The pathogenesis of cervical spondylomyelopathy (CSM) involves both static and dynamic factors. Traditionally, spinal cord compression was thought to be a key factor leading to the signs of CSM. Interestingly, two recent magnetic resonance imaging (MRI) studies found that 25 to 30% of clinically normal Dobermans have clinically silent spinal cord compression. 1, 2 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). 1 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.1, 3, 4

A key mechanistic difference between normal and CSM-affected Dobermans that explains why disc-associated spinal cord compression does not necessarily lead to neurological signs is the vertebral canal stenosis. 1 Vertebral canal stenosis was consistently present throughout the entire cervical spine of CSM-affected Dobermans, even at C2 and C7-T1 regions. 1 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. 5 In humans a smaller vertebral canal is considered the most important static factor for the development of cervical spondylotic myelopathy. 6-8 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 neurological signs) or a relative vertebral stenosis, which by itself does not lead to myelopathic signs, but predisposes the patient to develop myelopathy. 1 Despite some degree of overlap, the pathophysiology of the spinal cord compressions can be divided into osseous or disc-associated compression.9

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 symmetrical or asymmetrical. 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. 10 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. 11 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 three times more torsion than the cranial cervical spine,12 confirming the findings of a morphometric study. 13 Torsion is the main biomechanical force leading to intervertebral disc degeneration in nonchondrodystrophic dogs, more so than axial compression.14 Additionally, a recent study found that Dobermans with CSM have larger intervertebral discs compared with clinically normal Dobermans. 1 This difference would cause a larger volume of disc protrusion into the vertebral canal. Therefore, three 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 appears 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). The cause of the compression appears to be 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 are uncommon.

**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 two 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 upon 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 physiological 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 ligament flavum, annulus fibrosus, and posterior dura. At the same time, extension increases the spinal cord area by 9 to 17%. This 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. \(^{33-35}\) When subjectively evaluated in dogs with CSM, instability was thought to be either absent or rarely present. \(^{36, 37}\) Nonetheless, specific investigations are needed to define the specific role of dynamic lesions in dogs with CSM, and to assess the presence 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.

References