Cervical stenotic myelopathy (CSM) is one of the most common causes of spinal ataxia in young horses. The layman’s term for CSM is wobbler syndrome. Alternative scientific terms to describe the condition include cervical vertebral malformation/instability and cervical compressive myelopathy. In this condition, neurologic gait deficits result from compression of the spinal cord by malformed cervical vertebrae. Developmental orthopedic diseases of the appendicular skeleton, such as physitis, joint effusion, osteochondrosis, and flexural limb deformities, occur more frequently in horses with CSM. A direct cause-and-effect relationship between osteochondrosis and CSM has not been identified; however, the associative relationship indicates that the pathophysiology of these two conditions is similar. The cause of CSM is likely multifactorial, consisting of genetic and environmental influences. It is unlikely that CSM is heritable by simple Mendelian dominant/recessive patterns. The mode of inheritance more likely involves multiple alleles and variable penetrance, which determine genetic predisposition to CSM. A high plane of nutrition, micronutrient imbalance, rapid growth, trauma, and abnormal biomechanical forces likely contribute to the development of CSM in genetically predisposed individuals.

Spinal cord compression can be dynamic or static in horses with CSM. Dynamic compression results from vertebral instability and produces intermittent spinal cord compression during ventroflexion of the neck; compression is relieved when the neck is in the neutral position. Static compression is defined as continuous spinal cord impingement regardless of cervical position. Both static and dynamic compression are associated with narrowing of the vertebral canal from C3 to C6, regardless of the site of spinal cord compression, indicating that generalized vertebral canal stenosis is an important factor in the pathophysiology of CSM.

CSM has been increasingly recognized as a cause of spinal ataxia and cervical pain in horses older than 10 years. Spinal cord compression in this age group primarily results from osteoarthritic enlargement of the caudal cervical articular facets, with or without some degree of vertebral instability. Affected horses appear neurologically normal until osteoarthritic enlargement of the articular facets impinges on the cervical spinal cord. Not every horse with osteoarthritis of the caudal cervical vertebrae develops spinal cord compression. Similar to humans who develop spinal cord compression secondary to articular facet osteoarthritis in middle age, horses that develop neurologic gait deficits are predisposed due to an existing narrowed vertebral canal.

**DIAGNOSTIC CRITERIA**

**Historical Information**

- Most horses develop clinical signs at 6 months to 3 years of age.
- Males are more frequently affected than females (3:1).
• Thoroughbreds are particularly predisposed (prevalence: 2%), although CSM has been reported in most light and draft breeds.

• Affected horses grow rapidly and are large for their age.

• Some owners report acute onset of clinical signs in their horses, whereas others indicate that their horses have become increasingly clumsy.

**Physical Examination Findings**

• Symmetric spinal ataxia; hindlimb deficits are typically one neurologic grade worse than forelimb deficits (Box 1).

• Neurologic examination should be performed at a walk. Circling, elevating the head, and maneuvering over obstacles/inclines accentuate neurologic deficits.

• Circumduction, truncal sway, base-wide stance, delayed responses to proprioceptive positioning, toe dragging, and knuckling are common findings on the neurologic examination.

• When prompted to back up, the horse may stand base wide, lean backward, drag its hindlimbs, and step on a hind foot with a front foot.

• Moderately to severely affected horses have lacerations on their heel bulbs and have hooves that are chipped, worn, or squared at the toe.

• The musculature of the neck may appear disproportionately thin compared with the rest of the body.

• Horses with arthrosis of the caudal cervical spine may exhibit signs of cervical pain during manipulation of head position.

• The results of the cranial nerve examination are unremarkable, and mentation is normal.

• Affected horses may have concurrent developmental orthopedic disease of the appendicular skeleton (i.e., osteochondral fragments, physitis).
Laboratory Findings

- Complete blood count and serum chemistry analysis results are unremarkable.

- The results of a cytologic evaluation of the cerebrospinal fluid (CSF) are unremarkable in most cases. Horses with acute onset of clinical signs may have mild to moderate elevation in CSF protein (80 to 100 mg/dL; normal: ≤80 mg/dL).

Other Significant Diagnostic Findings

Radiographic Examination of the Cervical Spine

Determination of Sagittal Ratio

A precise lateral radiograph of the cervical vertebrae is required to obtain accurate values. The sagittal ratio is obtained by dividing the minimum sagittal diameter of the vertebral canal by the width of the corresponding vertebral body (Figure 1). The minimum sagittal diameter is measured from the dorsal aspect of the vertebral body to the ventral border of the dorsal laminae, and the vertebral body width is measured perpendicular to the vertebral canal at the widest point of the cranial aspect of the vertebral body. The sagittal ratio eliminates error due to magnification because the vertebral canal and vertebral body are in the same anatomic plane. The sagittal ratio should exceed 52% from C3 to C6 and 56% at C7 in horses heavier than 704 lb (320 kg). The intervertebral sites most frequently affected by dynamic compression are C3 to C4 and C4 to C5. Static compression occurs predominately in the caudal cervical region: the C5–C6 and C6–C7 articulations.

