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Surgical Conditions of the Spine Mini Series

Session Two: Wobblers and Lumbosacral Disease

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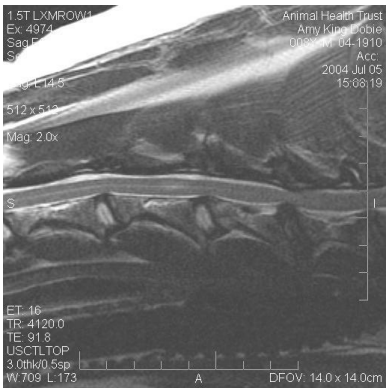

CERVICAL SPONDYLOMYELOPATHY

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Cervical spondylomyelopathy (CSM) is a multifactorial disease characterised by cervical spinal cord compression secondary to vertebral canal stenosis. The stenosis results from vertebral malformation associated or not with secondary degenerative changes of structures surrounding the spinal cord. The aetiology of CSM is not fully understood. Nutritional factors have been advocated in horses and in Great Dane as well as breed conformation, head weight and carriage in the Doberman pinscher. As it stands, we have a limited understanding of what we are treating. Many questions remain unanswered about the pathogenesis of CSM, the natural progression of the disease, and the role of medical treatment as opposed to the various types of surgical options considered for this condition.

Pathogenesis

- The role of cervical stenosis

An important feature that distinguishes normal from CSM-affected dogs is that CSM-affected dogs have a consistent stenosis of the cervical vertebral column. Two basics groups of CSM-affected dogs are recognised. The first one includes immature or young adult large-breed dogs such as Great Danes or Doberman pinschers which present with vertebral canal malformation, stenosis, or both. These developmental abnormalities are directly responsible for the spinal cord compression and multiple sites of compression are not uncommon. The second category includes middle-aged or older dogs which have degenerative changes of the vertebral column, ligaments structures and joints (osteoarthrosis of the articular processes, articular facet synovial hypertrophy and/or cyst formation, dorsal longitudinal and ligamentum flavum hypertrophy, osteophytes production, dorsal or lateral disc extrusion or protrusion) with subsequent acquired stenosis of the vertebral canal. These changes can also be added to the above mentioned developmental abnormalities and progressively induce clinical decompensation. The spinal cord compression due to the stenosis can be static (no worsening according to the head position) or dynamic (worsening or diminution of the cord compression according to the neck extension/flexion position). Adult Doberman pinschers seem to be most commonly affected by these degenerative changes characterised by ventral compressive lesions from hypertrophy of the dorsal part of the annulus fibrosus. The dorsal longitudinal ligament contributes slightly to the compressive force. If dorsal compression is present, it results from the ligamentum flavum or synovial facet proliferation.

The primary vertebral abnormalities which can be observed in CSM-affected dogs include:

- Vertebral body malformation found in 25% of CSM-affected Doberman pinschers. The vertebral body appears misshapen with rounding of the cranio-ventral aspect and a prominent cranio-dorsal ridge, which encroach into the ventral funiculi of the spinal cord. This vertebral body malformation can directly cause vertebral canal stenosis and cord compression in young dog or may predispose to secondary disc protrusion in adult dog. In one study, a third of Doberman Pinschers examined had abnormally shaped caudal cervical vertebrae before 16 weeks of age giving credence for the congenital hypothesis of cervical stenosis in this breed. However, no long-term follow-up was available to establish whether those vertebral changes caused clinical signs later in life.

- Impaired growth of the cranial vertebral pedicles resulting in cranial vertebral stenosis with cranial orifice narrower than the caudal orifice. The cord development is rapidly impaired and clinical signs appear after a few months of life. A recent morphometric ex-vivo study demonstrated that the height of the cranial aspect of the vertebral canal of large breed dogs is significantly smaller than those of small breeds, resulting in a funnel-shaped vertebral canal particularly in the caudal cervical vertebrae; among the large-breed dogs studied, this funnel shaped appearance of the vertebral canal was most pronounced in Doberman Pinschers.

- Malformed articular processes (overgrowth) resulting in encroachment into the dorso-lateral funiculi of the spinal cord. These alterations in the articular facets are common in Great Danes. Osteoarthrosis of the articular processes, proliferation of capsule of articular processes with or without synovial cyst formation can ensue and cause dorso-lateral compression in the adult dog.

- Relative stenosis of the cervical vertebral canal has recently been recognised in a morphologic and morphometric MRI study of Doberman Pinschers with and without clinical signs of CSM (da Costa et al. 2006 AJVR). The vertebral canal of CSM-affected dogs appears to be stenotic throughout the cervical portion of the vertebral column, and not just at the caudal cervical region were most clinical lesions have been detected. Since this stenosis is not responsible alone for the clinical signs, it is considered as a relative and not absolute stenosis. This relative stenosis associated with a space-occupying lesion such as intervertebral disc disease or articular facet proliferation, could then lead to the appearance of clinical signs.

- Abnormal orientation of the articular facets has recently been incriminated as a potential underlying cause for the high incidence of disc degeneration in the caudal cervical spine of large

breed of dogs with CSM. A recent ex-vivo osteological study in dogs showed a significant higher incidence of concave articular facets in the caudal cervical spine of large breed dogs compared to small dogs. Concave articular facets facilitate axial rotational motion or torsion. These torsional forces are the main forces leading to intervertebral disc degeneration in nonchondrodystrophoid dogs by causing concentric fissures or tears in the outer annular lamellae leading ultimately to fibrocartilaginous degeneration of the nucleus pulposus. However, in vivo kinetic and biomechanic studies of normal and abnormal dogs are needed to investigate if this different anatomic conformation leads to an altered and potentially harmful force distribution on the vertebral column and in particular the intervertebral disc.

