Review

Current insights and controversies in the pathogenesis and diagnosis of disc-associated cervical spondylomyelopathy in dogs

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Disc-associated cervical spondylomyelopathy (DA-CSM) is the most common cause of cervical spondylomyelopathy in dogs. In this condition, progressive caudal cervical spinal cord compression is typically caused by protrusion of one or more intervertebral discs. This disc-associated compression is sometimes seen in combination with mild vertebral abnormalities and dorsal compression resulting from ligamentum flavum hypertrophy. The intervertebral disc space between the sixth (C6) and seventh (C7) cervical vertebrae is most commonly affected. Although several large breed dogs can be affected, the adult to older dobermann is overrepresented. Clinical signs vary from cervical hyperaesthesia to tetraplegia. Dogs can present with a chronic progressive or an acute onset of clinical signs. Many aspects of this multifactorial neurological syndrome are not completely understood and are the subject of controversy and debate. Although several factors have been proposed, the underlying pathology and aetiology remain unknown. Recently, new insights have been gained in the pathogenesis, diagnosis and treatment of this challenging neurological syndrome. This review outlines current controversies and new developments concerning the pathogenesis and diagnosis of DA-CSM.

CERVICAL spondylomyelopathy (CSM) is a complex and incompletely understood neurological syndrome. It can be considered to be a collection of disorders causing cervical spinal cord compression, mainly affecting large and giant breed dogs. Many lesions have been attributed to this disorder (Sharp and Wheeler 2005), leading to a multitude of reported terms to refer to this neurological syndrome (Parker and others 1973, Trotter and others 1976, Read and others 1983, Lippsitz and others 2001). Based on signalment and imaging findings, more separate syndromes have been recognised over the years (Jeffery and McKee 2001, McKee and Sharp 2003). It is unclear if these reflect distinct disease entities or spectra of the same disorder. Probably the most common and typical of these syndromes is disc-associated cervical spondylomyelopathy (DA-CSM), also commonly referred to as disc-associated wobbler syndrome (Van Gundy 1988, Jeffery and McKee 2001).

Unfortunately, the term DA-CSM is not strictly defined. It is generally considered a multifactorial disorder in which caudal cervical spinal cord compression is caused by protrusion of one or more intervertebral discs. This is sometimes seen in combination with vertebral abnormalities and ligamentum flavum hypertrophy (Van Gundy 1988). The intervertebral disc spaces between the sixth and seventh (C6-C7) and the fifth and sixth (C5-C6) cervical vertebrae are most often affected (da Costa and others 2012). It is currently unclear if a diagnosis of DA-CSM should be reserved for dogs with caudal cervical intervertebral disc protrusion(s) in combination with other imaging abnormalities or also for dogs of particular dog breeds with one or more caudal cervical intervertebral disc protrusions without further obvious imaging abnormalities. This ambiguity might complicate the comparison of studies with different inclusion criteria. A diagnosis of DA-CSM can be established by myelography, CT myelography or MRI (Sharp and Wheeler 2005). Classically, it has been considered that approximately 20 to 25 per cent of dogs present with more than one site of spinal cord compression (McKee and Sharp 2003). However, more recent studies suggest that 59 to even 50 per cent of dogs have more than one site of spinal cord compression at the time of diagnosis (De Decker and others 2009, da Costa and others 2012). Although recent studies have provided new insights into the pathogenesis, diagnosis and treatment of this controversial neurological syndrome, many questions remain unanswered. This review will focus on our current understanding of and controversies in the pathogenesis and diagnosis of DA-CSM.

Aetiology and pathogenesis

Although several factors have been proposed, the exact aetiology and pathogenesis of DA-CSM remains unknown.

