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## **Spondylosis Deformans** ( 1-Jan-1985 )

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Spondylosis deformans is a generalized disease of aging that is secondary to the degeneration of intervertebral disks that affects the vertebral bodies of all animals. It is characterized by the formation of bony spurs or, less commonly, complete bony bridges around the diseased disk and thus reestablishes stability to the weakened amphiarthrodial joint. The rate and type of the disk degeneration therefore effect the occurrence of spondylosis deformans. It should be noted that the spurs do not originate from the end-plate but arise from the circumference of the end-plate and are thus "periarticular" in nature.

The formation of the osteophytes in spondylosis deformans is without clinical signs. However, as the spurs create pressure on exiting spinal nerve roots, the possibility of resulting neurologic deficit must be considered. While the osteophytes typically do not project into the spinal canal, this possibility with its resulting cord pressure must also be recognized.

Instability of the intervertebral disks may result from other conditions, including congenital vertebral deformities, following disk surgery, following trauma, and following disk or vertebral body infection. The vertebral spurs that form around these individually injured or weakened disks are better considered separately and are not included within the generalized condition of spondylosis deformans. Histologic changes within the developing bony spurs are identical, however, regardless of the etiology. Much of the information that follows in the sections Review of the Literature and Pathogenesis is taken directly from an earlier monograph.(19)

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### **Review of the Literature**

Disagreement on specific pathogenesis of spondylosis deformans has permitted use of a varied nomenclature. The individual bony growth has been referred to as a bony spur, spondyle, and a vertebral osteophyte. The condition has been known as spondylitis deformans traumatica, spondylitis ossificans deformans,(23) spondylarthritis,(28) ankylosing spondylitis,(3) spondylosis deformans,(10) spondylitis deformans, spinal osteoarthritis,(9) morbus Bechterew,(30) ankylosing spondylitis,(1) syndesmitis ossificans,(18) and spondylitis.(14) Many of these terms are incorrect, since they imply an inflammatory etiology, while others are autoimmune in nature.

Attempts have been made to relate the incidence of spondylosis deformans in the dog to sex, age, and breed. The condition has been described in all ages with a higher incidence noted in older animals.(6,8-10,14,15,17,19,20,23,25,27, 28) The number of affected dogs has been shown to increase with age, as has the degree of involvement in tends of both the magnitude of the bony response as well as the number of disks involved. (19,20,28) Most reports have indicated equal occurrence of spondylosis deformans in both sexes,(10,15,18-20,23) although a higher incidence has been reported in both males,(5,8) and

females.(21) It has been proposed that there is a predisposition in the boxer(9,10,17,19,28,30) and in the larger breeds; (3,6,8,15,16,18,21,23) however, it must be appreciated that the osteophytes will be larger in larger breeds and thus will be more easily identified. The dachshund has been reported to have a lower incidence.(8,14,23,24) This finding is probably influenced by the small size of the reactive spur, although contradicting studies are available.(1) The reported incidence of involvement of at least one disk in the vertebral column has ranged from 9% to 75% in dogs (7,9,10,15,16,21,23,27) One report cites the incidence of involvement of at least one disk as 67.6% in English cats and 34% in Swedish cats,(27) but this is probably age dependent, since the English cats were much older.

Most reports of incidence have been based on radiographic studies that are now known to be an inadequate technique for evaluation.(20) it appears that all dogs and cats, regardless of breed or sex, will develop a degree of spondylosis deformans if they live long enough.

Various etiologies and pathogenesis of spondylosis have been suggested. Pommer(23) reported that stresses on the ligaments and periosteum were significant and that the stresses could be normally present in active dogs or could be due to degenerative disks, trauma, or repeated pregnancies. Ipolyi (15,16) thought that intervertebral disk softening due to trauma or muscle or ligament weakening must always precede vertebral changes. Resulting motion and disk herniation exerting pressure on the periosteum were important in his hypothesis. However, he discounted the importance of the longitudinal ligament in pathogenesis.

