# One-year clinical and magnetic resonance imaging follow-up of Doberman Pinschers with cervical spondylomyelopathy treated medically or surgically

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**Objective**—To evaluate progression of clinical signs and magnetic resonance imaging (MRI) findings in dogs with cervical spondylomyelopathy (wobbler syndrome) treated medically or surgically.

**Design**—Prospective cohort study.

**Animals**—12 Doberman Pinschers.

**Procedures**—Neurologic examinations and MRI were performed before medical (n = 9) or surgical treatment (ventral slot, 3) and a minimum of 12 months later.

**Results**—Mean follow-up time was 14.5 months. Clinically, 2 dogs improved after surgical treatment and 5 improved after medical treatment. Magnetic resonance imaging of surgically treated dogs revealed adequate spinal cord decompression. Spinal cord signal changes were seen in 2 dogs before surgery, both of which had new signal changes at the same and adjacent sites during follow-up examination. One dog treated surgically developed 3 new areas of spinal cord compression. In the medically treated dogs, the severity of spinal cord compression at the time of follow-up examination was unchanged in 4 dogs, worse in 2 dogs, and improved in 3 dogs, but spinal cord atrophy was observed on transverse images. Four medically treated dogs had changes in spinal cord signal initially, but none developed new signal changes or compressions.

**Conclusions and Clinical Relevance**—Medical and surgical treatment improved or stabilized the clinical condition of most dogs. Surgical treatment appeared to hasten the development of additional areas of spinal cord compression and lesions in dogs with preoperative cord changes; however, the clinical importance of these changes was not determined. The progression of pathologic MRI abnormalities was notably less in medically treated dogs, compared with surgically treated dogs. (*J Am Vet Med Assoc* 2007;231:243–250)

Cervical spondylomyelopathy is a common disease of the cervical portion of the spine in large- and giant-breed dogs. Many questions remain unanswered about the pathogenesis of CSM, the natural progression of the disease, and the efficacy of various treatments.[1] In particular, the natural course of the disease and the role of medical treatment still need to be established. Long-term deterioration has been reported to occur in most dogs treated surgically,[2] but the cause of such deterioration is usually not investigated. Magnetic resonance imaging is a highly sensitive diagnostic technique that provides superior diagnostic information, compared with myelography, in dogs with CSM.[3] Several studies[4-8] in which MRI or computed tomography was used to investigate short- and long-term changes after medical or surgical treatment of humans with cervical spondylotic myelopathy have been published. To our knowledge, however, there are no published studies in which the long-term effects of medical or surgical treatment in dogs with CSM were examined prospectively by means of MRI. The purpose of the study reported here, therefore, was to prospectively investigate the progression of clinical signs and MRI findings over a period of 1 year in Doberman Pinschers with CSM treated medically or surgically. The present report represents 1 of a series of studies[3,9,10,a] investigating the pathogenesis, diagnosis, and treatment of CSM in dogs.

**Abbreviations**

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| CSM | Cervical spondylomyelopathy |
| MRI | Magnetic resonance imaging |

## Materials and Methods

Twelve Doberman Pinschers with CSM (5 males and 7 females; mean age, 5.7 years [range, 3 to 8 years]) were enrolled in the study. The study was conducted at the University of Guelph Ontario Veterinary College

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between June 2003 and December 2005 and was performed in accordance with the guidelines of and upon the approval of the University of Guelph Animal Care Committee.

A clinical history was obtained and physical and neurologic examinations, biochemical testing (ie, CBC and serum biochemistry profile), and MRI were performed in all dogs at the time of study enrollment. In all dogs, results of electrocardiography and echocardiography performed at the time of study enrollment were normal.

Neurologic status at the time of study enrollment was graded from 1 to 5 on the basis of modifications of a previously published grading scale.[3] Grade 1 was defined as cervical hyperesthesia only. Grade 2 was defined as mild pelvic limb ataxia or paresis with or without thoracic limb involvement. For dogs with grade 2 involvement, paw placement was generally consistent throughout most of the evaluation, but the thoracic limbs typically had a short-strided or spastic gait with a floating appearance. Grade 3 was defined as moderate pelvic limb ataxia or paresis with thoracic limb involvement as described for grade 2. This grade was characterized by consistent and obvious pelvic limb ataxia from the beginning of the gait evaluation. The dog would often misposition its pelvic limbs, with abduction or adduction being commonly seen throughout the gait evaluation. Grade 4 was defined as marked pelvic limb ataxia or paresis with thoracic limb involvement as described for grade 2. This grade was characterized by a severely abnormal gait, with consistently abnormal foot placement during gait evaluation, usually associated with marked pelvic limb weakness. Characteristically, the dog would be lower to the ground on its hind limbs than on its forelimbs and would sink on its hind limbs when it stood. Grade 5 was defined as nonambulatory tetraparesis. In all dogs, the gait evaluation was videotaped for later review.

