

## TVS, CAUDA EQUINA SYNDROME, AND SPONDYLOSIS

by Fred Lanting

Some website and magazine editors have asked for a treatment of orthopedic and non-orthopedic spinal disorders, specifically the syndrome (meaning a collection of symptoms) called cauda equina or CES. While not all involve bones and articulating joints directly, there are several problems that yield symptoms that could be mistaken for those of HD (or other disorders that produce lameness) and the miscellaneous vertebral malformations that compress the spinal cord. Several, including dural ossification, are mentioned in the chapter on spinal disorders in my up-coming book on canine orthopedic disorders. Three are discussed in this article, although CES might actually comprise several disorders instead of just one.

### TRANSITIONAL VERTEBRAE, TVS

There has been a flurry of questions in recent years about conditions known as TVS and APA (asymmetrical pelvic attachment), which makes me wonder if it is the incidence or the diagnosis that is on the rise.

A malformation of vertebrae, usually the last lumbar or first sacral vertebra (sometimes more than one per case) is simply and aptly called “Transitional Vertebrae”. This congenital and inherited defect begins in that stage of embryo development when

differentiation is nearly complete, but a vertebral body “can not make up its mind whether it wants to be part of the lower lumbar vertebral column or part of the sacrum”, as colorfully described by OFA’s Dr. Greg Keller, and it winds up taking on bony characteristics of *both* lumbar vertebrae and sacrum. Less common are the transitional vertebrae seen at the thoraco-lumbar junctions. Wherever it is found, the affected part is called TVS, Transitional Vertebral Segment.

Veterinary-science observer Barbara Nibling describes mild cases this way: “The processes are just a bit odd, one set of processes looking as if the T13 (the 13<sup>th</sup> thoracic vertebral segment) belongs to the lumbar group, while the other side looks like a normal T13.” Many go unnoticed unless there is a rib on one side but not on the other, in which case the difference leaps out at you. The affected part is called TVS, Transitional Vertebral Segment. It apparently can happen anywhere along the spinal column, but in the lumbo-sacral area most often. If it results in less symmetry there, the condition is usually referred to as APA.

The transitional vertebral body takes on some of the characteristics of both, and is an abnormality that may have a causative role in the severity or nature of HD although, by itself, it seems not to have any bad effects. Its developmental and inherited nature is extremely probable, having been identified as familial and being similar in many ways to other known genetic defects. Frequency of transitional vertebrae runs from quite low (zero in Saints, 3% in Labs) to somewhat higher (8-11% in German Shepherd Dogs), and most cases (69%) are associated with HD. That is, most but not all cases are found in dysplastic dogs. This is one reason why the frequency of asymmetrical HD is as high as it is (30% in the GSD, for example — more than in other breeds).

The trait is probably polygenic; at least it cannot be said to be simple. It is definitely familial. For example, Borzoi breeder Bonnie Dalzell reports around 40% of the dogs that were radiographed in a program she participated in had lumbo-sacral transitional vertebrae! She described them as “much slower gallopers at field trials than the dogs with normal anatomy” or having reduced interest in running at a fast gallop. Top racers, she said, can go 33 mph, while dogs with “weird backs” generally top out at 23 to 24 mph. She also surmised that many cases might be missed, if the transitional vertebrae were further toward the front of the dog, because of the great size of her breed — the crest of the ilium of the pelvis and the last lumbar vertebrae might not be included in the standard 17-inch film, and thus any TVS not observed. Field trial performance, for which she is well known and highly respected, is affected, so she naturally pays more attention to searching for the anomaly than most people might. She has had eager coursing dogs with TVS, and found them not as fast as the ones without it. Her meticulous observation has uncovered other little-known “symmetry anomalies” such as one in which there is an extra lumbar vertebra. This would be highly unusual in dogs, although there is a breed of pigs with an extra pair of ribs, and differences in number of cervical vertebrae between animals such as giraffes and squirrels (the numbers may surprise you!). Dogs with extra vertebrae in the loin and other lumbo-sacral anomalies seem to be prone to having symptoms of something that vaguely resembles “Wobblers”, also displayed neurological deficiencies in the hind legs, and were relatively poor gallopers, according to Dalzell. Another fairly location of transitional vertebrae is in the thoraco-lumbar region

, in which, she says, “the dog may have one less rib on one side than the other. This does not seem to affect performance at a gallop.”

