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Aetiology
The cause of neurological disease is compression of the cervical spinal cord, hence the term compressive myelopathy. The compression may be static, that is the compression is present constantly with the neck in a neutral position, or dynamic and only present intermittently when the neck is either flexed or extended. The second situation is often referred to as cervical vertebral instability. The aetiology of equine cervical stenotic myelopathy (CSM) in most cases is not known. Several basic syndromes of compressive myelopathy, based on age of occurrence, are recognised:

- Equine stenotic myelopathy (ESM) in immature horses (<3 years of age, depending on breed) that is often associated with developmental joint disease in the axial and appendicular skeleton. The fundamental underlying defect appears to be a narrow diameter of the cervical vertebral canal or structural instability of the vertebral column.
- Compressive myelopathy in mature horses, >4 years (usually >7 years) of age, associated with osteoarthritis of the articular facets of the caudal cervical vertebrae, with subsequent impingement of the vertebral canal by bony and soft tissue proliferative lesions associated with osteoarthritis.
- Miscellaneous causes of cervical cord compression by neoplasia (melanoma, sarcoma, lymphoma), trauma (cervical vertebral fractures), arachnoid or synovial cysts or, rarely, discospondylitis.

Epidemiology
Occurrence
The disease in young horses occurs sporadically throughout the world. The disease in young horses is sometimes endemic on farms or studs, and in particular lines of horses. There is a suggestion of a familial tendency for the disease, although this has not been well documented. The morbidity rate may be as high as 25% of each foal crop on individual Thoroughbred farms, although prevalence in Thoroughbred horses is estimated at 0.5–2.0%. Eighty-three of 4319 horses (2%) examined in Normandy by necropsy had a diagnosis of CVCM.

The disease in young horses is commonly recognised in Thoroughbred, Standardbred and Warmblood horses as overrepresented, but other breeds can be affected. Affected horses are usually younger. The disease occurs in horses less than 4 years of age, with most cases occurring in 1–3-year-old horses. Males are more commonly affected than are females.

Pathogenesis
The disease is attributable to injury to the spinal cord as a result of compression by either soft tissue (joint capsule, intervertebral ligaments or, rarely, intervertebral disk material) or cartilage and bone.

Constant or intermittent pressure on the spinal cord causes necrosis of white matter and neurons at the site of compression, and degeneration of fibres of ascending and descending tracts caudal to the site of compression. The ascending tracts are those associated with general proprioception whereas the descending tracts are upper motor neurones. These tracts are located superficially in the dorsolateral aspect of the cervical spinal cord and damage to them results in signs of ataxia and weakness. Tracts from the caudal limbs are more superficial, and therefore more easily injured, than are tracts associated with the cranial limbs. Consequently, clinical signs are usually more severe in the hindlimbs. The spinal cord lesions are usually, but not always, bilaterally symmetrical, as are the clinical signs. Proprioceptive pathways are disrupted, causing the signs of ataxia (incoordination) typical of the disease. Clinical signs vary depending on the site of the lesion.

Clinical findings
The onset of clinical signs is sometimes acute in young horses and there may be a history of trauma, such as falling. However, the onset of clinical signs in ESM in both young and mature horses is usually gradual and insidious and in mildly affected horses the nervous disease may be mistaken for lameness of musculoskeletal origin. Affected horses are bright and alert and have a normal appetite. There may be evidence of pain on manipulation of the neck, especially in mature horses, or on firm pressure over the lateral facets.

The severity of clinical signs varies from barely detectable to recumbency. There are no defects of cranial nerves, with the exception of the cervicofacial reflex.

Mildly affected horses may have deficits that are difficult to detect and only apparent under saddle or at high speed. The owner may complain of poor performance of a racehorse or dressage animal, of an animal that frequently changes leads or that is poorly gaited. Careful examination may reveal excessive circumduction of the hind feet, stumbling, and pacing when the head is elevated.

Moderately affected animals have truncal sway, the body of the horse and hind quarters swaying laterally when the horse is walked in a straight line, and excessive circumduction of the hind feet. Having the horse move in a very tight circle about the examiner often causes the circumduction to become worse in the outside hind leg and the horse to place one foot on top of the other. Affected horses will sometimes pace when walked in a straight line with the head elevated. Blindfolding the horse does not exacerbate the signs. Affected horses will stumble when walked over low objects, such as a kerb, and will knuckle at the fetlocks and stumble when walked down a steep hill.

Severely affected horses often fall easily when moved or are unable to stand. The horses are bright and alert, but anxious, and display marked truncal sway and ataxia. When standing they will often have their legs in markedly abnormal positions.

Mature horses with disease secondary to arthritis of the articular facets can have hypalgesia of the skin overlying those regions and atrophy of the cervical musculature.

Horses with lesions in the cervical spinal cord cranial to C6–C7 have signs in both fore- and hindlimbs. The hindlimbs are more severely affected and the signs are usually, but not always, bilaterally symmetrical. Lesions of the cervical intumescence (C6–T2) may cause signs that are more severe in the forelimbs than in the hindlimbs. Lesions at this site may also cause signs typical of brachial plexus injury. Focal muscle atrophy is not characteristic of CVCM or CVI and there are never signs of cranial nerve, cerebral or cerebellar disease.
After initial progression the clinical signs usually stabilise or partially resolve. However, complete spontaneous recovery is very unusual. Death is unusual unless it is by misadventure, although many affected animals are subjected to euthanasia for humane reasons.

**Radiographic examination**

Radiographic examination of cervical vertebral column of affected horses reveals narrowing of the spinal canal - whether measured as intra- or inter-vertebral ratios. This has diagnostic utility, e.g. a ratio of spinal canal to vertebral body diameter of less than 50% for C4 is associated with a 28-fold increase in the probability of ESM. Other measures of spinal canal diameter are useful in the detection of stenosis and compressive myelopathy. Other radiographic signs consistent with ESM or CVI include:

- Encroachment of the caudal vertebral physis dorsally into the spinal canal ("ski jump lesion")
- Extension of the arch of the vertebra over the cranial physis of the next vertebra
- Sclerosis of the spinal canal
- Kyphosis between adjacent vertebra
- Degenerative joint disease of the articular facets.

However, these signs are also common in normal horses and have poor predictive value.

Myelography has been considered to provide the definitive ante mortem confirmation of spinal cord compression. However, the sensitivity of this technique, using a 50% reduction in the width of the dorsal dye column as a cut-off for diagnosis of the disease is 53% (95% confidence interval 34–72%, n = 22) and the specificity is 89% (95% confidence interval of 84–93%, n = 228). These results indicate that a positive finding on myelography is highly suggestive of the disease, but that a negative finding does not eliminate the possibility of the disease. The false positive rate is increased to 12–27% for compression at mid-cervical sites during neck flexion. Myelography is superior in diagnosing compressive lesions at C6–C7 than at more proximal sites. Occasionally the compression is lateral rather than dorsoventral and is not readily apparent on routine myelography.

**Further reading**