For MRI, dogs were anesthetized and positioned in dorsal recumbency. Imaging was performed with a 1.5-Tesla magnet[b,c] and cervical spine array coil. T1- and T2-weighted images were obtained in the sagittal plane. T1-, T2-, and gradient echo-weighted images were obtained in the transverse plane. Magnetization transfer was performed with IV administration of gadolinium[d] (0.1 mmol/kg [0.045 mmol/lb]). Details of the MRI protocol have been published.[9]

Following the initial evaluation, 9 dogs were treated medically and 3 were treated surgically. Eight of the dogs treated medically were given dexamethasone (0.25 mg/kg [0.11 mg/lb], PO, q 24 h on the first day, followed by 0.1 mg/kg, PO, q 24 h for 5 to 7 days), and the remaining dog received no medication. Two dogs developed diarrhea after receiving the first dose of dexamethasone, and the drug was discontinued. Ranitidine and sucralfate were administered PO in conjunction with dexamethasone. In 6 of the dogs treated medically, prednisone (1.0 mg/kg [0.45 mg/lb], PO, q 24 h for 7 days; 0.5 mg/kg [0.22 mg/lb], PO, q 24 h for 7 days; 0.5 mg/kg, PO, q 48 h for 7 days) was administered for an additional 3 weeks after treatment with dexamethasone was discontinued. After the initial 4 weeks of corticosteroid treatment, administration of prednisone was discontinued and dogs were reevaluated. In 2 dogs, administration of prednisone was continued for an additional 4 weeks.

In the 3 dogs treated surgically, a ventral slot procedure was performed through a standard surgical approach.[11] No implants or grafts were used, and in all 3 dogs, the spinal cord appeared grossly to be decompressed following completion of the ventral slot procedure. None of the dogs developed complications during or after surgery. All 3 dogs had been treated with dexamethasone, prednisone, or both prior to surgery, but these were not used postoperatively.

Owners of all dogs were clearly instructed to restrict their dog's activities to walks and to avoid free running or rough play during at least the initial 4 to 6 weeks of treatment. Long-term restriction and monitoring of activity were also suggested, but cage rest was not recommended. Use of a body harness, instead of a neck collar, was strongly advised.

All dogs had neurologic examinations repeated as needed and the MRI repeated a minimum of 12 months after the initial evaluation. The gait evaluation was videotaped, and videotapes of the initial and follow-up examinations were used in association with the neurologic examination to determine the follow-up neurologic score.

The initial and follow-up MRI studies were examined to determine the cause and severity of spinal cord compression and other abnormalities. The main lesion was established by examining sagittal and transverse images to identify the region with the smallest spinal cord diameter, smallest vertebral canal diameter, and most severe changes in spinal cord signal. Changes in the spinal cord signal were classified as hyperintense or hypointense by comparison with signal intensity of adjacent areas of spinal cord. Each intervertebral disk was classified as normal, partially degenerated, or completely degenerated on the basis of signal intensity characteristics on midsagittal T2-weighted images. The presence and severity of intervertebral foraminal stenosis were evaluated on transverse gradient echo-weighted images centered on the disk space that were obtained with magnetization transfer following administration of gadolinium. Bone proliferation and signal changes in the cranial and caudal end plates were identified on sagittal T1- and T2-weighted images. The transverse area of the spinal cord and of the vertebral canal at the site of the main lesion was measured on T2-weighted images. Area measurements were performed on a single transverse image at the site of the most severe spinal cord compression, as described,[9] with software for image analysis.[e] The morphometric results are presented descriptively as mean and SD. Images obtained during initial and follow-up MRI were evaluated side by side with the assistance of a commercial software program.[f]

## Results

Mean time between initial and follow-up neurologic examinations and MRI was 14.5 months (range, 12 to 18 months). Historically, the onset of clinical signs prior to the initial evaluation was acute in 5 dogs, chronic in 6 dogs, and chronic with acute worsening in 1 dog. Dura-

tion of clinical signs prior to the initial evaluation ranged from 1 day to 2 years (mean, 19.6 weeks; median, 7.5 weeks). For the 9 dogs treated medically, initial neurologic status was classified as grade 1 (3 dogs), 2 (2 dogs), 3 (1 dog), or 4 (3 dogs). For the 3 dogs treated surgically, initial neurologic status was classified as grade 3 (1 dog) or 4 (2 dogs). Eight of the 12 dogs had historical or clinical findings indicative of cervical hyperesthesia, such as low head carriage, restricted cervical motion, or vocalization upon cervical movement.

