# Perception of Equine Practitioners Regarding the Occurrence of Selected Equine Neurologic Diseases in the Northeast Over a 10-Year Period

**Bethany A. DeVilbiss, BS, Hussni O. Mohammed, BVSc, PhD, and Thomas J. Divers, DVM , Dipl ACVIM, ACVECC**

## ABSTRACT

A survey was developed to examine the perception of equine practitioners regarding the occurrence of five equine neurologic diseases in the northeastern United States over the 10-year period between June 1, 1997 and June 1, 2007. This information was then compared with trends at Cornell University's Equine Hospital during the same time span, which in general agreed with practitioners' opinions. Equine herpes virus-1 (EHV-1) neurologic disease, equine motor neuron disease (EMND), and equine protozoal myelitis (EPM) have historic and current relevance. Results showed that the frequency of EMND and EPM has remained relatively stationary or decreased somewhat, whereas the frequency of the neurologic strain of EHV-1 may have increased slightly over the last decade. Less historical information on clinical disease associated with *Borrelia burgdorferi* infection (Lyme disease) and *Parelaphostrongylus tenuis* exists; however, results suggest that *P. tenuis* in the equine is presently emergent. Opinions regarding the existence and rate of occurrence of clinical borreliosis in horses appear divided. A better understanding of the frequency with which these diseases occur, as well as possible associated positive risk factors, will aid the equine practitioner in making an appropriate diagnosis in cases of neurologic disease in their equine patients.

**Keywords:** Equine herpes virus-1; Equine motor neuron disease; Equine protozoal myelitis; Lyme disease; *Parelaphostrongylus tenuis*

## INTRODUCTION

Significant findings germane to equine neurologic diseases have occurred, making these conditions an important part of equine practice. Notably, a neurotropic strain of equine herpes virus-1 (EHV-1) has been characterized,[1,2] the risk factors associated with equine motor neuron disease (EMND) have been identified[3,4] and the life cycle of equine protozoal myelitis (EPM) has been described.[5] The existence of clinical signs associated with *Borrelia burgdorferi* infection (Lyme disease) in the equine is currently being investigated,[6] and recently, the first case of *Parelaphostrongylus tenuis* in the horse was confirmed.[7]

Improved understanding of these conditions has influenced their diagnosis and treatment, and equine neurologic diseases remain of great clinical relevance. The objective of this investigation was therefore to examine the perceptions of equine practitioners regarding the occurrence of EHV-1, EMND, EPM, Lyme disease, and *P. tenuis* in the northeastern United States over a 10-year period and compare these perceptions with the observed cases among horses referred to Cornell University's Equine Hospital during the same time.

### Equine Herpes Virus-1 (Neurologic Strain)

EHV-1 is a typical herpesvirus consisting of an envelope and double-stranded DNA genome. EHV-1 was first reported as a cause of neurologic disease in horses as early as the mid-1960s; however, this virus is more frequently associated with respiratory signs and abortion.[8-10] Transmission is by inhalation or ingestion of infective droplets, with the virus being shed in nasal and ocular discharges, saliva, and products of abortion.[11] From the respiratory epithelium, the virus travels to regional lymph nodes and then enters the blood, where it circulates in mononucleated cells, causing a cell-associated viremia. Though unclear, it is speculated that virus crosses from peripheral blood mononuclear cells to brain endothelial cells. The result is vasculitis and vessel thrombosis, leading to ischemic damage of adjacent central nervous system (CNS) tissue.[9]

The tendency of certain EHV-1 strains to cause myeloencephalopathy appears to be related to a high level of viremia and endothelial tropism rather than neurotropism.[12] There is a point mutation difference between the abortigenic and most neuropathogenic EHV-1 stains.[1]

*From the Cornell University College of Veterinary Medicine, Department of Clinical Sciences, Ithaca, NY.*
*Reprint requests: Thomas J. Divers, DVM, Dipl ACVIM, ACVECC, Cornell University College of Veterinary Medicine, Department of Clinical Sciences, Ithaca, NY.*
*0737-0806/$ - see front matter*

*doi:10.1016/j.jevs.2009.03.002*

Diagnosis of EHV-1 is usually made by polymerase chain reaction (PCR) on nasopharyngeal swabs or peripheral blood leukocytes. If a cerebrospinal fluid (CSF) tap is performed, xanthochromia or an elevated protein concentration without pleocytosis is suggestive of EHV-1 infection.[9]

Disease prevention should focus on good biosecurity and quarantine of new horses entering the premises. Although not routinely done, testing could be performed to detect horses that are currently shedding. Recrudescence of latent infections may serve as a source of disease in closed populations, although this has rarely been documented in EHV-1 neurologic disease outbreaks; most outbreaks have been associated with arrival of a new horse to the facility or transportation of horses from and to the farm.[13] Killed and modified live vaccines against EHV-1 are available. Although EHV-1 vaccination is recommended, and the modified live vaccine has been shown to decrease nasal shedding,[14] these vaccines are not highly efficacious in preventing neurologic disease associated with EHV-1 infection.

