Examination of frozen cross sections of cervical spinal intersegments in nine horses with cervical vertebral malformation: lesions associated with spinal cord compression

S. S. Trostle, R. R. Dubielzig, K. A. Beck

Abstract. Nine horses with clinical and radiographic findings of cervical vertebral malformation that were necropsied and examined using frozen cervical spinal cord cross sections were reviewed. Only cases with actual distortion of the spinal cord due to compression were selected. The goal of the study was to determine the morphologic features responsible for narrowing of the spinal canal and compression of the spinal cord. In individual cases, bony changes are associated with osteochondrosis and osteomyelitis of the dorsal articular facets and osteosclerosis of the dorsal cervical lamina. Soft tissue pathology associated with spinal cord compression included ligamentum flavum hypertrophy, joint capsule swelling and hypertrophy, and synovial cysts. In most cases, a combination of abnormalities was found in horses with spinal cord compression.

Determination of the cause of ataxia secondary to neurologic impairment in horses based on physical and neurologic examinations is difficult. Impingement on the cervical spinal cord is the leading cause of ataxia in the horse. Terms used to describe the syndrome that results in constant or intermittent impingement of the cervical spinal cord include wobblers, equine sensory ataxia, cervical vertebral instability, cervical stenotic myelopathy, and cervical vertebral malformation/malarticulation (CVM). The signalment, history, and clinical and radiographic signs of horses with CVM have been previously reported.

The pathogenesis of CVM in horses remains unclear but appears to be multifactorial. Two general theories have evolved regarding the etiopathogenesis of CVM in horses. The first theory focuses on excessive and/or abnormal biomechanical forces, resulting in pathologic changes of the cervical vertebral canal that cause CVM. The second theory describes a generalized dysmaturity of cartilage and bony development of the cervical vertebrae, leading to CVM. Lesions associated with equine CVM include osteosclerosis of the dorsal cervical lamina, degenerative joint disease, and osteochondrosis (OCD) of the dorsal articular facets. Hypertrophy also occurs in the soft tissues surrounding the cervical vertebral canal, including the ligamentum flavum, joint capsule, and synovial cyst formation. The purpose of this report was to demonstrate by examination of frozen cross sections of cervical spinal intersegments the pathologic changes in horses with CVM that cause spinal cord compression. This technique allows the observer to record the extent of abnormality in both osseous and soft tissues that make up the spinal canal and to assess the relative importance of each component in the cause of spinal cord compression.

Materials and methods

Criteria for selection of cases. Cases were selected for inclusion in this study if they were examined at necropsy using frozen cross sections of the cervical spine and if the frozen sections actually demonstrated distortion of the spinal cord as proof of compression. All cases were examined clinically and radiographed prior to euthanasia. Nine horses that demonstrated cervical spinal cord compression suggestive of CVM were selected for the study.

Horses. Horse ages ranged from 12 months to 6 years (X = 34 months). Four of the 9 horses were Quarterhorses, 2 were Thoroughbreds, and the remaining 3 were represented individually by the following breeds: Tennessee Walking Horse, Trakhner, and American Paint Horse. Of the 9 horses, 5 were intact males, 2 were geldings, and 2 were females. Clinical histories varied from an acute to a slowly progressive onset. Most acute presentations were associated with a traumatic event. All horses had rear limb gait abnormalities (dysmetria, weakness, ataxia), and for those with both fore- and rear limb gait abnormalities, the problems were more pronounced in the rear limbs. Hyperesthesia was noted on dorsiflexion or lateral flexion of the head or neck in 3 of 9 horses.

Postmortem evaluation of frozen spine. In each case, the horse was suspended with its head and neck down immediately after euthanasia. The head and neck were removed by incising at the scapulohumeral joint, which is the most

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caudal area in which removal of the cervical spine is practical. In most cases, the C₆-₇ interspace was still intact. The severed head and cervical spine were suspended from a wooden pole inserted through the musculature dorsal to the spine in the cervical region. The head and neck were positioned in an empty 220-liter drum, placing the severed end up in an effort to minimize loss of cerebral spinal fluid or blood from the sinus vessels. In all cases, the forehead rested on the bottom of the drum so that the head was in flexion. Care was also taken to prevent the cervical spine from twisting when positioning the head and neck in the drum. The suspended head and neck were rolled into a walk-in freezer maintained at -20°C. After several days, the head and neck tissue were solidly frozen, and the frozen tissues were sectioned into rectangular blocks containing the spine. The blocks were then processed as controls (Fig. 1).