At the time of the follow-up evaluation 12 to 18 months after initial treatment, 5 of the 9 dogs treated medically had improved clinically (from grade 2 to clinically normal, from grade 1 to clinically normal, from grade 4 to 3, from grade 4 to 2, and from grade 3 to 2), 2 dogs had become clinically worse (from grade 1 to 2 and from grade 1 to 3), and 2 dogs were clinically unchanged (1 dog that was grade 2 and 1 that was grade 4). Two of the 3 dogs treated surgically had improved clinically (from grade 4 to 3 and from grade 3 to 2). The remaining dog treated surgically had reportedly improved during the first 3 months after surgery, but then its condition had markedly deteriorated. At that time, the dog was treated medically, and its condition improved so that at the time of follow-up evaluation, it was clinically unchanged from the preoperative state (grade 4).

Examination of images obtained during the initial MRI study in the 3 dogs treated surgically revealed spinal cord compression in all 3. The area of compression was located at C6-7 in 1 dog and at C5-6 in 2 dogs. The compression was ventrally positioned and associated with the disk in all 3 dogs. All 3 dogs had evidence of intervertebral disk degeneration affecting at least 1 disk in the cervical portion of the spine, and 2 dogs had foraminal stenosis.

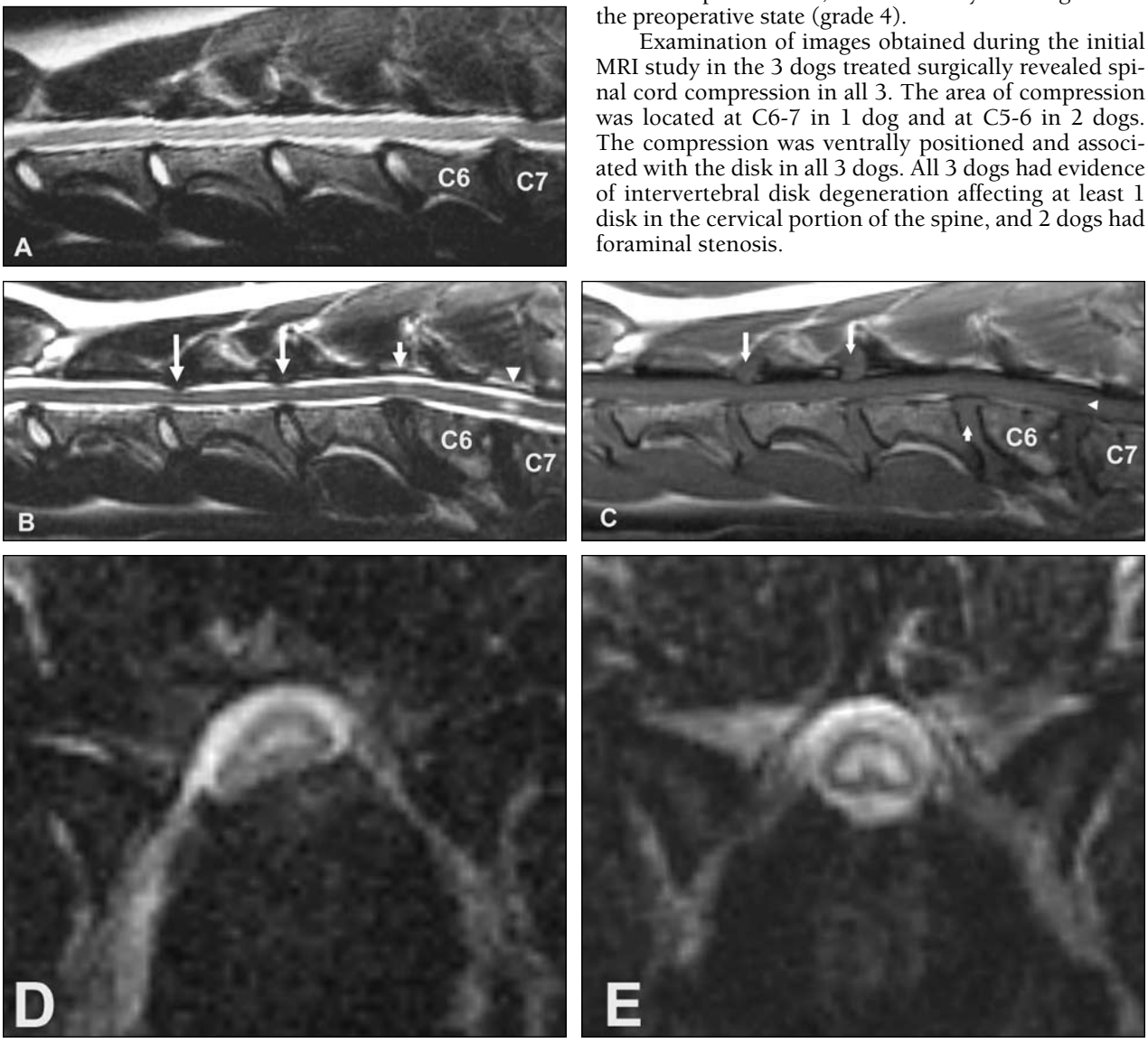


Figure 1—Magnetic resonance images of the cervical portion of the spine in a 7-year-old Doberman Pinscher with CSM and marked ataxia (grade 4). A—Midsagittal T2-weighted image obtained prior to surgery; notice the spinal cord compression and spinal cord hyperintensity at the C6-7 space. B—Midsagittal T2-weighted image obtained 14 months after a ventral slot procedure at C6-7. Notice that spinal cord compression is no longer visible at the C6-7 space; however, the area of spinal cord hyperintensity is more apparent, although slightly smaller (arrowhead). There are new areas of spinal cord compression ventrally at C5-6 and dorsally at C3-4 and C4-5 (long arrows). Mild spinal cord hyperintensity can be seen associated with the compression at C5-6 (short arrow). The vertebral body of C6 is triangular and rotated dorsally. The C5-6 intervertebral disk is also degenerated, compared with the preoperative appearance. C—Midsagittal T1-weighted image obtained 14 months after surgery. Notice the area of hypointensity in the spinal cord at C6-7 (arrowhead), minimal bone proliferation between the vertebral bodies of C6 and C7, and the intermediate signal of structures compressing the spinal cord dorsally (long arrows) and ventrally (short arrow). D—Transverse T2-weighted image at the level of C6-7 obtained before surgery. E—Transverse T2-weighted image at the level of C6-7 obtained 14 months after surgery. Adequate spinal cord decompression is seen with marked spinal cord hyperintensity and atrophy.