- The role of instability

Instability is defined as “the loss of ability of the cervical spine under physiologic loads to maintain relationships between vertebrae in such a way that there is neither initial nor subsequent damage to the spinal cord or nerve roots, and in addition, there is neither development of incapacitating deformity nor severe pain”. The importance and role of vertebral instability associated with the developmental abnormalities is still controversial. Furthermore, there is still a lot of confusion between the terms ‘instability’ and ‘dynamic’. The fact that a lesion is dynamic (i.e. degree of compression changing with the position of the neck) does not imply that instability is involved in the pathophysiology of this lesion. Although it has been commonly proposed in the literature as mechanism implicated in the pathogenesis of CSM, no study has objectively examined this hypothesis. The evaluation of instability has only been done subjectively based on radiographic or pathological studies. The idea of instability was initially proposed in the pathogenesis of CSM because of the misalignment of the cervical vertebrae or spinal cord compression that was evident on radiographic views of the cervical portions of large-breed dogs’ vertebral columns during flexion and extension. However, this does not mean that instability is present. This slippage of cervical vertebrae represents a natural pattern of motion in dogs during flexion or extension of the neck. Aside the ‘extreme’ and rare cases where a macroscopic instability can be identified, further studies are necessary to document what represents ‘beyond’ physiological variation in term of slippage of cervical vertebrae.

A primary instability is difficult to evaluate in the presence of compensating secondary degenerative changes including spondylosis and ligament hypertrophy. However, it seems reasonable to consider vertebral body or facet malformation as a possible predisposing factor for the previously mentioned degenerative processes. As it stands, it appears that a restricted rather than an excessive intervertebral motion is more likely to take place at the sites of advance disc degeneration. It has been found in human that the overall and segmental stiffness of the cervical vertebral column increased with increasing severity of disc degeneration. Data collected in an

MR imaging study to investigate the relationship between disc degeneration and the conventional plain radiographic evaluation of cervical segmental instability in 260 humans patients suggested that instability was associated with early intervertebral disc degeneration but normal or moderate to severely degenerated discs were stable (Dai Spine 1998). Finally, experimental study in dogs also suggested that with progressive fibrocartilaginous degeneration of the intervertebral disc, the increased fibrosis of the nuclear region result in progressive stiffening of the disc (and therefore segmental stiffness causing restricted vertebral motion at the site of disc degeneration) which would point again against instability as a mechanism involved in canine CSM. Although the above evidences do not strongly suggest instability in the pathogenesis of CSM, again in vivo kinetic and biomechanical studies of normal and CSM-affected dogs are needed to investigate this controversial issue.

- The role of dynamic factors

The simple pathoanatomic concept that a narrowed spinal canal causes compression of the enclosed cord, leading to local tissue ischemia, injury, and neurological impairment, often fails to explain the entire spectrum of clinical findings observed in CSM. The pathophysiology of CSM likely involves static factors, which result in acquired or developmental stenosis of the cervical canal and dynamic factors, which involve repetitive injury to the cervical spinal cord. These mechanical factors in turn result in direct injury to neurons and glia as well as a secondary cascade of events including ischemia, excitotoxicity, and oligodendrocyte apoptosis.

In any cases, constant progressive or recurrent spinal cord compression is responsible for spinal cord parenchymal ischemic lesions (mostly affecting the gray matter) associated with demyelination and ultimately Wallerian-type degeneration affecting the ascending and descending white matter tracts.

Diagnosis

The diagnosis of cervical spondylomyelopathy is based on the demonstration of extradural cord compression by diagnostic imaging studies (myelography, computed tomography or MRI). However, signalment (breed, age) and patient history (age of onset, progression) are the first steps of the diagnostic approach.

CSM is mainly observed in large or giant breed dogs. Doberman pinschers are over-represented with a first peak incidence around 7 years. A second peak is observed at 6 months to 1 year old in large or giant breed dogs such as Great Dane as well as Doberman pincher presenting with vertebral malformation.

The most common presentation of CSM is a gait disturbance. Truncal ataxia and progressive tetraparesis are classically observed. Initially the forelimbs may appear less severely affected than the hind limbs. A short stride and a stiff gait may appear later. Neurological examination most often is consistent with a C6 – T2 spinal cord segments presentation. Usually the cranial part of the intumescence is involved, inducing lower motorneuron signs of the shoulders. Muscular palpation may reveal atrophy of the supraspinatus, infraspinatus and biceps brachii muscles. When the lesion is localised at C5-C6 intervertebral space in a post-fixed type cervico-thoracic intumescence (segment C5 not contributing to the nerve roots of the brachial plexus), the neurolocalisation may refer to an upper motorneuron C1-C5 presentation with a 'floating' thoracic limb gait. Cervical pain is usually mild or absent and tetraplegia is an uncommon presentation. Clinical manifestation is often characterised by paraspinal muscle rigidity and reluctance to extend or laterally flex the neck. In case of nerve roots compression, pain is usually more evident, especially on forelimb extension. Neurological deterioration can also be observed acutely in some animals.

Differentials diagnosis for CSM (slowly progressive gait abnormality on all 4 limbs) should include spinal/spinal cord tumour, meningo-myelitis, discospondylitis and epidural abscess, cervical disc herniation, degenerative myelopathy, leukodystrophies, synovial and spinal arachnoid cyst, atlanto-axial subluxation and syringohydromyelia.

- Survey radiographs

Plain radiographs of the cervical spine are useful in order to rule-out other differentials such as discospondylitis, fracture/luxation, vertebral neoplasm. Radiographic changes suggestive of cervical spondylomyelopathy are:

- Vertebral body malformation with rounding of the cranio-ventral aspect and prominent cranio-dorsal ridge which encroach on the vertebral canal
- Vertebral misalignment secondary to dorsal vertebral body tilting. This abnormality does not always seem to be correlated with the myelographic findings. In some cases the compression is localised to the adjacent caudal intervertebral space
- Cranial stenosis of the vertebral canal: in general without clinical consequence when difference between the cranial and caudal vertebral canal diameter is less than 3 mm
- Narrowing of the inter-vertebral spaces (spaces C5-C6 or C6-C7 in Doberman pinscher), nucleus pulposus calcification (rare) with or without disc herniation, ventral spondylosis
- Increased opacity and loss of the joint space between facets which may be associated with extensive new bone formation on the facets (common in Great Danes)

In some affected dogs, plain radiographs can be normal despite the fact that myelographic evaluation will reveal significant extra-dural spinal cord lesion.