Given the breed predisposition for the dobermann, an underlying genetic aetiology has been suggested (Wright and others 1973, Selcer and Oliver 1975, Mason 1977). Research groups, evaluating the pedia-
gree information of dobermann populations in England (Lewis 1991) and New Zealand (Burbidge and others 1994), did not find conclusive evidence for a hereditary basis. However, the more recent unravelling of the canine genome has created a large opportunity to evaluate disease-related genes in dogs (Lindblad-Toh and others 2005), including candidate gene sequencing, linkage mapping and genome-wide association studies (Karlsson and others 2007). Therefore, until well-designed studies have been performed, little evidence exists to accept or reject the hypothesis of an underlying or contributing genetic predisposition. The overrepresentation of the dobermann has further led to the hypothesis of possible breed-specific conformational factors. Large heads in combination with long, straight necks (Lewis 1989) and cervical hyperextension (Sharp and others 1995) have been suggested as possible risk factors for the development of DA-CSM. However, no correlation was found between several conformational dimensions and radiographic evidence of CSM in adult dobermanns (Burbidge and others 1994). Although nutritional factors have also been suggested as a possible cause for CSM (Hazewinkel and others 1985), there is currently little evidence to support this hypothesis (Burbidge and others 1999).

**Predisposition of spinal cord compression in the caudal cervical vertebral column**

Large breed dogs have a tendency to demonstrate the degeneration and protrusion of the more caudal cervical intervertebral discs (Cherrone and others 2004). The predilection for caudal cervical intervertebral disc degeneration in large breed dogs can be explained by the biomechanical properties of the cervical vertebral column (Breit and Kunzel 2002, Johnson and others 2011). More concave shaped caudal articular facets have been demonstrated in the cervical spine of large breed compared to small breed dogs. Additionally, more concave-shaped caudal articular facet joints have been demonstrated in the caudal compared to the cranial cervical vertebral column (Breit and Kunzel 2002). Concave-shaped articular facet joints allow more axial rotation (Breit and Kunzel 2002), which has been suggested as an important biomechanical force contributing to intervertebral disc degeneration (Farfan and others 1970). These findings were confirmed recently in a biomechanical study, demonstrating a higher amount of axial rotation in the caudal compared to the cranial cervical vertebral column in large breed dogs (Johnson and others 2011).

Recent imaging studies have demonstrated cervical spinal cord compression in 25 to 50 per cent of clinically normal dobermanns and English foxhounds, two breeds with a similar body conformation (da Costa and others 2006a, De Decker and others 2010). Spinal cord compression together with other imaging abnormalities, commonly seen in dogs with DA-CSM, did not occur more frequently in the group of clinically normal dobermanns compared to English foxhounds (De Decker and others 2010). Since none of the studied dogs developed clinical signs during a variable follow-up period, it was suggested to consider these abnormalities in clinically normal dogs irrelevant and not necessarily preclinical (De Decker and others 2010). In agreement with several human studies (Boden and others 1990, Lehto and others 1994, Abdullahkarim and others 2003), cervical intervertebral disc degeneration and spinal cord compression in clinically normal subjects is associated with higher age and the more caudally located intervertebral discs. Therefore, spinal cord compression in the caudal cervical region of clinically normal older dogs is suggested to be part of the normal process of age-related spinal degeneration (De Decker and others 2010).

**The role of relative vertebral canal stenosis**

If caudal cervical spinal cord compression is considered part of the common process of age-related spinal degeneration, the question remains why some dogs tolerate spinal cord compression, while other dogs do not (da Costa and others 2006a, De Decker and others 2011a). Several studies have compared the morphometric dimensions of the cervical vertebral column between dobermanns and other dog breeds, and between clinically normal and clinically affected dobermanns (Breit and Kunzel 2001, da Costa and others 2006a, De Decker and others 2011b, 2012a). The results of these studies suggest a potential role of pre-existent relative vertebral canal stenosis for the development of DA-CSM. In relative vertebral canal stenosis, the vertebral canal is smaller than what would be expected normally. This results in a decreased space between the spinal cord and the vertebral canal. This carries an increased risk of becoming clinically relevant on the development of space-occupying conditions of the vertebral canal (Bailey and Morgan 1992), such as age-related intervertebral disc protrusions. There is evidence that vertebral canal stenosis in clinically affected dobermanns is not limited to the caudal cervical vertebral canal only. Their vertebral canal is narrow over the entire cervical and even the cranial thoracic region, suggesting a more generalised vertebral canal stenosis (da Costa and others 2006a). Cervical spondylotic myelopathy is considered the human counterpart of CSM in dogs (Sharp and others 1992). Also, in this human condition, relative vertebral canal stenosis is considered an important static risk factor for the development of clinical signs (Hayashi and others 1997, Baptiste and Fehlings 2006).