The abnormal attachment of these segments causes the pelvis to be rotated a little in a longitudinal fashion, which means the pelvis is tilted so one femoral head gets more coverage by the acetabulum than the other side does, and this tends to draw laxity or joint ligament weakness into subluxation. Yet it is not so much the fact that transitional vertebrae are there, as what effect they have on pelvic attachment that is important. TVS does not participate in *causing* HD unless it does so through intermediately causing APA. Asymmetry in pelvic attachment is highest in Labs and GSDs (around 7-8%) and 79% of these cases are associated with HD. While transitional vertebrae and asymmetrical pelvic attachment may not cause all cases of unilateral HD, they do appear to uncover it or make bilateral laxity look like unilateral HD, at least on the hip-extended view. An important conclusion of this ten-year study is that the quality of hip joints is not independent of the nature of pelvic vertebral attachment.

The L7 to S1 region of the spinal column is where some see what they call “block vertebra”, referring to a somewhat unusual shape. Not only are affected coursing sighthounds slower, according to some in the sport, but most dogs upon very careful motion analysis will evidence an asymmetric gait. The last lumbar vertebra can be fused on one side to either the first sacral vertebra, or its process (“wing”) can bridge over to near the top of the ilium. In any case, the dog has more articulation, and range of movement and flexion/extension on one side than the other. Many have noticeably shorter stride, but this can also be the result of other problems, such as arthroses in any of the four limbs’ joints.

More acronyms are not what I enjoy, but these abbreviations are helpful. In orthopedic lingo, the condition of asymmetrical (without symmetry) pelvic attachment of the last vertebrae before the sacrum is called APA. The condition of transitional vertebral segments is referred to as TVS. The fact that TVS and APA are associated with unilateral HD or differences in the severity of one side vs. the other, is why the OFA looks for these signs and requires a full picture of the pelvis be included on each hip radiograph submitted for evaluation/certification. The incidences of both APA and TVS in “normal” dogs are quite low, but these conditions do exist independently of HD in a small number of dogs with otherwise normal hips. For example, APA was 2.8% and TVS 1.4% in Labrador Retrievers with normal hip joints, but 13.8% and 5.2%, respectively, in dysplastic Labs. There are similar or perhaps identical conditions in humans. In people, TVS is an indicator of probable future problems in the lower back.

Is it a serious problem? Many think not, but I believe their lack of concern is based on old information that indicated TVS was not a significant problem. In a conversation between the OFA and GSD fancier Rita Ledda (via e-mail chat list) about transitional vertebrae, she was told that it is not a big problem; yet OFA recommends an affected dog not be bred to a mate that also has transitional vertebrae. They say that normally, TVS or APA by itself (without concurrent HD or another disorder) does not affect the dog. There might be some concern about dogs that engage in some type of pulling competition, a popular sport in Malamutes, American Bulldogs, and American Pit Bull Terriers. The stress may cause irritation at the sacrum joint. It must be kept in mind that there could be a relationship between TVS and other disorders, too. Morgan *et al* found that in German Shepherd Dogs, at least, the presence of lumbosacral transitional vertebrae is a predisposing cause of cauda equina syndrome; this disorder will be treated below.

Another GSD fancier, trainer, and writer named Ricardo Carbajal reported (on an e-mail list) a December 1996 conversation with OFA in which Dr. Keller said that TVS is a developmental problem. When the puppy is developing *in utero*, some of the lumbar vertebrae attach firmly or tenuously to the sacrum. You usually see bony changes that are more similar to the sacral area; one of those changes is that the transverse process (the side wing) deforms or “bends” and fully or partially fuses to the ilium. Some times you also see that the inter-vertebral space is diminished or absent. Usually this problem does not affect the health of the individual. But it does tend to run in families, so it should be considered a genetic defect and given whatever amount of serious attention you decide it deserves.

Morgan has done much work in this area, and says that transitional vertebrae “can occur at the site of junction of the major portions of the vertebral column, i.e., occipito-atlanto-axial, cervico-thoracic, thoraco-lumbar, and lumbosacral. The spinal segments at these sites are altered so they have features in common with those either cranial or caudal” [to them] When we speak of transitional vertebrae, we should also describe the specific nature of the change, such as where the attachment is directed, and whether it is unilateral or bilateral. Some TV are symmetrical, equal on both sides. Others are asymmetrical (APA); “for example, a lumbar transverse process on one side and a heavy wing on the other that attaches to the sacrum.” Transitional lumbo-sacral vertebra (TLV) was found in 38% of German Shepherd Dogs diagnosed with cauda equina syndrome. Is there a biochemical or genetic connection, or are such dogs selected for by sloppy breeding practices in general?