The evaluation of the sections for narrowing of the spinal canal, osteosclerosis, osteophyte formation, and hypertrophy of the joint capsule and ligamentum flavum were made subjectively by comparing the affected spines with those of control animals.

The histologic sections were prepared by decalcifying the entire slab and embedding it in paraffin. The larger decalcified sections were cut on an electrically driven sledge microtome, mounted on jumbo glass slides, and stained with hematoxylin and eosin. In most cases, the quality of the histologic sections of the spinal cord tissue after freezing and thawing were not sufficient to identify subtle compressive lesions. Therefore, only cases where compression was seen grossly were included, and we did not attempt to correlate compressive lesions with spinal cord histopathology.

Results

The signalment, history, and clinical, radiographic, and pathologic examination findings of the 9 horses diagnosed with CVM are summarized in Table 1. Postmortem examination of frozen cervical spinal segments demonstrated various lesions associated with CVM in horses. Osteosclerosis and subsequent narrowing of the intervertebral canal (Fig. 2) was noted in all 9 horses. Osteophyte formation indicative of degenerative changes of the dorsal articular facets (Figs. 2-5) was present in horse nos. 3-7 and 9. In 5 of these 6 horses, multiple vertebral interspaces from C3-4 to C6-7 were involved. Osteophyte formation was always affiliated with other pathology, such as joint capsule and ligamentum flavum hypertrophy (horse nos. 3-7, 9) and synovial cysts (horse nos. 3, 7). Abnormalities of the bone and cartilage interface or OCD was found on the dorsal articular facets (Figs. 6, 7) in horse nos. 1, 2, and 4 at intersegments C2-3, C5-6, and C3-4, respectively. Horse no. 2 had a bilaterally symmetric OCD lesion; the other 2 horses had unilateral OCD pathology. Osteochondrosis was associated with narrowing of the spinal canal in all 3 horses. Osteomyelitis of the dorsal articular facet (Fig. 3) was seen in horse no. 8 at C5-6 and was associated with osteophyte formation and synovial membrane hypertrophy. No growth was obtained on culture, and the organism responsible for the osteomyelitis was unknown.

Ligament flavum hypertrophy (Figs. 4, 5) was demonstrated in horse nos. 5, 6, and 9 and was associated with marked degenerative joint disease of the dorsal articular facets and narrowing of the cervical spinal
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Figure 3. Horse no. 8. Cross section of C,,. There is a 2-cm irregular lytic lesion (a) in the ventral caudal dorsal articular facet associated with osteophyte formation, new bone proliferation, and spinal cord compression in an adjacent section. Histologically, the lytic bone lesion was associated with inflammation and was presumed to be a subchondral cyst secondary to osteomyelitis.

canal in all horses. Ligamentum flavum hypertrophy occurred in multiple cervical vertebral interspaces in horse nos. 5 and 6 but not in horse no. 9. There was joint capsule hypertrophy (Figs. 4, 5) in horse nos. 3, 4, 6, 7, and 9 in conjunction with synovial cysts (horse nos. 3, 7) and OCD (horse no. 4). Joint capsule hypertrophy was seen in multiple cervical vertebral interspaces in 4 of the 5 horses. Synovial cysts (Figs. 3, 4) were found in horse nos. 3 and 7. In both horses, the lesions were located at C6-7 and were associated with marked osteophyte formation and joint capsule thickening extending 3-4 intervertebral disc spaces cranially.

