP, Ljunggren G, Read R. Spondylosis deformans (vertebral osteophytosis) in the dog. A radiographic study from England, Sweden and U.S.A. J Small Anim Pract 1967; 8:57-66.

247. Romatowski J. Spondylosis deformans in the dog. Compend Contin Educ Pract Vet 1986; 8:531-535, 536.

248. Kornegay JN. Vertebral diseases of large breed dogs. Neurologic disorders. Neurologic Disorders: Contemporary Issues in Small Animal Practice. New York: Churchill Livingstone, 1986. 197-215. 39 ref. 1986.

249. Langeland M, Lingaas F. Spondylosis deformans in the boxer: estimates of heritability. J Small Anim Pract 1995; 36:166-169.

250. Eichelberg H, Wurster H. Course of ossification in Boxer dogs with spondylosis deformans. [German]. Kleintierpraxis 1983; 28:393-396.

251. Eichelberg H, Wurster H. Spondylosis deformans in the Boxer. [German]. Kleintierpraxis 1982; 27:59-72.

252. Larsen JS, Selby LA. Spondylosis deformans in large dogs - relative risk by breed, age and sex. J Am Anim Hosp Assoc 1981; 17:623-625.

253. Crawford MA. Challenging cases in internal medicine: What's your diagnosis. Vet Med 1989; 84:20-26.

254. Morgan JP. Spondylosis derformans in the dog. A morphologic study with some clinical and experimental observations. Acta Orthop Scand 1967:7-87.

255. Dallman MJ, Moon ML, Giovannitti-Jensen A. Comparison of the width of the intervertebral disk space and radiographic changes before and after intervertebral disk fenestration in dogs. Am J Vet Res 1991; 52:140-145.

256. Prata RG. Diseases of the lumbosacral spine. In: Bojrab MJ, Smeak DD and Bloomberg MS, eds. Disease mechanisms in small animal surgery. 2nd ed. Philadelphia: Lea & Febiger, 1993; 987-998.

257. Gillett NA, Gerlach R, Cassidy JJ, et al. Age-related changes in the beagle spine. Acta Orthop Scand 1988; 59:503-507.

258. Morgan JP, Hansson K, Miyabayashi T. Spondylosis deformans in the female Beagle dog: a radiographic study. J Small Anim Pract 1989; 30:457-460.

259. Wright JA. Spondylosis deformans of the lumbo-sacral joint in dogs. J Small Anim Pract 1980; 21:45-58.

260. Breit S, Kunzel W. The position and shape of osteophyte formations at canine vertebral endplates and its influence on radiographic diagnosis. Anat Histol Embryol 2001; 30:179-184.

261. Viateau V, Preault H, Moissonnier P, et al. [Characterization of biomechanical behavior of the lumbosacral spine in dogs. Characteristics related to spondylosis and disk degeneration]. Chirurgie 1994; 120:94-99.

262. Breit S, Kunzel W. Breed specific osteological features of the canine lumbosacral junction. Ann Anat 2001; 183:151-157.

263. Eichelberg H, Schon W, Loeffler K, et al. [Immunogenetic studies of spondylosis deformans in boxers]. Berl Munch Tierarztl Wochenschr 1988; 101:236-239.

264. Morgan JP. Spondylosis deformans in the dog: its radiographic appearance. J Am Vet Radiol Soc 1967; 8:17-22.

265. Hoskins JD, Kerwin SC. Musculoskeletal system. Joint and vertebral column diseases. Vet Clin North Am Small Anim Pract 1997; 27:1433-1449.

266. Vaughan LC. Orthopaedic problems in old dogs. Vet Rec 1990; 126:379-388.

267. Langeland M, Stigen O. Spondylosis deformans in the Boxer. Evaluation of the inclusion of oblique projections in radiographic studies. [Norwegian]. Norsk Vet 1994; 106:297-303.

268. Koppel E, Rein D. Lumbosacral instability in dogs. A contribution to the cauda equina compression syndrome. Tierarztl Prax 1992; 20:637-645.

269. Gaschen L, Lang J, Haeni H. Intravertebral disc herniation (Schmorl's node) in five dogs. Vet Radiol Ultrasound 1995; 36:509-516.

270. Lang J, Hani H, Schawalder P. A sacral lesion resembling osteochondrosis in the German Shepherd Dog. Vet Radiol Ultrasound 1992; 33:69-76.

271. Hanna FY. Lumbosacral osteochondrosis: radiological features and surgical management in 34 dogs. J Small Anim Pract 2001; 42:272-278.

272. Jensen VF, Christensen KA. Inheritance of disc calcification in the Dachshund. J Vet Med (Series A) 2000; 47:331-340.

273. Baum F, de Lahunta A, Trotter EJ. Cervical fibrotic stenosis in a young Rottweiler. J Am Vet Med Assoc 1992; 201:1222-1224.

274. Breit S, Kunzel W. Osteological features in pure-bred dogs predisposing to cervical spinal cord compression. J Anat 2001; 199:527-537.

275. Drost WT, Lehenbauer TW, Reeves J. Mensuration of cervical vertebral ratios in Doberman pinschers and Great Danes. Vet Radiol Ultrasound 2002; 43:124-131.

276. Jeffery ND, McKee WM. Surgery for disc-associated wobbler syndrome in the dog-an examination of the controversy. J Small Anim Pract 2001; 42:574-581.

277. Mayhew PD, Kapatkin AS, Wortman JA, et al. Association of cauda equina compression on magnetic resonance images and clinical signs in dogs with degenerative lumbosacral stenosis. J Am Anim Hosp Assoc 2002; 38:555-562.

278. Tidwell AS, Specht A, Blaeser L, et al. Magnetic resonance imaging features of extradural hematomas associated with intervertebral disc herniation in a dog. Vet Radiol Ultrasound 2002;43:319-324.

279. Liptak JM, Allan GS, Krockenberger MB, et al. Radiographic diagnosis: intramedullary extrusion of an intervertebral disc. Vet Radiol Ultrasound 2002;43:272-274.

280. Lamb CR, Nicholls A, Targett M, et al. Accuracy of survey radiographic diagnosis of intervertebral disc protrusion in dogs. Vet Radiol Ultrasound 2002;43:222-228.

281. Ferreira AJ, Correia JH, Jaggy A. Thoracolumbar disc disease in 71 paraplegic dogs: influence of rate of onset and duration of clinical signs on treatment results. J Small Anim Pract 2002;43:158-163.

282. Davis GJ, Brown DC. Prognostic indicators for time to ambulation after surgical decompression in nonambulatory dogs with acute thoracolumbar disk extrusions: 112 cases. Vet Surg 2002;31:513-518.

283. Jones JC, Shires PK, Inzana KD, et al. Use of computed tomographic densitometry to quantify contrast enhancement of compressive soft tissues in the canine lumbosacral vertebral canal. Am J Vet Res 2002;63:733-737.

284. Quance-Fitch FJ, Schachter S, Christopher MM. Pleural effusion in a dog with discospondylitis. Vet Clin Pathol 2002;31:69-71.

285. Brockman DJ, Trout NJ. Epidural migration of a suspected pharyngeal foreign body in a dog. Vet Rec 1991;128:210-211.

286. Rayward RM. Acute onset quadriparesis as a sequela to an oropharyngeal stick injury. J Small Anim Pract 2002;43:295-298.

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