For a long time, it was generally thought that these abnormal vertebral segments and attachments did not cause pain and therefore had little clinical or breeding significance throughout a dog’s lifetime. Currently, the feeling is that such lesions at the

lumbosacral junction predispose the dog to further weakness, instability, excessive motion around that disc, creation of cauda equina syndrome, and possibly a worsening of (already-present, even if covert) hip joint laxity or instability. TVS can weaken an already unstable sacro-iliac joint and put additional stress on the joint between the sacrum and the last lumbar vertebra.

## **CES: CAUDAL EQUINA SYNDROME**

### **Description, Causes, and Symptoms**

At the caudal (rearmost) end of the spinal cord in the sacral and coccygeal (tail) area, there are roots of several nerves named for their resemblance to a horse's tail. They emanate from the vertebral column and branch out to various parts of the hindquarters. These include the seventh lumbar nerve, the first three sacral nerves, and the first five caudal nerves. The cord itself terminates around the sixth lumbar vertebra in most dogs (Dachshunds are a bit different, with the termination being one or two vertebrae distant). Some CES is actually secondary and due to environment such as lumbosacral disc degeneration. Sometimes people refer to the major manifestation of the syndrome as Degenerative Lumbosacral Stenosis, as it generally involves a compression of the nerve roots in the lower back, at or near the junction of the sacrum with the last lumbar vertebra.

Beyond the sacrum, the vertebral bodies of the tail extend further than the main cord does. If any of the vertebrae from L5 back toward the tail are diseased, it can result in canine CES, which typically involves some compression, destruction, or displacement of the nerve roots which form the cauda equina. CES is called a *syndrome* instead of a single traditional disease because there is no sole cause or malady. When you have a number of disorders that have similar signs and location they are lumped together (in both common and medical parlance) as a syndrome. And of course, that means there can be a number of different causes for this set of disorders. Thus, while there may be various causes for the collection of symptoms, we see similar reactions — namely, the filling in or stenosis (narrowing) of either the cord canal or other “holes” through which pass veins, arteries, and nerves. Just as a stenosis of the vertebral canal further toward the head can produce Wobbler syndrome and other disorders described in my book, compression of the cord in the lumbosacral region or of these post-cord nerves as they exit from between the last vertebrae of the spinal column before the last three quarters of the tail can give both sensory and motor (feeling and moving) nerve dysfunction. Even the blood vessels can be compressed and fail to deliver nutrients or remove waste efficiently from the rear limbs below. The impingement can take place in the vertebral canal, or where the nerves exit the cord through the *dura mater* (the fibrous covering of the cord), or between there and where a nerve exits the canal between two adjacent vertebrae. If so, it is probably caused by bone and cartilage changes in vertebral end plates where the discs are attached. Wherever they are pinched, the nerves, arteries, and veins lose part of their functions and produce clinical signs. Most specialists restrict the term *cauda equina* to symptoms arising from lumbo-sacral area defects.

Congenital causes include spondylolisthesis (slipping of one vertebra over an adjacent one somewhat similar to Wobbler syndrome), malformations of the vertebrae, spina bifida, perhaps spondylosis deformans, and stenosis of unknown origin. Cauda equina syndrome can also be the result of acquired conditions such as cancers, infections, disc disease or prolapse/protrusion, and others. Cauda equina syndrome (entrapment and pressure on the nerve root) can also be the result of arthritis or acquired conditions such as cancers, infections, disc disease or prolapse, and others. Some dogs will have more than one of these conditions at once. Osteoarthritis can make CES worse or it can produce signs similar to the syndrome by itself. Exercise can bring on the signs because blood vessels dilate when one exercises, and dilated vessels can press on the nerve roots; conversely, rest or tranquilizers may help calm the dog and avoid pain until the condition improves, if it does at all. Some CES cases involve a bone fragment from the sacrum that detaches and moves into the spinal canal to press against the cord; some call this “osteochondrosis of the sacrum”. In many of these, there is an appearance of misalignment similar to what we see in “Wobblers”. Many of these dogs have multiple spinal abnormalities, which may be related to CES symptoms. Some have a persistent disk in the sacrum, which means that the sacral segments did not fuse, as they normally would have.

Many also have transitional vertebral segment (TVS), but given that the GSD breed is represented by so many of these cases, this may possibly be somewhat coincidental. It also may be that research facilities just see more GSDs than other breeds. Breed incidence varies, but the GSD, at least in America, apparently does have a higher incidence of spinal column disorders in the lumbo-sacral area than do other breeds. Dr. Joe Morgan at Davis told me that his study showed that dogs with cauda equina syndrome more often than expected also have lumbo-sacral transitional vertebral segments (TVS or APA), and that this combination was seen in the GSD more than in dogs in general. Most affected dogs tend to be members of large, “athletic” breeds.