- Myelography

Myelography should always be preceded by plain radiographic evaluation. In any case, CSF should be collected in order to rule-out myelitis/meningo-myelitis. Myelographic abnormalities that could be seen in CSM include:

- Ventral extradural compression centred on an intervertebral space suggestive of disc extrusion or protrusion
- Dorsal extradural compression suggestive of ligamentum flavum hypertrophy and/or osteocartilaginous proliferation
- Dorso-lateral extradural compression in general associated with abnormally positioned and/or degeneration of the articular facets and/or synovial cyst formation
- Annular extradural compression suggestive of cranial vertebral canal stenosis secondary to impaired growth of the pedicles
- Ventral extradural compression secondary to the dorsally tilted cranial part of the vertebral body

Several views of the cervical spine should be obtained after contrast injection: neutral lateral, flexed lateral, extended lateral, lateral with linear traction of the neck and ventro-dorsal. Compressive lesion can be classified as 1) static or positional (degree of compression influenced by the position of the neck in flexion and/or extension) and 2) traction-responsive or traction non-responsive (improve or not with linear traction of the neck). Plain radiographs obtained with flexion of the neck may enhance the appearance of vertebral misalignment. However, these modifications may be observed in normal patients. The flexed post-myelographic views will show a decreased ventral compression because of ligament and dorsal annular distraction. Post-myelographic extension views make ligaments and dorsal annulus redundant; this will increase ventral cord compression. They may be useful in order to detect lesion that could have been overlooked in neutral view (positional lesion). If two lesions are present, extended myelographic views can be used to determine which one of the lesion is the most significant in terms of compression. These views must be performed with caution as cord compression is exacerbated in this position. Traction views are used to classified the lesion as traction-responsive (often see with annulus fibrosus and/or ligamentous hypertrophy) or not (bony malformation, abnormally positioned and/or degeneration of the articular facets) and should therefore be obtained for surgical planning (decompressive versus distraction procedures). No standardised method has been set for defining a lesion as traction-responsive, since the amount of force required to significantly reduce cord compression has not been quantified. Furthermore, it still remains unclear how much distraction the spinal cord and chosen implant can tolerate. Further studies are necessary to establish the distraction efficacy of different weights and traction methods.

Myelography is essential to make a definitive diagnosis. It helps to localise accurately the site and the type of compression (static or positional and traction-responsive or not). Each case should be assigned a definite subtype because therapy should be aimed at correcting the abnormality in that individual case, rather than treating every case of CSM the same way.

- CT or MRI

It is often difficult to choose between these two techniques, especially when the results of myelography are not conclusive. CT remains the technique of choice in order to evaluate anatomical osseous details (vertebral malformation, abnormally positioned and/or proliferation of the articular facets) allowing transverse views. Post-myelographic CT studies are used to detect spinal cord atrophy (enlargement of the subarachnoid space). This evaluation may be important in term of prognosis in surgical candidates although no study has been done to correlate this finding with the surgical outcome. MRI also offers the advantage of giving better resolution of soft tissue (ligament structures, compression or parenchyma malformation, spinal cord atrophy) as well as 3-dimentional views.

One question that remains to be answered in veterinary medicine is the effect of intramedullary signal intensity changes seen in MRI of some CSM-affected dog on the surgical outcome. The evidences in human medicine suggest that the presence of intramedullary signal changes on T1- as well as T2-weighted sequences on MRI in CSM-affected patients indicate a poor prognosis whereas T2-weighted hyperintensity alone reflects pathologically reversible changes. Correlation between these MRI changes and histopathology has been established in humans. Areas of hyperintensity in T2-weighted images alone were characterized by slight loss of nerve cells, gliosis, and edema in the grey matter, as well as demyelination, edema, and Wallerian degeneration in the white matter while the combination of T2 hyperintensity and T1 hypointensity was characterized by severe histologic changes such as necrosis, myelomalacia, and spongiform changes in the gray matter, as well as white matter necrosis.

Treatment of CSM

CSM continues to present a challenge with regard to decision on optimal form of treatment and on the role of conservative management. Considering the heterogeneity in the type of compressions encountered in CSM it is not surprising that no single surgical technique can be applied effectively to all forms of CSM. Currently, there is no strong evidence that shows one surgical procedure to be superior over the others or in some instances better than nonsurgical management.

Two basic types of surgical interventions are: 1) direct decompression techniques such as ventral slot, dorsal laminectomy or facetectomy, and 2) indirect decompression techniques via distraction/fusion. All of these methods of surgical treatment claim an initial favourable outcome in 70 to 80% of dogs. However 20% of successful surgeries appear to have significant recurrence after long-term follow-up. Currently there are multiple procedures that are used clinically in dogs, and the recommended therapy is dependent on the type of compression present and the experiences and opinions of the attending veterinarian.

- Is there an optimal time to treat?

Intuitively, it would be logical to assume that an early surgical intervention may result in a better outcome by stopping progression of spinal cord degeneration while waiting for more severe signs could result in a poorer outcome due to irreversible loss of neural tissue. The basis for neurological improvement is stopping further injury to the spinal cord and remyelination of those axons that have so far been spared. If majority of lesion is predominantly axonal disruption then surgical therapy could not reasonably be expected to result in improvement. Unfortunately, there is so far no diagnostic modality that would allow the clinician to assess reversibility/irreversibility of spinal cord lesions associated with CSM and give an indication on expected degree of recovery.

- What is the literature telling (or not telling) us so far?

The majority of studies focused primarily on traction-responsive, disc-associated soft-tissue compressions with fewer studies on the surgical treatment of the osseous form of CSM. This exclusion ultimately creates a bias and makes interpretation of the results challenging

They are only limited number of studies on treatment of CSM with even fewer studies comparing different techniques. When reviewing the literature critically, many flaws in study design make interpretation of results and comparison between studies difficult to achieve. These flaws include variable details on the inclusion criteria resulting in most studies in a very heterogenous population of CSM, variable rate of onset, duration of clinical signs prior to diagnosis, severity of neurologic dysfunction at the time of diagnosis, type of work-up and clinical follow-up. For example, certain studies evaluate all type of CSM without making any distinction between osseous versus soft-tissue form of the syndrome or never made a distinction between dynamic and static lesions. In addition, the definition of long-term follow-up and methods of follow-up differed amongst studies and even within a study itself between weeks and years. The definition of successful outcome is not homogeneous between studies and range from return to normal

neurological function, improvement in at least 1 grade, to lack of deterioration. While some dogs may undergo re-examination at a set time following surgery, other follow-up may only be based on client surveys or telephone interviews.

