Neurologic Examination in the Horse and Medical and Surgical Approaches for Treating Cervical Myelopathies

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I. NEUROLOGIC EXAMINATION

Diseases affecting the central nervous system of the horse are common and often interfere with performance. Although gait deficits secondary to neurologic disease are often obvious, some conditions can lead to only subtle gait changes that may be difficult to recognize as being secondary to neurologic disease. The gait deficits in these cases may either be mistaken for musculoskeletal disease or may even occur simultaneous with musculoskeletal disease. If the two co-exist, diagnosis of either neurologic or musculoskeletal disease is significantly complicated. The key to a successful neurologic examination is to do the examination in a systematic and organized fashion.

The goals of the neurologic examination are to 1) determine whether disease of the neurologic system exists; 2) localize the lesion to a particular area of the nervous system (Table 1); and 3) describe and record the responses as a baseline for future evaluations. There are many good sources that describe how to complete a neurologic examination in the horse. Here, I will provide a brief overview of some of the critical components of a neurologic examination. First one needs to know the signalment of the horse since it is well known that there are risk factors for certain diseases based on breed, gender, and age of the animal (e.g. cervical vertebral stenotic myelopathy, cerebellar abiotrophy, equine motor neuron disease, equine protozoal myeloencephalitis). The history of the disease is important to learn, in particular when the onset of clinical signs occurred and whether the disease is static, progressive, or recurrent. Some conditions may be present since birth and are progressive, whereas others may be associated with a move from one location to another, or may be secondary to trauma. A good physical examination will allow the examiner to find unusual and or unexpected abnormalities such as tachycardia or an elevated temperature. In addition, a good physical examination may point out lesions associated with an abnormal gait or areas of asymmetry (head, muscles).

The neurologic examination starts with an evaluation of the horse’s behavior and mentation, preferably in an area the horse is comfortable in (stall, paddock). Abnormal mentation includes depression, obtundation and coma. Abnormal behaviors that may be discovered include seizure activity, epilepsy, headpressing, circling, cortical blindness, narcolepsy, or headshaking. At this time the horse’s stance and posture can also be evaluated. Once the horse is in a safe area for further evaluation, one can walk around the horse to look at other aspects of stance and posture, look for the presence of proprioceptive deficits, and determine symmetry of muscle development. The author prefers to start the remainder of the neurologic examination by working from head to tail and starts with evaluation of cranial nerve function before evaluation of sensation and finally gait. Reflexes can be evaluated in foals or recumbent horses.

Careful assessment of cranial nerve function is important since there are a number of diseases that may result in dysfunction of those nerves in addition to abnormalities found elsewhere. This can be found in particular with diseases such as *polyneuritis equi* and *equine protozoal myeloencephalitis*. Furthermore, if there are deficits noticed in multiple cranial nerves, there may be central disease for example in the area around the brainstem since that is where most cranial nerves originate. Deficits of the afferent pathways (sensory) would include reduced smell, taste, vision, hearing, or balance and specific proprioception. Deficits of the efferent pathways (motor) would include reduced ability to change pupil diameter, lesions of eyeball movement, reduced muscle of mastication mass, altered facial expression, reduced ear play, problems with swallowing, vocalization, and reduced tongue movement or tone. The most commonly seen deficits of cranial nerves in the horse include facial nerve paralysis, head tilt, laryngeal dysfunction, and dysphagia.