**The role of spinal cord compression**

In agreement with human studies (Fenning and others 1986, Teneri and others 1987), it seems that the cross sectional area of the spinal cord should be decreased to a critical value before clinical signs of DA-CSM will occur (De Decker and others 2012a). Although this finding indicates that the degree of spinal cord compression is important for the development of DA-CSM, it was also demonstrated that the degree of intervertebral disc protrusion and dorsoventral spinal cord flattening are...
not key features for the development of clinical signs. Therefore, it has been suggested that as long as the cross sectional area of the spinal cord is preserved, clinical signs of DA-CSM do not necessarily occur in cases where the spinal cord is clearly deformed (De Decker and others 2012a). It seems plausible that in the presence of intervertebral disc protrusion, the cross sectional area of the spinal cord is maintained for longer in dogs with a relative wider vertebral canal.

Wider intervertebral discs have been demonstrated in dobermanns with CSM (da Costa and others 2006a). It was suggested that these wider intervertebral discs could potentially be at higher risk of herniation and that the volume of disc protrusion into the already narrowed vertebral canal would be relatively higher than that of clinically normal dogs (da Costa and others 2006a); however, this could not be confirmed in another study (De Decker and others 2012b).

Spinal cord compression is probably not the only cause of clinical signs in dogs with CSM. In agreement with human beings (Fehlings and Shaf 1995, Sohn and others 2004), it is believed that intervertebral foraminal stenosis with associated nerve root compression can be a contributing factor or even a primary cause of clinical signs in dogs with CSM (da Costa and others 2006a). Nerve root pathology has been demonstrated in dogs with DA-CSM (De Decker and others 2012c).

**Shape of the vertebral canal and vertebral body**

Dobermanns have a more pronounced funnel-shaped caudal cervical vertebral canal than other dog breeds (Breit and Kunzel 2001) and dobermanns with clinical signs of DA-CSM have a more funnel-shaped vertebral canal at the level of C7 compared to clinically normal dobermanns (De Decker and others 2011b). The vertebral canal of both clinically normal and affected dobermanns becomes more funnel-shaped from the cranial to the more caudal cervical vertebrae (Lewis 1991, De Decker and others 2011b). These data suggest that a funnel-shaped vertebral canal at the level of C7, resulting in a narrowed cranial orifice, can be considered an additional risk factor for dobermanns to develop spinal cord compression at this site.

Dobermanns with DA-CSM have more square-shaped vertebral bodies compared to clinically normal dogs (De Decker and others 2011c). It is possible that differences in vertebral body shape may alter the biomechanical properties of the cervical vertebral column (Breit and Kunzel 2004). It is suspected that a decrease in the length of the cervical vertebral bodies results in an increased range of motion during flexion and extension (Graf and others 1995, Wilke and others 1997).

**Role of vertebral column instability and dynamic spinal cord compression**

Although traditionally considered a key feature (Trotter and others 1976, Read and others 1983, Shores 1984), the role of vertebral column instability in the pathogenesis of CSM has been questioned (da Costa 2010). Vertebral column instability has never been objectively evaluated. Attempts have been made to measure the variation in alignment of the ventral surface of the vertebral canal using flexed, extended and neutral lateral radiographic views (Wright 1977, Lewis 1991). However, a wide variation in angulation between flexion and extension views was also demonstrated in normal dogs.

Human studies have demonstrated that early intervertebral disc degeneration is associated with instability, while more severe disc degeneration is associated with an increased stiffness of the vertebral column (Dai 1998, Kumaeran and others 2001). Because most dogs with DA-CSM have advanced disc degeneration, it seems unlikely that cervical vertebral column instability can be demonstrated when dogs are presented with clinical signs (da Costa and others 2006a). This finding does not, however, exclude the possibility of instability being present earlier in the degenerative process, before disc degeneration and clinical manifestation are evident. Unfortunately, biomechanical evaluation of the cervical spine of dogs in the preclinical stage would be very difficult to achieve (as one would never know if the dogs would, if alive, develop the disease).