Treatment of neurologic signs associated with EHV-1 infection focuses on supportive care. Corticosteroids may be of some benefit in severely affected horses but are not without risk. Valacyclovir and aspirin are also believed to be beneficial in some cases. Prognosis is fair to good for mildly affected horses. Even recumbent horses may occasionally recover, but often they have permanent neurologic deficits.

## Equine Motor Neuron Disease

The first case of EMND was described in the mid 1980s,[15] and in the decade before 1997, the number of EMND cases reported annually to Cornell University increased each year. After 1997, a marked decline in the diagnosis of EMND at Cornell occurred. Although in the United States, the incidence of EMND has historically been highest in the New England states, EMND does occur throughout the country and has also been reported in Great Britain, Canada, Ireland, Switzerland, Belgium, Japan, Brazil, and the Netherlands.[16]

Identified risk factors for EMND include Quarter Horse breed, residence on the same premises for at least the 2 years before disease onset, age (reported range of 2 – 23 years in the United States), low dietary vitamin E, and an excess of dietary pro-oxidant factors such as iron (Fe) and copper (Cu).[16-18]

Diagnosis of the disease can be made presumptively based on a history of inadequate green forage being fed, especially in horses older than 2 years of age. Elevations in plasma creatine kinase and aspartate aminotransferase, as well as low plasma vitamin E, are supportive, but not definitive or specific. Evidence of denervation demonstrated by muscle or nerve biopsies is more conclusive.[16] The treatment of choice for clinical EMND remains vitamin E supplementation; however, response to treatment is variable, and prognosis may be poor in many cases.

## Equine Protozoal Myelitis

Although the first case of EPM was diagnosed as early as 1964, it was not until 1991 that *Sacrocystis neurona* was definitively identified as the causative organism.[19] The life cycle of *S. neurona* has recently been described, representing a significant advancement in the management of this disease in the horse.[5]

*S. neurona* is a coccidian parasite with a typical two-host life cycle. The opossum serves as the definitive host for the parasite, and the sexual phase of reproduction takes place in this animal. Although the intermediate hosts in the *S. neurona* lifecycle remained unclear for many years, we now know that the raccoon, skunk, armadillo, sea otter, and domestic cat all support asexual reproduction of the parasite and may serve as intermediate hosts for the organism.[5]

In the normal life cycle, opossums shed infective sporocysts into the environment. These sporocysts are ingested by the intermediate host, leave the gastrointestinal tract as sporozoites, travel as merozoites in the blood stream, and eventually form sarcocysts within the muscle tissue. The life cycle is completed when the opossum consumes sarcocyst-containing muscle of an infected intermediate host.[5]

Horses are considered dead-end hosts of *S. neurona*, becoming inadvertently affected after consuming infective sporocysts shed into the environment by opossums. Neurologic disease associated with *S. neurona* infection in the equine occurs after penetration of the CNS. The parasite does not enter the CNS of the intermediate or definitive host during the normal life cycle, and the mechanism of CNS penetration in the equine is unknown.[20]

Presently, the ante-mortem diagnosis of EPM in the horse is complicated by a high seroprevalence in the population. Although a positive result on a Western blot of CSF is considered one of the methods of choice, antibodies against *S. neurona* have been demonstrated in the CSF of healthy horses. Variations of the Western blot, as well as concurrent testing of serum antibody levels, have improved sensitivity and specificity. Other available tests include an enzyme-linked immunosorbent assay (ELISA) and indirect fluorescent antibody testing (IFAT). Values obtained by IFAT of either serum or CSF have been used to determine probability of the disease when both prevalence and clinical signs are considered.[21]

Prevention of EPM involves clearing pasture and stable areas of opossums and intermediate hosts. A conditionally approved vaccine was used for several years, but its efficacy was unproven, and it is no longer available. Ponazuril, nitazoxanide, and pyrimethamine/sulfa have some evidence of treatment efficacy. Intermittent or continual administration of ponazuril was demonstrated to decrease disease incidence or CSF seroconversion, but it does not eliminate

clinical signs and is likely cost prohibitive on a continuous-use basis for most horses.[22,23]

### Equine *Borrelia* Infection

The existence of clinical disease associated with infection by the spirochete bacteria *B. burgdorferi*, and neuroborreliosis in particular, in the equine is controversial. Lyme disease is transmitted through the bite of a tick vector, *Ixodes scapularis* (black-legged tick or deer tick) in the eastern and mid-western United States and *Ixodes pacificus* in the West.[24]

A case described in the literature reported a horse with neurologic signs, elevated *B. burgdorferi* titers and spirochetes identified in the brain on post-mortem examination.[25] Clinical signs of equine Lyme disease are nonspecific, however, and this, coupled with a high seroprevalence in the northeastern United States, has caused some practitioners to argue the reality of clinical borreliosis in the equine.[26] Signs that have been blamed on Lyme disease in the horse include chronic weight loss, sporadic lameness, laminitis, low-grade fever, swollen joints, muscle tenderness, and anterior uveitis. Recently, horses and ponies with neurologic signs and CSF ELISA antibody levels equal to serum have been examined, and a tentative diagnosis of Lyme neuroborreliosis was made by one of the authors (T.J.D.). Other seropositive horses have had CNS signs indicative of spinal cord or nerve root disease and at necropsy had lymphocytic meningitis.