As you may have suspected, age is a factor in CES, with middle aged or older dogs being more often afflicted. This causes a dilemma regarding decisions to operate or euthanize or just wait to see how long the dog can put up with the disease. Surgery seems to give benefit for no more than two years in most cases. One of my correspondents had a 14½-year Boxer that was “going downhill” rapidly, was on Rimadyl™ for a couple of months, and enthusiastically dragged herself to the cookie jar for a treat. With a bright mind, such a dog makes it very difficult for the owner to decide. This Boxer was not “showing” any pain, according to the owner. It also had a concurrent case of severe spondylosis, and was certainly not the only such case of Boxers with both ventral spondylosis and dorsal bony overgrowth/stenosis. Although most CES cases

are in older dogs, I have a friend in New Mexico who lost a 14-month old German Shepherd Dog puppy to the disorder.

## Diagnosis

To accurately diagnose, expert radiography is highly recommended, almost essential, but is only part of the whole work-up. Diagnosis can be difficult, and may involve many ways of looking at the problem: clinical signs (symptoms), neurological signs, radiography, surgery, and deductive reasoning. In an early stage or in a more sensitive dog, the only symptom might be progressive, sharp pain. However, this pain or discomfort can show in a number of ways. Intermittent lameness in one or both hind limbs or a stilted gait like that of a Chow-Chow, more and more difficulty in rising to a standing position, reluctance to jump, and perhaps sudden but temporary lameness immediately after doing either of those things. Strenuous activity may exacerbate these signs, just as it does in hip-dysplastic dogs. The dog may express pain by as little as a moan or whimper, or as much as a yelp or howl when moving or being touched on the croup. As the Southern California Veterinary Referral Group has stated, many dogs “are very tolerant of discomfort until it eventually overrides their character and results in clinical manifestations”. Sooner or later, even a Pit Bull Terrier or Rottweiler (noted for being stoic) will indicate the burning sciatic pain of the syndrome’s nerve root entrapment. Bowel and bladder incontinence is common in advanced cases. Paresis or full paralysis may occur.

As you see, symptoms are many, with some resembling those of “Wobbler syndrome”, some being mistaken for signs of HD. Its clinical signs may also resemble degenerative myelopathy, axonal neuropathy, disc protrusion, and tumors of the spine. It reportedly has its highest incidence in GSDs and those breeds with high incidences of hip dysplasia — which might not have any inherent connection. Not all dogs with cauda equina syndrome have all the same symptoms, but these may include straining with defecation, partial paralysis, fecal or urinary incontinence, lameness, bunny hopping, and standing with a hind leg drawn up. Leg pain is probably very similar to what we humans call sciatica, judging from the identical locations of the pinched nerves. Some probably have what humans refer to as a “falling asleep” of the legs. This tingling may be a reason why some dogs inflict damage to their tails and rear paws, in the form of chewing, lick granulomas and other dermatoses. A similar thing happens to many dogs with DM, but without pain.

Some may show relatively early signs with a “dead” tail carriage. From SV judge and then-chief-Körmeister Leonhard Schweikert regarding the bitches at the huge GSD Sieger Show in Bremen, 2000 we have this comment: “With respect to the evaluation of the rear, I noticed in more than one case a lifeless or nearly lifeless tail. This is a very worrisome fact, as this is one of the first indications of a cauda equina syndrome (CES). We must look into this topic in the near future in more depth.” (Translation by Susanne Stramm.)

Neurologic examination usually begins with gait analysis. Then the dog is tested for pain and neurologic dysfunction to confirm the suspected site of the lesion. Physical exam of the standing dog reveals an exaggerated response to the pain of extending the hip joint, which in turn extends the lumbosacral joint. This is very subjective; dogs with moderate to severe HD will often show a mild response to hip extension, but dogs with lumbosacral disease such as CES will often complain more when the vet extends the hip and presses on the croup (lumbosacral junction). This can also be seen if the dog attempts to “stretch” or straighten (extend) its back, because part of the disc and a ligament are pushed into the spinal canal space occupied by the nerves. Manipulating and lifting the tail typically elicits an exquisite pain response. Pain is also caused by deeply palpating the muscles over the croup and loin. Spinal reflexes such as the perineal reflex (beneath anus and vulva or scrotum) and anal tone are tested. Diagnosis is sometimes much a matter of differentially distinguishing this syndrome, i.e., ruling out other disorders by a process of elimination. Since cauda equina syndrome affects mostly German Shepherds, the vet is challenged to decide whether the symptoms are those of GSD myelopathy instead, which he can do by determining whether there is pain and where the motor neurons are affected, high or low.