Standing cervical vertebral radiographs had been taken of all 9 horses, and myelograms had been made for horse nos. 2, 4, and 7. All 9 horses had radiographic bony irregularities of the dorsal articular facets and all cervical vertebral interspaces, C2-3 to C7-T1, had irregularities of the dorsal articular facets. Multiple vertebral interspaces were involved in all horses except horse no. 1. Vertebral interspaces C4-5, C5-6, and C6-7 were affected in 7 horses. Subluxation or malalignment of cervical vertebral bodies was noted in horse nos. 1, 3, and 4-6. Three of 5 horses had singular subluxation lesions at C2-3, C5-6, or C6-7. The other 2 horses had multiple lesions that involved C6-7 and C7-T1 (horse no. 5) or C4-5, C5-6, and C6-7, (horse no. 6). The most common subluxation site was C6-7 (3 horses). Plain radiographs revealed a narrowed vertebral canal in horse nos. 1-4, 6, and 8. Five of the 6 horses with narrowing of the vertebral canal were affected at a single site. Three horses were affected at C5-6, 2 horses at C6-7, and 1 each at C3-4 and C2-3. Myelograms demonstrated multiple sites of spinal cord compression in all 3 horses (nos. 2, 4, and 7). Vertebral interspace C6-7 was compressed in all 3 horses, C3-4 in horse nos. 2 and 4, and C4-5 in horse no. 7.

Discussion

Examination of cross sections of frozen specimens allowed documentation of the extent and nature of disease affecting both the bone and the surrounding soft tissues of the spinal cord and correlation of the disease process with the cause of spinal cord compression. The 2 main theories involving the pathogenesis of CVM are 1) excessive or abnormal biomechanical forces resulting in instability24 and 2) a dysmaturity in the cartilage and bony development of the cervical spinal canal.29 This limited series of cases demonstrated osteosclerosis of the dorsal lamina, degenerative joint disease, OCD and osteomyelitis of the dorsal articular facets, synovial cysts, and thickening of the ligamentum flavum and joint capsule. All of these changes, with the exception of osteomyelitis of the dorsal articular facets of cervical vertebrae, have been previously reported in association with spinal cord compression, but in this series we were able to document how each pathologic change contributed to spinal cord compression.

Osteosclerosis of the dorsal lamina has been reported in horses,15,24 dogs31 and humans6 with CVM. The cause of osteosclerosis in the dorsal lamina is unclear. Some authors have reported that this condition is secondary to suppression of normal osteocytic osteolysis and is exacerbated by normal compressive forces.15 The investigators hypothesized that this condition may
## Table 1.
Clinical, radiographic, and pathologic examination of horses with cervical vertebral malformation (CVM).