The treatment of choice for Lyme disease remains tetracycline, doxycycline, or ceftiofur.[27] A tentative diagnosis is based on clinical signs and response to antibiotic therapy. A positive titer (whole cell ELISA, C6 ELISA, Western blot, or IFAT) has low specificity for diagnosing Lyme disease because of the high seroprevalence. This is further confounded because of the apparent long-term persistence of antibody in most seropositive horses.

### Parelaphostrongylus tenuis

*P. tenuis* is the natural nematode parasite of the white-tailed deer (*Odocoileus virginianus*). In the normal life cycle, adult worms reside and lay eggs in the meninges of the deer. The eggs pass into venous circulation and travel to the lungs, where they hatch into first stage (L1) larvae. The L1 stage is coughed up, swallowed, and passed in the feces. Once shed into the environment, larvae invade or are ingested by terrestrial gastropods and develop to the third (L3) larval stage over 3 to 4 weeks. Infected gastropods are ingested, and the L3 stage migrates to the dorsal horn gray matter of the spinal cord, where they mature. From here, the life cycle is completed by parasitic migration to the subdural space and then to the brain through the dura mater and cranial venous sinuses. The prepatent period in the white-tailed deer is 82 to 92 days.[7]

In aberrant hosts, third-stage larvae migrate aimlessly within the CNS, leading to clinical disease. Although commonly associated with neurologic signs in South American camelids, a parasite believed to be *P. tenuis* was recently found in horses with a syndrome of acquired dorsal gray matter myelitis and acute-onset cervical scoliosis. This suggests that *P. tenuis* is capable of penetrating the equine CNS, and that parasite migration may cause cervical scoliosis in some cases.[7,28]

## MATERIALS AND METHODS

A questionnaire was developed with questions addressing five different equine neurologic diseases that may have temporal changes in prevalence in the northeast region of the United States and was sent to practitioners in the target area. The selected diseases were: (1) the neurologic strain of EHV-1, (2) EMND, (3) EPM, (4) equine *Borrelia,* and (5) *P. tenuis.* A photograph of a typical case of *P. tenuis* scoliosis was included in the survey, because it was believed that some practitioners may not be aware of this particular disease in horses. Practitioners were selected to receive a survey if they had referred at least one case of any of the five diseases of interest to Cornell University's Equine Hospital over the 10-year period between June 1, 1997 and June 1, 2007. A computer query of hospital records on all horses admitted to the Cornell University Hospital for Animals during the same time period for the five diseases was performed. The diagnosis listed on the medical record may have been determined by either definitive or presumptive diagnosis of each disease based on clinical, laboratory, and in some cases necropsy findings. Results from the veterinarian surveys were compiled and compared with trends at Cornell based on a computer query for the frequency of the five diseases during the selected time. The summary statistics were performed using the Statitix *.1 software (Anaylytical Software, Tallahassee, FL).

## RESULTS

Results are reported as a percentage of all practitioners that returned surveys, unless otherwise noted.

### Practitioner Demographics

Demographic data on the practitioners enrolled in this study are shown in Table 1. Surveys were returned from 51 practitioners in New York and surrounding states as well as one practitioner from Ontario, Canada. Of the veterinarians surveyed, 76.5% had been practicing in the same region for 15 or more years, with 66% reporting a caseload that was more than 90% equine. Only 10% of those surveyed reported a caseload composed of 40% or less equine patients.

### Equine Herpes Virus-1 (Neurologic Strain)

Data on the occurrence of the neurologic strain of EHV-1 are reported in Table 2. Most of those surveyed reported

**Table 1.** Practitioner demographics

| State(s) of Primary Practice | Number | Percent of Total |
|---|---|---|
| CT | 3 | 5.9% |
| DE | 1 | 2.0% |
| MA | 3 | 5.9% |
| NH | 2 | 3.9% |
| NJ | 1 | 2.0% |
| NY | 24 | 47.1% |
| OH | 1 | 2.0% |
| PA | 6 | 11.8% |
| VA | 2 | 3.9% |
| CT & NY | 1 | 2.0% |
| ME & NH | 1 | 2.0% |
| NJ & NY | 1 | 2.0% |
| CT & MA & NY | 1 | 2.0% |
| MA & NH & VT | 1 | 2.0% |
| ONT | 1 | 2.0% |
| Did not report | 2 | 3.9% |
| **Number of Years Practicing in the Same Region** | **Number** | **Percent of Total** |
| <5 | 2 | 3.9% |
| 5 – 15 | 10 | 19.6% |
| 15 – 25 | 16 | 31.4% |
| 25 – 35 | 14 | 27.5% |
| >35 | 7 | 13.7% |
| Did not report | 2 | 3.9% |
| **Percentage of Caseload That Is Equine** | **Number** | **Percent of total** |
| <15 | 2 | 3.9% |
| 15 – 40 | 3 | 5.9% |
| 40 – 65 | 4 | 7.8% |
| 65 – 90 | 6 | 11.8% |
| >90 | 34 | 66.7% |
| Did not report | 2 | 3.9% |