Ordinary radiographs are not very useful in diagnosing syndromes such as CES and distinguishing it from (or ruling out association with) infection, trauma, severe arthritis or bone cancer. Special x-ray procedures may be required for a definitive diagnosis. Myelograms and/or epidurograms (contrast dye studies of the spinal canal) can help to confirm the location of the lesion and the position of any ruptured disc in relation to entrapped nerve roots as the spine is flexed and extended. The Southern California Veterinary Referral Group that I mentioned earlier says, “With new gas anesthetics, advanced monitoring equipment, and modern contrast agents for the dye study, the myelogram and epidurogram are now common and safe diagnostic procedures when performed under the proper conditions. In difficult cases, MRI or CT scans are available and are of exceptional diagnostic value. Electromyography (EMG) may be of value in substantiating the diagnosis and the severity and symmetry of nerve root entrapment.” I know that in the many cases of human disk disease that I have had opportunity to ask about because of a possibility of back surgery for myself, the epidural is extremely uncomfortable (people are generally not knocked out for the insertion of the needle into the nerve!) and if used for pain relief, is only temporary; if used to inject dye, it is not only painful for an extended time, but also is reportedly risky. MRI or CT is very expensive, and very few vets have the equipment.

A Morgan, *et al* study divided hundreds of study dogs into 3 groups segregated by whether or not the dogs had diagnosed degenerative disc disease (DDD) involving lumbosacral discs and transitional vertebral segments

(TVS), and whether the dog had CES. Their data suggested an association between TVS and CES and an association between DDD and CES: more CES when DDD and TVS were both evident. Some CES seems to be partially dependent on the presence of TVS. They also say that TVS is probably an inherited condition.

Morgan believes that GSDs have a higher frequency of TVS than other breeds do, and that may partially explain the higher incidence of CES in GSDs. TVS therefore should be part of the criteria in selection of breeding stock. The study size is small, but a warning sign nevertheless.

### **Treatment of Cauda Equina**

Rest and anti-inflammatory/analgesic medications are often prescribed for the first episode, when mild pain only is experienced, and when patient age or client's monetary situation indicates that to be the best course. Treatment with medications such as analgesics, steroids, chloramphenicol, tranquilizers, and sedatives have temporary effect at best; surgery has variable prognosis depending on the cause(s) of the syndrome. Neurologic signs, continued, frequently recurring, or considerable pain that does not respond to conservative treatment, call for surgical treatment. To relieve pressure on the entrapped roots, a dorsal laminectomy is one of the choices. Removal of parts of the vertebrae or fusion of two vertebrae together are some of the procedures chosen. In some cases, the vertebral segment is sliced horizontally, the top lifted, and extra space thereby created. If this is done on only one side, it is called a hemi-laminectomy. Unfortunately, scar tissue can make the situation as bad or worse in a year or more afterwards; thickening of ligaments or cartilage may be part of the body's way of trying to compensate and stabilize. But in general, surgery is far preferable to medication. Exposing the nerve roots in a more-or-less conservative surgical approach allows one to safely retract them and expose the disc space. "The cauda equina is gently retracted to one side with blunt nerve hooks, exposing the herniated discs as a large dome on the floor of the spinal canal. The herniated disc is excised, compressive osteophytes removed, and foramenotomies (opening the nerve root canals) performed to relieve root entrapment. Once the pressure is relieved, the neurologic function gradually returns as the nervous tissue heals in its decompressed environment."

Considering treatment? Shop around. Don't spend too much money. Use common sense in spending your common cents.

### **Postoperative Care**

Rest for at least 6 weeks is recommended post-surgery. That means, cut out the strenuous activity. Short walks on leash at first, then gradually increase the amount of exercise. Keep the dog lean and fit; obesity exacerbates most diseases and prolongs recuperation. Prognosis depends on severity of signs before surgery. Dogs with chronic neurologic dysfunction may never recover to a satisfying lifestyle, but at least they will enjoy a pain-free life.

### **Similar Signs and Conditions**

One consulting client described her dog thus: "Her back is hunched, she wobbles and she acts as if she has HD but her x-ray showed that her hips are normal." I told her that she should ask her vet to investigate the possibilities of spondylosis, TVS, and CES. Spondylosis deformans is a condition in which bridges are formed along the ventral (bottom) parts of the vertebrae, starting with the last lumbar segment and, with time, extending to progressively more forward segments. There is no spinal cord compression in this disorder, but pain may come from encirclement or pressure on nerve roots leading out from the cord to peripheral nerves. Differential diagnosis should rule out discomfort from concurrent arthritis, cauda equina syndrome, or other problems. If one of these bony bridges breaks, there could be considerable pain and inflammation in the surrounding soft tissues.

