Cervical Spondylomyelopathy (Wobbler Syndrome) in Dogs

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Cervical spondylomyelopathy (CSM) is a common disease of the cervical spine of large and giant breed dogs. CSM is characterized by dynamic and static compressions of the cervical spinal cord, nerve roots, or both, leading to variable degrees of neurologic deficits and neck pain. CSM is a controversial disease. There are few diseases in veterinary medicine that have been referred to by 14 different names. Wobbler syndrome, caudal cervical spondylomyelopathy, cervical spondylopathy, cervical spondylopathy disc associated compression, cervical vertebral instability, cervical malformation/malarticulation syndrome, cervical spondyloolisthesis, cervical stenotic myelopathy, disc-associated wobbler syndrome, cervical spinal stenosis, cervical subluxation, cervical vertebral instability-malformation syndrome, and cervical spondylotic myelopathy are all terms that have been used to describe the disease.1–13 The pathogenesis, diagnosis, and treatment of CSM are also controversial. No fewer than 21 surgical techniques have been proposed to treat CSM. This diversity of treatment approaches reflects the lack of understanding of the basic mechanisms of CSM. Fortunately, recent studies have aimed at understanding the mechanisms leading to CSM. Only with a thorough knowledge of the disease will the treatment of CSM evolve.

Palmer and Wallace,1 in 1967, were the first to describe CSM in young Basset hounds. Later in the 1970s, Great Danes appeared as the most commonly affected breed.2,14 Since the early 1980s, Dobermans have accounted for the majority of the reported cases.4,9,15–17 Although no prevalence study has been performed, it is likely that CSM is the most common disease of the cervical spine of large and giant breed dogs. Canine CSM bears similarities with the cervical spondylotic myelopathy of humans and the Doberman breed has been proposed as a natural model to study this disease in humans.18 Not surprisingly, much more is known about cervical...
spondylotic myelopathy than its canine counterpart. Therefore, when applicable, parallels between the 2 diseases are emphasized herein.

ETIOLOGY

The etiology of CSM is still unknown. Proposed etiologies include genetic, congenital, body conformation, and nutritional.

Genetic

Many investigators have proposed a genetic origin.\textsuperscript{1,19–21} The first description of the syndrome in Basset hounds suggested that the disease was inherited, but only 6 dogs (all males younger than 6 months of age) were studied and the specific mode of inheritance could not be established.\textsuperscript{1} A Swiss study in Borzois suggested that CSM was inherited with an autosomal recessive pattern, but no explanation was given why only females were affected.\textsuperscript{22} Other studies proposed a hereditary or familial basis,\textsuperscript{14} but the methodology to support such conclusions was not reported. Two large studies with more than 370 Dobermans failed to demonstrate an inheritable trait.\textsuperscript{16,23} However, no well-designed prospective study has yet specifically evaluated the genetic aspects of CSM in Dobermans or Great Danes.

Congenital

A study of neonatal Dobermans investigated the computed tomography (CT) features of the cervical spine in 27 dogs, comparing it to the cervical spine of neonatal dogs of other breeds.\textsuperscript{24} Stenosis of the cranial aspect of the vertebral canal and asymmetry of the vertebral body were identified in the fifth, sixth, and seventh cervical vertebrae of Dobermans. The most severely affected vertebra was the seventh. The findings of the study indicate that Dobermans are born with congenital vertebral canal stenosis.\textsuperscript{24}

Body Conformation

Body conformation has been proposed as a predisposing factor since the early study by Wright and colleagues\textsuperscript{3} in 1973. The abnormal forces exerted by a large head in a long neck and the association with a rapid growth rate were proposed to lead to abnormal stresses in the vertebral bodies, causing vertebral changes and spinal cord compression. However, a study found no correlation between body conformation (head size, neck length, body length, and height at withers) and radiographic evidence of CSM in 138 Dobermans aged 1 to 13 years.\textsuperscript{16} It seems unlikely that body conformation has a significant role in the development of CSM.

Nutritional

Dietary factors, including overfeeding and excessive dietary calcium, were implicated as contributory factors in Great Danes,\textsuperscript{25,26} but these factors do not seem to have the same importance in Dobermans.\textsuperscript{27} Even in Great Danes, their importance is questionable because the practices of overfeeding and calcium supplementation have been abandoned for many years, and the disease is still commonly seen.

PATHOPHYSIOLOGY

As stated in the definition of CSM, the disease involves both static and dynamic factors. Traditionally, spinal cord compression was thought to be a key factor leading to the signs of CSM. Of note, 2 recent magnetic resonance imaging (MRI) studies found that 25% to 30% of clinically normal Dobermans have clinically silent spinal cord compression.\textsuperscript{28,29} Similarly, other spinal changes previously thought to be associated with CSM have
been found in a high percentage of clinically normal Dobermans. Such abnormalities are intervertebral disc degeneration (75% of dogs), intervertebral disc protrusion (100% of dogs), and intervertebral foraminal stenosis (68% of dogs). These findings in normal dogs called into question the traditional assumptions of CSM and prompted consideration of other mechanisms potentially involved in the pathogenesis of the disease.

A key mechanistic difference between normal and CSM-affected Dobermans that explains why disc-associated spinal cord compression does not necessarily lead to neurologic signs is the vertebral canal stenosis. Vertebral canal stenosis was consistently present throughout the entire cervical spine of CSM-affected Dobermans, even at C2 and C7-T1 regions. Clinically normal Dobermans have a larger vertebral canal. A narrow canal lowers the threshold at which the cumulative effects of various structures encroaching on the spinal cord cause signs of myelopathy. In humans a smaller vertebral canal is considered the most important static factor for the development of cervical spondylotic myelopathy.

All dogs with CSM have some degree of vertebral canal stenosis. It may be an absolute vertebral canal stenosis (which then causes direct spinal cord compression and neurologic signs) or a relative vertebral stenosis, which by itself does not lead to myelopathic signs, but predisposes the patient to develop myelopathy. Despite some degree of overlap, the pathophysiology of the spinal cord compressions can be basically divided into osseous- or disc-associated compression.