Evaluation of the gait in horses suspected to have neurologic disease aims at specifically identifying the presence of ataxia, paresis, dysmetria, and spasticity. Ataxia is considered a lack of coordination of muscle movements and in the horse typically
<table>
<thead>
<tr>
<th>Neuroanatomic Localization</th>
<th>Predominant Clinical Signs</th>
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<tbody>
<tr>
<td>Brain – Cranial to foramen magnum</td>
<td>Cerebral cortex: Postural deficits, seizures, altered mentation, blindness</td>
</tr>
<tr>
<td>Brain stem</td>
<td>Ataxia, paresis, dysmetria, dysphagia, anisocoria, or dilated pupils</td>
</tr>
<tr>
<td>Vestibular system</td>
<td>Ataxia, head tilt, pronounced postural deficits</td>
</tr>
<tr>
<td>Cerebellum</td>
<td>Ataxia, intention tremors</td>
</tr>
<tr>
<td>Cranial Nerves</td>
<td>Ataxia, paresis, dysmetria, spasticity</td>
</tr>
<tr>
<td>Spinal Cord</td>
<td>C1 – C5: All 4 limbs, worse in pelvic limbs, +/- Horner’s</td>
</tr>
<tr>
<td></td>
<td>C6 – T2: All 4 limbs, worse in thoracic limbs, +/- Horner’s</td>
</tr>
<tr>
<td></td>
<td>T3 – L3: Pelvic limbs</td>
</tr>
<tr>
<td></td>
<td>S3 - S5: Urinary incontinence, fecal retention, hypalgesia tail and perianal areas</td>
</tr>
<tr>
<td></td>
<td>Coccygeal: Decreased tail tone, hypalgesia caudal to lesion</td>
</tr>
<tr>
<td>Peripheral nervous system</td>
<td>Weakness predominates; within 10 – 20 days muscle atrophy</td>
</tr>
<tr>
<td></td>
<td>Radial n.: Inability to flex shoulder, extend the limb and fix elbow</td>
</tr>
<tr>
<td></td>
<td>Suprascapular n.: ‘Sweeney’ – shoulder slip: Supraspinatus and Infraspinatus muscle atrophy, abduction of limb, inability to advance shoulder</td>
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<tr>
<td></td>
<td>Femoral n.: Inability to extend stifle</td>
</tr>
<tr>
<td></td>
<td>Obturator n.: Inability to adduct pelvic limbs, ‘splay-legged’</td>
</tr>
<tr>
<td></td>
<td>Sciatic n.: Limb hangs behind the horse with stifle and hock extended, foot cannot be advanced</td>
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Table 1. Description of Neuroanatomic Localization Based on Predominant Clinical Signs Seen

arises from lesions of the vestibular system, cerebellum, or deficits of the ascending sensory systems. Paresis, or weakness typically originates from lesions in either upper motor or lower motor neuron tracts, or can be secondary to muscle disease or weakness. Dysmetria is frequently associated with ataxia and the term is used to describe whether there is excessive (hypermetria) or reduced (hypometria) movement of joints. Hypermetria can be seen with spinocerebellar disease and hypometria typically is seen as stiffness or a tin soldier gait. Although dysmetria and spasticity can be difficult to differentiate clinically, spasticity results from reduced inhibition of extensor motor neurons. In the horse, sensory ataxia is the predominant feature of spinal cord disease in which proprioceptive input to the cerebellum is compromised.

In equine neurology, scoring of ataxia is necessary for diagnostic and prognostic purposes and for determination of efficacy of therapeutic regimens. Currently the severity of ataxia assessed during a neurologic examination is graded using a modified ordinal grading scale (modified Mayhew scale; Table 2) where grade 0 is assigned to horses without neurologic deficits and grade 4 is assigned to horses that stumble, trip, and fall spontaneously.\(^1\,2\) Grade 5 is reserved for horses that are recumbent. A recent study, however, has shown that there is poor agreement, even between skilled observers of equine gait abnormalities when performing a neurologic examination, especially when clinical signs are subtle\(^3\); however, scoring is an important part of the examination to help determine what level of improvement has occurred. Watching the horse in motion is standard practice for both neurological and lameness examinations; however, it has become evident that these subjective scoring systems do not suffice for reliable and accurate assessment of horses with gait deficits, in particular when deficits are mild.\(^4\,6\)

II. CERVICAL MYELOPATHIES

Cervical vertebral stenotic myelopathy (CVSM) is the result of developmental orthopedic disease or abnormal mechanical stresses on the cervical column that affect bone development, growth plates, joints, cartilage and connective tissue.\(^7\,8\) CVSM is caused by malformations or structural changes of cervical vertebrae leading to narrowing/stenosis of the vertebral canal, malalignment of vertebral bodies due to changes at the joints between vertebral bodies (facets), osteochondrotic lesions at the facets, and attempts of the body to stabilize the instabilities (through production of excessive connective tissue). Subsequent compression of the spinal cord in the neck leads to clinical signs of disease.
CVSM typically affects young horses, with an increased incidence in certain breeds, including Thoroughbreds, Tennessee Walking Horses, Quarter Horses and Warmbloods. There is a well-documented gender ratio of males over females with reports ranging from 3:1 up to 23:1. CVSM is also seen in older horses that may be affected more by the biomechanical stresses on the vertebral column and these horses develop CVSM later in life. CVSM is considered a multifactorial disease with genetics, high planes of nutrition, alterations in copper and zinc ratios, rapid growth rates and trauma thought to play a potential role in the etiology and pathogenesis in young animals, and abnormal mechanical stresses and forces on the vertebral column in older horses. This disease is often recognized as having an acute onset (most likely secondary to a fall during which minor neck trauma may exacerbate insidious compressive lesions), but it is not uncommon for it to be reported as having a more gradual development, with the horse having appeared ‘clumsy’ during walking and handling for a longer time.