**Table 2.** Perception of practitioners' regarding the occurrence of EHV-1 neurologic strain

| Number of Cases of Neurologic EHV-1 Evaluated in the Past 12 Months | Number | Percent of Total |
|---|---|---|
| 0 | 40 | 78.4% |
| 1 – 2 | 8 | 15.7% |
| 2 – 5 | 1 | 2.0% |
| 5 – 10 | 0 | 0% |
| >10 | 0 | 0% |
| Did not respond | 2 | 3.9% |
| **Number of Outbreaks of Neurologic EHV-1 Evaluated in the past 12 months** | **Number** | **Percent of Total** |
| 0 | 36 | 70.6% |
| 1 – 2 | 3 | 5.9% |
| 2 – 5 | 0 | 0% |
| 5 – 10 | 0 | 0% |
| >10 | 0 | 0% |
| Did not respond | 12 | 23.5% |
| **Frequency of Neurologic EHV-1 over the Last 12 Months in Comparison with the Last 5 Years** | **Number** | **Percent of Total** |
| Less than is typical | 2 | 3.9% |
| Fairly typical | 38 | 74.5% |
| More than is typical | 1 | 2.0% |
| Did not respond | 10 | 19.6% |
| **Frequency of Neurologic EHV-1 over the Past 10 Years** | **Number** | **Percent of Total** |
| Decreased substantially | 0 | 0% |
| Decreased somewhat | 1 | 2.0% |
| Remained relatively unchanged | 29 | 56.9% |
| Increased somewhat | 6 | 11.8% |
| Increased substantially | 4 | 7.8% |
| Did not report | 11 | 21.6% |

no cases (78.4%) or outbreaks (70.6%) of the neurologic strain of EHV-1 over the previous 12-month period. A total of 64.7% reported that they had not had more than one experience with an outbreak of the neurologic strain of EHV-1 over the past 10 years. The common perception among the surveyed practitioners (74.5%) was that this was fairly typical in comparison with the previous 5 years. Fifty-seven percent of the practitioners indicated that the frequency of the neurologic strain of EHV-1 over the past 10 years had remained relatively unchanged, and 20% perceived that the frequency of EHV-1 over the past 10 years had increased.

Practitioners who perceived that the neurologic strain of EHV-1 had increased over the 10-year period of the survey attributed the observation to emergence of a neurotropic form of the virus, increased mobility of the equine population, increased travel, stress, contact between horses from different areas, heightened awareness of the disease, overcrowding, or increased levels of mutated virus.

No cases of the neurologic strain of EHV-1 were admitted to Cornell University's Equine Hospital in the 12-month period between June 1, 2006 and June 1, 2007. A total of six cases of the disease were admitted in

**Table 3.** Cornell University's Equine Hospital historical data

| | Total | NY | CT | FL | PA | OH | NJ | MA | KY |
|---|---|---|---|---|---|---|---|---|---|
| EHV-1 cases 06/01/06 –06/01/07 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| EHV-1 cases 06/01/02 – 06/01/07 | 6 | 5 | 0 | 0 | 1 | 0 | 0 | 0 | 0 |
| EHV-1 cases 06/01/97 – 06/01/07 | 10 | 8 | 0 | 0 | 2 | 0 | 0 | 0 | 0 |
| | Total | NY | CT | FL | PA | OH | NJ | MA | KY |
| EMND cases 06/01/06 – 06/01/07 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| EMND cases 06/01/02 – 06/01/07 | 3 | 1 | 0 | 0 | 2 | 0 | 0 | 0 | 0 |
| EMND cases 06/01/97 – 06/01/07 | 27 | 19 | 2 | 1 | 4 | 1 | 0 | 0 | 0 |
| | Total | NY | CT | FL | PA | OH | NJ | MA | KY |
| EPM cases 06/01/06 – 06/01/07 | 8 | 6 | 0 | 0 | 1 | 0 | 1 | 0 | 0 |
| EPM cases 06/01/02 – 06/01/07 | 36 | 24 | 0 | 0 | 7 | 0 | 2 | 2 | 1 |
| EPM cases 06/01/97 – 06/01/07 | 129 | 97 | 1 | 2 | 20 | 0 | 6 | 2 | 1 |
| | Total | NY | CT | FL | PA | OH | NJ | MA | KY |
| *Borrelia* cases 06/01/06 – 06/01/07 | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| *Borrelia* cases 06/01/02 – 06/01/07 | 6 | 3 | 1 | 0 | 0 | 0 | 1 | 1 | 0 |
| *Borrelia* cases 06/01/97 – 06/01/07 | 11 | 6 | 1 | 0 | 0 | 0 | 3 | 1 | 0 |
| | Total | NY | CT | FL | PA | OH | NJ | MA | KY |
| *P. tenuis* cases 06/01/06 – 06/01/07 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| *P. tenuis* cases 06/01/02 – 06/01/07 | 4 | 4 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| *P. tenuis* cases 06/01/97 – 06/01/07 | 5 | 5 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |

the 5-year period between June 1, 2002 and June 1, 2007; 10 cases were admitted in the 10-year period between June 1, 1997 and June 1, 2007. This indicates a possible increase in the frequency of the neurologic form of EHV-1 diagnosis at Cornell over the 5 years before the survey when compared with the previous 5 years. The distribution of cases of EHV-1 referred to Cornell by state over the years is shown in Table 3. Most of the cases were from New York State, with a smaller number of cases being from Pennsylvania.

## Equine Motor Neuron Disease

Data on the occurrence of EMND are reported in Table 4. Among those surveyed, 76.5% reported no cases of EMND over the previous 12-month period. Most practitioners reported that this was fairly typical (49%) or less than typical (29%) in comparison with the previous 5 years. A total of 53% of the surveyed practitioners indicated that the frequency of EMND over the past 10 years had remained relatively unchanged.

Practitioners who perceived that EMND had decreased over the 10-year period of the survey (25%) attributed the decline to increased administration of vitamin E in unpastured horses, client education, difficulty of diagnosis, previous overdiagnosis, nutrition, genetic selection, or early disease recognition and treatment of the disease. Four percent of the surveyed practitioners perceived that the occurrence of EMND had increased over the 10-year period of the survey, citing greater awareness of the disease as a possible reason for the increase. According to those surveyed, the survival time of horses after EMND diagnosis was highly variable: between less than 2 weeks to greater than 3 years. Survival likely depends on the severity of disease at the time of initial presentation.

No cases of EMND were referred to Cornell University's Equine Hospital in the 12-month period between June 1, 2006 and June 1, 2007. A total of three cases were referred in the 5-year period between June 1, 2002 and June 1, 2007 and 27 cases in the 10-year period between June 1, 1997 and June 1, 2007. These observations indicate a decrease in the frequency of EMND diagnosis at Cornell over the 5 years before the survey when compared with the previous 5 years.

The distribution of cases of EMND referred to the Cornell by state over the years is shown in Table 3. Most of the cases were from New York State, whereas the other cases were from Connecticut, Florida, Ohio, and Pennsylvania, respectively.

## Equine Protozoal Myelitis

Data on the occurrence of EPM are reported in Table 5. Forty-one percent of the surveyed practitioners reported seeing at least one case of EPM in the previous 12 months, whereas 22% indicated they had seen between two and five cases. Most practitioners reported that this was fairly typical

**Table 4.** Perception of practitioners' regarding the occurrence of EMND

| Number of EMND Cases Evaluated in the past 12 Months | Number | Percent of Total |
|---|---|---|
| 0 | 39 | 76.5% |
| 1 – 2 | 7 | 13.7% |
| 2 – 5 | 2 | 3.9% |
| 5 – 10 | 1 | 2.0% |
| >10 | 0 | 0% |
| Did not respond | 2 | 3.9% |
| **Frequency of EMND over the Last 12 Months in Comparison with the Last 5 Years** | **Number** | **Percent of Total** |
| Less than is typical | 15 | 29.4% |
| Fairly typical | 25 | 49.0% |
| More than is typical | 0 | 0% |
| Did not respond | 11 | 21.6% |
| **Frequency of EMND over the Past 10 Years** | **Number** | **Percent of Total** |
| Decreased substantially | 8 | 15.7% |
| Decreased somewhat | 5 | 9.8% |
| Remained relatively unchanged | 27 | 52.9% |
| Increased somewhat | 1 | 2.0% |
| Increased substantially | 1 | 2.0% |
| Did not report | 9 | 17.6% |
| **Average Survival Time of EMND Patients after Diagnosis** | **Number** | **Percent of Total** |
| <2 weeks | 2 | 3.9% |
| 2 weeks – 2 months | 5 | 9.8% |
| 2 – 6 months | 5 | 9.8% |
| 6 months – 1 year | 3 | 5.9% |
| 1 – 3 years | 3 | 5.9% |
| >3 years | 5 | 9.8% |
| Did not report | 28 | 54.9% |