Examination of images obtained during follow-up MRI of the 3 dogs treated surgically revealed adequate decompression of the spinal cord in all 3, with minimal residual compression in 1 dog. In 1 dog, follow-up MRI performed 14 months after the initial evaluation revealed 3 areas of spinal cord compression cranial to the surgical site at C6-7 (1 ventrally and 2 dorsally) that were not present on the initial images (**Figure 1**). This dog also had evidence of tipping of C6 and disk degeneration cranial and caudal to the surgical site not seen on initial images. Changes in spinal cord signal characterized by hyperintense areas on T2-weighted images were observed on initial images obtained from 2 of the dogs treated surgically. On follow-up images, these areas were hypointense on T1-weighted images and larger than the areas seen on the initial images. One of these dogs also had a new area of spinal cord hyperintensity on T2-weighted images at C5-6 that was associated with ventral spinal cord compression, whereas the other had a new area of spinal cord hyperintensity on T2-weighted images caudal to the surgery site without evidence of spinal cord compression. This dog had worsening of foraminal stenosis on the right side, compared with initial images. The extent of bone proliferation and bone marrow changes at the site of surgery evident on follow-up images varied markedly among the 3 dogs. One dog had minimal bone proliferation with no apparent bone fusion. The 2 other dogs had moderate to marked bone marrow changes and fusion with osteophyte formation.

Examination of initial magnetic resonance images revealed spinal cord compression in 8 of the 9 dogs treated medically. In 7 of the 8 dogs, the compression was predominantly ventral and associated with the disk, whereas in 1, bilateral dorsolateral spinal cord compression caused by medial proliferation of the articular facets was evident. One dog with cervical hyperesthesia (grade 1) had evidence of bilateral foraminal stenosis without spinal cord compression. The main lesion was located at C5-6 in 5 dogs and at C6-7 in 3 dogs. The remaining dog had compression at both C5-6 and C6-7, with the former being worse. Foraminal stenosis was observed in 7 of the 9 dogs, and intervertebral disk degeneration was seen in all 9. On follow-up images, 4 of the 9 dogs treated medically had no change in the severity of spinal cord compression (**Figure 2**). All but one of these dogs had improved clinically, whereas clinical status in the remaining dog had remained unchanged. In 2 of the 9 dogs, spinal cord compression worsened, and clinical status had worsened in both of these dogs. In the remaining 3 dogs, the compression seemed to have regressed on sagittal follow-up images (**Figure 3**). However, examination of the transverse images revealed spinal cord atrophy. Two of these 3 dogs had improved clinically, whereas clinical status in the third dog had remained unchanged. None of the dogs treated medically developed new sites of spinal cord compression. Four of the 9 dogs had areas of spinal cord hyperintensity on initial T2-weighted images. On follow-up images, areas of hyperintensity remained unchanged in 2 dogs and had become more prominent in the other 2. None of the dogs developed new areas of spinal cord signal changes or hypointensity on T1-weighted images. Three of the 9 dogs treated medically had new areas of intervertebral disk degeneration (1 disk in each dog). One of the 9 dogs had unilateral worsening of intervertebral foraminal stenosis.