Clinical signs are symmetrical ataxia and dysmetria in thoracic and pelvic limbs. Lesions at C3 – C5 usually result in greater pelvic limb involvement (usually a grade 1 worse). Lesions at C5 – Th1 tend to affect the thoracic and pelvic limbs more equally. Involvement of the cervical intumescence (LMN for thoracic limbs) may also cause weakness and decreased reflex activity in thoracic limbs. Diagnostic criteria have been established for ante mortem diagnosis using standing cervical lateral radiographs and myelography. Thresholds have been established using anatomical measurements converted to intravertebral and intervertebral sagittal diameter ratios to identify presumptive areas of canal stenosis both at the intervertebral joint and within vertebrae. Similarly, criteria have been established to determine spinal cord compression using level of attenuation of dorsal and ventral contrast columns during myelography (dural diameter reductions). While both methods are important diagnostic aids, controversy remains with regards to the accuracy and the levels of false positives and false negatives seen.

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description</th>
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<tbody>
<tr>
<td>0</td>
<td>No neurologic deficits</td>
</tr>
<tr>
<td>1</td>
<td>Neurological deficits just detected at normal gait, but worsened by backing,turning, loin pressure, or neck extension</td>
</tr>
<tr>
<td>2</td>
<td>Neurological deficits easily detected at the walk an exaggerated by backing, turning, loin pressure, or neck extension</td>
</tr>
<tr>
<td>3</td>
<td>Neurological deficits prominent at the walk with a tendency to buckle or fall with backing, turning, loin pressure, or neck extension; Postural deficits noted at rest</td>
</tr>
<tr>
<td>4</td>
<td>Stumbling, tripping, and falling spontaneously at a normal gait</td>
</tr>
<tr>
<td>5</td>
<td>Horse recumbent</td>
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</table>

Table 2. Current Ataxia Grading Scale Used in Horses. Modified Mayhew Scale

With the development of more sophisticated diagnostic imaging modalities, we may start looking at the equine neck in a more comprehensive fashion in the near future. It has been well-established that neck lesions can lead to spinal cord compression with subsequent ataxia, but neck lesions can also be much more subtle and considered a cause for thoracic limb lameness when pain cannot be localized to the limb. It is unclear what aspects of the neck are painful in those situations. There are no reports in the literature of nerve root impingement causing radicular or referred pain in the horse. However, there is one report that compared traditional myelography to post-mortem obtained contrast enhanced computed tomography in 6 horses that demonstrated the presence of nerve root compression by a malformed articular facet, at the level of the proximal intervertebral foramen in 2 horses. Nerve root compression was associated with osteophytes that projected ventrally to encroach upon the proximal intervertebral foramen at 2 sites, and with multiple fractures of a severely malformed facet resulting in obliteration of the proximal intervertebral foramen at 1 other site. These horses had unilateral atrophy of the paravertebral musculature, longissimus cervicis. Also, concurrent central compressive lesions were present at all 3 of these peripheral compressive sites. We expect that with advanced imaging and electrodiagnostics some of these pain syndromes can be better explained.

III. TREATMENTS FOR CVSM AND OUTCOME

A large multicenter case-control study that reported findings on 270 horses with CVSM showed that 66% (173 of 263) horses died or were euthanatized prior to discharge from the hospital. Of the 173 nonsurviving horses, 1 died (< 1%) and 6 (3%) were euthanatized for financial reasons. The remaining 166 horses were euthanatized due to poor prognosis. Only 2 case horses that did not survive until discharge from the hospital underwent vertebral column surgery. Thirty-four percent (90 of 263) survived to discharge from the hospital, of which 27 (30%) had undergone surgical treatment. Horses that survived until discharge from the hospital had significantly lower grades of ataxia at admission than did non-survivors.