**Disc-Associated Compressions**

Disc-associated compression is typically seen in middle-aged large breed dogs. This form of CSM is commonly seen in Doberman Pinschers and most studies have focused on this breed. Disc-associated CSM is primarily associated with ventral spinal cord compression. This compression may be symmetric or asymmetric (Fig. 1). It can also be complicated by dorsal compressions caused by either vertebral canal stenosis or hypertrophy of the ligamentum flavum. Affected dogs are apparently born with a congenital relative vertebral canal stenosis. This relative vertebral canal stenosis per se does not lead to clinical signs, but predisposes to the development of signs. The vast majority of the disc-associated spinal cord compressions are located in the caudal cervical spine, affecting the discs C5-6 and C6-7. The biomechanical features of the caudal cervical spine explain the high incidence of caudal cervical disc lesions. The caudal cervical spine was recently shown to experience 3 times more torsion than the cranial cervical spine, confirming the findings of a morphometric study. Torsion is the main biomechanical force leading to intervertebral disc degeneration in non-chondrodystrophic dogs, more so than axial compression. In addition, a recent study found that Dobermans with CSM have larger intervertebral discs than clinically normal Dobermans. This difference would cause a larger volume of disc protrusion into the vertebral canal. Therefore, 3 factors act in combination to explain the pathophysiology of disc-associated CSM: relative vertebral canal stenosis, more pronounced torsion in the caudal cervical spine leading to intervertebral disc degeneration, and protrusion of larger volume of disc material in the caudal cervical spine.

**Osseous-Associated Compressions**

The pathophysiology of osseous or bony-associated CSM is different. Osseous-associated CSM is seen predominantly in young adult giant breed dogs. Because the disease is seen at an earlier age, a congenital cause seems likely. Affected dogs have severe, absolute vertebral canal stenosis secondary to proliferation of the vertebral arch (dorsally), articular facets (dorsolaterally), or articular facets and pedicles (laterally) (Fig. 2). The cause of the compression appears to be
Fig. 1. Disc-associated CSM. (Top) Ventral spinal cord compression and nerve root compression at C5-6 caused by intervertebral disc protrusion. Dorsally, hypertrophy of the ligamentum flavum causes mild spinal cord compression. (Bottom) (A) Transverse section at the level of the C4-5 disc region showing normal spinal cord and vertebral canal. (B) Ventral compression at C5-6 region caused by intervertebral disc protrusion and hypertrophy of the dorsal longitudinal ligament (yellow) and ligamentum flavum (causing mild dorsal compression). (C) Asymmetric intervertebral disc protrusion at C6-7 causing spinal cord and nerve root compressions. (Courtesy of The Ohio State University; with permission.)
Fig. 2. Osseous-associated CSM. (Top) (A) Severe dorsolateral spinal cord compression at C2-3 caused by osseous malformation and osteoarthritic changes. (B) Normal C3-4 disc region. (C) Bilateral compression at C4-5 caused by osteoarthritic changes and medial proliferation of the facets, leading to absolute vertebral canal stenosis and foraminal stenosis, respectively. (Bottom) Dorsal spinal cord compression at C3-4 caused by lamina malformation and hypertrophy of the ligamentum flavum. Osteoarthritic changes are also shown at C2-3.

(Courtesy of The Ohio State University; with permission.)
a combination of vertebral malformations and osteoarthritic changes of the articular facets. Even though most giant breed dogs have osseous compressions, occasionally these compressions are complicated by disc protrusion in older dogs. Extradural synovial cysts may also be present secondary to degenerative arthritic facet changes, leading to uni- or bilateral axial compression. Large breed dogs also have purely osseous compressions, but not as commonly as disc-associated compressions.

Ligamentous compression (ligamentum flavum) may be part of the disease in giant and large breed dogs, but pure ligamentous compression as the single source of compression does not appear often.

**Dynamic Compressions**

An important mechanism to explain the development of clinical signs in dogs with either disc- or osseous-associated CSM is the concept of dynamic lesions. Confusion appears in the literature regarding the concepts of instability and dynamic lesions. These 2 concepts are completely distinct. Instability in cervical myelopathies 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.” A dynamic lesion is one that worsens or improves with different positions of the cervical spine. The fact that the spinal cord appears to be compressed on neck flexion or extension on a myelogram does not necessarily mean that there is instability. Variations on the degree of spinal cord compression are expected because it is a physiologic pattern of motion in dogs and humans. Cervical extension in healthy humans causes an 11% to 16% reduction of the area of the vertebral canal due to infolding of the ligamentum flavum, annulus fibrosus, and posterior dura. At the same time, extension increases the spinal cord area by 9% to 17%. This fact explains why cervical extension or dorsiflexion causes worsening of cord compression and clinical signs in dogs. Neck flexion generates the opposite effect in the spinal cord, stretching the cord between C2 and T1 for up to 17.6% of its length in humans, with the maximal stretch occurring in the caudal cervical region. With spinal cord stretch, a ventrally positioned space-occupying lesion, such as a protruded intervertebral disc, will cause more severe ventral spinal cord compression. Continuous flexion and extension of the cervical spine can lead to spinal cord elongation, causing axial strain and stress within the spinal cord, both of which are considered key mechanisms of spinal cord injury in cervical spondylotic myelopathy in humans.

Instability as previously defined is unlikely to be present in dogs with CSM, and the evidence currently available does not support it as a factor in the pathogenesis of CSM. A study compared the amount of intervertebral disc distraction between normal and CSM-affected Dobermans and found no difference between groups. In addition, it appears that restricted, rather than excessive, intervertebral motion is more likely to occur at the sites of disc degeneration. When subjectively evaluated in dogs with CSM, instability was thought to be either absent or rarely present. Nonetheless, specific investigations are needed to define the specific role of dynamic lesions in dogs with CSM, and assess the presence or absence of instability.

In summary, the pathogenesis of CSM involves an association of static and dynamic factors independent of the cause and direction of the compressive spinal cord lesion.

**Distribution and location of compressive lesions**

Two recent studies of 118 dogs with CSM indicated that approximately 50% of large breed dogs with CSM have a single site of spinal cord compression, whereas the other
50% have 2 or more sites of compression. Other studies have found lower prevalence of multiple compressive lesions (approximately 25%). The prevalence of single compressive lesion in large breed dogs contrasts with that seen in giant breed dogs, in which a single site of compression was seen in 20% of dogs, with multiple sites of spinal cord compression observed in 80%. Others have reported an even higher incidence of multiple compressive sites in giant breeds.