Schick,(29) Fankhauser,(7) and Hansen(10) correctly associated disk changes with the presence of spondylosis deformans. Schick noted in a radiographic study that affected vertebral bodies were sclerotic and disk spaces narrowed. Hansen had earlier described two types of disk protrusion in the dog. Type I protrusions were caused by a total rupture of the anulus fibrosus and were massive in scope, whereas type II protrusions were more limited and were caused by only a partial rupture of the anulus fibrosus with a resulting bulging of the dorsal surface of the disk. He concluded that spondylosis deformans and the two types of disk protrusion were separate morphologic expressions of disk degeneration. Disk degeneration resulting in type II protrusions was similar to that associated with spondylosis deformans. The simultaneous presence of spondylosis deformans in type II protrusions in the cervical region of the dog, however, was rarely found. Hansen also found that the attachments of the ventral longitudinal ligament might have played a role in the development of osteophytes if changes in the disk were minimal.

Martin,(17,18) however, observed that intervertebral disks associated with vertebral osteophytes were normal in appearance. If there were changes in the disks, he interpreted them as appearing later than the osteophytes. He seemed to have been influenced by descriptions of ankylosing spondylitis in humans and thus suggested an association of changes in the dog with prostatic disease and sacroiliac change. He introduced the name syndesmitis ossificans into veterinary literature, incorrectly recognizing a primary ossification of vertebral ligaments.

Schnitzlein (29,30) and Martin (30) also stressed the role of the ligaments and incorrectly described the vertebral changes as morbus Bechterew, a condition synonymous with spondyloarthritis ankylopoietica or ankylosing spondylitis in humans. Archibald and Cawley (1) also considered that the condition satisfied the definition of a rheumatoid spondylitis as it occurs in humans and referred to it incorrectly as ankylosing spondylitis.

Throughout the literature, the alternate use of the terms spondylitis and spondylosis was obvious. Hansen (10) appeared to be the first in the veterinary literature to correctly use the term spondylosis, implying a non-inflammatory disease. Hoerlein (12,13) returned to the use of spondylitis to support his description of the condition as inflammatory with complete ossification of vertebral ligaments. In 1965 (14) he considered spondylosis deformans to be similar to the bony response associated with specific osteomyelitis of vertebrae such as those due to mycotic infection and *Spirocerca lupi*. Glenney(9) also defined the condition as an inflammation of the vertebra.

Morgan(19) has offered the most in-depth study to date as to pathogenesis of spondylosis deformans. He found that the lesions were non-inflammatory and were initiated following degeneration of the anulus fibrosus. He described the nucleus pulposus as playing a secondary role in the disease process. Formation of the vertebral osteophytes took place in an effort to stabilize the resulting disk instability. The pattern of change was observed in patients examined clinically and was confirmed experimentally. Surgical procedures that created minimal damage to the anulus fibrosus only and to the anulus fibrosus and ventral longitudinal ligaments were performed in such a way that the nucleus pulposus was not affected. Another experimental technique created a puncture wound through the anulus fibrosus so that mucoid nuclear material exuded. All of the young dogs with these types of surgically created disk injury developed classic vertebral osteophytes within 10 months or less. Additional insight into the pathophysiology of spondylosis deformans is gained by observing vertebral osteophyte formation in patients with disk fenestration, fracture/luxation injuries with disk involvement, diskospondylitis, and congenital vertebrae in which instability of the intervertebral disks was evident. In these cases, vertebral osteophytes form around the injured, unstable disk space.

Clinical significance has been attributed to spondylosis deformans from the time of the early research and has been encouraged by the high incidence of easily detected bony osteophytes that have been noted radiographically in patients who have shown clinical signs of musculoskeletal or neurologic disease. In the absence of more sophisticated diagnostic techniques, these clinical signs have frequently been attributed to the very obvious bony spurs.