Transitional Vertebral Segment, TVS, has quite a few of the same symptoms as spondylosis and CES, but they are more subtle; in fact, TVS is highly unlikely to give any signs at all. Only when accompanied by HD or the dog is routinely radiographed for a pelvic picture do people generally know that their dogs have it. However, remember that Morgan found that in German Shepherd Dogs, at least, the presence of lumbosacral transitional vertebrae seems to be a predisposing cause of cauda equina syndrome. He says that transitional vertebrae can occur at the lumbosacral portion of the vertebral column. Some TV are symmetrical, others are asymmetrical pelvic attachments (APA); "for example", he says, "a lumbar transverse process on one side and a heavy wing on the other that attaches to the sacrum." TVS can weaken an already unstable sacro-iliac joint and put additional stress on the joint between the sacrum and the last lumbar vertebra.

Congenital spinal stenosis, according to Morgan, is a primary narrowing of the canal with resultant compression of the cord. It's like it is strangling it and preventing proper transmission of the electrochemical neural impulses. He says such a constriction is a cause of CES when it involves the last lumbar or the first couple of sacral vertebral segments. However, it may be seen without the typical symptoms of CES. The word *primary* in this context means a disorder that has no apparent origin in a different disorder; i.e., it is not secondary to another disease state or syndrome. Primary stenosis is possible anywhere, but is more often seen at the area where cervical and thoracic vertebrae meet, and at the lumbosacral junction. Bone tissue looks normal except the shape of the segment is different, with shortened parts and narrower canal space.

While I have tried to present most of the disorders in bones and joints in my book on orthopedic disorders, it must be

remembered that there are many less known and infrequently encountered. In the case of the spinal column, for example, there are also such aberrations as failure of sacral vertebrae to fuse, sometimes referred to as lumbarization of the first sacral segment. This can be considered as a variant of TVS, though slightly different than what we are used to.

Osteochondrosis of the sacrum is found on the cranial end plate of the sacrum, on the lumbar side, is similar in appearance to osteochondrosis in joints, and superficially resembles avulsion fractures.

## Breeding

Is it wise to use a CES dog in breeding? Unless there is associated HD and osteoarthritis starting to be evident in the hip joint (rim, femoral neck, pectineal eminences, etc.), or there are several in your dog's family that have the syndrome, or the signs are severe enough to cause real discomfort and interfere with breeding, some people tell us not to worry about it. They say that you can look instead to whether the dog has enough other qualities to keep him in the gene pool. I believed that for a while, but then when I saw the increase in number of cases reported, I decided we should not be that complacent. Kitty Porter of Duke University had a CES dog, and *preferred* to think of TVS as not "pathological" (by which she meant serious and inherited). Perhaps we need to reevaluate our preferences from time to time.

CES might not be the worst problem in your breed, but still, it is not wise to perpetuate the sort of weakness that allows defects such as CES to be perpetuated, however minor the ailment may seem at first. There are enough dogs that have had to be euthanized for us to take this disorder seriously and assume genetic components to exist. Transitional lumbo-sacral vertebra (TLSV) was found in 38% of German Shepherd Dogs diagnosed with cauda equina syndrome. Is there a biochemical-genetic connection, or are such dogs selected for by sloppy breeding practices in general? For a long time, it was generally thought that these abnormal vertebral segments and attachments did not cause pain and therefore had little clinical or breeding significance throughout a dog's lifetime. Currently, the feeling is that such lesions at the lumbosacral junction predispose the dog to further weakness, instability, excessive motion around that disc creation of cauda equina syndrome, and possibly a worsening of hip joint laxity or instability.

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## SPONDYLOSIS DEFORMANS

by Fred Lanting

Spondylosis deformans is a condition in which bridges are formed along the ventral (bottom) parts of the vertebrae. It has been diagnosed in man, domestic cats (68%, yet no symptoms!), bulls, and even whales as well as in dogs.

It is suspected that bulls on high-calcium diets may have increased susceptibility. The term "spondylitis" literally means "an inflammation of the spine", especially the bone, and spondylosis is sometimes used as a synonym as well as for describing types of ankylosis. One of these types is a bone proliferation, usually on the ventral surfaces of adjacent vertebrae, producing a bridge from one to the other. This condition is best known as spondylosis deformans.