The concept of dynamic lesions should be considered distinct from the concept of vertebral column instability (da Costa 2010). A dynamic lesion is one that worsens/improves with different positions of the neck. Repetitive flexion and extension in dogs with DA-CSM is believed to cause intermittent pressures and spinal cord elongation, leading to axial strain and stress within the spinal cord (McKee and others 1990, da Costa 2007). In DA-CSM, spinal cord compression is usually aggravated by dorsal extension and improved by ventral flexion of the neck (McKee and Sharp 2003, Sharp and Wheeler 2005). Cervical extension in healthy people causes an increase in the spinal cord length combined with a reduction in vertebral canal area (Reid 1960, Wäld 1967). Ventral flexion of the neck generates the opposite effect (Reid 1960).

**Clinical presentation**

Dogs affected with DA-CSM are usually between four and eight years old. Several large breed dogs can be affected, but dobermanns are overrepresented. Although male dogs are more often affected in some studies, there is no confirmed sex predisposition for DA-CSM in dogs (Denny and others 1977, Read and others 1983, Dixon and others 1996, da Costa and others 2008, De Decker and others 2009).

Clinical signs can vary from cervical hyperaesthesia to tetraplegia (Sharp and Wheeler 2005). The most common presentation is a gait disturbance. A wide-based ataxia and/or paresis of the pelvic limbs...
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FIG 4: (a) Transverse CT myelogram image (b) at the level of C6-C7 and sagittal reconstruction of an eight-year-old dobermann with ambulatory tetraparesis (same dog as Fig 1a). (a) Attenuation ventral subarachnoid space with dorsal displacement and deformation spinal cord is seen. The vertebral endplate demonstrates a mixed hyperattenuate signal. Hypoattenuating lesion indicating vacuum phenomenon, suggesting intervertebral disk degeneration (black arrow) is also seen and spondylosis deformans ventral to intervertebral disk space (white arrow). (b) Collapsed intervertebral disk space and extradural spinal cord compression at C6-C7 with increased attenuation in vertebral endplates and vertebral bodies are usually noted. Thoracic limb involvement with a short stilted gait can occur. Affected dogs often show a characteristic ‘two-engine’ gait, with the thoracic and pelvic limbs advancing at different rates. Although a gradual onset is noticed most commonly, clinical signs can also occur or exacerbate more acutely (Van Gundy 1988, Sharp and Wheeler 2005).

The wide variety of presenting clinical signs has resulted in a high number of reported classification systems to grade severity of neurological deficits in dogs with DA-CSM (McKee and others 1996, Queen and others 1998, Rosbrid and others 1998, De Ruio and others 2002, da Costa and others 2006a, Voss and others 2006, Bergman and others 2008, Shamir and others 2008, De Decker and others 2009, 2011a). The lack of an established grading system for dogs with DA-CSM limits the objective comparison of results between different clinical studies (Levine and Fosgate 2009). A recent study assessed but failed to demonstrate a significant correlation between the type of presenting clinical signs, degree of spinal cord compression, electrophysiological evaluation of the cervical spinal cord function and the outcome in dogs treated medically for DA-CSM (De Decker and others 2012c). To allow comparison of outcomes, prospective (instead of retrospective) scoring of affected animals should be considered and more detailed gait grading systems for dogs with DA-CSM are required. Alternatively, the use of objective, computerised systems could also be used. Studies evaluating the use of kinematic and force plate gait analysis to quantify neurological deficits in dobermanns with CSM have been recently performed and could be useful in the future (Foss and da Costa 2012, Foss and others 2012).

