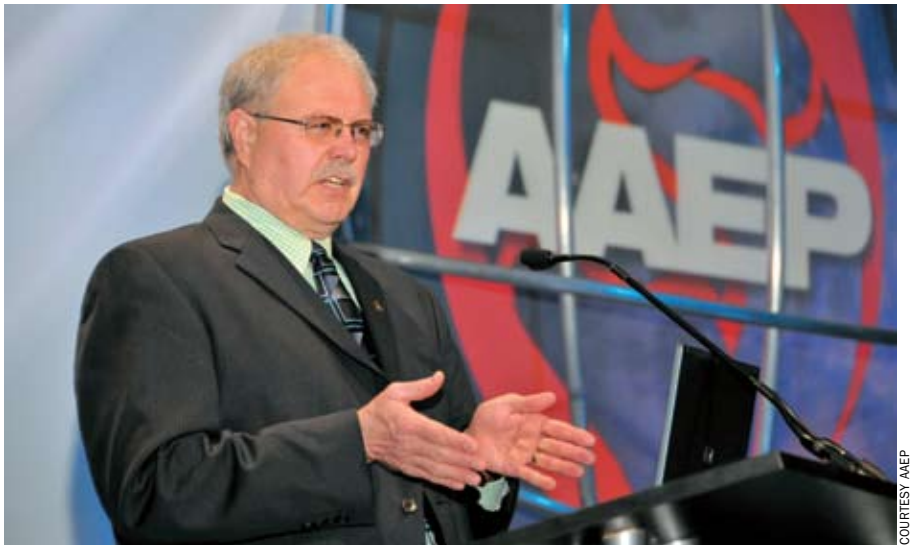
Milne: Neurologic Disease

NANCY S. LOVING, DVM

When faced with a horse exhibiting neurologic disease, the importance of a thorough physical exam and diagnostic testing cannot be emphasized enough. Stephen Reed, DVM, Dipl. ACVIM, of Rood & Riddle Equine Hospital in Lexington, Ky., described selected equine neurologic diseases during the prestigious Milne Lecture.

Some elements of the exam include evaluating proprioception (the horse's awareness of where his feet are in space), gait changes, the presence of unusual gaits, and identifying the neuroanatomical location of an abnormality. He explained that proprioceptive deficits are the first signs of compressive lesions in the spinal cord, while deep pain sensation is the last function lost.

Reed relies on a full exam, beginning with evaluating the horse's behavior and mental status, examining the head and cranial nerves and evaluating gait. He records all findings, characterizes signs, and attempts to localize the lesion to help determine the cause.



COURTESY AAEP

Dr. Stephen Reed discussed the importance of a thorough physical exam and diagnostic testing for a horse exhibiting signs of neurologic disease in his Milne State of the Art Lecture.

Wobbler Syndrome

The first condition Reed discussed was cervical vertebral stenotic myopathy (CVM), also known as wobbler syndrome. This can be a developmental problem in young light-breed horses, or it can be an acquired problem in older horses (over 10 years) from osteoarthritis of the neck vertebral articular process joints (facets). Elongation of the dorsal laminae (the bony plates that form the roof of the vertebral canal) into the intervertebral space (between the vertebrae) can cause stenosis (narrowing), requiring surgical correction. Compression most commonly occurs between cervical vertebrae 6 and 7 (C6-C7).

Osteochondrosis dissecans (OCD) of the facets is a common developmental cause of wobbler syndrome, and growth plate abnormalities (vertebral epiphysitis) can also narrow the canal. The vertebral column is sensitive to dietary imbalances, particularly of copper, which influences vertebral development, or excess energy, which causes rapid skeletal growth. This rapid growth influences vertebral development and the propensity for OCD. Often, wobbler syndrome changes in the axial skeleton occur in conjunction with developmental orthopedic disease in the limbs. Reed said conservative treatment of diet (protein and energy) and exercise restrictions might prove successful for young wobbler horses.

Malalignment of vertebrae can also cause CVM, especially at C2-C3. Such malalignment is unusual, but it tends to be associated with the fixed, high arch of head and neck seen in Saddlebreds or some Warmbloods.

Reed described the tenets of the approach used by Barrie Grant, DVM, MS, Dipl. ACVS, a veterinarian in Bonsall, Calif., known for his CVM treatments:

- Keep the horse alive, treating the underlying cause when possible (this might result in an amazing recovery).
- Take an aggressive approach to achieving an accurate diagnosis, using spinal taps and myelograms (which involve injecting a solution—that shows up on X rays—into the spinal canal to reveal any compressive lesions of the spinal cord) as necessary.
- Perform standing cervical (neck) radiographs; pay careful attention to vertebral anatomy and measurements.
- A mildly ataxic (incoordinated) horse might perform okay, and a lame horse might perform okay, but a horse that is both ataxic and lame neither can perform, nor is he safe to ride.