Pommer (23,24) described patients both with and without clinical signs. However, it would be difficult to determine the exact role disk herniation played in his research. In his study of calcinosis and enchondrosis intervertebralis,(25) he stated that the clinical appearance resulting from disk herniation was very similar to that caused by spondylitis ossificans. Ipolyils (15,16) described clinical signs in dogs progressing to paralysis but noted that 50% of his patients with spondylosis deformans were asymptomatic. He found no relationship between degree of spur formation and symptoms. Frequently, the differential diagnosis for paraplegia or paresis in the dog has included spondylosis deformans. (3,6,7-9,11,12,22)

Debard (6) described pain as a result of vertebral spurs putting tension on vertebral ligaments. He also reported pain due to pressure on spinal nerve roots. Hoerlein (12) briefly described vertebral osteophytes in a discussion of clinical conditions affecting the vertebral column of the dog. Later, he attributed the pain to resulting pressure on spinal nerves or the spinal cord. Belkin (2) also described involvement of the spinal cord or nerves as a cause of clinical symptoms. Morgan,(19) however, was unable to establish a relationship between spondylosis deformans and associated clinical signs. Vertebral osteophytes are often seen in dogs in whom there are no clinical signs pointing to the vertebral column.

Many of the causes of neurologic deficit in the hind legs are not clearly evident on survey radiographs. These include noncalcified disk herniation, osteoarthritis of the true vertebral joints, primary or secondary tumors of the cord and spinal nerve roots, occlusive vascular disease affecting the spinal cord, degenerative myelopathy, inflammatory myelitis or meningitis, and peripheral nerve disease. None of the muscular diseases that might cause hindquarter pain or instability are evident on survey radiographs. The use of myelography assists in identifying only those diseases that create a mass lesion within the cord, involving the meninges, or are located in an extradural space.

The following problem thus exists: All dogs and cats will develop spondylosis deformans if they live long enough, since they all will develop degenerative disk disease. In most dogs, this degeneration will be evident by 10 years of age; the disease becomes evident somewhat later in cats. In the aging dog and cat, clinical signs of paresis or paralysis that may be spinal cord or spinal nerve related are frequently seen. Thus, every one of these patients who is radiographed will offer the clinician the opportunity to assume that the cause of the clinical signs may be the very prominent bony spurs of spondylosis deformans. Unfortunately, this assumption is often made rather than pursuing the true cause of the pain, weakness, or paresis.

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## Pathogenesis

### ANULUS FIBROSUS

Changes within the anulus fibrosus play a major role in the development of vertebral osteophytes. In many affected disks, the earliest changes are focal lesions in the anulus that progress to major intradisk fissures. These changes are noted more commonly in the ventral aspect of the disk and are a frequent finding in disks with associated osteophytes. However, the same annular changes are also found in disks without osteophytes and dorsally in disks with osteophytes. The size and shape of the osteophyte do not always correspond to the extent and degree of degenerative change within the anulus.(19)

The more severe disk changes consist of further damage to already ruptured anular lamellae and a noticeable increase in the amount of debris within the disk. In advanced cases, the disk tissue almost disappears, and bone of adjacent vertebrae is ground and appears almost polished. The narrowing of the disk space appears to have followed rather than preceded the early formation of the osteophytes. (19)

Hansen (10) had earlier described changes in the anulus fibrosus that were related to dorsal disk protrusions. These changes were similar to the ones currently described. However, intradisk fissures within the ventral lamella were not described in relation to spondylosis deformans in the dog.

The outer annular fibers consistently appear to have been affected to a lesser degree than the remaining portions of the anulus. The reason for this is unknown. (19)

### NUCLEUS PULPOSUS

The nucleus pulposus does not seem to play as important a role in the pathogenesis of spondylosis deformans as the anulus

fibrosus. Still, changes in the nucleus similar to those described by Hansen (10) were commonly noted in the disks with concurrent spondylosis deformans. The nuclear changes consisted of chondroid and fibroid metaplasia within the chondrodystrophoid and nonchondrodystrophoid breeds, respectively.(19)

Contrary to the findings of Hansen, a slight positive correlation was observed by Morgan (19) in the intrasegmental relationships between type I protrusions and the presence of vertebral osteophytes in chondrodystrophoid breeds. Again, this may have reflected the effect of age on the material. Further examination of intrasegmental pairs within the chondrodystrophoid breed group indicated almost no correlation between degeneration and calcification of the nucleus and vertebral osteophytes. The degeneration of the nucleus pulposus in the chondrodystrophoid breeds occurred at a younger age and preceded the damage to the anulus. This lack of correlation suggested that degeneration of the nucleus in the form of calcification did not appear to stimulate the formation of vertebral osteophytes.