The most commonly affected intervertebral region in large breed dogs is C6-7, followed closely by the C5-6. The main lesion is located in 1 of these 2 sites in 90% of dogs. In giant breed dogs, the C6-7 region is also the most commonly affected, followed by C5-6 and C4-5. The main spinal cord compression is located in 1 of these 3 regions in approximately 80% of cases. The remaining 20% have involvement of C2-3 and C3-4 regions. A recent CT study of 58 dogs identified lesions affecting the T1-T2 and T2 regions in 14% of giant breed dogs, and the C7-T1 region in 22% of dogs. Although these lesions were not considered the primary source of compression, their identification is important in the decision-making process of treatment planning. The cranial thoracic spine should therefore always be examined, and the need to investigate this area should be considered when selecting an imaging modality for diagnosis.

**DIAGNOSIS**

**Signalment**

CSM can affect dogs of all ages and breeds, even small dogs, albeit uncommonly. The majority of Dobermans and large breed dogs (Weimaraners, Dalmatians) with CSM are presented after 3 years of age. The mean age for Dobermans with CSM is 6.8 years, whereas for all large breeds it is 7.9 years. Earlier reports described Dobermans younger than 1 year old, but this presentation is currently uncommon. Although most affected Dobermans tend to be middle-aged, Great Danes and giant breeds (Mastiffs, Rottweilers, Bernese, and Swiss Mountain dogs) are usually younger. The mean age of giant breed dogs with CSM is 3.8 years, and the disease may be seen in dogs just a few months old.

The author has recently seen several middle-aged to old German shepherds with osseous-associated CSM. This illustrates that large and giant breeds may have either osseous- or disc-associated forms of the disease, with a great overlap of clinical presentations.

Both males and females are affected by CSM, and many studies report a similar incidence between males and females. Studies having a higher proportion of giant breed dogs usually report a higher incidence in males.

**History and Clinical Signs**

A chronic progressive history (several weeks to months) is typical. Acute presentations are usually associated with neck pain. Occasionally, acute decompensation of a chronic lesion is observed.

Neck pain or cervical hyperesthesia is a common historical finding, but typically is not the main reason for presentation. Neck pain is part of the clinical findings in approximately 65% to 70% of Dobermans, and in 40% to 50% of other breeds, but it is the chief complaint in only 5% to 10% of dogs with CSM. Forceful manipulations of the cervical spine are unnecessary to document the presence of neck pain, and can lead to severe neurologic decompensation. Careful assessment of posture and evaluation of voluntary range of motion (side-to-side, ventrally, and dorsally) using a food treat is recommended to assess cervical pain. Deep palpation of the transverse
processes can also assist in the identification of neck pain. Supraspinatus muscle atrophy is frequently observed in large and giant breed dogs with CSM, and reflects involvement of the suprascapularis nerve or cell bodies of the sixth spinal cord segment. Elbow abduction with internal rotation of the digits ("toe-in posture"), is seen in approximately one-third of Dobermans with CSM (Fig. 3).61

Gait evaluation is the most important component of the examination in dogs suspected of having CSM because it reliably identifies proprioceptive ataxia, even in the absence of conscious proprioceptive deficits.62,63 Proprioceptive ataxia is seen in most dogs with CSM. Dogs with lesions in the cranial or midcervical spine tend to present with ataxia affecting all 4 limbs more equally. However, affected dogs typically have obvious pelvic limb ataxia with milder abnormalities in the thoracic limbs. In some cases, the thoracic limb ataxia or weakness may be very mild in comparison with the pelvic limbs signs, making the thoracic limb abnormalities go unnoticed. The thoracic limb gait can appear short-strided or spastic with a pseudo-hypermetric ("floating") appearance.62 This pseudo-hypermetria is the result of upper motor neuron release causing stiffness/spasticity, and it differs from the true hypermetria whereby the limbs show the "high-stepping" secondary to hyperflexion of the thoracic limb joints. Occasionally, thoracic limb lameness can be seen, suggesting nerve root entrapment. The pelvic limb gait is often wide-based (abducted) and markedly uncoordinated. The stride length of the pelvic limbs is prolonged, causing the swaying movements of the hind end typical of the disease. Scuffing of the pelvic or

Fig. 3. Elbow abduction with internal rotation of digits ("toe-in posture") in a Doberman pinscher dog with CSM.
thoracic limb toes/nails can also be seen. A short-strided thoracic limb gait, with a wide-based, long-strided pelvic limb gait has been called a “2-engine” gait. In general, the faster the dog is walked, the less obvious the abnormalities are, so the dog should be walked at a slow pace. In severely affected dogs, weakness may be pronounced and they may collapse in their pelvic, thoracic, or all 4 limbs. Postural reaction deficits (proprioceptive positioning deficits) are seen in most dogs with CSM, but may not be evident in those with a chronic history despite the presence of proprioceptive ataxia. The reason for this discrepancy is that different tracts carry the pathways for conscious and unconscious proprioception.\textsuperscript{62,63} Mildly asymmetric neurologic signs are seen in approximately 50% of dogs with CSM.\textsuperscript{61} Approximately 10% of dogs with CSM present initially with nonambulatory tetraparesis.\textsuperscript{17,61} Evaluation of the spinal reflexes in dogs with CSM will indicate a lesion located either at C1-5 spinal cord segments (normal to increased spinal reflexes in all 4 limbs, with neurologic signs as described above) or C6-8 spinal cord segments. A C6-8 myelopathy is typical because the osseous and disc lesions are concentrated in the C5-6 and C6-7 regions. In these cases, the gait is affected in all 4 limbs but more severely in the pelvic limbs. Evaluation of the spinal reflexes in the thoracic limbs will show a decreased flexor (withdrawal) reflex indicating involvement of the musculocutaneous nerve from C6-8 spinal cord segments, with normal to increased extensor tone suggesting an upper motor neuron lesion and release of the radial nerve from spinal cord segments C7, C8, T1, and mostly C8-T1. The pelvic limb reflexes will be normal to increased.