Diagnosis

Establishing a final diagnosis of DA-CSM requires myelography, CT myelography or MRI. There is considerable variability in interpretation by using either of these frequently employed imaging techniques for assessment of DA-CSM. Therefore, it seems difficult to consider one of these modalities as absolutely superior and they should probably be considered complementary to each other for making a diagnosis of DA-CSM in dogs (De Decker and others 2011d). Although most recent studies have focused on the use of MRI in dogs with DA-CSM, it should be emphasised that in several of these studies (e.g., De Decker and others 2011d), MRI was performed with low-field instead of high-field MR units. It is currently unclear if results retrieved from low-field MR units can be reliably compared with or extrapolated to those from high-field MR units. Several randomised, blinded studies in human beings have demonstrated that the use of high-field MRI units results in images with higher signal-to-noise ratios, contrast-to-noise ratios and better subjective global quality of the images (Mabon and others 1999, Merl and others 1999). However, these studies did not demonstrate a significant difference in diagnostic accuracy between low-field and high-field MR units.

Survey radiographs

Some radiographic findings may be suggestive of DA-CSM, but they are not diagnostic (Sharp and Wheeler 2005). Radiographic abnormalities in dogs with DA-CSM include vertebral body malformations, consisting of varying degrees of loss of the cranioventral border to an almost triangular-shaped vertebral body, an abnormal position of the vertebral body with craniodorsal tilting into the vertebral canal, spondylosis deformans, narrowing or collapse of the intervertebral disc space (Van Gundy 1992, Lewis 1991, Sharp and others 1992) and the vertebral canal may appear stenotic, with the cranial orifice being narrower than the caudal orifice (Sharp and others 1992). Presence or absence of changes on survey radiographs do not always correlate with evidence of spinal cord compression on more advanced imaging techniques (Read and others 1983, Lewis 1991).

Absolute measurements on survey radiographs have been used to evaluate the presence of vertebral canal stenosis (Wright 1977, 1979, Morgan and others 1986). However, these absolute measurements are influenced by radiographic magnification (Ravi and Rampersaud 2008). Radiographic magnification can be avoided by using ratios of different measurements (Pavlov and others 1987, Herzog and others 1991). Several vertebral ratios have been developed and used in human beings and horses to assess vertebral canal stenosis (Pavlov and others 1987, Herzog and others 1991). Several vertebral ratios have been developed and used in human beings and horses to assess vertebral canal stenosis (Pavlov and others 1987, Herzog and others 1991, Moore and others 1994, Hahn and others 2008). Cervical vertebral ratios, representing vertebral canal size, vertebral canal shape and vertebral body shape have recently been investigated in dobermanns, with and without clinical signs of DA-CSM. These studies indicated a smaller mid-sagittal vertebral canal height, a more funnel-shaped caudal cervical vertebral canal, and more square-shaped vertebral bodies in clinically affected compared to clinically normal dobermanns (De Decker and others 2011b, 2011c). Although these results suggest different vertebral column dimensions in dobermanns with DA-CSM, these vertebral ratios proved to be of little diagnostic value to differentiate clinically affected from clinically normal dobermanns (De Decker and others 2011b). Moreover, it has been suggested that vertebral canal to vertebral body ratios may not reliably predict relative vertebral canal stenosis in dogs (De Decker and others 2011c). More recently, intervertebral ratios (e.g., vertebral canal to body ratios between two adjacent vertebrae) were also investigated. Intervertebral ratios were proposed in horses as being more sensitive in the diagnosis of cervical malformation than the standard intravertebral (i.e., vertebral canal to body ratio within the same vertebra) ratios (Hahn and others 2006). Unfortunately, intervertebral ratios were not useful in differentiating dobermanns with and without DA-CSM (da Costa and Johnson 2012).

Myelography

Previously, myelography has been the method of choice to diagnose DA-CSM (Sharp and Wheeler 2005). Currently, myelography is largely being replaced by more advanced imaging techniques such as MRI (da
FIG 6: Sagittal T2-weighted MR image of a six-year-old dobermann with ambulatory tetraparesis and cervical hyperesthesia. Hyperintense intramedullary signal at the level of C6-C7. Abnormal, almost triangular-shaped vertebral body at C7 and complete intervertebral disk degeneration and collapsed intervertebral disk space at C6-C7 is seen.