**Table 5.** Perception of practitioners regarding the occurrence of EPM

| Number of EPM Cases Evaluated in the Past 12 Months | Number | Percent of Total |
|---|---|---|
| 0 | 7 | 13.7% |
| 1 – 2 | 21 | 41.2% |
| 2 – 5 | 11 | 21.6% |
| 5 – 10 | 6 | 11.8% |
| >10 | 3 | 5.9% |
| Did not respond | 3 | 5.9% |
| **Frequency of EPM over the Last 12 Months in Comparison with the Last 5 Years** | **Number** | **Percent of Total** |
| Less than is typical | 11 | 21.6% |
| Fairly typical | 33 | 64.7% |
| More than is typical | 5 | 9.8% |
| Did not respond | 2 | 3.9% |
| **Frequency of EPM over the Past 10 Years** | **Number** | **Percent of Total** |
| Decreased substantially | 7 | 13.7% |
| Decreased somewhat | 9 | 17.6% |
| Remained relatively unchanged | 24 | 47.1% |
| Increased somewhat | 4 | 7.8% |
| Increased substantially | 3 | 5.9% |
| Did not report | 4 | 7.8% |

(64%) or less than typical (22%) in comparison with the previous 5 years. Forty-seven percent of the practitioners indicated that the frequency of EPM over the past 10 years had remained relatively unchanged.

Practitioners who perceived that the occurrence of EPM had decreased over the 10-year period of the survey (31%) attributed the decline to difficulty and cost of definitive diagnosis, previous overdiagnosis, immunity, vaccination, or better ability to definitively diagnose similar diseases.

Fourteen percent of the practitioners perceived that the occurrence of EPM had increased over the 10-year period of the survey, citing increased awareness of the disease, better protocol for EPM evaluation, better diagnostics, warmer winters, and opossum migration into new regions as possible reasons for the increase.

A total of eight cases of EPM were diagnosed at Cornell University's Equine Hospital in the 12-month period between June 1, 2006 and June 1, 2007, 36 cases in the 5-year period between June 1, 2002 and June 1, 2007, and 129 cases in the 10-year period between June 1, 1997 and June 1, 2007. These observations indicate a decrease in the frequency of EPM diagnosis at Cornell over the 5 years before the survey when compared with the previous 5 years. The distribution of cases of EPM referred to the Cornell by state over the years is shown in Table 3. Most of the cases were from New York State, whereas the other cases were from Connecticut, Florida, Kentucky, New Jersey, Massachusetts, and Pennsylvania, respectively.

### Equine *Borrelia* Infection

Data on the distribution of the occurrence of equine *Borrelia* infection and associated clinical signs as perceived by the surveyed practitioners are shown in Tables 6 and 7. When

**Table 6.** Perception of practitioners regarding the occurrence of equine *Borrelia* infection

| Belief about Equine *Borrelia* Infection (Lyme Disease) | Number | Percent of Total |
|---|---|---|
| **(1)** *Borrelia* infection in horses is common in my practice | 14 | 27.5% |
| **(2)** *Borrelia* infection is horses is absent or rare in my practice | 23 | 45.1% |
| **(3)** Lyme disease (clinical disease related to *Borrelia* infection) does not exist in horses | 1 | 2.0% |
| **(4)** Lyme disease (clinical disease related to *Borrelia* infection) is common in horses in my practice | 4 | 7.8% |
| **(5)** Did not report | 3 | 5.9% |
| Selected both **(1)** & **(2)** | 1 | 2.0% |
| Selected both **(1)** & **(3)** | 1 | 2.0% |
| Selected both **(1)** & **(4)** | 4 | 7.8% |

asked to select what best described what their belief about equine *Borrelia* infection in horses, opinions were divided among practitioners, with 45.1% indicating they thought *Borrelia* infection in horses was absent or rare in their practice. A total of 27.5% of those surveyed indicated they thought that infection in horses was common in their practice. Only 16% of those surveyed believed that Lyme disease, defined as clinical disease related to Borrelia infection, was common in horses in their practice.

When asked about the following clinical signs of *Borrelia* infection, opinions varied widely; however, according to the majority of practitioners, stiffness (31.4%) and behavior change (37.3%) were observed fairly often, whereas ataxia (35.2%) was never seen. Lameness, swollen joints, hyperesthesia, and muscle wasting were sometimes observed. Other clinical signs of disease related to *Borrelia* infection were reported by 17.6% of those surveyed, including weight loss, intermittent anorexia, depression and listlessness of 1 to 2 weeks' duration, popping and cracking of joints when moving, decrease in appetite, shifting leg lameness, loss of energy, diarrhea, fever, poor performance, and reaction at the tick bite site. Many of these signs were reported by more than one practitioner. Cornell University's Equine Hospital diagnosed 11 cases of equine borreliosis in the 10-year period between June 1, 1997 and June 1, 2007. In 45% of these cases, lameness was the principal clinical sign. Other clinical signs included stiffness, swollen joints, hyperesthesia, behavior change, muscle wasting, ataxia, uveitis, and facial nerve paralysis. The distribution of cases of *Borrelia* referred to the Cornell by