To this date, most comparative studies are retrospective, not randomized and consider overall a very heterogenous group of CSM-affected dog without focusing on one specific type of CSM (e.g. soft-tissue traction responsive). Most studies are concentrating on a single type of surgery in a very heterogenous group of dogs as opposed to comparing two surgical techniques in a homogenous group (same type of CSM, duration of signs and neurological impairment) of CSM affected dogs. Until a well designed, prospective, double-blinded randomized study based on a large number of dogs is established, most studies on surgical techniques so far published should only be viewed as descriptive. The lack of knowledge of the natural history of the disease makes treatment recommendation for this disease difficult

- Direct decompression techniques

Direct decompression techniques are usually reserved for CSM lesions that do not significantly improve with linear traction based on advanced imaging. Direct decompression typically is done through a ventral slot, facetectomy or dorsal laminectomy

Dorsal decompression

In general, decompression of a dorsal or lateral, osseous or soft tissue compression (ligamentum flavum hypertrophy, joint capsule proliferation, extradural synovial cyst, dorsal osteocartilaginous proliferation) requires a dorsal laminectomy. Partial (medial) or complete (unilateral) facetectomy is indicated for overgrowth of the articular processes.

Ventral decompression

The ventral soft-tissue, traction non-responsive lesion requires direct decompression through a ventral slot. The surgical treatment of the ventral, soft-tissues, traction-responsive is most controversial. Both direct and indirect decompressions have been described. No study has so far evaluated both methods of decompression on dog with only soft-tissue traction responsive lesion.

Argument against direct ventral decompression in these dogs would be that many of them also present dorsal compression due to hypertrophy or folding of the ligamentum flavum which would not be addressed by direct ventral decompression. Ventral decompression may also result in further collapse of the disc space with exacerbation of spinal cord compression by additional infolding of the ligamentum flavum and remaining part of the dorsal annulus as well as worsening

of foraminal stenosis. When combining results of published reports (Rusbridge et al 1998, Chambers et al 1986, Bruecker et al 1989), ventral slot has a long-term success rate of 72% unfortunately not distinction was made between traction-responsive versus traction non-responsive lesion and the definition of 'long-term' outcome differed between these case series.

- Indirect decompression techniques

Intuitively, a significant traction-responsive lesion suggests that surgical therapy should involve stabilization of the vertebrae. Maintenance of distraction in these cases would in theory immediately and effectively relieve the extradural spinal cord compression caused by the redundant dorsal annulus and/or ligamentum flavum, relieve the spinal cord ischemia caused by compression of the ventral spinal artery and reopen the narrowed intervertebral foramina decompressing the nerve roots/spinal nerves.

In addition, some surgeons argue that stabilization should be performed in all forms of CSM on the basis that increased vertebral stability can result in intervertebral disc and ligament atrophy as well as osseous regression, which can lead to eventual to resolution of spinal cord compression.

However, other surgeons would argue against stabilization because of the reported implant failure or domino lesions.

The most important concept to understand is that the goal of stabilization is to treat instability in the short-term, but long-term stability requires bony fusion, or the implant can fail which has been the most common cause of failure in distraction-fusion techniques.

Implants and grafts

Various implants can be used to maintain distraction including Steinmann pins or cancellous bone screws and PMMA, a PMMA-interbody plug, screw and washer, locking plate such as SOP or Synthes cervical spine locking plate, intervertebral tantalum spinal fusion blocks… Whichever technique is used, the development of spinal fusion should remain the ultimate goal as failure is a time-dependent certainty in the absence of an associated bony union.

Bone grafts are therefore recommended and perform two functions: structural support and biologic enhancement of fusion. Two types of grafts can be used (sometime in combination): autograft (e.g. cancellous autograft harvested from proximal humerus) or allograft (cortical ring allograft taken from a cadaver long bone such as humerus or tibia or cancellous bone allograft). Whether spinal fusion ever occurs, within what time frame and to what extent in the stabilized

patient remains unclear. Bony proliferation on radiographic follow-up may suggest bony fusion and stabilization but no concurrent histopathological and biomechanical studies have proven this to be true.

A ¾ ventral slot is performed. The metal implants used can be two Steinmann pins, threaded pins, or bone screws. Positive-profile pins provide more rigid fixation than smooth pins. Positive profile pins are also preferred to the partially threaded ones as the thread junction can act as a stress riser. The author prefers to use cortical screws. Two 3.5 to 4.5 mm screws are implanted in each vertebral body. The cortical screws (self tapping titanium alloy cortical screw or stainless steal cortical screw) are directed from the ventral midline of the vertebral body in a diverging path and at a safe reported angle of 34.21° at C5 (range, 30– 351), 36.61° at C6 (range, 30–401), and 47.51° at C7 (range, 45–551)**.** The screws have better purchase if they penetrate two cortices but there is less risk of penetrating the vertebral canal or damaging nerve roots if they do not. A recent cadaveric study has shown that monocortical screw fixation (with tip of the screw contacting the inner cortex) was biomechanically equivalent to bicortical screw fixation. The screw should be allowed to protruded by 12 to 15 mm from the vertebral body ventral surface to allow incorporation of the PMMA. The disc space is then distracted with a modified Kaspar distractor. Alternatively, distraction can be maintained by a rope around the maxillary teeth. Then an autograft from the ilium is wedged into the slot to maintain distraction prior to cement application. Use of such a graft has the additional advantage that it will promote an osseous union. The screws are then incorporated in PMMA while maintaining distraction.

A new motion sparing technique has recently been proposed using artificial replacement disc (Adamo spinal disc®**).** This prosthesis is made of a titanium alloy and consists of two end-plates, with a range of movement of 30° between the plates and is designed for large dogs, ranging from 22Kg to 45Kg.

Complications

Complication associated with indirect decompression techniques include implant failure (subsidence, fracture of the implant), limited bony fusion (pseudoarthrosis), iatrogenic spinal cord injury during placement of the implant, surgical site infection, and adjacent segment disease.