**Radiography**

Survey radiographs cannot confirm a diagnosis of CSM but often are used as a screening test to rule out other differential diagnoses for cervical myelopathies such as osseous neoplasia, trauma, vertebral osteomyelitis, and discospondylitis.\textsuperscript{64} Radiographic findings seen in disc-associated CSM are primarily changes in the shape of the vertebral body (assuming a triangular shape in severe cases), narrowing of the intervertebral disc space, and vertebral canal stenosis.\textsuperscript{64} Osteoarthritic, sclerotic changes of the articular facets are the radiographic hallmarks in giant breed dogs with osseous compressions, and can be seen on lateral and ventrodorsal projections (Fig. 4). Some of the radiographic findings seen in dogs with CSM (eg, vertebral

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**Fig. 4.** Radiographs of a 1-year-old Mastiff with CSM. (A) Lateral radiograph. Observe severe osteoarthritic changes in the articular processes of C4-5, C5-6, and C6-7 regions (white arrows). The black arrowhead indicates C6 vertebra. (B) Ventrodorsal radiograph shows medial proliferation of the enlarged/arthritic facets (arrows).
tipping), also are seen in normal dogs. Studies in Dobermans indicate that approximately 20% to 25% of clinically normal dogs have radiographic changes comparable with those seen in dogs with CSM.16,23,28 Cervical vertebral ratios are used routinely in the diagnosis and screening of humans and horses with cervical stenosis.55–67 Two recent studies have revisited the use of vertebral ratios in dogs, more specifically in Dobermans.68,69 Although the methodologies were different, the results of one of these studies indicate that cervical vertebral ratios may be useful as a screening test for dogs with CSM.68

**Myelography**

For many years, and up to recently, myelography has been considered the method of choice to diagnose CSM.70 Myelography defines the site or sites and direction (ventral, dorsal, lateral) of the spinal cord compression, and allows stress myelographic studies.4,64 Lateral and ventrodorsal views should be obtained, and oblique views may increase the diagnostic sensitivity of myelography. Stress myelography is defined as the radiographic examination of the cervical spine in various positions (ventral flexion, dorsal extension, and linear traction). Due to the risk of severe neurologic decompensation after myelography, only traction views are routinely used. For the last 30 years, traction myelographic views have been extensively used to distinguish dynamic from static lesions.4 This differentiation has since been considered fundamental for surgery planning. Surprisingly, guidelines have not been established for performance or interpretation of traction myelography. Some clinicians indicate that a dynamic lesion is one that improves with traction, whereas others consider a lesion dynamic when it reduces completely.71 The technique of traction also has never been standardized. The original description of grasping and holding the dog’s head violates radiation safety and cannot be standardized.4 A recent study proposed using a cervical harness and traction forces not greater than 25% of the dog’s weight.61 Comparative myelographic and MRI studies have shown how the concept of static and dynamic lesions is subjective.61 A lesion that appears dynamic on myelography may appear static on MRI (Fig. 5). It appears that any compressive lesion will improve with traction (Fig. 6).61 Others have also indicated that the concept of dynamic and static lesions is highly subjective and dependent on personal opinion.71 As such, caution should be used when trying to apply this concept clinically. In dogs with multiple sites of spinal cord compression, it may be difficult to establish the clinically relevant site on myelography. Postmyelographic seizures and temporary deterioration of the patient’s neurologic status are important adverse effects in large and giant breed dogs with CSM.72–74

**Computed Tomography**

CT is a rapid test that allows visualization of transverse sections of the cervical spine. CT is used mostly after myelography in dogs with CSM, and has been found to be complementary to standard myelography in the evaluation of Dobermans with CSM.60 CT provides superior visualization of the direction and severity of the spinal cord compression (Fig. 7) and more precise identification of the most severely affected site when compared with myelography alone. An advantage of CT myelography is the visualization of spinal cord atrophy,60 which has been identified in CT myelographic studies of 20%–30% of dogs with CSM.38,60 Atrophy is identified on CT myelography as a widening of the subarachnoid space surrounding the spinal cord, with the cord assuming a triangular shape.60 It is currently unknown whether spinal cord atrophy is associated with a poor prognosis in affected dogs. Disadvantages
Magnetic Resonance Imaging

MRI is the gold standard test for evaluation of dogs suspected of having CSM. The main advantage of MRI is that it detects signal changes in the spinal cord and thus allows assessment of the spinal cord parenchyma.11,61 These signal changes are seen in approximately 50% of dogs with CSM, and allow precise identification of the site most severely affected.39 A recent study compared myelography and MRI in the diagnosis of CSM and concluded that MRI was more accurate in predicting the site, severity, and nature of spinal cord compression (see Figs. 5 and 6).61

The presence of spinal cord signal changes, namely hyperintensity on T2-weighted images (T2WI), seems to be associated with severity of clinical signs, seen most commonly in dogs with moderate to severe neurologic deficits and a chronic history.61 Although not yet documented in naturally occurring canine CSM, the correlation of spinal cord signal changes on MRI and histopathology has been well described in...
Hyperintensity in T2WI and isointensity in T1WI were characterized by slight loss of nerve cells, gliosis, and edema in the gray matter, as well as demyelination, edema, and Wallerian degeneration in the white matter. The combination of T2 hyperintensity and T1 hypointensity was characterized by severe lesions such as necrosis, myelomalacia, and spongiform changes in the gray matter, as well as white matter necrosis. Hyperintensity on T2WI does not appear to correlate with prognosis in dogs, but preliminary evidence suggests that the combination of hyperintensity on T2WI and hypointensity on T1WI may be associated with a worse prognosis. Current evidence in humans suggests that multilevel hyperintensity on T2WI and hypointensity on T1WI are associated with a poorer prognosis.
The observed MRI changes are dependent on the cause of spinal cord compression. Osseous compressions are observed as hypointense proliferations associated with the articular processes, lamina, and pedicles in both T1- and T2-weighted images. Transverse and dorsal images are useful to assess the degree of vertebral canal stenosis in these cases. On disc-associated CSM, disc hypointensity or isointensity relative to the vertebral bodies on T2WI is seen along with variable degrees of protrusion. Even when not clearly visible on imaging, relative vertebral canal stenosis is present in dogs with disc-associated CSM. The disc-associated compression leads to ventral spinal cord compression and displacement. Intervertebral foraminal stenosis can be an important cause of cervical spinal pain, and it is important to evaluate the foraminal diameter in all dogs with CSM. T2WI are more useful for assessment of foraminal size. Synovial spinal cysts may be seen in giant breed dogs as hyperintense regular areas associated with the articular processes on T2WI.

In some cases, the degree of spinal compression is minimal relative to the severity of clinical signs. Dynamic spinal cord compressions are assumed to be present in such cases. Testing for dynamic compression using traction MRI can be performed, and guidelines for testing have been published.