There is no spinal cord compression, but the spine is immobilized in that location. If the condition continues to spread, there may be several such bridges, "welding" a series of vertebrae into an inflexible backbone. It is seen fairly easily via lateral radiography. Because of different degrees seen in different breeds, I believe there to be more than one genetic determinant for this disorder, though nutrition may play a modifying role. I know full well the familial line in a significant portion of American German Shepherd Dogs with this problem, but there are some "German" lines with it, too. As in Wobbler Syndrome, much growth of osteophytes can occur, and encirclement or pressure on nerve roots leading out from the cord to peripheral nerves may be part of the reason some dogs have been reported to show a little pain, but generally the animal does not appear to be suffering and such discomfort more likely comes from concurrent arthritis, cauda equina syndrome, or other problems. Males seem more at risk than females.

It is not completely clear how this disorder progresses, but it may start with a breakdown of Sharpey's fibers, which are the fibers making up the annulus or outer portion of the intervertebral disks. Subsequently, inner disk material protrudes, stretching the longitudinal ligament, and promoting the appearance of osteophytes which grow out from the vertebral bodies in such a way that one cannot tell where the original bone ends and the osteophytic growth begins. Before that happens, though, separate ossification centers can be seen forming a few millimeters from the vertebral bodies; they later fuse and grow toward the adjacent vertebral segment. Eventually, and depending on breed and family history, the disk spaces between particular segments are bridged. True ankylosis (complete fusion into a continuous bony bridge between vertebrae) is far less common than the near-junction of these osteophytes, and is much more likely on the last lumbar segments. Very seldom do the osteophytes grow upward or in such a way as to pinch the spinal cord or otherwise cause neurological signs, so spondylosis deformans might be considered a relatively benign disorder when compared with HD, elbow dysplasias, wobbler syndrome, etc.

All the dogs I have known with ventral spondylosis have gradually become inflexible in the spinal column, and required ramps instead of stairs, for example. But pain is not a usual part of the spondylosis pathology, unless the bridges

fracture, which is highly uncommon. Many affected dogs live satisfactory lives, though somewhat limited in flexibility and range of motion. Fortunately, by the time spondylosis deformans becomes noticeable in clinical signs, the dog may be considered “retired” from his duties of running around, jumping, and doing the other things expected of a youngster. In some individuals, it will get worse suddenly rather than continue in a gradual worsening. Possibly, trauma may bring fracture of the bridge created in the development of spondylosis, which crack may spread to the arch and body, thus pinching the cord.

Often, spondylosis will be discovered on radiographs incidentally while the vet is looking for something else, such as a cause for lameness. In some of these cases, he may be tempted to make his diagnosis right then, and not to look further for the actual main cause, which may include HD, osteochondrosis in other joints, tumors, and others. Osteoarthritis of the spine (inflammation of the joints between vertebrae) is not the same disorder, nor is true spondylitis (an inflammation of the vertebrae themselves, brought on by either trauma or infection). Remember that “-itis” means inflammation, and spondylosis deformans in itself is a non-inflammatory degenerative disease.

Owners and their veterinarians should distinguish between what I call “classical” spondylosis and a somewhat different form. In a Mastiff breeders’ 2003 Internet chat group, Canadian vet Dr. Claire Duder says, “Not all spondylosis is created equal. Most dogs with spondylosis will have radiographic lesions on one or a few intervertebral joints, and in the majority of cases, the dogs will act perfectly normal. On the other hand, some dogs with spondylosis of the lumbo-sacral joint (where the spine and the pelvis meet) can experience severe pain. This latter subset of dogs may well benefit from surgery. Surgical treatment for L-S spondylosis should only be considered for those dogs who demonstrate narrowing of the spinal canal (actually, the spine ends a bit above this area, but there are still lots of large spinal nerves going through) via myelogram or MRI. Sometimes this narrowing is only present radiographically in certain positions. Surgery is designed to reduce pressure on the spinal nerves, and is usually done in specialty centers only.”

She was talking about surgery for lumbo-sacral disease only. She told me she had never referred a dog for surgical repair of spondylosis anywhere *except* at the L-S joint — and that it does not seem to be common practice even there. If a myelogram or an MRI showed narrowing of the spinal canal or foramina, she supposed it would be an option. As in CES, the surgical approach is dorsal (going in from the back instead of via the abdominal cavity), and the objective is to reduce pressure on the spinal nerves by removing all or some of the “roof” of bone over the affected area. The technique of cutting and removing part of that bony roof, often including the spinous process that juts upward like the mast or chimney of a ship, is called a dorsal laminectomy (hemi-laminectomy, if you only do one side).