Hansen (10) thought correctly that spondylosis deformans was a morphologic expression of more advanced disk degeneration. There have been others who have also considered the pathogenesis of spondylosis deformans to be directly related to disk degeneration. However, Martin (18) described early spondylosis in the dog associated with disks that were normal in gross and microscopic appearance.

The findings of Morgan (19) strongly suggested that changes in the anulus fibrosus were far more important in the pathogenesis of spondylosis deformans than changes in the nucleus pulposus. Hansen(10) had suggested that changes in the nucleus pulposus were primary causes of all pathologic changes in the intervertebral space. Whether the anular changes described were preceded by nuclear changes or not was difficult to determine. Experimental work by Morgan (19) certainly showed that osteophytes would form with injury only to the anulus.

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## Origin and Growth of Osteophytes

The exact location of the origin of the osteophyte varies slightly but is usually adjacent to the intersection of the cortex of the vertebral body with the bony end-plate. Multiple foci of fibrocartilage that subsequently undergo dystrophy calcification form and unite with the vertebra at an early age. Ingrowth of blood vessels is followed by destruction of the calcified cartilage and formation of mature-appearing trabecular bone. This slow, orderly growth is similar to that noted in endochondral ossification and leads to a final stage in which there is removal of the preexisting vertebral cortex and a continuous blending of the trabecular pattern and marrow spaces between the vertebra and the osteophyte, with a shifting of the vertebral cortex to include the new bone growth. (19)

The mass of connective tissue formed between developing osteophytes continually provides the network on which the new bony tissue is formed. This collagenous and fibrotic tissue appears as an extension of the outer anular fibers. In some cases it may include nuclear material. The multiple foci of fibrocartilage and calcified cartilage often remain unattached within the collagenous tissue that forms outside the anulus fibrosus. Bone may form within these foci. Many of these bony segments reach considerable size and attain a mature appearance but still remain unattached to the vertebrae. Other free bony fragments join advancing osteophytes. Gross radiographic studies suggest the possibility that these bony segments are fracture fragments. However, histologic study makes it possible to show the manner in which these free bony fragments are formed and excludes the possibility that they are a result of trauma. In fact, histologic examination shows the bony tissue in the vertebral osteophytes to be stronger than that present in the original vertebrae.(19)

Formation of the osteophytes is similar in both chondrodystrophoid and nonchondrodystrophoid breeds. Microscopic studies have shown which osteophytes and specifically which part of the osteophytes have a growth potential. This is done through tetracycline labeling of the bone and shows growth occurring on the leading edge of the osteophyte. The calcified cartilage as well as bone on the edge of the osteophyte takes up high levels of the tetracycline label. Not all osteophytes show tetracycline labeling, indicating that the osteophyte is not always in the stage of growth but can become quiescent at any time during development. The resulting signs of maturity include a blending of trabecular pattern with that of the vertebral body and disappearance of calcified cartilage on the surface of the osteophytes with replacement by an intact cortex. Since maturity can occur at any stage, this suggests that the formation and growth of the osteophyte is governed by some functional requirement. When sufficient size is reached to provide the reinforcement required to the diseased disk, growth of the osteophyte apparently ceases. Whether or not new stimuli can initiate renewal of growth is unknown, but this seems probable.(19)

Contrary to earlier suggestions, the development of the vertebral osteophytes has been shown to have no association with possible remnants of the physal growth plate of the vertebral body.

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## Clinical Significance

Spondylosis deformans is the non-inflammatory bony response to disk degeneration as it occurs in dogs and cats. The fact that the growing osteophyte may cause no clinical signs was demonstrated by Morganl (9) when he reradiographed 22 dogs with spondylosis deformans and showed growth of osteophytes that were present on the first examination and/or formation of new osteophytes in 15 of the 22 dogs. None of these dogs had clinical signs during the intervals between the two examinations, which ranged from 11 to 27 months.

If clinical signs are present they must be related to encroachment of the osteophyte on an emerging spinal nerve root, they must be causing pressure on the spinal cord, or they must be causing a meningitis/myelitis.