**Table 7.** Perception of practitioners regarding the clinical signs of equine *Borrelia* infection

| Lameness—Frequency | Number | Percent of Total |
|---|---|---|
| Never | 3 | 5.9% |
| Rarely | 13 | 25.5% |
| Fairly often | 10 | 19.6% |
| Almost every time | 4 | 7.8% |
| Did not report | 21 | 41.2% |
| **Stiffness—Frequency** | **Number** | **Percent of Total** |
| Never | 0 | 0% |
| Rarely | 4 | 7.8% |
| Fairly often | 16 | 31.4% |
| Almost every time | 11 | 21.6% |
| Did not report | 20 | 39.2% |
| **Swollen Joints—Frequency** | **Number** | **Percent of Total** |
| Never | 7 | 13.7% |
| Rarely | 15 | 29.4% |
| Fairly often | 5 | 9.8% |
| Almost every time | 2 | 3.9% |
| Did not report | 22 | 43.1% |
| **Hyperesthesia—Frequency** | **Number** | **Percent of Total** |
| Never | 6 | 11.8% |
| Rarely | 13 | 25.5% |
| Fairly often | 7 | 13.7% |
| Almost every time | 1 | 2.0% |
| Did not report | 24 | 47.1% |
| **Behavior Change—Frequency** | **Number** | **Percent of Total** |
| Never | 2 | 3.9% |
| Rarely | 5 | 9.8% |
| Fairly often | 19 | 37.3% |
| Almost every time | 4 | 7.8% |
| Did not report | 21 | 41.2% |
| **Muscle Wasting—Frequency** | **Number** | **Percent of Total** |
| Never | 12 | 23.5% |
| Rarely | 12 | 23.5% |
| Fairly often | 5 | 9.8% |
| Almost every time | 0 | 0% |
| Did not report | 22 | 43.1% |
| **Ataxia—Frequency** | **Number** | **Percent of Total** |
| Never | 18 | 35.2% |
| Rarely | 8 | 15.7% |
| Fairly often | 2 | 3.9% |
| Almost every time | 1 | 2.0% |
| Did not report | 22 | 43.1% |
| **Other Clinical Signs Observed** | **Number** | **Percent of Total** |
| Yes | 9 | 17.6% |

**Table 8.** Cornell University's Equine Hospital historical data on equine *Borrelia* infection

| Clinical Sign | Number | Percent of Total |
|---|---|---|
| 1. Lameness | 5 | 45.0% |
| 2. Stiffness | 1 | 9.1% |
| 3. Swollen joints | 0 | 0% |
| 4. Hyperesthesia | 0 | 0% |
| 5. Behavior change | 0 | 0% |
| 6. Muscle wasting | 0 | 0% |
| 7. Ataxia | 1 | 9.1% |
| 8. Uveitis | 1 | 9.1% |
| 9. Facial nerve paralysis | 0 | 0% |
| 1, 3, 4 & 6 | 1 | 9.1% |
| 1 & 8 | 1 | 9.1% |
| 7 & 9 | 1 | 9.1% |

**Table 9.** Perception of practitioners regarding the occurrence of *P. tenuis*

| Observed a Young Horse with Acute Onset of Cervical Scoliosis and the Classic Signs of *P. tenuis* | Number | Percent of Total |
|---|---|---|
| Yes | 5 | 9.8% |
| No | 43 | 84.3% |
| Did not report | 3 | 5.9% |
| **Among Those Practitioners That Had Seen a Case of *P. tenuis*, Number of Cases Seen over the Last 5 Years** | **Number** | **Percent of Those Practitioners That Had Seen a Case of *P. tenuis*** |
| 0 | 0 | 0% |
| 1 – 2 | 5 | 100% |
| 2 – 5 | 0 | 0% |
| 5 – 10 | 0 | 0% |
| >10 | 0 | 0% |
| Did not report | 0 | 0% |

state over the years is shown in Table 3. Information on associated clinical signs is shown in Table 8.

### Parelaphostrongylus tenuis

Data on the perception of practitioners regarding *P. tenuis* are shown in Table 9. Most of those surveyed (84.3%) reported that they had not seen young horses with acute onset of cervical scoliosis and the classical signs of *P. tenuis*. Among the other practitioners who reported seeing at least one case of *P. tenuis*, none of them reported seeing more than two cases in the previous 5-year period.

No cases of *P. tenuis* was admitted to the Cornell University's Equine Hospital in the 12-month period between June 1, 2006 and June 1, 2007, whereas four cases of the disease were diagnosed in the 5-year period between June 1, 2002 and June 1, 2007 and five cases in the 10-year period between June 1, 1997 and June 1, 2007. These data indicate an increase in the frequency of *P. tenuis* diagnosis at Cornell University College of Veterinary Medicine over the 5 years before the survey when compared with the previous 5 years. Information on cases of *P. tenuis* referred to Cornell over the years is shown in Table 3. All cases were from New York State.