When examining lateral survey radiographs, the clinician may also measure the intervertebral sagittal ratios. Sites of measuring for intervertebral sagittal ratios are described by the minimum distance between the most craniodorsal aspect of the vertebral body to the most caudal aspect of the vertebral arch, divided by the widest point of the cranial aspect of the vertebral body (Figure 2). Because compression may occur between adjacent vertebrae, the intervertebral sagittal ratio provides an additional diagnostic tool when cervical radiographic images are interpreted. Intervertebral sagittal ratio cut-off values have recently been determined by Hahn et al to be ≤0.440 at C2 to C3, ≤0.505 at C3 to C4, ≤0.510 at C4 to C5, ≤0.470 at C5 to C6, and ≤0.550 at C6 to C7.

Subjective Evaluation of Bony Malformation

Five categories of malformation are subjectively assessed during the interpretation of cervical radiographs. The presence of characteristic vertebral malformations supports a diagnosis of CSM:

- Subluxation (malalignment) between adjacent vertebrae
- Flare of the caudal physis of the vertebral body (ski-slope lesion)
- Abnormal ossification patterns
- Caudal extension of the dorsal laminae
- Degenerative arthropathy

Subluxation and caudal epiphyseal flare are the most valuable parameters to differentiate affected and unaffected horses. Degenerative arthropathy is the most frequent and severe malformation in horses with CSM but also occurs in 10% to 50% of nonatactic horses and is the most frequent and severe vertebral malformation in horses without CSM. Therefore, degenerative arthropathy, in the absence of stenosis or other bony malformations, should be interpreted cautiously.
Myelographic Examination
Criteria for Spinal Cord Compression
Spinal cord compression has traditionally been defined as a 50% reduction of the dorsal contrast column at the site of compression compared with the dorsal contrast column just cranial to the site. This criterion may be too conservative, resulting in false-positive results. On the other hand, the alternative diagnostic criterion—a dorsal contrast column of less than 2 mm—may result in false-negative results. To reduce false-negative and false-positive results, van Biervliet et al suggest a new criterion described as a 70% reduction of the dural diameter (entire contrast column) compared with adjacent intravertebral dural diameter measurements. Because of the difficulty of obtaining dorsoventral radiographic views, myelographic examination may result in false-positive results if lateral projections (osteoarthritic articular processes) compress the spinal cord.

Classification of Lesions
Dynamic spinal cord compression is the narrowing of the dorsal and ventral contrast columns with the neck in the flexed position. It is relieved with the neck in the neutral position. Static spinal cord compression is continuous spinal cord compression regardless of cervical position.

Summary of Diagnostic Criteria
Cervical radiographs are obtained to determine the likelihood of spinal cord compression. The clinician should classify the patient in one of the following categories:

- Low sagittal ratio (<48%; moderate to severe bony malformation): Perform a myelographic examination
to identify sites of spinal cord compression and classify lesions as static or dynamic.

- **Marginal sagittal ratio** (48% to 56%; mild to moderate bony malformation): Perform a myelographic examination to confirm or rule out CSM.
- **High sagittal ratio** (>56%; minimal bony malformation): Pursue other diagnostic differentials.

Myelographic examination must be performed to confirm the diagnosis, identify the site(s) of spinal cord compression, and classify the lesion(s) as static or dynamic. A 70% reduction in dural diameter has been suggested as the new diagnostic criterion.

**Differential Diagnosis**

- **Equine protozoal myeloencephalitis**: Neurologic examination may reveal multifocal or asymmetric neurologic gait deficits, although symmetric spinal ataxia can occur in horses with equine protozoal myeloencephalitis. The results of cytologic analysis of CSF may be normal or reveal mild inflammation (mononuclear pleocytosis). Immunoblot analysis for antibody against *Sarcocystis neurona* in CSF shows positive results. An indirect fluorescent antibody test may be conducted on whole blood or CSF to detect antibodies to *S. neurona*. The test maintains validity for CSF samples in the presence of blood contamination up to 100,000 erythrocytes/µL.

- **Equine herpes myelitis**: Neurologic examination may reveal urine dribbling, poor tail tone, and progressive ascending myelitis. CSF analysis often demonstrates xanthochromia and albuminocytologic dissociation (high CSF protein, normal cell count). Real-time polymerase chain reaction testing has been developed for rapid identification of the neuropathic strain of equine herpesvirus 1. Samples of blood, respiratory secretions, and nerve tissue can be analyzed within 24 hours using real-time polymerase chain reaction testing.