Cerebrospinal Fluid Analysis

Cerebrospinal fluid abnormalities are uncommon and nonspecific in dogs with CSM. Mild pleocytosis (<12 cells/µL) was seen in approximately 20% of dogs with CSM, mainly in those with acute onset of signs. Mild increases in protein concentration (<40 mg/dL) also were observed in 27% of dogs with CSM, primarily in dogs with a chronic history.

Electrodiagnostics

Electromyography (EMG) may be used to document neurogenic muscle atrophy in dogs with supraspinatus muscle or thoracic limb atrophy. However, the EMG may be normal in these dogs even when obvious muscle atrophy is present. Transcranial magnetic motor-evoked potentials (MEPs) are a sensitive way to assess spinal cord function. All imaging modalities provide anatomic, but not functional information. MEPs of the cranial tibial muscle were shown to correlate with neurologic and MRI findings in dogs with CSM. An advantage of the MEP is that is a rapid test that
can be performed under mild sedation. Transcranial magnetic MEPs can provide an objective way to assess treatment outcome in dogs with CSM. Additional Diagnostic Tests

Many large breed dogs suffer from concurrent medical conditions that can potentially increase anesthetic or surgical risk, or affect long-term prognosis. The following tests should be considered in addition to the minimum database and previously mentioned tests:

Thyroid function: Hypothyroidism is very common in Doberman pinschers and has been identified in a high percentage of dogs in association with CSM. Hypothyroidism can interfere with neurologic function and anesthetic recovery.

von Willebrand status: Deficiency of von Willebrand factor can lead to severe hemorrhage. A prevalence of 73% of von Willebrand disease has been found in Dobermans. Buccal mucosal bleeding time is a rapid and efficient method to evaluate von Willebrand status.

Cardiac function: Electrocardiogram (5-minute ECG or Holter monitor) and echocardiogram are recommended before surgical treatment. General anesthesia can worsen cardiac function and lead to decompensation in dogs with occult dilated cardiomyopathy.

Differential Diagnosis

Other differentials should be considered in large and giant breed dogs with neck pain, tetraparesis, and/or proprioceptive ataxia. Many neurologic diseases can cause at least one of these signs, and the primary differentials to consider are spinal neoplasia, intervertebral disc disease, trauma, discospondylitis, vertebral osteomyelitis, meningitis or meningoarachnitis, synovial or subarachnoid spinal cysts, fibrocartilaginous embolic myelopathy, and polyneuropathies or polymyopathies. Orthopedic conditions frequently occur in older large and giant breed dogs and can coexist with neurologic disturbances, but no matter how severe a musculoskeletal disease may be, it will never cause proprioceptive ataxia.

TREATMENT

Conservative (Medical) Treatment

Traditionally, medical treatment for CSM has been considered a temporary measure to alleviate clinical signs. Without surgery, the disease was thought to be progressive, and euthanasia would have to be contemplated. The only evidence to support these statements came from a study of primarily Great Danes that essentially received no treatment, more than 30 years ago. Medical management for CSM was recently revisited in 2 studies. A study compared the outcome of dogs treated medically and surgically and found that 54% of dogs treated medically improved and 27% were unchanged in a long-term follow-up. Therefore, the clinical signs of CSM are either improved or stable in 81% of dogs managed medically. Of note, the overall percentage of improvement and the owner’s perception of the dog’s quality of life were similar between dogs treated medically and those treated surgically. Although many surgical techniques offer a higher success rate (approximately 80%), the index of improvement or stabilization of clinical status seen with medical management is quite acceptable, and may be preferred by some owners because of financial constraints or concerns about anesthetic and surgical risks, predominantly in Dobermans due to the high incidence of dilated cardiomyopathy in the breed.
A key component of medical management is exercise restriction to minimize high-impact activities that would exacerbate the dynamic component of spinal cord compression. Dogs can be leash walked but free, unsupervised activity is strongly discouraged. A body harness should be worn instead of a neck collar.

Corticosteroids appear to benefit dogs with CSM, and anti-inflammatory dosages of prednisone often are used (0.5–1.0 mg/kg every 12–24 h), progressively tapering the dosage over the course of 2 to 3 weeks, even though no scientific evidence is available to support their use. In some patients, dexamethasone appears to elicit a better response, and so can be used for more severely affected patients or as a rescue therapy for dogs with sudden deterioration. Only low doses of dexamethasone should be used, never more than 0.25 mg/kg every 24 hours. No therapeutic benefit is gained by the higher dosage, and the risk of adverse effects is higher. The severe complications reported with dexamethasone use were seen mainly when much higher dosages were used (1–2 mg/kg/d). When using dexamethasone, the author uses a dosage of 0.2 to 0.25 mg/kg every 24 hour (avoiding doses higher than 8 mg/dog) initially for 1 to 3 days, depending on the severity of clinical signs, and then continues with 0.1 mg/kg every 24 hours. Corticosteroids, particularly dexamethasone, improve neurologic function in chronic spinal cord compression predominantly by decreasing vasogenic edema. Other proposed mechanisms include protection from glutamate toxicity and reduction of neuronal and oligodendroglial apoptosis. Despite the potential benefits associated with corticosteroid therapy, the use of corticosteroids, particularly for long periods, can be associated with important adverse effects. Gastrointestinal ulceration, colonic perforation, iatrogenic hypoadrenocorticism, pulmonary thromboembolism, risk of infections, overt diabetes mellitus, and behavioral changes all have been associated with corticosteroid treatment. Due to the possibility of gastrointestinal complications, omeprazole (0.7 mg/kg every 24 hours) or famotidine (0.5 mg/kg every 12–24 hours) often are used in conjunction with corticosteroid therapy. Nonsteroidal anti-inflammatory drugs (NSAIDs) can be used in place of corticosteroids if neck pain appears to be a main component of the syndrome or if the adverse effects of the corticosteroids cannot be tolerated. Although many NSAIDs can be effectively used, the author often uses meloxicam (0.2 mg/kg initially, followed by 0.1 mg/kg every 24 hours). Independent of the NSAID used, corticosteroids and NSAIDs should never be used in combination. The response to medical management (corticosteroids and exercise restriction) can be used to indirectly assess the degree of reversible spinal cord lesions.

One reason for the success with medical management is the slow progression of spinal changes associated with the disease (Figs. 8 and 9). Surviving demyelinated axons also may remyelinate with treatment. Remyelination has been shown in the spinal cords of horses and humans with cervical myelopathy treated medically.