<table>
<thead>
<tr>
<th>Horse no.</th>
<th>Age, sex, breed</th>
<th>History and clinical exam†</th>
<th>Radiographic exam‡</th>
<th>Pathologic exam§</th>
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<td>#1 12 mo, F, TWH</td>
<td>HX: acute ataxia of 2 days GA: 2/4 fore- and 3/4 hind limbs PE: reluctance to dorsoventral flexion of the head and neck</td>
<td>SCR: Dorsal subluxation at C23, with marked narrowing of the vertebral canal and periaxial osteophyte formation of the dorsal articular facets at C23. Radiographic interpretation was CVM at C3.</td>
<td>Marked malalignment and malformation of dorsal articular facets at C23, and caudal dorsal articular facet of C2 had lesions typical of OCD. Dorsoventral flattening of spinous canal with spinal cord compression. Pathologic diagnosis was CVM secondary to OCD of dorsal articular facets with spinal cord compression at C3.</td>
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<tr>
<td>#2 12 mo, M, QH</td>
<td>HX: 2-wk history of large flocculent mass over left gluteal region and progressive rear limb lameness/ataxia. Mass was a seroma secondary to an antibiotic injection. Mass resolved after 5-day hospital stay, but ataxia persisted. GA: 2/4 hind limb</td>
<td>SCR: Periarticular osteophyte formation on the dorsal articular facets of C14, C45, C56, C67, with decreased diameter of vertebral canal at C14 and C45. Myelography: Loss of dorsal and ventral dye columns at C14 and C45 with ventral subluxation at C67. Radiographic interpretation was CVM with multiple areas of spinal cord compression, the most severe at C45.</td>
<td>Profound asymmetry of dorsal articular facets of C4, with bilateral OCD lesions of caudal dorsal articular facets of C4. Dorsoventral asymmetrical narrowing of spinous canal and flattening of spinous canal at C5. Pathologic diagnosis was CVM secondary to OCD of dorsal articular facets with spinal cord compression at C45.</td>
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<tr>
<td>#3 34 mo, M/C, QH</td>
<td>HX: 7-mo history of hind limb lameness that progressed to ataxia GA: 1/4 fore- and 2/4 hind limbs</td>
<td>SCR: Hypertrophic degenerative change of the dorsal articular facets at C14, C45, and C67, with decreased diameter of cranial spinous canals at C1 and C4 and ventral malalignment of C4 in relation to C1. Radiographic interpretation was CVM at C45.</td>
<td>Osteophyte formation on dorsal articular facets and joint capsule thickening at C45, C67, and C5, accentuated at C3, where synovial cyst produced dorsoventral flattening of the spinous canal. Pathologic diagnosis was CVM with spinal cord compression secondary to synovial cyst formation at C45.</td>
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<tr>
<td>#4 13 mo, F, Trak</td>
<td>HX: 2-day history of acute ataxia, recumbent at admission GA: 3/4 fore- and 4/4 hind limbs</td>
<td>CSR: Bulbous enlargement of the articular facets at C14, C45, C67. Mild ventral subluxation noted between C5 and C6. Slight cranial narrowing of the vertebral canal moving caudocranially through the vertebral body of C5. Myelography: Flexed views demonstrated loss of ventral and dorsal contrast column at C14 and mild thinning at C45. Relaxed views showed thinning of both dorsal and ventral contrast columns at C67. Radiographic interpretation was CVM with spinal cord compression during flexion at C45.</td>
<td>Considerable asymmetry and narrowing of spinous canal at C45, small OCD lesion on cranial dorsal articular facet of C5 with spinal cord compression. Spinal cord compression associated with impingement of canal by thickened joint capsule and osteophyte formation from dorsal articular facets. Pathologic diagnosis was CVM secondary to OCD with spinal cord compression at C45.</td>
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<tr>
<td>#5 2 yr, M, Thb</td>
<td>HX: 3-day history of progressive neurologic signs after stumbling out of a racing gate GA: 1/4 fore- and 2/4 rear limbs PE: reluctance to dorsoventral flexion of the head and neck</td>
<td>SCR: Bulbously enlarged, irregular articular facets with periaxial osteophyte formation at C14, C45, C67, C7T1. Mild ventral subluxation between C6 and C7T1. Radiographic interpretation was CVM at C45 and C7T1.</td>
<td>Instability with osteophyte formation and eburnation of articular surface of dorsal facets and thickening of ligamentum flavum at C45, C5, C67. C45 was most severely involved, with dorsoventral narrowing of spinous canal with thickening of ligamentum flavum and spinal cord compression. C45 and C67 had similar but less severe lesions. Pathologic diagnosis was CVM secondary to DJD with spinal cord compression at C45.</td>
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be the result of rapid growth rates and metabolic imbalance. Others have suggested that osteocytic osteolysis is not important in bone matrix turnover, based on the normal size and appearance of lacunae, that abnormal and/or repeated loading stresses on the dorsal lamina result in osteosclerosis. In humans, osteosclerosis is reported to be the result of degenerative changes of the articular facets and/or intervertebral disks, resulting in abnormal stresses on the dorsal laminae. Studies have demonstrated that increased cyclic loading strain results in the deposition of new bone on existing surfaces and internally. Immature bone is even more responsive to repeated cycling load strains; many horses with CVM are <2 years of age.

Osteosclerosis of the dorsal lamina results in a generalized and stenotic vertebral canal. This condition has been quantified by measuring the minimal sagittal diameter (MSD) of the vertebral canal from radiographs. The MSD of horses with CVM is less than that of normal horses. Horses with CVM have a

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Table 1. Continued.