A low-protein, low-carbohydrate diet combined with stall rest appeared to improve clinical signs in adolescent Thoroughbred foals with CVSM. These older studies showed that 15 of 18 horses (83%) that were less than 1 year of age, and were diagnosed with CVSM based on clinical signs and a radiographic score, went on to have at least 1 racing start. In these experiments, a diet formulated to meet 65–75% of the 1978 National Research Council recommendations for crude protein and energy was fed to foals with neurologic signs of spinal cord disease or radiographic evidence of spinal cord compression, and adjustments were made at 3–4 month intervals through their 1st year of life. All animals were reported to have improvement in their neurologic or radiographic signs, which suggested that dietary management from an early age might play an important role in CVSM. A recent review of 103 Thoroughbred horses that underwent medical management for CVSM showed that 30% (21 of 70)
went on to race. Treatment in these cases was geared at decreasing inflammation, decreasing concussion to the spinal cord, and when possible, regulating the growth rate to prevent continued progression of developmental orthopedic disease. For this particular population, specific dietary recommendations for horses less than 12–14 months of age included feeding only grass hay or pasture with no grain and supplemented with a ration balancer to ensure adequate protein and mineral intake. However, dietary changes had no significant effect on whether or not horses with CVSM returned to racing in this study. A caveat here is that the horses in this study typically presented as older yearlings or 2-year-olds that had just entered into race training, so the window for which dietary modification may have had an impact on changing the rate of growth may have been missed. Horses that went on to race had a significantly lower grade of ataxia, with a median of 1.0 in the thoracic limbs and 2.0 in the pelvic limbs. 

Surgical treatment of CVSM has been performed since 1979 when the use of Cloward’s intervertebral fusion technique was first reported. Briefly, a homologous bone dowel was implanted into a 16 mm hole drilled from ventrally into the 2 affected cervical vertebrae at their articulation. The technique was soon modified by replacing the dowel with a 25 mm diameter stainless steel basket (Cloward Bagby basket) filled with an autogenous cancellous bone graft and placed into a 25 mm hole drilled into the 2 vertebrae. In a more recent further modification of the technique, the basket has been replaced by an open-ended, threaded, stainless steel cylinder (Kerf Cut Cylinder) which offers more secure fixation of the implant and allows the preservation of a central core of vascularized bone within it. Initial follow up studies showed that 57 of 72 (79%) horses were in use. Twenty of these horses were used for pleasure riding or breeding only, 29 were in training for racing or showing, and 8 were actually racing or showing. A second study showed that neurologic function was improved in 56 of 73 horses (77%) that underwent either cervical vertebral interbody fusion (n = 63) or dorsal laminectomy (n = 10), and 17 of these (23%) went on to either train for or return to racing. Another study showed a complete recovery in 15 of 27 horses. A review of 12 surgically managed triple level cervical cord compression cases, 5 of which were Thoroughbreds, showed that 1 of these horses (20%) went on to have at least 1 racing start. A more recent series out of Europe showed that 17 of 28 horses followed-up regained a normal gait. Twelve of these had returned to normal use and 2 of the 17 were not yet in full work.

The current surgical technique for cervical stabilization is not widely performed because it is costly, technically challenging, and results in long convalescence. Recently, a novel, porous intervertebral fusion device with titanium screw and rod construct was evaluated for fusion of C3-C4 in 4 clinically normal horses and in 3 horses diagnosed with CVSM through myelography. The use of an intervertebral fusion device with a titanium pedicle screw and rod system has been evaluated in a sheep model and in vitro in the horse, resulting in constructs that resist load to failure testing up to 5000N. This system is easy to apply, requires less disk space removal, and results in immediate stability in compression and tension. The procedure was performed safely in all 7 horses, with minimal postoperative complications. In three horses, seroma formation was noted approximately 10 days following surgery. No neurologic deficits were seen in any of the normal horses at any time point. Neurologic evaluation at 6 months post-surgery revealed improvement of neurologic deficits (by 1-2 grades) in the 3 clinically affected horses. In one horse, the neck stiffness and pain that was present at admission had also significantly improved. This technique appears to be a promising alternative to current surgical procedures for the treatment of CVSM.

Overall it appears that most surgeons seem to achieve a success rate, measured by the patient returning to use, between 45 and 60%. Considering that euthanasia is the alternative for surgical candidates for cervical fusion, experience has shown that the procedure is worthwhile. Case selection for surgical intervention with CVSM cases remains critical. Surgical experience with the procedure seems to improve success rate and lower post-operative morbidity. Recovery may take over 12 months and an exercise program during convalescence is considered beneficial.