Physical therapy also has been reported in the treatment of dogs with severe cervical myelopathies, and can also be used in the treatment of dogs with CSM. A study also described the use of electroacupuncture in the treatment of CSM. Anecdotally, protein and calorie reduction have been used in the treatment of CSM in young dogs with osseous lesions. No controlled studies are currently available, but as nutrition seems to have a questionable role in CSM, the efficacy of this therapy is uncertain.

As previously mentioned, some dogs with CSM have concurrent hypothyroidism (8 out of 12 dogs in a recent study). Hypothyroid dogs with CSM may show remarkable improvement in strength and energy when thyroid supplementation is started. In these cases, improvement usually is noticeable within a week.
SURGICAL TREATMENT

The decision to recommend surgical treatment should be based on several factors such as severity of neurologic signs, degree of pain, type and severity of compressive lesion(s), response (or lack of response) to medical management, short- and long-term expectations of the owner, and presence of other concurrent neurologic or orthopedic problems or extraneurologic diseases such as dilated cardiomyopathy that would affect the long-term outcome. Once a decision has been made that surgery is the ideal method of treatment, the selection of the specific method of surgical treatment to be used can be complicated. Few diseases in veterinary medicine have had so many proposed surgical techniques as CSM. Direct decompressive techniques reported include dorsal laminectomy, dorsal laminoplasty, ventral slot, inverted cone slot, and hemilaminectomy. Indirect decompressive techniques typically are grouped into the distraction-stabilization category, and have been reported using bone grafts of several types, pins (smooth, threaded) or screws and polymethyl methacrylate (PMMA), interbody screws, washers, metallic spacers, metallic plates, plastic

Fig. 8. MR images of the cervical spine of a 5-year-old male Doberman with CSM treated conservatively. (A) Midsagittal T2W image showing spinal cord compression and mild cord hyperintensity at C6-7. (B) Midsagittal T2W image obtained 15 months after the first MRI. The area of spinal cord hyperintensity is still present, and adjacent discs do not show degenerative changes. Clinically the dog had improved and was stable. The spinal cord compression appears less severe but the dog actually had spinal cord atrophy on the follow-up MR images. (From da Costa RC, Parent JM. One-year clinical and magnetic resonance imaging follow-up of Doberman Pinschers with cervical spondylomyelopathy treated medically or surgically. J Am Vet Med Assoc 2007;231(2):247; with permission.)
plates, k-wire spacer, Harrington rods, interbody PMMA plug, and fusion cage. All of these techniques have been combined either with discectomy or with partial or complete ventral slots.\textsuperscript{12,14,15,57,59,70,84,106–118} Intervertebral disc fenestration also has been used, and more recently, motion-preserving techniques, using disc arthroplasty or artificial disc replacement, have been proposed.\textsuperscript{119–121} With so many choices, the decision on which technique to use may be difficult.

In general, as the source and direction of compression can be broadly divided into disc-associated or osseous-associated, treatment recommendations can be made as follows.

**Disc-Associated CSM**

Disc-associated CSM is the most common form of CSM and the one with the largest number of surgical techniques proposed to treat it. Many, if not most, of the surgical techniques have been based on the concept of static or dynamic lesions following stress or traction myelography which, as discussed in the diagnosis section, is highly subjective. Nevertheless, the outcome for most surgical techniques is quite similar and generally positive.\textsuperscript{8} Ventral static compressions are usually treated with the traditional ventral slot or the inverted cone slot. Dynamic compressions can be treated with distraction-stabilization techniques, and the PMMA plug or pins/screws combined with PMMA, are commonly used. Multiple compressive sites can be treated with distraction-stabilization techniques, the most common being distraction with a PMMA plug.\textsuperscript{70} Dorsal laminectomy is an alternative for multiple ventral compressions.\textsuperscript{103}

**Osseous Compressions**

These compressions typically are thought to be primarily static and, as such, direct decompression of the affected sites is recommended\textsuperscript{105}; this is typically achieved by dorsal laminectomy, but can also be achieved by cervical hemilaminectomy.\textsuperscript{58,104}

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**Fig. 9.** Transverse T2W MR images of a 4-year-old male Doberman with CSM with mild ataxia and neck pain treated conservatively. (A) Bilateral spinal cord deformation caused by medial proliferation of the articular processes is observed (arrows). (B) MR image obtained 12 months after the first MRI. The spinal cord compression was unchanged. Clinically the dog still had mild ataxia but no neck pain. Identical cord area to the first MRI was confirmed by morphometry. (From da Costa RC, Parent JM. One-year clinical and magnetic resonance imaging follow-up of Doberman Pinschers with cervical spondylomyelopathy treated medically or surgically. J Am Vet Med Assoc 2007;231(2):246; with permission.)
Another way to treat osseous lesions is by distraction-stabilization of the affected segments ventrally. Stabilization and fusion of the affected segments does not directly decompress the affected sites, but eliminates the dynamic component of the spinal cord compression. It also may allow regression of the osseous and ligamentous lesions over time. The technique used in these cases was the PMMA plug.

**Pure Ligamentous Compressions**

Ligamentous hypertrophy is usually combined with either disc- or osseous-associated compressions. Pure ligamentous compression (hypertrophy of the ligamentum flavum) is currently a rare presentation. Surgical treatment can be achieved either by decompressing the affected sites (dorsal laminectomy) or by using the PMMA plug technique.

**Surgical Techniques**

**Direct decompressive techniques**

**Ventral slot** Ventral slot is primarily indicated for single ventral static compressions. As the determination of a static or dynamic lesion is subjective, it can be considered for any ventral compression, as there are no data indicating that it is less successful or carries a worse outcome with dynamic lesions. Two slots (preferably using the inverted cone technique) can be performed but the risk of complications is higher. Ideally, the slot should not exceed one-third of the length and width of the vertebral bodies (Fig. 10). Care should be exercised to avoid injuring the internal vertebral venous plexus, which can cause very severe hemorrhage. All disc protrusion and ligament hypertrophy should be removed to effectively decompress the spinal cord. The ventral slot technique offers adequate spinal cord decompression, and fusion is expected to occur at the slot site 8 to 12 weeks postoperatively (Fig. 11). Serious complications can occur after a ventral slot procedure, including respiratory compromise, cardiac dysrhythmias, vertebral subluxations, and hemorrhage. These complications were reported to occur in 14.9% of dogs in one study. The reported long-term success rate of the ventral slot procedure is 72%. An alternative to the traditional ventral slot procedure is the inverted cone technique (see Fig. 10C). This modification aims at minimizing bone removal, and therefore reducing the risk of vertebral subluxation, and hemorrhage. The decompression window resembles an inverted cone in which the base of the cone lies adjacent to the ventral vertebral canal, allowing maximal surgical access cranially, caudally, and laterally.