- **Equine degenerative myeloencephalopathy**: Diagnosis of exclusion. Age at onset of neurologic signs is 6 months to 2 years. Neurologic examination reveals symmetric spinal ataxia; hyporeflexia may be detected. Unremarkable results of CSF analysis; negative results of a myelographic examination. Serum vitamin E concentrations may be low.

- **Traumatic injury**: Horses with traumatic injury have cervical pain and resist cervical manipulation. Cervical radiographs should be obtained to differentiate traumatic injury from developmental malformation.

**TREATMENT RECOMMENDATIONS**

**Surgical Treatment**

**Ventral Stabilization**

- Fusion of the vertebral bodies at the affected site in the extended position using a Bagby basket or partially threaded titanium baskets.
- Provides immediate decompression of dynamic compressive lesions and prevents repetitive spinal cord trauma.
**The Way It Was**

In 1938, Errington coined the term wobbler to describe any horse with spinal ataxia. In 1978 in *The Cornell Veterinarian*, Mayhew et al presented a comprehensive investigation differentiating the causes of equine spinal ataxia. Since then, veterinarians have been discouraged from using the terms wobbler syndrome or true wobbler, which are now reserved to describe horses with spinal cord compression due to stenosis of the cervical vertebrae.


- Provides delayed decompression of static compressive lesions (weeks to months) due to remodeling of soft tissue and bony structures after stabilization.
- Improves the neurologic status in 44% to 90% of horses, and 12% to 62% return to athletic function. Projected improvement in neurologic status is one or two neurologic grades.
- Dynamic compressive lesions have a more favorable postoperative prognosis than static compressive lesions; caudal cervical lesions (C6 to C7) carry a less favorable prognosis than midcervical lesions (C3 to C5).
- Correction of triple-level lesions is discouraged based on the prognosis.
- Postoperative complications include implant failure, vertebral body fracture, spinal cord edema, and seroma formation.

**Dorsal Laminectomy**

- Removal of portions of the dorsal lamina, ligamentum flavum, and joint capsule overlying the site of compression provides immediate relief of static compressive lesions.
- Improves the neurologic status of 40% to 75% of horses with static compressive lesions.
- Postoperative complications include vertebral body fracture, spinal cord edema, and seroma formation.
- This procedure is rarely performed because of its difficulty and postoperative complications.

**Conservative Treatment**

**Dietary management** is indicated for horses in which surgical treatment is not an option and euthanasia is being considered. Dietary management is commonly used when conservative therapy is chosen. The goals are
to retard bone growth, enhance bone metabolism, and allow the vertebral canal diameter to enlarge to relieve spinal cord compression. This approach is recommended for horses 1 year of age or younger at the time of diagnosis. This treatment approach is controversial; whether the following approach can achieve the above treatment goals has not been proven.

• Restrict dietary energy and protein (65% to 75% of National Research Council [NRC] recommendations).
• Maintain balanced vitamin and mineral intake (minimum: 100% NRC recommendations).
• Supplement vitamins A and E at three times the NRC recommendations.
• Supplement selenium to 0.3 ppm.
• Provide roughage by pasture or low-quality (6% to 9% crude protein) grass hay.
• Reduce activity to avoid repetitive spinal cord injury from dynamic instability.

**Supportive Treatment**

- Antiinflammatory therapy is indicated for horses with acute onset of clinical signs.
- Phenylbutazone (2.2 mg/kg PO or IV q12h for 5 to 7 days).
- Cervical articular process joint injection (40 mg of methylprednisolone and 125 mg of amikacin) may be performed with ultrasound guidance (Figure 3). This may provide temporary pain relief and decrease inflammation associated with osteoarthritis of the articular facets.

**Patient Monitoring**

- Neurologic examination and cervical radiography should be conducted every 90 days in horses receiving dietary management.
- Neurologic examination should be conducted every 60 days after surgical intervention.
- Horses that develop cervical pain (implant instability) or worsening in neurologic status should be reevaluated immediately.

**Postoperative Recovery**

- The duration of convalescence and rehabilitation following surgery is approximately 6 to 12 months.
- Depending on the capability and neurologic status of the horse, an individualized exercise program should be designed to promote muscular strength.
- Extended exercise at slow speed, including ponying and lunging on inclines, is recommended during rehabilitation.
- Neurologic examination can help determine the point at which the horse is competent to return to athletic function following surgery.
- Significant improvement in neurologic status is unlikely beyond 1 year after surgery.

**PROGNOSIS**

**Favorable Criteria**

Horses should demonstrate an improvement in neurologic status within 90 days after the initiation of treatment (dietary or surgical).

- Horses with neurologic gait deficits for less than 1 month before surgery are more likely to return to athletic function than are horses with neurologic signs for more than 3 months.

**Unfavorable Criteria**

Horses that fail to improve in neurologic status within 6 months of the initiation of treatment (dietary or surgical) are unlikely to respond significantly to treatment.

**RECOMMENDED READING**