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<tr>
<th>Horse no.</th>
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<td>#6</td>
<td>6 yr, M/C, QH</td>
<td>HX: 3-wk history of progressive ataxia</td>
<td>SCR: Mild irregularity of the dorsal articular facets of C₂₄, C₃₄, C₄₄; marked irregularity at C₂₄. Caudodorsal portions of C₁, C₂, and C₃ vertebral bodies had dorsal deviations of the ventral aspects of vertebral canal and narrowing of spinal canal at C₂₄, joint space. Radiographic interpretation was CVM at C₂₄.</td>
<td>Osteophyte formation and etuburation of dorsal articular facets, joint capsule and ligamentum flavum thickening at C₂₄, C₃₄, C₄₄. Dramatically decreased vertebral canal space and dorsoventral flattening of spinal cord at C₂₄. Pathologic diagnosis was CVM secondary to DJD and spinal cord compression at C₂₄.</td>
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<tr>
<td>#7</td>
<td>38 mo, M, QH</td>
<td>HX: 48-hr history of mild abdominal pain and ataxia</td>
<td>SCR: Irregular dorsal articular facets at C₂₄, C₃₄, C₄₄. Myelography: Flexed views demonstrated loss of the ventral contrast column at C₂₄ and C₃₄ by cranial vertebral bodies of C₁ and C₂, respectively. Radiographic interpretation was CVM secondary with spinal cord compression at C₂₄, C₃₄.</td>
<td>Irregularity of dorsal articular facets with osteophyte production at C₂₄, C₃₄, with mild proliferation of joint capsule and bulging of joint capsule into spinal canal. Most severe abnormality was at C₂₄, asymmetry of oral articular facets with dramatic thickening of joint capsule and synovium extending to ligament flavum. 1-cm-diameter pocket of synovial fluid (synovial cyst) extended medially and ventrally underneath spinal cord, resulting in distortion of spinal cord. Pathologic diagnosis was CVM secondary to synovial cyst and spinal cord compression at C₂₄.</td>
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<tr>
<td>#8</td>
<td>34 mo, M, Paint</td>
<td>HX: 3 wk of progressive ataxia after falling on icy spot in pasture</td>
<td>SCR: Osteophyte formation on the dorsal articular facets at C₂₄ and C₃₄, with mild narrowing of the cranial vertebral canal of C₂. Radiographic interpretation was CVM at C₂₄ and C₃₄.</td>
<td>Red lytic lesion at C₂₄ of right ventral articular surface, with synovial bulging and osteophyte proliferation. Lesion demonstrates replacement of subchondral bone by dense, fibrovascular connective tissue and extensive bony lysis. Pathologic diagnosis was CVM secondary to low-grade osteomyelitis at C₂₄.</td>
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<tr>
<td>#9</td>
<td>27 mo, Thb</td>
<td>HX: 6 wk of progressive ataxia</td>
<td>SCR: Osteophyte formation on dorsal articular facets at C₂₄, C₃₄, C₄₄, with narrowing of the spinal canal at C₂₄. Radiographic interpretation was CVM at C₂₄, C₃₄, C₄₄, with spinal cord compression at C₂₄.</td>
<td>Joint capsule hypertrophy at C₂₄, C₃₄, C₄₄. Degenerative changes of dorsal articular facets at C₂₄, C₃₄, C₄₄. Pathologic diagnosis was CVM secondary to DJD with spinal cord compression at C₂₄.</td>
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* F = female; M = male; M/C = gelding. QH = Quarterhorse; TWH = Tennessee Walking Horse; Thb = Thoroughbred; Trak = Trakhner, Paint = Paint of America.
† HX = history; GA = gait abnormality; PE = physical exam.
‡ SCR = standing cervical vertebral radiographs.
§ OCD = osteochondrosis; DJD = degenerative joint disease.
generalized decreased MSD rather than a focal lesion, supporting the theory of cervical vertebral canal dysmaturation. However, a decreased MSD in horses may result in altered biomechanical forces and subsequent CVM.

All of the horses in our case series had lesions of osteosclerosis associated with an increase in the dorsal lamina bone contributing to the loss of space in the vertebral canal. Osteosclerosis was apparently not a primary lesion in the animals examined because it was always associated with other abnormalities, such as OCD (3 horses), degenerative changes of the dorsal articular facets (6 horses), and osteomyelitis (1 horse).

Degenerative joint disease of the dorsal articular facets has been reported in horses, humans, and dogs with CVM. Osteophyte formation is believed to be the result of abnormal biomechanical forces at an articulation leading to instability; however, the severity of degenerative changes has not been correlated with the clinical signs in horses. Retention of the cartilage matrix in the dorsal lamina was reported in 82% of the horses with increased fibrocartilage at the attachment site of the ligamentum flavum to the dorsal lamina.

Six horses in the series had changes associated with degenerative joint disease. In all horses with degenerative changes, there was reorganization of the dorsal articular facets due to new bone formation associated with ligamentous and joint capsule attachment sites.