A number of factors should be considered in an attempt to reduce the incidence of acute implant-related complications:

- Excessive vertebral distraction should be avoided. Unfortunately, no standardised method has been set for defining a lesion as traction-responsive, since the amount of force required to significantly reduce cord compression has not been quantified. Furthermore, it still remain

unclear how much distraction the spinal cord tissue can tolerate. Finally, one should not assume that the degree of distraction achieved during imaging study could be exactly re-created at surgery.

- Although the cancellous bone should be exposed to the graft to promote its vascular in-growth, preservation of intact end plates is critical (load sharing). Any violation of the end plates predisposes to end plates collapse and exposes the cancellous bone of vertebral bodies which is totally incapable of resisting compressive loads.

- Ideally, the interbody material used should have an elastic modulus compatible with vertebral bone to limit the risk of crushing or perforating the adjacent end plates. This is unlikely to be achieved with interbody material such as cement plug, washer and other metallic spacers or cages.

- Interbody material should be biologically compatible to facilitate vertebral fusion for long-term stability. Again this is unlikely to be achieved with cement plug or metallic interbody materials. A recent experimental study in sheep concluded that intervertebral cement plug stabilisation did not maintain distraction of the disc space and bony union between vertebrae was not achieved.

- Conservative treatment

Results of medical management of CSM have varied in the literature. Two studies have recently been published. The first study by da Costa et al. in JAVMA (2008) looked at 104 dogs, half of which non-Dobermann breed, and considered all types of CSM. This study showed no significant differences in owner-reported percentage of improvement and score for quality of life between surgical and conservative treatment. 54% of dogs improved and 27% remained unchanged with conservative treatment but 81% were reported to improve with surgery. Unfortunately, this study looked at a very heterogenous group of CSM affected dog in term of type of CSM. The second study published by De Becker et al. in JSAP (2009) looked at 51 dogs with disc-associated wobbler syndrome (DAWS) treated conservatively. Successful outcome was achieved in only 45%. Furthermore, a successful outcome was defined as not only dogs that had improved but also dogs that did not show any worsening of clinical signs. While this study looked at a fairly homogenous group of CSM affected dog (DAWS), no comparison was made with outcome of surgical treatment in a similar population.

Both of these studies and likely results of future studies on conservative treatment should be interpreted with caution. In absence of randomisation, it is possible that the above referred dogs represented a patient group with severe clinical signs and poor prognosis as many dogs with mild clinical signs are rarely admitted to a referral hospital. It is also important to realize that cases treated successfully in first opinion practice with medical therapy may not form part of the literature. Therefore, it is possible that success rate with medical management is higher than reported. Ideally, a prospective randomized controlled study involving a very large number of dogs with the same type of CSM and similar neurological grading undergoing either conservative or the same surgical treatment and long-term MRI and clinical follow-up could help us to resolve the question of the role of conservative management in CSM affected dogs.

LUMBOSACRAL DISEASES

CAUDA EQUINA SYNDROME, LUMBO-SACRAL DISEASE & DEGENERATIVE LUMBO-SACRAL STENOSIS

The cauda equina lies within the lumbosacral canal and is defined anatomically as the seventh lumbar, sacral, and caudal spinal cord segments and their respective nerve roots. The clinically important peripheral nerves that arise from the spinal nerve roots of the cauda equina are the sciatic, pudendal, parasympathetic pelvic and caudal nerves. Cauda equina syndrome (CES) is a definitive term describing sensory and/or motor neural dysfunction that results in compression, inflammation, destruction, displacement, or vascular disruption to the nerve roots of the cauda equina. Any disease affecting the caudal lumbar vertebrae, sacral vertebrae or the first five caudal vertebrae can potentially lead to CES and deficits of function. Terminology regarding cauda equina dysfunction is confusing, as lumbosacral disease has been used synonymously with cauda equina syndrome. Lumbosacral disease is a collective term encompassing many diseases that can lead to pathologic changes of the cauda equina. Lumbosacral disease is often caused by stenosis of the lumbosacral vertebral canal. The term lumbosacral vertebral canal stenosis encompasses a spectrum of disorders that cause a narrowing of the vertebral canal or intervertebral foramina with compression of the cauda equina nerve roots or their blood supply. Degenerative lumbosacral stenosis (DLSS) is a common cause of cauda equina syndrome in dogs. However, CES may result from numerous causes other than lumbosacral vertebral canal stenosis. DLSS is characterized by intervertebral disc degeneration, Hansen type-2 disc herniation, osteophyte formation at the vertebral endplates and facet joints, and hypertrophy of the interarcuate ligament and joint capsules leading to a narrow vertebral canal or intervertebral foramina. The term lumbosacral instability is a misleading term, as instability is not demonstrated consistently in association with lumbosacral vertebral canal stenosis.

CAUSES OF CAUDA EQUINA SYNDROME

Disorders of the cauda equina that result in CES can be either congenital or acquired, or may be a combination of both these categories. Disorders that result in clinical signs of cauda equina dysfunction in dogs include:

- Congenital disorders
  Congenital idiopathic stenosis of the vertebral canal
  Spinal dysraphism
  Transitional vertebrae
  Dysgenesis of lumbosacral vertebrae
  Spina bifida
- Acquired disorders
  Discopondylitis and spinal epidural empyema
  Primary or metastatic vertebral tumour and malignant nerve sheath tumour
  Intervertebral disc disease
  Spinal fracture and/or luxation
  Ischaemic myelopathy
  Granulomatous meningomyelitis
  Infectious meningomeylitis
  Cauda equina neuritis
- Combined disorders
  Combination of congenital and acquired disorders (e.g., disc degeneration and lumbosacral vertebral canal stenosis)

PATHOGENESIS OF DEGENERATIVE LUMBOSACRAL STENOSIS

Many similarities have been noted between vertebral and soft tissue alterations seen in dogs with DLSS and cervical stenotic myelopathy. As it stands, we have a limited understanding of what we are treating. Many questions remain unanswered about the pathogenesis of DLSS, the natural progression of the disease, and the role of medical treatment as opposed to the various types of surgical options considered for this condition. A number of degenerative changes may combine to cause compression of the cauda equina: Hansen type II disc herniation at L7/S1, osteophytosis of the articular processes, articular facet synovial hypertrophy or cyst formation, soft tissue proliferation of the ligamentous structures. Large breed dogs, especially GSD, are most commonly affected. Breed-specific differences in the anatomic conformation of the

lumbosacral region and type of motion at the discovertebral junction have been suspected to play an important role in the development of this condition.