Overall, 6 of the 12 dogs (4 treated medically and 2 treated surgically) had MRI evidence of spinal cord hyperintensity on T2-weighted images obtained during the initial evaluation. All of these dogs had a history of chronic signs, and none of the 5 dogs with acute signs had areas of hyperintensity. Spinal cord hyperintensity was observed in all 5 dogs with marked ataxia and in 1 of the 2 dogs with moderate ataxia. Spinal cord hyperintensity was not observed in dogs with cervical hyperesthesia only or with mild ataxia.

All 6 dogs with areas of spinal cord hyperintensity had concurrent spinal cord atrophy on follow-up magnetic resonance images. Only 1 dog developed spinal cord atrophy not associated with spinal cord hyperintensity.

For the 3 dogs treated surgically, mean ± SD spinal cord area was 52 ± 10.5 $mm^2$ at the time of initial MRI

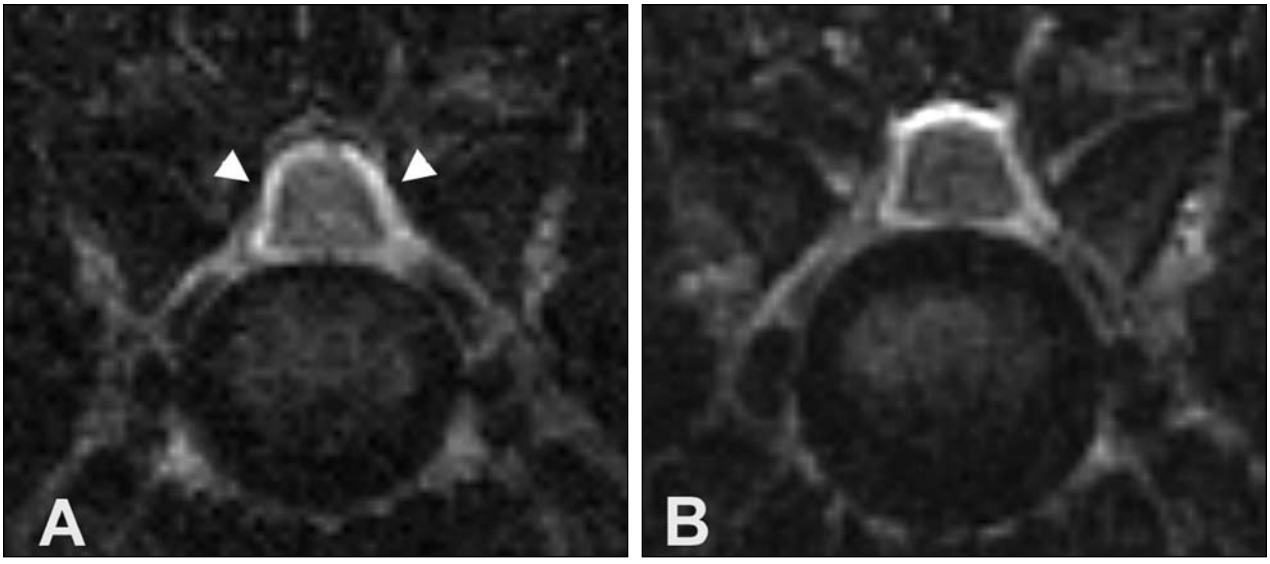


Figure 2—Transverse T2-weighted magnetic resonance images at the level of C5-6 in a 4-year-old Doberman Pinscher with CSM and mild ataxia (grade 2) that was treated medically. A—Image obtained prior to treatment; notice the bilateral spinal cord deformation caused by medial proliferation of the articular processes (arrowheads). B—Image obtained 12 months later; the dog had been treated with corticosteroids. The severity of spinal cord compression is unchanged. Morphometrically, the area of the spinal cord and area of the vertebral canal were also unchanged.