Figure 5. Horse no. 6. Gross (left) and histopathologic (right) cross section of C6-7. There is degenerative joint disease with joint capsule swelling, osteophyte formation, thickening of ligamentum flavum, and a synovial cyst (a) dorsal to the spinal cord compressing the spinal cord dorsoventrally. Bar = 1 cm.

Figure 6. Horse no. 1. Histopathologic cross section of C2-3. The diameter of the spinal canal is extremely small. The dorsal articular facet demonstrates osteochondrosis (black arrows) with a severe disorganized osteochondral surface. Bar = 1 cm. 
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Figure 7. Horse no. 2. Gross (left) and histopathologic (right) cross section of C5. There is profound asymmetry and instability of the dorsal facets with osteochondrotic lesions (black arrows on histopathologic section) seen on both the left and the right articular facets, but most prominently on the right. The spinal canal is extremely small. Bar = 1 cm.

The retention of the fibrocartilage matrix is a disorder in endochondral ossification or OCD. Osteochondrosis has been reported in horses 19,29 and dogs 5 with CVM. In one study, the severity of OCD was greater in horses with CVM, but the frequency was not in osteochondrotic versus nonosteochondrotic horses. 29 Also, there was an increase in frequency and severity of OCD lesions in the appendicular skeleton and an increase in severity of vertebral OCD in horses affected with CVM. 29 Some authors believe that degenerative changes of the articular facets in horses with CVM is secondary to a primary OCD lesion. 15,19,24,29

Osteochondrosis was the sole cause of spinal cord compression in 2 horses and was associated with degenerative changes of the dorsal articular facet in 1 horse with spinal cord compression. All 3 horses had severe osteochondrotic and developmental abnormalities and were 3 of the youngest horses (12-13 months of age) in the study. Unfortunately, there was no record of examination of the appendicular skeleton for other OCD lesions.

Osteomyelitis of thoracolumbar vertebral bodies with spinal cord compression has been reported in horses. 13 Vertebral osteomyelitis is generally found in younger horses, and a nidus for infection (lung, umbilicus, or muscular abscess) has often been identified. Most of these lesions have been demonstrated in the thoracolumbar spine rather than the cervical spine. However, osteomyelitis of the dorsal articular facets of the cervical spine has not been reported in the literature. Mycobacterium bovis 10 and Brucella abortus 14 have been implicated in vertebral body osteomyelitis of the adult horse. In foals, Streptococcus zooepidemicus, Escherichia coli, Salmonella typhimurium, Rhodococcus equi, and Staphylococcus spp. have been isolated from vertebral body osteomyelitis. 14

Osteomyelitis of cervical vertebral articular facets has not been reported previously. The lack of growth on culture may be a disadvantage of using frozen cervical spinal intersegments to examine lesions of CVM. The inciting cause of the osteomyelitis is unknown, but this condition may be secondary to an intramuscular injection of the neck.

Increased stresses by overriding of adjacent dorsal laminae can cause excessive stretching and tension of the soft tissue support structures of the vertebral column. Horses with CVM exhibit hypertrophy of the ligamentum flavum and joint capsule both grossly and histologically. 24 Ligamentum flavum hypertrophy has been reported in humans 1 and dogs 30,31 with CVM. Pathology of the ligamentum flavum is the result of fibrovascular and fibrocartilaginous proliferation. 24 The increase in fibrocartilage attachments of the ligamentum flavum to the dorsal lamina may be associated with active osteoblastic activity at the site of attachment to bone. The increase in osteoblastic activity may result in the osteophyte formation that occurs at ligamentous attachment sites in degenerative joint disease.

Six horses had thickening of the soft tissue that surrounds the spinal cord. The joint capsule alone was thickened in 1 horse, and the joint capsule and the ligamentum flavum were both thickened in 5 horses. Soft tissue hypertrophy was associated with various other lesions.

Synovial cysts can cause spinal cord compressions in horses 8 and humans. 11 These cysts are believed to be the result of degenerative joint disease of the articular facets with secondary herniation of synovial tissue from the joint cavity. Two horses in the series had synovial cysts. In both cases, there was joint capsule and ligamentum flavum thickening and osteophyte for-
mation, indicating coexisting degenerative joint disease. In the 2 horses in this study and 1 horse in a previous study, all 3 lesions were located at C6-7. Prevalence for synovial cyst formation at this site remains unclear at this time. Frozen cross sections of the control horses demonstrated no variation in synovial morphology at C6-7.