Costa 2010. In lateral views, abnormalities are seen both in the ventral and dorsal aspects of the vertebral canal. Ventral extradural compression related to one or more intervertebral discs is the most common finding. Dorsal extradural compression caused by hypertrophy of the ligamentum flavum can also be seen in dogs with DA-CSM (Sharp and others 1992) (F2). Compared with CT myelography and low-field MRI, myelography is reported to be more reliable to assess many of the abnormalities commonly seen in dogs with DA-CSM, but less reliable to assess articular facet abnormalities and intervertebral foraminal stenosis (De Decker and others 2011d).

Another disadvantage of myelography is its rather invasive nature (Sharp and Wheeler 2005). Seizures and transient neurological deterioration are the most important complications following myelography (Sharp and others 1992), and have been reported to occur in 27 per cent (da Costa and others 2006b, De Decker and others 2011d) and 14 per cent (De Decker and others 2011d) of dogs with DA-CSM.

Most dogs with DA-CSM have been reported to be at higher risk of experiencing myelographic complications, compared to dogs with other cervical spinal cord disorders (Lewis and Hosgood 1992). However, this observation was probably made because of the breed predisposition to DA-CSM. A recent study found that all large breed dogs have a higher risk for postmyelographic seizures owing to their large size, primarily if they have cervical disease (particularly CSM), and injection of larger volumes of contrast medium into the cerebellomedullary cistern (da Costa and others 2011).

The value of applying traction during myelography has been discussed (Van Gundy 1993, Sharp and others 1992, Rusbridge and others 1998, da Costa and others 2006b). Lesions are termed ‘static’ when the degree of compression remains the same, whereas ‘traction-responsive’ lesions improve after performing linear traction to the head (Sharp and others 1992) (F3). Different methods have been suggested to perform linear traction (Van Gundy 1982, Penderis and Dennis 2004, da Costa and others 2006a). This subdivision has been suggested to give information concerning the nature of the lesion and the preferred type of surgery to perform (Sharp and Wheeler 2005). Most dogs with DA-CSM have traction-responsive lesions. It has traditionally been suggested that traction-responsive lesions will benefit from distraction-stabilisation, while static lesions will benefit from direct decompressive surgical techniques (McKee and Sharp 2003, Sharp and Wheeler 2005). However, there is currently no strict definition of a traction-responsive lesion, standardisation and objectivity of the technique is not ideal, little is known about the optimal amount of traction required (da Costa and others 2006b) and a retrospective study demonstrated similar outcomes after distraction-stabilisation or direct decompressive surgery in dobermanns with traction-responsive lesions (Rusbridge and others 1992).

The degree of compression can also change as the neck is moved between flexed, neutral and gently extended positions. Extension usually exacerbates and flexion usually relieves spinal cord compression in dogs with DA-CSM (McKee and Sharp 2003) (Fig 3). Performing flexion or extension is not without risk and should be done either with extreme care or not at all (Lewis 1991).

CT myelography

Relatively few studies have discussed the use of CT myelography for the diagnosis of DA-CSM in dogs (Sharp and others 1992, 1995, De Decker and others 2011d, da Costa and others 2012). The most characteristic CT myelographic finding in dogs with DA-CSM is an attenuation of the ventral subarachnoid space with a dorsal displacement of the spinal cord. These abnormalities are caused by protruding annulus fibrosus (Sharp and others 1995) (F4). CT myelography is suggested to be the most reliable imaging modality to assess articular facet abnormalities and intervertebral foraminal stenosis in dogs with DA-CSM (De Decker and others 2011d).

Traction studies can also be performed by CT myelography (Adiego da Silva and others 2010). Although an optimal CT myelography study is performed with a lower dose of contrast medium than a conventional myelographic study (Yu and others 1986), this technique still carries the risk of complications such as postmyelographic seizures (da Costa and others 2012b). Cranial thoracic lesions were identified in eight per cent of dogs with CSM using CT myelography (da Costa and others 2011), and as such, it is recommended that the field of view of CT and MRI studies include the cranial thoracic region.