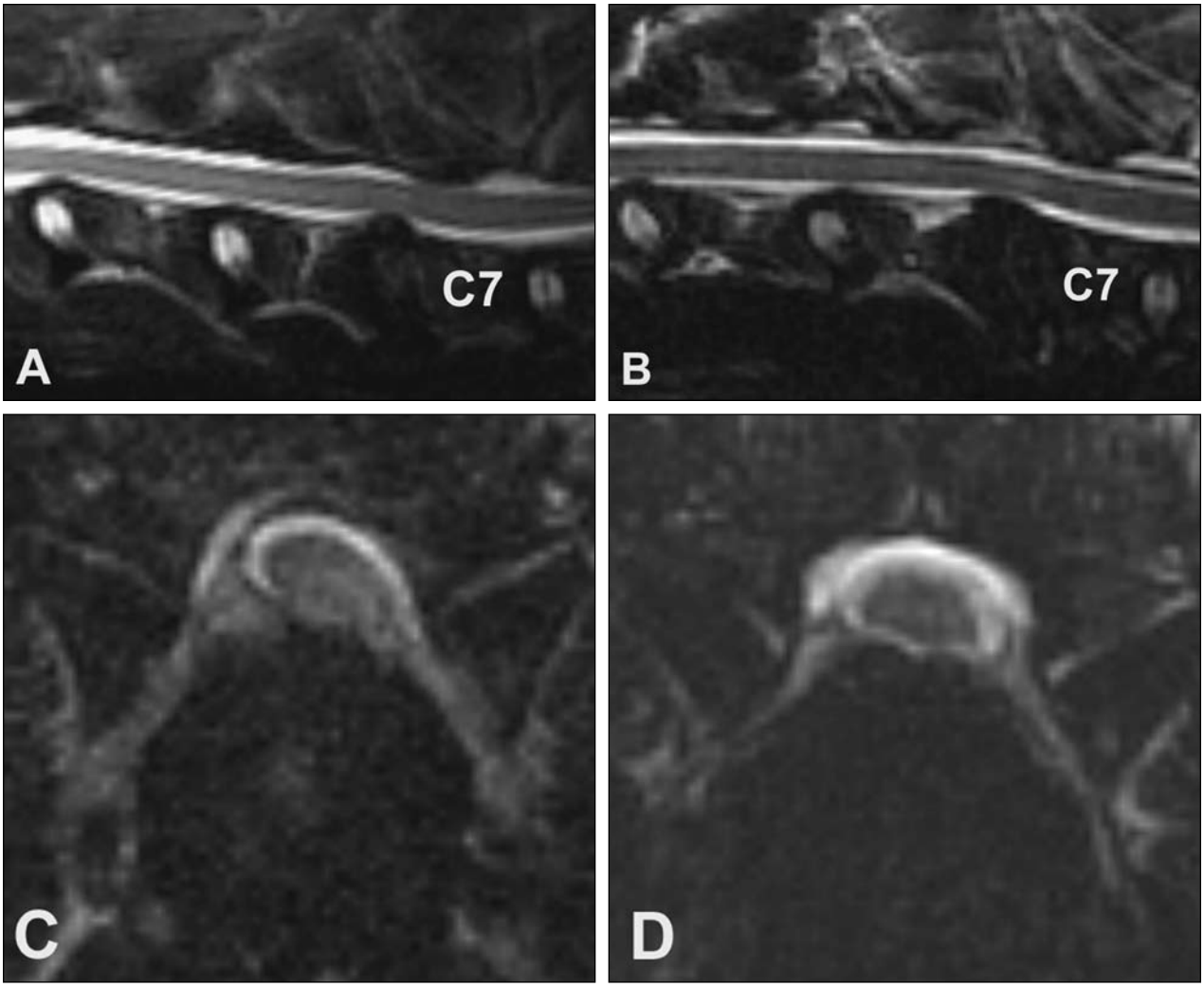


Figure 3—Magnetic resonance images of the cervical portion of the spine in a 5-year-old Doberman Pinscher with CSM and marked ataxia (grade 4) that was treated medically. A—Midsagittal T2-weighted image obtained prior to treatment; notice the spinal cord compression and mild spinal cord hyperintensity at C6-7. B—Midsagittal T2-weighted image obtained 15 months later. The area of spinal cord hyperintensity is still present, and adjacent disks are not dehydrated. C—Transverse T2-weighted image at the level of C6-7 obtained prior to treatment. D—Transverse T2-weighted image obtained 15 months later. The compression seems less severe, but spinal cord atrophy is present. Spinal cord area is 28% smaller than area on the initial image, whereas vertebral canal area is unchanged.

evaluation and 49 ± 12.2 $mm^2$ at the time of the follow-up evaluation. At the time of the follow-up evaluation, spinal cord area was smaller in 2 dogs and unchanged in 1. Mean vertebral canal area in the 3 dogs treated surgically was 88 ± 17.3 $mm^2$ at the time of initial evaluation and 103 ± 12.4 $mm^2$ at the time of follow-up evaluation. At the time of follow-up evaluation, vertebral canal area was increased in 2 dogs and unchanged in 1.

For the 9 dogs treated medically, mean ± SD spinal cord area was 49.7 ± 4.6 $mm^2$ at the time of initial MRI evaluation and 45.2 ± 8.7 $mm^2$ at the time of follow-up evaluation. At the time of follow-up evaluation, spinal cord area was smaller in 5 dogs and unchanged in 4. Mean vertebral canal area in the 9 dogs treated medically was 102.1 ± 13.9 $mm^2$ at the time of initial evaluation and 101.7 ± 13.7 $mm^2$ at the time of follow-up evaluation. At the time of follow-up evaluation, vertebral canal area was increased in 2 dogs, unchanged in 6, and decreased in 1.