CVM has been divided into 3 categories. Type I CVM is a severe malformation in which the spinal cord is fixed in flexion at a given point. This form of CVM is rare and is usually present at birth and predominantly located at C2-3 Type II CVM, commonly called cervical vertebral instability, occurs with malformation of the dorsal articular facets. The malformation leads to malarticulation, which can produce or exacerbate spinal cord compression when the head is in flexion. Type II lesions occur commonly in horses between 8 and 18 months of age and primarily at the C3-4 and C4-5 vertebra articulations. Type III CVM, commonly termed cervical static stenosis, is characterized by enlargement of the dorsal articular facets and surrounding soft tissue structures, resulting in a dorsal or dorsolateral spinal cord compression regardless of neck position. Type III CVM lesions primarily involve cervical vertebrae from C5 to C7 and usually occur in horses > 2 years of age. A similar syndrome has been reviewed in dogs and humans. The variations in clinical, radiographic, and pathologic findings may represent different stages of the inciting causes and development of CVM.

CVM has been reported more commonly in male than female horses and with greater incidence in Quarterhorses and Thoroughbreds. Some authors have suggested that CVM has a genetic predisposition in faster growing “performance” animals, but others have refuted this theory. These findings may be biased by economics, management, and the intended use of these horses and/or an over-representation of these breeds at referral hospitals capable of performing advanced diagnostic and surgical treatments.

The signalments of the 9 horses with CVM in our study were similar to those reported for other horses with CVM. However, 8 of the 9 horses were <38 months of age. Six of the 9 horses were either Quarterhorses or Thoroughbreds, and 7 of the 9 horses were either intact males or geldings. The onset of clinical signs of CVM can be insidious, but owners/trainers sometimes report acute clinical signs, often following a traumatic incident? Neurologic signs may stabilize and even regress for a period of time; however, complete spontaneous recovery is rare, and most cases of CVM progressively deteriorate. Initial clinical signs may vary from a subtle lameness to recumbency. Horses with CVM typically have a wide-based stance to both fore- and rear limbs because of proprioceptive deficits. Gait abnormalities consist of weakness, ataxia, dysmetria, and spasticity. CVM results in gait abnormalities that are generally more noticeable in the rear limbs, but often the forelimbs are involved to a lesser extent. In most cases, the clinical signs are symmetric; however, lateralizing, asymmetric clinical signs can occur.

Many papers have been written on the protocols and interpretation of standing radiographic and myelographic studies of the cervical spine. Survey radiographs of the cervical vertebrae are helpful for identifying proliferative changes and vertebral body malalignment but are inefficient in comparison to myelography for the diagnosis of compressive spinal cord lesions. In a study of 306 ataxic horses undergoing radiography and myelography, the most common sites of spinal cord compression were (in order) C3-4, C6-7, C5-6, and G-5. Of the horses with spinal cord compression, myelography revealed that 29% were affected at multiple sites. The recent advances in surgical techniques to treat CVM have made it imperative to identify the site(s) of the lesion(s) before attempting surgical intervention. A study utilizing equine cadavers and contrast-enhanced computed tomography identified generalized stenosis of the cervical spinal canal in horses with CVM. Computed tomography also demonstrated the false-positive identification of spinal cord compression by myelography. In that same study, lateral compressive lesions of the peripheral nerve roots by malformed articular facets that encroached on the intervertebral foramen were documented.

The pathologic changes of generalized decreases in vertebral canal diameter and OCD in 3 horses support the theory of dysmaturation of the cervical vertebral canal. The pathologic changes of degenerative joint disease and ligamentum flavum and joint capsule hypertrophy in 6 horses support the theory of abnormal or excessive biomechanical forces. One horse had changes consistent with both dysmaturation and excessive biomechanical forces. Osteochondrosis is a factor in the pathogenesis of CVM because it causes gross abnormalities in the development of the spinal canal or contributes to instability and increases the risk of degenerative joint disease. Five horses had changes of degenerative joint disease of the dorsal articular facet without signs of OCD, which suggests that OCD is not a universal factor in the pathogenesis of CVM.

References