- Chronic degenerative disc disease

Most cases of acquired lumbosacral stenosis appear to be related to intervertebral disk degeneration at L7 - S1, especially Hansen type 2 protrusion with dorsal bulging of the dorsal annulus into the vertebral canal, intervertebral foramina or both. The presumed sequence of pathophysiological events in DLSS is that mechanical stress or other factors result in early degeneration of the intervertebral disc. The loss of shock-absorbing and stabilizing function of the intervertebral disc is presumed to elicit 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. The end result is DLSS with compression of the cauda equina.

- The role of lumbosacral motion

Range of motion is influenced by the condition of the discovertebral junction and the dorsal elements, which include the facet joint capsules, ligamentum flavum, laminae, and pedicles of the vertebrae. These structures are stretched during flexion. As degeneration develops, these elements become rigid and flexion is reduced. Results of studies with vertebral columns of human indicate that lumbar disc degeneration in an early stage caused segmental instability that mainly affected translational motion. However, in severely degenerated discs, motion is reduced. Many studies have examined lumbosacral conformation and mobility in normal and affected dogs with many question remaining unanswered regarding the role of lumbosacral motion in the pathogenesis of DLSS. The lumbosacral joint has the highest mobility of the lumbar spine with considerable transfer of forces between the 7^{th} lumbar vertebrae and the sacrum. This high mobility may predispose to high wear and tear and may be a risk factor for disc degeneration. Flexion and extension are the main movement of the LS joint, but lateral and rotational movements also occur. Lumbosacral angle and range of motion during flexion – extension and vertebral alignment have been assessed as possible cause for disc degeneration in dogs with clinical signs of cauda equina compression. However, in these studies survey radiographs were used and the degree of disc degeneration was not known. Recent ex-vivo 3-dimensional motion pattern studies in dogs suggest that while GSD are predisposed to DLSS, mobility in the lumbosacral junction is significantly smaller in that breed compared to other breeds (Benninger et al. AJVR 2004). These findings indicate that the amount of motion alone does not explain the high prevalence of DLSS in GSDs.

The significance of the ventrodorsal translation of the sacrum in the pathogenesis of DLSS in dogs remains as well unknown. In humans, the term spondylolisthesis refers specifically to a forward (anterior) movement of a lower lumbar vertebra relative to a lumbar vertebra or sacrum directly below it. The term “retrolisthesis” has been proposed to describe this “reverse spondylolisthesis” of dogs. Some authors consider this as a clinical instability which could lead to disc degeneration, however, ventrodorsal translation of the sacrum is not always associated with clinical signs of cauda equina compression or intervertebral disc degeneration. Whether increased ventrodorsal translation is a primary problem leading to abnormal shearing forces and lumbosacral disc degeneration, or whether disc degeneration precedes increased translation, is not yet known.

- The role of anatomic conformation of the lumbosacral junction

Although GSDs are most commonly affected by degeneration of the lumbosacral disc, vertebral columns in that breed appear to have less mobility at L7 – S1 than in dogs of other breeds. Therefore, in addition to mechanical load, other factors have to be considered as causes of disc degeneration. The orientation and shape of canine articular process joints and their association with disc degeneration have also been investigated. Articular process joint tropism, defined as an asymmetry of left and right articular process joints, was analysed as a possible cause of abnormal axial rotation and increased torsional stress on the intervertebral disc (Benninger et al. 2004 Am J Vet Res). However, the higher degree of tropism in GSD was not associated with the severity of disc degeneration. Different shapes of the facet joint have also been described, and differences have been found between levels within an individual, within the same breed, and also between breeds (Rossi et al 2004 Vet Rad & Ultrasound). In GSD, straighter facet joint were found, whereas those dogs of other breeds were more commonly round. The study by Rossi et al. supported the hypothesis that a different anatomical conformation of the articular process plays an important role in the pathogenesis of degenerative lumbosacral stenosis. A positive association between articular process orientation (articular process joint angle difference) and MR-imaging stage of disc degeneration in the caudal lumbar spine was found in GSD. In this group, vertically oriented articular process joints at L6 – L7 with a sudden change to more horizontally oriented articular process joints at the lumbosacral junction were associated with a higher degree of disc degeneration.

Seiler et al. (2002) found that large facet joint angles were also more commonly associated with disc degeneration. It was proposed that disc degeneration could be a consequence of increased axial rotation as a result of large facet joint angles. However, more recent cadaver studies by Benninger et al. (2006) revealed that larger, less sagittally oriented facet joint angles at L7-S1, were associated with mainly increasing flexion and extension and only a low influence on axial

rotation. This was in accordance with other studies on human vertebral column specimens where it was concluded that the facet joint angle was not correlated with axial rotation and was not a critical factor for disc degeneration. Although the facet joints act as a positive stop to axial rotation, different facet joint shape does not seem to influence the motion pattern and has to be regarded as polymorphism of the facets with no effect on the 3-D motion pattern (Benninger et al. 2006).

In vivo kinetic and biomechanic studies of normal and abnormal dogs are needed to investigate if this different anatomic conformation leads to an altered and potentially harmful force distribution on the vertebral column and in particular the intervertebral disc.

- The role of congenital and developmental vertebral anomalies

Predisposition to DLSS may be caused by congenital or developmental vertebral anomalies. If a dog is born with a relatively narrow vertebral canal, minimal degenerative changes may be sufficient to cause clinical signs. Vertebral malformations such as transitional vertebrae may initiate DLSS by altering spinal biomechanics. An association has been described between transitional vertebrae (vertebrae having anatomic features of two adjacent vertebral regions), cauda equina syndrome, and degenerative disc disease. Because transitional vertebrae may result in asymmetry of the lumbosacral junction including the disc space, altered mechanical stress on the disc could result in disc degeneration. Developmental vertebral anomalies such as sacral osteochondrosis, in which the attachment of the disc to the endplate is damaged, is always associated with disc degeneration. The GSD is over-represented in the population of dogs with sacral osteochondrosis and transitional vertebrae. Lateralised disc herniation on the side of the abnormal sacroiliac attachment is a common finding in dogs with asymmetrical, transitional, lumbosacral vertebral segments with the disc protrusion tending to occur on the opposite side from the broadest fusion of the vertebra with the ilium.