In human beings, CT myelography also provides prognostic information by detecting spinal cord atrophy (Badami and others 2001). Spinal cord compression, intervertebral disc degeneration, intraparenchymal signal changes and abnormalities commonly seen in dogs with DA-CSM (da Costa and others 2006a) (F5). Traction studies can also be performed by MRI (Penderis and Dennis 2004, da Costa and others 2006a).

A difficulty in evaluating spinal MRI studies is the possible occurrence of imaging abnormalities in clinically normal animals. Severe degenerative changes can be seen in clinically normal dogs, while some MRI studies of dogs with DA-CSM demonstrate only mild imaging abnormalities (da Costa and others 2006a, De Decker and others 2011a). In a recent study, 17 per cent of clinically normal dogs were categorised, based on the interpretation of low-field MRI studies, as suspected to be clinically affected and 10 per cent of dogs with DA-CSM were categorised as suspected to be clinically normal. This resulted in a sensitivity of 0.85 and specificity of 0.71 for low-field MRI to differentiate between dogs with and without clinical signs of DA-CSM (De Decker and others 2011a). This highlights the importance of evaluating imaging studies in the light of thorough neurological examination findings. Morphometric and morphological MRI variables have been investigated to differentiate between clinically relevant and irrelevant cervical spinal cord compressions (da Costa and others 2006a, De Decker and others 2011a, 2012a). A threshold value was identified for the ‘remaining spinal cord area’, a morphometric variable quantifying spinal cord compression, to differentiate clinically relevant from irrelevant cervical spinal cord compressions; however, a less than optimal interobserver agreement currently hampers extrapolation of this value to clinical practice (De Decker and others 2012a).

The occurrence of T2-weighted hyperintense intraparenchymal signal intensity (ISI) changes is currently the most reliable morphological variable to predict the presence of clinical signs (da Costa and others 2006a, De Decker and others 2011a) (F6). Although the exact meaning of T2-weighted ISI changes is currently unknown, it is suggested that they reflect a broad spectrum of reversible and irreversible spinal cord abnormalities, such as oedema, inflammation, ischaemia, gliosis and myelomalacia (Suri and others 2008). Hyperintense T2-weighted ISI changes are common in dobermanns with DA-CSM and they have been associated with a longer duration of clinical signs and more severe spinal cord compression (Faissler and Bildendecker 2008, da Costa 2012, De Decker and others 2012c). Variable results have been found considering the association of hyperintense T2-weighted ISI changes and...

When compared to myelography and CT myelography, low-field MRI is reported to be less reliable for the assessment of narrowed intervertebral disc spaces and spondylosis deformans in dogs with DA-CSM (De Decker and others 2011d).

Transcranial magnetic stimulation (TMS) is a non-invasive, painless and sensitive electrophysiological technique for stimulating the cerebral cortex in order to evaluate the functional integrity of the spinal cord motor pathways (Nollet and others 2003). Different TMS-values have been demonstrated between dobermanms with and without clinical signs of DA-CSM (da Costa and others 2006c) and between dobermanms with and without clinically relevant cervical spinal cord compressions (De Decker and others 2011e). Variable results have been reported considering the association of the type of presenting clinical signs and TMS in dogs with DA-CSM (da Costa and others 2006c, De Decker and others 2011e, 2012c).

Conclusions

Although several new insights have been gained in the pathogenesis and diagnosis of DA-CSM, several questions remain unanswered. DA-CSM may be a multifactorial pathogenesis with potential contributions of relative vertebral canal stenosis, the degree and dynamics of spinal cord compression and the shape of the vertebral canal and vertebral body. The contribution of dynamic compressions, the role of vertebral stability and an underlying genetic or hereditary component are currently unclear. The lack of an objective grading system for neurological evaluation is concerning and warrants further investigation. Although the specificity of MRI has been questioned, other diagnostic techniques have not compared clinically affected dogs with and without clinically relevant cervical spinal cord compressions (De Decker and others 2011e). Variable results have been reported considering the association of the type of presenting clinical signs and TMS in dogs with DA-CSM (da Costa and others 2006c, De Decker and others 2011e, 2012c).


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