**Dorsal laminectomy** Dorsal laminectomy is indicated for dorsal compressions associated with osteoarthritic changes of the articular facets, lamina malformation, or ligamentum flavum hypertrophy. It can be used to decompress one or multiple sites (Fig. 12). Dorsal laminectomy also has been recommended to treat multiple ventral spinal cord compressive lesions. No direct comparisons of dorsal laminectomy with other surgical techniques have been published. An important complication of dorsal laminectomy is worsening of neurologic status, which has been reported to occur in 70% of treated dogs postoperatively. Dogs that became or remained non-ambulatory after surgery took an average of 2.5 months to recover their ability to walk without assistance. Postlaminectomy membrane is a major problem after decompressive spinal surgery in humans, being responsible for 8% of all failures of spinal surgery. Although reported in one dog after dorsal cervical laminectomy, its true incidence is unknown. Typically a free fat graft is placed over the laminectomy defect to minimize the...
development of a postlaminectomy membrane. However, a study in dogs indicated a high incidence of graft failure and neurologic complications after the use of a free fat graft in the thoracolumbar spine.\textsuperscript{126} Whether or not the same occurs in the cervical spine, explaining the common neurologic deterioration seen postoperatively, is unknown at this time. Considering the high incidence of graft failure and the neurologic complications documented in dogs, the use of free fat grafts after dorsal laminectomy is not recommended at this time.

The success rate for dorsal laminectomies ranges from 79% to 95%.\textsuperscript{11,58,103} The mean time to reach optimal improvement is long (3.6 months), and in one report, 30% of dogs had recurrence of signs postoperatively.\textsuperscript{58} Dorsal laminoplasty is an alternative to dorsal laminectomy that has not been sufficiently explored in dogs.\textsuperscript{105}

**Cervical hemilaminectomy** A technique of lateral approach for cervical hemilaminectomy was used in the treatment of CSM in dogs with either disc- or

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**Fig. 10.** Ventral slot at C6-7. (A) Ventral view. (B) Transverse view. The slot width and length should ideally be kept at about one-third of the vertebral bodies. The internal vertebral venous plexus are represented by the blue vessels running in the ventral aspect of the vertebral canal. (C) Inverted cone slot; this is a modification of the traditional ventral slot that minimizes the risk of hemorrhage and subluxation. (Courtesy of The Ohio State University; with permission.)
The investigators reported no postoperative worsening of neurologic status, which is an attractive benefit for dogs with osseous lesions.\textsuperscript{104}

**Indirect decompression: vertebral distraction techniques**

**Pins and PMMA** This technique is recommended primarily for single ventral dynamic compressive lesions.\textsuperscript{107} It can be used for 2 affected segments, but the risk of failure

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**Fig. 11.** Magnetic resonance (MR) images of the cervical spine of a 4-year-old female Doberman dog with CSM treated with ventral slot decompression at C5-6. (A) Initial MR images show ventral spinal cord compression at C5-6 (arrow). (B) Follow-up MR image 12 months after the ventral slot shows effective decompression with residual compression. Bone proliferation at the slot site suggests that fusion developed at that site (arrow). Clinically the dog had improved after surgery and was stable.

**Fig. 12.** Dorsal laminectomy from C4 to C7. (Courtesy of The Ohio State University; with permission.)
increases. Either a partial or complete ventral slot is created. The metal implant can be either 2 Steinmann pins, threaded pins, or bone screws (Fig. 13). A recent study suggested that positive profile pins provide more rigid fixation than smooth pins. The original recommended angle of implant insertion was 30° to 35°. A recent study indicated that these angles are relatively safe for the vertebrae C5 and C6, but that an angle of approximately 45° should be used for implants at C7, which is very challenging at this location. The long-term success rate of this technique is 73%. Vertebral or transverse foramina penetration is a major risk of this procedure, and has been reported to occur in 25% to 57% of cases studied experimentally.

Several modifications of this procedure have been reported using different techniques and materials for distraction. A recent study reported the use of screws inserted into the transverse processes, using a U-shaped Steinmann pin that was wired to the screws and covered with PMMA. The advantage of this technique is that it avoids the risk of penetration into the vertebral canal or transverse foramina. A disadvantage of some of the distraction techniques is that the instrumentation used for vertebral body fixation can cause severe imaging artifacts, predominantly with MR imaging, making postoperative spinal cord visualization difficult or impossible.

**Distraction using the PMMA plug** Distraction using the PMMA plug is a popular technique and has been used for either single or multiple ventral and dorsal compressions,
static and dynamic. The original report focused on ventral disc-associated dynamic lesions, and the investigators did not recommend the technique for dorsal or ventral static lesions. However, others have indicated that it can be used for up to 3 ventral sites and can also be indicated to treat sites with dorsal osseous compressions. A discectomy is performed, traction is applied, and multiple holes (including an anchor hole) are drilled into the cranial and caudal end plates before application of PMMA (Fig. 14). Modifications of this technique have included a retention screw or pin ventral to the plug, and its application combined with a complete ventral slot. The reported long-term success with the PMMA plug technique is 82%.

**Locking plate**

Locking plates have been used with either partial ventral slots or discectomies. Screws of the locking plates are less likely to loosen than are conventional screws, and can be inserted monocortically with sustained stability, thus decreasing the risk of vertebral canal penetration and spinal cord injury. A variety of bone grafts was used in the reported cases including cancellous autograft, cortical allograft with cancellous autograft, and cancellous bone block graft. The success rate based on the follow-up information of 3 cases series is 73%. An important disadvantage of this system is the cost of the plate and screws.

**Motion-preserving techniques**

**Cervical disc arthroplasty**

Disc arthroplasty is an area of intensive investigation in humans with cervical spondylotic myelopathy. Fusion or distraction may increase the risk of adjacent segment disease or “domino” lesion by altering the biomechanics of the adjacent segments. Disc arthroplasty allows reestablishment of the normal disc space with motion preservation of the affected intervertebral segment.