IV. EQUINE PROTOZOAL MYELOENCEPHALITIS

Equine protozoal myeloencephalitis (EPM) is an important cause of neurologic disease in the horse. Two causative agents that have been identified are Sarcocystis neurona and Neospora hughesi. For highest accuracy in antemortem diagnosis, the examiner needs to demonstrate the presence of clinical signs consistent with EPM through a neurologic examination, ensure that other potential causes of disease are ruled out, and conduct immunodiagnostics testing of serum and CSF to confirm intrathecal antibody production against S. neurona or N. hughesi. The ratio of antibody in serum to CSF will reveal intrathecal antibodies in most cases of EPM. The SnSAG 2, 4/3 ELISA serum:CSF titer ratio and NhSAG1 ELISA serum:CSF titer ratio are the only tests currently offered commercially that provide information regarding intrathecal antibody production based on serum and CSF titers. For treatment of EPM, it is recommended that 1 of the FDA-approved anticoccidial drugs should be used to control infection. The current FDA-approved drugs are: a) Ponazuril (Marquis); b) Diclazuril (Protazil); and c) Sulfadiazine/ Pyrimethamine. Additional medical and supportive treatment should be provided based on the severity of neurologic deficits and complications arising from them. Duration of treatment is based on resolution of clinical signs. Horses that develop recurrent signs should be reassessed.

V. ROLE OF VITAMIN E IN TREATMENT OF MYELOPATHIES

Vitamin E’s role in peroxyl radical scavenging is as follows:

\[ \text{ROO}^• + \text{VitE} - \text{OH} \rightarrow \text{ROOH} + \text{VitE} - \text{O}^• \]

The tocopherol radical (VitE-•) then reacts with vitamin C [AH: hydrogen donors] to return to a reduced state:
VitE – O• + AH → VitE – OH + A•

In the absence of vitamin E, reactive oxygen species are repeatedly generated and oxidative damage ensues:

ROO• + RH → ROOH + R•

R• + O2 → ROO•

Reactive oxygen species serve as key signal molecules in physiological processes and also have a role in pathologic processes. Alpha-tocopherol is probably involved in maintaining normal axonal transport, and α-tocopherol deficiency could cause a defect of fast retrograde axonal transport, vesicular “turnaround”, or both, causing an accumulation of normal and abnormal cytoplasmic organelles at the axon terminal and subsequent blockage of normal axonal flow. The underlying molecular and cellular mechanisms that result in neuromuscular dysfunction after α-tocopherol deficiency have yet to be defined. Vitamin E deficiency on its own does not appear to reliably cause disease in horses. Studies examining the effect of vitamin E deficiencies in exercising or resting horses have revealed no apparent clinical signs resulting from vitamin E deficiency. There are, however, three specific diseases that consistently have been associated with α-tocopherol deficiency: equine motor neuron disease, neuroaxonal dystrophy/equine degenerative myelonecephalopathy, and vitamin E-deficient myopathy.

The difference between natural α-tocopherol and synthetic vitamin E in the horse was recently demonstrated, where CSF concentrations of α-tocopherol were significantly elevated above baseline values after supplementation with the natural vitamin E, but not after supplementation of a synthetic all-rac-α-tocopherol acetate at equivalent high dosages (10,000 IU/500 kg horse/day). It is currently recommended that the natural RRR-α-tocopherol (nonacetate) form of vitamin E be used to supplement deficient horses with equine motor neuron disease, neuroaxonal dystrophy/equine degenerative myelonecephalopathy, and vitamin E-deficient myopathy because this form is the most biologically available, most readily absorbed, and has the most potent antioxidant activity. At this time, there is no strong evidence to support supplemental α-tocopherol above the 2007 NRC dietary recommendations (1 – 2 IU/kg/day) unless the horse has been diagnosed with equine motor neuron disease, vitamin E-deficient myopathy, or is part of a neuroaxonal dystrophy/equine degenerative myelonecephalopathy genetically susceptible herd. Of note is that the NRC set the upper safe diet concentration at 20 IU/kg BW (10,000 IU/500 kg horse) based on 2% dietary intake. Above this level, coagulopathy and impaired bone mineralization have been reported.

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Declaration of Ethics

The Author has adhered to the Principles of Veterinary Medical Ethics of the AVMA.

Conflict of Interest

The Author declares no conflicts of interest.

REFERENCES