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## Radiography

Radiography can be used to illustrate the distribution of lesions along the vertebral column. Cats demonstrate lesions at any level of the spine, although the thoracic spine seems more likely to be affected. The highest incidence in English and Swedish dogs is at the T9-T10, L2-L4, and the lumbosacral interspace.(19,21) It must be appreciated that osteophytes may occur at any location along the circumference of the end-plate of the vertebral body except for that area that creates the floor of the spinal canal. Thus, lateral radiographs will clearly demonstrate only bony osteophytes that project ventrally (Fig. 61-1). The dorsoventral or ventrodorsal radiograph will show those osteophytes that project laterally; however, the problem of superimposed shadows from the abdominal contents often influences this evaluation (Fig 61-2). Therefore the majority of spondylosis deformans that occurs other than ventrally will not be identified on routine radiographic studies.(20)



FIG. 61-1 Spondylosis deformans is present in a dog having no clinical signs. Note the variable pattern of osteophytes ranging from small and interlocking fingerlike projections to massive ankylosing bridges.



FIG. 61-2 Lateral osteophytes (arrow) of spondylosis deformans are visible on a ventrodorsal view of a dog's lumbar spine. Lateral osteophytes can be mistaken for a calcified or herniated disk on a lateral radiographic view of the spine.



FIG. 61-3 A sagittal section of T12-L2 from a dog demonstrates the variety of appearances that osteophytes have in spondylosis deformans. (MorganJP: spondylosis deformans in the dog. Acta Orthop Scand, [Suppl 96] p 23, 1967)

The patterns of osteophyte formation vary. Osteophytes are found at numerous locations about the junction of the vertebral end-plate and vertebral body cortex. They may be single or multiple and may be located in an interrupted or non-interrupted pattern around the circumference of the end-plate. The opposing vertebral segment may have no bony growth, equal growth, or larger osteophyte formation. The spurs range in size from small, barely discernible nodules to massive ankylosing bridges (see Fig 61-1). Generally, the osteophyte appears as a "scoop" with the tip extending toward the disk. It usually presents a sloping and smooth ventral or lateral surface that blends gently with the cortex of the vertebral body. Bony ankylosis between opposing osteophytes is rare; and interlocking of fingerlike projections is more common. The width of the osteophyte is not related to its longitudinal development. Dorsolateral spurs were found frequently near the intervertebral foramina. These have the radiographic appearance of projecting into the spinal canal.(21)

Separate centers of ossification are found frequently in the ventral portion of the anulus and often appear as "fracture

fragments." It is possible for osteophytes forming on the cranial and caudal ends of a given vertebral body to become so large that they meet at the ventral midpoint of the vertebral body, causing an increase in dorsoventral diameter of the vertebral body (Fig. 61-3).

End-plates appear radiographically smooth and often have a sclerotic appearance. The associated intervertebral disk space may be of normal width but more commonly becomes narrow.

Osteophytes may involve one or more joining vertebrae or many separate areas within the vertebral column.

Vertebral osteophytes also present secondarily to instability of the intervertebral disk space due to diseases other than degenerative disk disease, including disk fenestration, previous vertebral fracture or dislocation, congenital anomaly of the vertebrae, spondylitis, and spondylolisthesis. While having the same radiographic appearance, the entire vertebral column can be examined radiographically in an effort to determine other radiographic patterns that assist in identifying the primary cause for the disk instability.

Vertebral osteophytes forming around a degenerating disk have the same radiographic appearance as those forming around a disk with instability due to another cause. Thus, the differential diagnosis is in the determination of the cause of the disk disease. Spondylosis deformans commonly is a generalized pattern and involves numerous disk spaces. The vertebral osteophytes that form due to another cause usually are limited in location and dictated by the particular cause of disk instability. It is obviously possible in the older animal to have diskospondylitis or fracture, dislocation that could be superimposed upon an existing spondylosis deformans. This would reestablish an instability and initiate further osteophyte formation.

Vertebral osteophytes of the bridging nature form in association with hypervitaminosis A in the cat. A more solitary form of vertebral osteophyte is noted in multiple osteochondromatosis in the dog, a condition in which the osteophyte may be present within the spinal canal. These lesions are noted at an early age and stop growing at the time of skeletal maturation. Multiple osteochondromatosis in the cat is a virus-induced disease in which the bony masses occur later in life and the bony growth assumes massive size with the possibility of spinal cord or nerve compression.

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