![Fig. 14. The PMMA plug distraction technique. The PMMA plug is in place (gray). Two anchor holes in the cranial and caudal endplates prevent plug displacement. The dorsal annulus is left intact. Multiple small holes should be drilled in the ventral aspect of the vertebral bodies to promote incorporation of the cancellous bone graft (dark red) and fusion. (Courtesy of The Ohio State University; with permission.)](image-url)
An artificial disc was recently introduced for treatment of CSM in dogs.\textsuperscript{120,121} The technique was combined with complete ventral slot allowing direct spinal cord decompression. Long-term results of large patient series are not yet available.

\textbf{Complications}

Several complications, including death, can occur with surgical treatment of CSM. The mean mortality rate associated with decompressive surgery on the cervical spine of 771 reported cases was 3\% (range 0\%–6.3\%).\textsuperscript{124,131–135} Common complications are postoperative worsening of the neurologic status (which is challenging in large and giant breed dogs), penetration of the vertebral canal or transverse foramina with implants, and implant failure, ranging from 7.5\% to 30\%.\textsuperscript{12,14,15,57,59,70,71,84,106–118}

\textbf{Adjacent segment disease or “domino” effect}

“Domino” effect or adjacent segment disease is a late postoperative complication after surgical treatment of CSM that occurs in approximately 20\% of dogs after surgery, mainly with distraction-stabilization techniques.\textsuperscript{8,112,136} Ventral slot techniques reportedly decrease the risk of “domino” lesion.\textsuperscript{8,71} The domino effect may occur secondary to bone fusion.\textsuperscript{70,106} Evidence in humans, however, suggests there is no difference in the incidence of adjacent segment syndrome in sites with or without fusion.\textsuperscript{137} The domino effect typically affects only one disc region, either cranial or caudal to the operated area.\textsuperscript{15,64,106,136} However, a recent study documented involvement of 3 sites after lack of fusion after ventral slot (Fig. 15).\textsuperscript{76} Experimentally, ventral slots always developed bone proliferation and fusion 2 to 3 months after surgery.\textsuperscript{101,123} Apparently this does not always occur in clinical patients, which then can lead to postoperative instability.\textsuperscript{76} Motion-preserving techniques potentially could decrease the incidence of domino lesions by preserving the local biomechanics, but this still needs to be proved. Questions have been raised about whether these domino lesions are simply part of the natural history of CSM.\textsuperscript{8} A recent study suggests that they are in fact a surgically induced phenomenon. A 1-year follow-up MRI study found no evidence of adjacent lesions in 9 dogs treated medically, whereas 2 of 3 dogs treated with ventral slots developed spinal cord lesions in the sites adjacent to surgery.\textsuperscript{76} Long-term MRI follow-up investigations of other surgical techniques have not yet been reported.

\textbf{Outcome and Prognosis}

The outcome of surgical treatment of disc-associated CSM is usually successful, with approximately 80\% (70\%–90\%) of dogs improving after surgery.\textsuperscript{8,17} No surgical technique stands out as being clearly superior, even for dogs with disc-associated CSM. Intervertebral disc fenestration is not recommended because the reported success rate was only 33\%.\textsuperscript{119} In contrast to older studies, new reports on medical management indicate an improvement rate of approximately 50\% (45\%–54\%).\textsuperscript{17,88} Several factors were recently investigated to assess if they would be associated with a successful or unsuccessful outcome after surgical or medical treatment in 104 dogs with CSM. Age, duration of clinical signs, presence or absence of neck pain, severity of ataxia, nonambulatory status, and location of the lesion all were deemed nonsignificant.\textsuperscript{17}

Considering the success rate of surgical and medical treatments for CSM, surgery more consistently leads to clinical improvement, and should always be considered in the treatment of dogs with CSM. Surgery, however, does not alter the long-term survival of dogs with CSM. The survival time of 76 dogs with CSM (33 dogs treated
surgically and 43 dogs treated medically) was reported recently. The median survival time of dogs with CSM was identical (36 months), regardless of whether the dog was treated medically or surgically.\textsuperscript{17} This finding indicates that CSM continues to progress independent of the method of treatment, and that the clinical deterioration seen months to years after treatment may not be due solely to failure of the surgery or development of adjacent segment disease, but also may occur secondary to other mechanisms, such as ischemia, apoptosis, or other molecular changes within the spinal cord.\textsuperscript{30}

**Fig. 15.** Magnetic resonance (MR) images of the cervical spine of a 7-year-old Doberman pinscher with CSM treated with ventral slot decompression at C6-7. (A) Midsagittal T2W image shows spinal cord compression and cord hyperintensity at C6-7. (B) Midsagittal T2W image obtained 14 months after the first MRI. Spinal cord compression is no longer visible at C6-7, but cord hyperintensity is more evident (arrowhead). There are new areas of spinal cord compression at C5-6 and at C3-4 and C4-5 dorsally (long arrows). Mild cord hyperintensity can be seen associated with the compression at C5-6 (short arrow). (C) Follow-up midsagittal T1W image. Observe an area of hypointensity within the spinal cord at C6-7 (arrowhead), minimal bone proliferation between the vertebral bodies of C6-7, and the intermediate signal of the structures compressing the spinal cord dorsally (long arrows) and ventrally (short arrow). Apparently bone fusion did not occur at this site. Contrast the images of this dog with the dog in Fig. 11. (D) Transverse T2W image at the level of C6-7 before surgery. (E) Follow-up transverse T2W image. Adequate cord decompression is seen with marked cord hyperintensity and atrophy. C6, C7, sixth and seventh cervical vertebrae. (From da Costa RC, Parent JM. One-year clinical and magnetic resonance imaging follow-up of Doberman Pinschers with cervical spondylomyelopathy treated medically or surgically. J Am Vet Med Assoc 2007;231(2):245; with permission.)
SUMMARY

Many advances have been made in the diagnosis and treatment of cervical spondylomyelopathy in recent years. However, much is still unknown on the mechanisms causing the disease. Molecular investigations aiming to unveil the causes of the CSM are needed to enable us to prevent rather than only treat the disease. Newer surgical techniques are proposed continuously, but the criteria for patient evaluation and outcome assessment often do not allow meaningful comparisons among different surgical techniques. Objective inclusion criteria and valid methods of outcome assessment are needed to facilitate assessment of treatments for CSM. Routine MRI investigation of dogs that deteriorate postoperatively is also important to address the mechanisms leading to late-onset postoperative deterioration. This could lead to refinement of treatment strategies and improvement in survival times beyond those currently available.

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