DIAGNOSIS OF DLSS

- Clinical presentation

DLSS affects middle-aged medium to large breeds, particularly GSD, with a male to female ratio close to 2:1 in most reports. The most common historical findings is pain in the caudal lumbar region and pelvic limb weakness manifested as a reluctance or difficulty in jumping, climbing, rising, or sitting. Affected animal often stands with the pelvic limb tucked under the caudal abdomen to flex the spine which lessen canal stenosis and nerve root compression. Lumbosacral hyperaesthesia can be differentiated from pain associated with hip dysplasia by transrectal palpation of the lumbosacral joint and by applying digital pressure on the spinous

processes of L7 and S1 with the dog standing and laying on its side. Neurologic deficits are lower motor neuron in nature and relate either to the sciatic nerve (conscious proprioceptive deficits, decreased hock flexion, or patellar pseudo-hyperreflexia) or to the pudendal, pelvic, or caudal nerves (urinary or faecal incontinence, motor or sensory deficits to the perineum or tail). Reflex dysfunction of the limbs commonly involves those muscles innervated by sciatic nerve (L6 – S1 nerve roots but L7 and S1 provide the major contribution) particularly the flexor and extensor muscles of the hock. The patellar reflex may be hyperreflexic due to loss of antagonism form the flexor muscles (pseudohyperreflexia).

In some animals with L/S disc herniation, one pelvic limb may be held in partial flexion or a repetitive "stamping" motion may be observed. These animals frequently show considerable amount of pain on manipulation of the limb and lumbosacral spine. This combination of signs is termed "root signature" and is believed to be associated with nerve root compression or entrapment by lateralised disc herniation or stenotic intervertebral foramen. The term "neurogenic intermittent claudication" is used to describe the occurrence of exercise-induced pain in some affected dogs. This condition is believed to be related to dilation of radicular vessels and subsequent compression of adjacent nerve roots in a stenotic intervertebral foramen or lateral recess of the caudal L7 vertebral foramen narrowed by a degenerative process.

Definitive diagnosis of DLSS is difficult because no one test has a 100% specificity and sensitivity, causing false positives and negatives.

- Conventional radiography

Interpretation of survey radiographs is difficult due to the highly complex anatomic region and the non-specific nature of commonly observed findings, leading to false positive conclusions. Survey radiographs help to identify conditions such as discospondylitis, vertebral tumour, traumatic spinal fracture/luxation, and to detect predisposing factors for DLSS such as sacral osteochondrosis or transitional vertebrae. Indirect evidence of DLSS such as end plate sclerosis, spondylosis deformans at L/S and narrowing of the L/S disc space may be seen. However, these findings are not specific and may be observed in clinically normal dogs. Ventral displacement of the sacrum with respect to L7 and diminished dorsoventral dimensions of the lumbosacral spinal canal may be seen; however, such findings must be interpreted with caution, as they may be seen in normal dogs in association with slight rotation of the vertebral column on lateral radiographs. Furthermore, the inability to assess soft tissue structures and therefore neural tissue compression limits the use of conventional radiography alone to assess patients with suspected CES.

- Positional radiography

The exact role of positional radiography in detecting CES is unclear. Several attempts to separate normal dogs from dogs with L/S vertebral canal stenosis by means of objective measurements made from positional radiographs have not been successful.

- Myelography

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 L/S junction. Myelography provides however a means to "screen" the entire spinal cord for abnormalities, particularly the lumbar enlargement, where a lesion may result in signs of CES. Cisterna magna injection is preferred to prevent epidural leakage which may hinder assessment of the lumbosacral vertebral canal. Myelography is not helpful in the diagnosis of DLSS when the dural sac ends cranial to the L/S junction (common in large breed dogs) or when compressive lesions are located in the intervertebral foramen or lateral recess through which spinal nerves travel. The use of "stressed" radiographs (flexion and extension projections) may be combined with myelography.

- Discography

Discography consists of radiography completed following the injection of contrast material into the nucleus pulposus of an intervertebral disc. This technique has special application to the lumboscral disc space. While it should not be possible to inject more than 0.3 mL of contrast medium in a normal disc, intradiscal accumulation and focal extravasation of contrast medium into the vertebral canal strongly suggest disc herniation.

- Epidurography

Alone, epidurography has been reported to be diagnostic in 78%-93% of dogs confirmed surgically. It is easier to perform than myelography and has less morbidity. The disadvantage is that filling of the epidural space may be incomplete because this space is poorly defined, contains fat and has multiple lateral openings.

- Electrophysiology

Electromyographic (EMG) studies can help to confirm neurological disease affecting the cauda equina as well as mapping out denervation. Unfortunately, a large number of dogs with L/S disease (particularly those presented with only pain) have normal findings on EMG. Although an abnormal EMG can aid in confirming a clinical suspicion of DLSS, it does not provide information on the etiology.

- Computed tomography

Computed tomography (CT) is useful in the investigation of DLSS as it shows clearly the vertebral canal, intervertebral foramina, lateral recess and articular processes in cross-sectional images. Individual nerve roots can be visualized directly because of the inherent contrast provided by the epidural fat. CT images of dogs with suspected DLSS are best obtained prior to injection of any contrast medium into the vertebral canal or subarachnoid space. Abnormalities observed on CT in dogs with DLSS include loss of epidural fat, bulging of the intervertebral disc, spondylosis, increased soft tissue opacity in the intervertebral foramen, thecal sac displacement, degenerative changes affecting the articular processes and facets.