Sheared heels can be a general sign of ataxia, but typically a horse with wobbler syndrome has symmetric ataxia, weakness, or spasticity, most notably in the rear limbs. Toe dragging, stumbling, and outward excursion of the rear limbs are also typical signs. Other musculoskeletal diseases can make a horse look ataxic, such as bilateral suspensory ligament desmitis or bilateral stifle OCD. It is possible for a horse to have several coexisting conditions.

Residual damage to neuromuscular tissue might not resolve, so address the situation early. Be patient, allowing at least 18-24 months to realize the treatment outcome, and employ physical therapy to improve outcome. Consider the horse's intended use when attempting treatment,

and always conduct follow-up exams to help ensure rider and horse safety.

Genetic influences of wobbler syndrome are still under investigation. Breeding wobblers to other wobblers results in an increase in limb OCD occurrence, but this research effort never reproduced a wobbler. This disease can affect all ages, but if the horse is older than 10 years, he likely has an acquired ostearthritic problem. Breed distribution of wobbler-affected horses is as follows: 37% Thoroughbred, 25% Warmblood, 16% Quarter Horse, 21% other (including a fair number of Tennessee Walkers), and 1% pony.

EPM

Next Reed discussed equine protozoal myeloencephalitis (EPM), a parasitic disease with neurologic consequences. The opossum (definitive host) eats the flesh of an intermediate host (skunk, armadillo, sea otter, or domestic cat) that has encysted stages of the protozoan parasite *Sarcocystis neurona*. The opossum excretes parasitic oocysts in its feces, which are inadvertently eaten by horses in contaminated feed or pasture. Opossums tend to be opportunistic feeders. If separate sources of water are available for horses and wildlife, there is lessened chance of infection.

Ingesting contaminated feces might result in parasitic development in the horse's central nervous system (CNS), leading to asymmetric ataxia with muscular atrophy. Horses exposed to *S. neurona* might not necessarily develop clinical disease. Therefore, blood testing is fraught with false positives.

A positive response in the CSF (cerebral spinal fluid) indicates that *S. neurona* entered the CNS and evoked an immune response; however, any blood contamination of the CSF tap can produce false positive results. False negatives are rare on blood or CSF, but they have been observed in the first seven to 10 days post-infection. Most importantly, the veterinarian should rule out all other possible causes of ataxia.

A horse is 50 times more likely to survive if there is notable improvement in clinical signs with treatment. Prevention involves keeping wild animals away from horses, removing carcasses of intermediate hosts, keeping definitive hosts (opossums) away from horse feed and water sources, and decreasing risk factors of stress and transport as much as possible.

Neurologic EHV-1

Equine herpesvirus-1 (EHV-1) can cause respiratory disease (rhinopneumonitis), abortion, severe neonatal disease, and a less-common neurologic form that is acute and progressive. EHV-4 also can cause neurologic disease.

EHV-1 can be a mutated or nonmutated strain that induces symmetric ataxia with ascending (moving forward) paralysis beginning with urinary incontinence, poor tail and anal tone, and rear limb ataxia. The virus attacks the blood vascular supply of nerves. Facial nerve signs, head tilt, and nystagmus (involuntary, rapid eye movement) are other manifestations, as well as dementia and other cerebral signs.

The 2003 EHV-1 neurologic outbreak in Ohio demonstrated the rampant communicability of this disease. Of 137 horses in a barn, 117 had fever; 46 of those showed neurologic signs, 14 of which died. Fever, cough, and nasal discharge preceded ataxia or recumbency by about five days. All horses had been previously immunized with rhinopneumonitis vaccines, which afforded no protection. Reed said the virus multiplies rapidly once the horse is exposed, thus increasing its contagion level.

The EHV-1 mutated strain also magnified the degree of virulence for longer. In 132 Thoroughbred broodmares, George Allen, PhD, found that while 46% harbored the wild-type strain, 8% harbored the mutant EHV-1 strain in their submandibular lymph nodes; the mutant strain comprised 18% of the total latent viral reservoir (horses not showing signs of illness). This high proportion of neurologic EHV necessitates implementing measures to control its spread.

Veterinarians must enforce diligent biosecurity measures in suspected neurologic outbreaks to limit the numbers of horses exposed. Horses older than 15 years of age and especially older than 20 seemed to be at the highest risk of infection; these are horses with less competent cell-mediated immunity. Stress plays a huge role in onset of clinical signs.

Reed encouraged further research.

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