Lumbo-sacral spondylosis is much more likely to be clinically significant than spondylosis in other areas of the spine; i.e., more likely to involve pain and influence movement. One deciding issue regarding surgery is whether the nerves are being compressed by structural changes of the L-S joint. As Dr. Duder says, “The bony overgrowths seen on radiographs are usually not the cause of the problem, but are themselves a symptom of, and a response to, the underlying instability of the articulation. It matters not if there is a big hunk of bone on the ventral surface of the joint, as long as the dorso-lateral surfaces, where the nerves exit, and the spinal canal are unaffected.” I agree, and have counseled friends to get a second opinion when surgery has been discussed. There really is no operation that has a high confidence rating or good prognosis for classical ventral spondylosis. It is probably better to keep the dog comfortable until euthanasia is needed, providing you have obtained a good diagnosis, and other disorders have been ruled irrelevant or absent. Analgesics are frequently prescribed (vets and M.D.s like to push pain medication almost as frequently and enthusiastically as they do antibiotics and steroids) but I have yet to see any reason or excuse for their use for typical spondylosis, backed up by a scientific study. Rimadyl, Deramaxx, and other analgesics might be somewhat effective initially in relieving symptoms of CES and dorsal lumbo-sacral disease, but not indicated for ventral spondylosis. I would not put much currency (defined both ways!) in any vet’s prescription for painkillers if he did not find hard evidence of pain, or something other than the ventral vertebral bridging of classical spondylosis on the radiographs!

An intervertebral inflammation resulting in fusion of the vertebrae has been seen in humans and is known as ankylosing spondylitis. It is related to both adult and juvenile rheumatoid arthritis, and the similarity to spondylosis deformans in the dog (minus the inflammation) once made me wonder if there is a common or similar genetic defect in the “immune systems” of man and beast. Although it has been misnamed ankylosing spondylitis in the past, spondylosis deformans in the canine is not that disorder, exactly. Senile ankylosing hyperostosis is a syndrome in humans that is considered to be a variation of osteoarthritis characterized by large osteophytes, also shows bridging between and on the anterolateral (front and side) surfaces of the vertebral bodies. In man, it appears mainly in males over 50 years of age, giving symptoms of minor to moderate back pain, stiffness, and lack of flexibility. Bone spurs and ossification in tendons and ligaments are common. Even intervertebral osteochondrosis may be a separate disorder; although

also a result of disk degeneration, it is characterized by reduced disk height and vertebral end-plate sclerosis, not seen in spondylosis deformans.

The genetic transmission of the tendency to develop spondylosis deformans is obvious to anyone who has watched it appear in offspring of certain dogs, generation after generation. But exactly how (the etiology) is not as sure. Perhaps there is an inherited weakness in how a dog's vertebrae respond to or withstand repeated microtraumas; perhaps in some lines, the blood vessels that serve the outer layers of the disks regress and disappear faster than the normal or expected three or four years. It seems to be a fairly natural consequence of aging, as 75% of dogs in some breeds are affected to some degree by 9 years, and half by 6 years. On the other hand, some work has indicated that spondylosis deformans is more a disease of middle age. Breed and family variables make the incidence figures vary tremendously. It became a very noticeable disorder in the German Shepherd Dog when, for a while, 90% of the "show" GSDs in the USA were allegedly descended from one very popular late-1960s American Grand Victor who had and passed along this disease in a severe form (estimate based on a pedigree study reported in a GSD magazine several years ago).

Great Danes are probably the most likely to develop this disorder, followed by mastino/mastiff types that are historically related. Boxers also appear to be at relatively high risk for spondylosis. Among the many that have corresponded with me was the owner in northern Michigan who reported a few things with her 6-year-old male that are not typical: panting, emaciation, and vomiting, in addition to limping and refusal to run. The vet "discovered his spine was fusing together and new bone was being formed over the top" and mentioned spondylosis. Unfortunately, this is like steering a car in two directions at once, since we think of spondylosis as bone proliferation on the *bottom* (ventral) surfaces of the vertebral segments, not the top. I suggested she also have the vets look for possible tumors/cancer on the spinal cord or associated tissues, heartworm, kidney failure, liver function insufficiency, CES, and Addison's Disease as potential causes for the other symptoms. Rimadyl had been administered, and the owner thought there might have been some temporary help. Another vet prescribed Deramaxx without even looking at the radiographs! This Boxer owner previously had his dam, who "developed the same symptoms when she was nearly nine. She could barely walk at the end and threw up frequently. The vet we had at the time didn't know what she had, although her white blood count was high and her spine was fused too." This is a strong indication that an inherited weakness should preclude such dogs from being bred.

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editor's note: Fred is one of only half a dozen in the Western Hemisphere who have SV conformation judging experience. He is an all-breed judge for several U.S. and foreign-countries' registries, wrote the definitive book on orthopedic disorders, and lectures worldwide on that and other topics. He can be reached at Mr.GSD@Juno.com or by mail:3565ParchesCoveRd,UnionGroveAL35175-8422.  
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