## Discussion

This study provides the first prospective clinical and MRI evidence of the evolution of CSM in Doberman Pinschers treated medically and surgically. In the present study, medical and surgical treatment improved or stabilized the clinical condition of most dogs. However, spinal cord and vertebral changes seen on follow-up magnetic resonance images were more severe in dogs treated surgically than in dogs treated medically. In particular, in contrast to findings for the dogs treated surgically, new compressive lesions and new spinal cord signal changes were not seen in dogs treated medically. Overall, the progression of MRI changes in the medically treated dogs was slow, with worsening of spinal cord compression in only 2 of the 9 dogs, both of which also had worsening of their neurologic status. Clinical progression in the 2 dogs that became worse following medical treatment also appeared slow, with signs progressing from cervical hyperesthesia to mild or moder-

ate ataxia over a 17-month period. Spinal cord atrophy occurred in 5 of the 9 dogs treated medically but also occurred in 2 of the 3 dogs treated surgically.

Despite the findings observed with medical treatment in the present study, surgical treatment has historically offered a higher chance of clinical improvement in dogs with CSM. A recent retrospective study,[a] for instance, found that 81% of patients improved with surgical treatment, whereas 53.5% improved with medical treatment. In that study, however, median survival time for dogs with CSM treated surgically (36 months) was identical to median survival time for dogs treated medically.

Despite the large number of previous studies describing surgical treatment for CSM, only a few have described results of follow-up diagnostic imaging. Two reports[12,g] described computed tomographic features of dogs that underwent ventral slot decompression or fusion, and 2 others[2,13] described results of follow-up myelography in a limited population of patients that deteriorated after surgery. A recent study[3] showed that MRI offers superior diagnostic information, compared with myelography. Therefore, in the present study, we elected to perform follow-up MRI in all dogs, independent of their clinical status. It was surprising to see the progression of pathologic changes in surgically treated dogs that, clinically, had responded well to surgery. The so-called domino effect, or adjacent segment disease, as it is known in humans, is reported to occur in approximately 20% of dogs[1] and is reported to occur more commonly with distraction-stabilization techniques and less commonly following a ventral slot procedure.[2,11,14] Most reports[12,15,16] in the veterinary and human literature suggest that the domino effect occurs secondary to bone fusion. However, recent evidence in humans suggests that there is no difference in the incidence of adjacent segment disease between sites with and without fusion.[17] One surgically treated dog in the present study developed 3 new areas of spinal cord compression in the 14 months after surgery. In addition, this dog developed intervertebral disk degeneration caudal and cranial to the surgical site and was the only one that did not have apparent bone fusion at the surgical site. In a previous study,[18] bone proliferation and vertebral ankylosis were identified in all dogs by 3 months after ventral slot surgery. One can hypothesize, therefore, that the 3 new areas of compression seen in one of the dogs treated surgically developed secondary to increased motion at the intervertebral disk space where the ventral slot procedure had been performed. A recent in vitro study[19] showed that ventral slot decompression increased the range of motion of the operated segment, which could cause clinical instability. Even though instability was identified in this in vitro study, if bone proliferation and fusion occur between 2 and 3 months after surgery,[18,20] the intervertebral instability would be short lasting. In all reports[13-15,21] found by the authors, the domino effect occurred in a single intervertebral disk cranial or caudal to the surgical site. In the dog described in the present report, 1 of the 3 new areas of spinal cord compression was likely a result of disk protrusion, but the other 2 were dorsal and likely caused by ligament flavum hypertrophy. This suggests that a lack of fusion following a ventral slot procedure can have effects on the entire cervical portion of the spine, rather than on only the adjacent segments.

In 6 of the dogs in the present study, areas of spinal cord hyperintensity were seen on T2-weighted images. This included 2 dogs treated surgically and 4 dogs treated medically. Interestingly, in the 2 dogs treated surgically, follow-up MRI showed not only enlargement of the initial area of hyperintensity but also hypointensity on T1-weighted images and new areas of hyperintensity on T2-weighted images adjacent to the surgical site in both dogs. Enlargement of the area of hyperintensity on T2-weighted images was seen in 2 medically treated dogs, but neither progression to hypointensity on T1-weighted images nor the appearance of new signal changes in adjacent areas was observed. In humans, an association between spinal cord signal changes on magnetic resonance images and histologic abnormalities has been well documented.[22] Areas of hyperintensity on T2-weighted images without changes on T1-weighted images were characterized by slight loss of nerve cells, gliosis, and edema in the gray matter and by demyelination, edema, and Wallerian degeneration in the white matter. The combination of hyperintensity on T2-weighted images and hypointensity on T1-weighted images was characterized by severe changes such as necrosis, myelomalacia, and spongiform changes in the gray matter and white matter necrosis.[22] To our knowledge, there are no published studies relating signal changes seen on magnetic resonance images from dogs with CSM with histologic abnormalities. However, experimentally induced chronic cervical myelopathy in dogs caused signal abnormalities and spinal cord changes characterized by motor neuron loss and gray matter necrosis and cavitation.[23]