- Magnetic resonance imaging

MRI is now considered as the best imaging modality to evaluate the lumbosacral vertebral canal by providing important information regarding soft tissue stenosis of this canal. MRI can clearly reveal soft tissue such as the cauda equina, epidural fat, and intervertebral disc, at the lumbosacral region. However, no correlation was found between severity of the clinical signs and the severity of cauda equina compression as assessed by MRI in one study (Mayhew et al. 2002 JAAHA). In humans, it has been proposed that MR imaging can lead to overdiagnosis of disc disease because many people without back pain have disc bulges or protrusions on MR imaging. In one series of people with clinical signs of disease in the lower back, >25% had various radiologic evidence of intervertebral disc herniation (Gorman et al. 1997 J Okla State Med Assoc). In another MRI study of 98 asymptomatic people, it was found that 52% had a bulge in at least one level of the vertebral column and 27% had a protrusion (Jensen et al. 1994 N Engl J Med). Special features of L7 foraminal nerve root (which exits between L7 and S1 vertebrae) involvement on MRI include: absent foraminal fat signal in transverse and sagittal images and dorsal STIR hyperintense signal of exiting L7 nerve root. These changes occur in combination with spondylosis deformans and degenerative disc disease.

TREATMENT OF DLSS

Both medical and surgical management have been advocated in the treatment of DLSS. Historically, dogs with less severe clinical signs or those with contraindications for surgery receive medical treatment alone, resulting in inherent bias in patient selection and results found in the literature. Reciprocally, severely affected dogs such as those with faecal or urinary incontinence or marked motor and sensory deficits are surgical candidates, resulting in a similar treatment bias.

Conservative treatment consists of the use of NSAIDS, a change in exercise pattern, and body weight reduction. Lumbosacral epidural injections of corticosteroids (methylprednisolone acetate 1 mg/kg injected at day 1, day 14 and day 42) have recently been reported as a treatment method in dogs showing improvement in 79% of the patients with no neurological signs. Conservative treatment does not cure the underlying problem but may result in sufficient pain management.

Dorsal laminectomy

Dorsal laminectomy at the lumbosacral junction allows good visualisation of the cauda equina, L7/S1 dorsal annulus, articular processes and surrounding tissues.
The dog is placed in sternal recumbency with the pelvic limbs drawn forward. The spine is extended over a sandbag, opening the dorsal space between the vertebral laminae.

The skin is incised from the spinous process of L5 and extended to the caudal margin of the sacral spinous process on the dorsal midline. The superficial and deep lumbosacral fascia is incised. The epaxial musculature is elevated from the spinous processes and sharp dissection is used to remove the musculature attachments to the articular processes of L7/S1. Dorsal laminectomy is performed with a motorized burr. The caudal two-thirds of the L7 laminar bone is removed, leaving a cranial laminar bridge. Bone is removed as far lateral as possible, including sublaminar extensions of the interarcuate ligament extending under the caudal facet of L7. The ligamentum flavum is removed to expose the cauda equina. The cauda equina may be retracted gently to visualise underlying structures, such as dorsal annulus, as well as to assess the L7 nerve root as it enters the intervertebral foramen. Disc protrusion, ligamentous hypertrophy and secondary compressive osteoarthritis can be treated by decompressive dorsal laminectomy, with or without stabilisation. Partial discectomy is performed by performing a dorsal fenestration (or annulectomy) and is continued with a nuclear pulpectomy (or nucleotomy). A small bone spoon or curette is used to remove degenerated disc material. Following decompression, a free subcutaneous fat graft is harvested and is transplanted dorsally to the laminectomy site to prevent dural adhesions and new bone formation.

Foramenotomy/facetectomy

Decompression of the neuroforamen can be achieved by partial removal of the pedicle cranial to the neuroforamen or via facetectomy. The latter is generally not recommended if bilateral decompression is necessary. Facetectomy can be followed by stabilisation (see below) if instability is a concerned.

Lumbosacral stabilisation

Several stabilisation techniques of the L7/S1 articulation have been reported including cross pin fixation, transarticular screw fixation combined with dorsal laminectomy, fixation/fusion using pedicle or vertebral body screws fixed with a bone cement bridge, pedicle screw-rod fixation or intervertebral titanium bolt placed between vertebra providing distraction and pins and PMMA-bars. In view of the malarticulation and ventral displacement of the sacrum relative to L7, a distraction-fusion technique has been advocated for treatment of some cases of lumbosacral disease. However, clear indications for stabilisation are lacking. Distraction and fusion of the lumbosacral spine is generally performed in patients with severe dynamic lumbosacral pain and transitional displacement of S1 in respect to L7 > 4 mm in extension or flexion of the spine. Depending on the size of the patient, 3.5 to 4.5 mm cortical screws are positioned in the vertebral bodies of L7 and S1 respectively, stabilisation is achieved with bone cement and fusion is promoted by cancellous bone graft.

The author uses either pedicle or vertebral body screw fixation implanted at L7 and S1. For L7, the entry point for the pedicle screw is the intersection between the line crossing the caudal border of the facet joint and the craniocaudal line crossing the base of the transverse process. For S1, the entry point is situated halfway between the caudal border of the cranial articular process and the intermediate sacral crest. Optimal implantation corridor runs is a dorsocaudolateral to ventrocraniomedial oblique direction from the dorsal cortex of the lamina to the ventral cortex of the vertebral body, without perforating the ventral cortex, the medial pedicle wall, or the lateral pedicle wall. Optimal screw anchorage is achieved by involving, but not perforating, as many cortices as possible.

Dorsal laminectomy is performed across the caudal aspect of L7 and the cranial aspect of the sacrum. The ligamentum flavum is removed to expose the cauda equina. The dorsal spinous process of L7 is partially preserved. The laminectomy used is narrower than reported for decompressive laminectomy in an attempt to maintain base strength of the articular facets. Protruded annulus fibrosus is resected if present.

Articular cartilage on both surfaces of the facet joint is removed using a scalpel blade at a 30-45° angle through the centre of the L7 facet in a craniodorsal to caudoventral direction to traverse the L7-S1 articular facets into the body of the sacrum to thus maintain the distracted position. Cancellous bone graft obtained from an ilial wing is placed into and around the L/S facet joint.
An autogenous fat graft, harvested from the subcutis is placed over the dorsal laminectomy site. Fusion is promoted by placing a cancellous bone graft over the dorsal lamina. Fixation is

achieved by embedding screw heads or pin ends in PMMA, which functions as an internal fixator along the dorsal aspect of the lumbosacral spine.