Controversy exists as to whether signal changes have prognostic importance in humans with CSM. Some have proposed that hyperintensity on T2-weighted images was associated with a poor outcome,[24,25] whereas others found no association between hyperintensity and poor outcome.[26-28] More recently, it has been proposed that hyperintensity on T2-weighted images reflects pathologically reversible changes, whereas the combination of hyperintensity on T2-weighted images and hypointensity on T1-weighted images suggests irreversible spinal cord changes and a poorer prognosis.[29,30]

Findings of the present study do not suggest that spinal cord hyperintensity on T2-weighted images implies a poor prognosis because clinical improvement was observed in 4 of 6 dogs with such changes and clinical status was unchanged in the other 2. The combination of hyperintensity on T2-weighted images and hypointensity on T1-weighted images is more intriguing. One must question why the 2 surgically treated dogs that initially only had areas of hyperintensity subsequently developed areas of hypointensity if the spinal cord was adequately decompressed. The most plausible explanation relies on an ischemic pathogenesis.[31] A large body of experimental evidence has shown a major role for vascular disturbances in the pathogenesis of chronic spinal cord injury.[23,32-34] Without spinal cord compression, we hypothesize that the progression of spinal cord lesions could be related to foraminal stenosis. Unilateral worsening of foraminal stenosis was seen in 1 dog that developed an area of hyperintensity on follow-up mag-

netic resonance images; this area was caudal to the site of the ventral slot procedure and not associated with spinal cord compression. Foraminal stenosis has been proposed as an important factor in the ischemic pathogenesis of CSM in humans.[35] The ventral slot procedure may have caused a collapse of the craniocaudal diameter (width) of the intervertebral foramina, which then may have caused or contributed to partial spinal cord ischemia. The foraminal width cannot be seen on transverse magnetic resonance images, in which the mid foraminal height is evaluated.[9] Evaluation of foraminal width on parasagittal images was difficult because positioning was not always perfect. It is also possible that foraminal stenosis was exacerbated by movement. Cervical extension in healthy humans has been shown to reduce foraminal area by 22% to 25% and occasionally even more in individuals with stenotic foramina.[36,37] Against this theory is the fact that foraminal stenosis occurs commonly in Doberman Pinschers, even clinically normal ones.[9] The long-term importance of these postoperative signal changes remains to be clarified.

Some reports[12,38,39] have proposed that the main cause for failure following a ventral slot procedure in dogs is incomplete spinal cord decompression. In contrast, the ventral slot procedure effectively decompressed the spinal cord in all 3 dogs treated surgically in the present study, and the neurologic deterioration seen in 1 dog was caused by multiple secondary compressions, rather than residual compression.

Spinal cord atrophy can only be objectively determined by means of morphometry. Evaluation of the spinal cord area is recommended because it is a better reflection of spinal cord size.[9] In severe cases of spinal cord atrophy, widening of the subarachnoid space was seen on transverse magnetic resonance images. In the present study, 7 dogs had a decrease in spinal cord area, suggestive of spinal cord atrophy, on follow-up magnetic resonance images. All but one of these dogs also had spinal cord signal changes. The spinal cord atrophy was more pronounced in the medically treated dogs. At first, spinal cord atrophy gave a false impression of a reduction in the degree of spinal cord compression on sagittal follow-up magnetic resonance images. Experimentally, chronic spinal cord compression has been shown to cause a significant reduction in the number of axons and a higher proportion of axons with thinner myelin than normal.[40] These axonal changes, along with the pathologic changes associated with the signal changes, likely account for the spinal cord atrophy. Because spinal cord atrophy was observed in all dogs with signal changes, one can expect these processes to be related. The presence of spinal cord atrophy on follow-up magnetic resonance images from most dogs in the present study suggests that the underlying pathologic process of CSM continues despite clinical improvement after medical or surgical treatment.

In summary, we conclude, on the basis of clinical and MRI findings after a minimum of 1 year's follow-up in Doberman Pinschers with CSM, that the progression of clinical signs and MRI abnormalities is slow in most dogs treated medically. In contrast, decompression by means of a ventral slot procedure appeared to hasten the development of additional areas of spinal cord compression and lesions in dogs with preoperative spinal cord signal changes, but was clinically effective. Further studies with a larger population of surgically treated dogs and longer follow-up times are needed to determine the incidence and clinical importance of these postoperative signal changes.

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