## DISCUSSION AND CONCLUSION

Practitioners indicated they believe that the frequency of disease associated with the neurologic strain of EHV-1 has remained relatively unchanged or increased somewhat during the last 10 years. This could be real or perceived because of the large numbers of articles written on EHV-1 in veterinary and horse journals in the past 4 years. Cornell University's Equine Hospital medical records support a slight increase in the diagnosis of the neurologic strain of EHV-1 over the 10-year period of the survey. An increase in EHV-1 myelitis could be explained by the increased prevalence in the mutant neurologic form of EHV-1 as previously reported.[29] Herpes myeloencephalitis is most common in middle-aged or older[12,13] sport horses, and increased travel during the past decade of those horses as well as mixing of horses from different areas also might explain an increased incidence of the disease.

The opinion of most practitioners surveyed regarding the frequency of EMND and EPM is that the incidence of these diseases has remained relatively stable or decreased somewhat over the last 10 years. A computer query of Cornell University's Equine Hospital medical records supported a decrease in the frequency of EMND and EPM diagnosis over the 10-year period of the survey.

The historical onset of EMND in the early 1990s may reflect changes in management practices about that time, such as increased stabling of horses and decreased access to pasture in addition to improved neurodiagnostics including electromyography and neuropathology. If decreases in disease incidence over the last decade have occurred, they are likely attributable to improvements in diet, including vitamin E supplementation in unpastured horses, because vitamin E deficiency has been directly associated with EMND.[30] Survival time after initial diagnosis of EMND by a surveyed veterinarian was variable, which may be explained by differences in severity and duration of the clinical disease in addition to owner economics. Variable progression in the clinical disease EMND has been previously reported.[16]

Possible decreases in EPM over the last 10 years could be attributed to previous overdiagnosis coupled with

improved ability to interpret serologic tests for EPM and to definitively diagnose similar-appearing neuromuscular diseases. The identification of the definitive host for *S. neurona* and decreased exposure of horse feed to the host could likely result in decreased incidence of EPM.

Survey results indicate varying beliefs regarding *Borrelia* infection causing clinical signs in horses. Opinions regarding the typical signs, if any, that may be associated with *Borrelia* infection in the equine are also disparate, although stiffness and behavior change appear to be among those most frequently observed, and thus equine *Borrelia* infection and Lyme disease should be considered for a differential diagnosis for horses displaying these signs. Evaluation of such horses at Cornell University's Equine Hospital did find high serologic titers in most of the horses referred for Lyme disease and positive fluid or synovial PCR for *Borrelia* in three of the cases. Although these findings document *Borrelia* infection, they do not prove that the infection is the cause of the clinical signs. Two cases of Lyme neuroborreliosis were tentatively diagnosed at Cornell based on clinical signs, serology, and the postmortem finding of a lympho/plasmacytic meningitis. Confirming the diagnosis by response to treatment is further complicated because doxycycline has been shown to have anti-inflammatory effects and inhibit cytokine-stimulated metalloproteinases in equine synoviocytes.[31]

Most practitioners surveyed indicated that they never diagnosed a case of *P. tenuis* in a horse; however, Cornell University's Equine Hospital medical records indicate that although the disorder is not common, a substantial increase in the diagnosis of *P. tenuis* in the equine has occurred over the last 10 years. This increase is thought to be attributable, at least in part, to heightened awareness of the disease at this institution, and it would be prudent for practitioners to consider this as a differential for neurologic disease in the equine, particularly in those patients exhibiting the classic signs of acute onset of cervical scoliosis that can be manually straightened temporarily without pain, analgesia of the neck on the convex side, and mild or no ataxia.

In conclusion, because the conditions discussed herein remain of great clinical relevance, knowledge of the frequency with which they occur as well as an understanding of possible associated risk factors are crucial in enabling the equine practitioner to make an appropriate diagnosis in cases of neurologic disease in their patients. A general knowledge of prevalence of diseases such as EPM or Lyme disease in a geographic area is important in assessing the predictive value of serologic test. In both diseases, seropositive horses are common in some Northeastern areas; however, the positive predictive values of the tests are greatly affected by current prevalence of the clinical disease.

## REFERENCES


1. Nuggent J, Birch-Machin I, Smith KC, Mumford JA, Swann Z, Newton JR, et al. Analysis of equid herpesvirus 1 strain variation reveals a point mutation of the DNA polymerase strongly associated with neuropathogenic versus nonneuropathogenic disease outbreaks. J Virol 2006;80:4047–4060.
2. Goodman LB, Loregian A, Perkins GA, Nugent J, Buckles EL, Mercorelli B, et al. A point mutation in a herpesvirus polymerase determines neuropathogenicity. PLoS Pathog 2007;3:e160.
3. de La Rua-Domenech R, Mohammed HO, Cummings JF, Divers TJ, de Lahunta A, Valentine B, et al. Incidence and risk factors of equine motor neuron disease: an ambidirectional study. Neuroepidemiology 1995;14:54–64.
4. de la Rua-Domenech R, Mohammed HO, Cummings JF, Divers TJ, de lahunta A, Summers BA. Intrinsic, management, and nutritional factors associated with equine motor neuron disease. J Vet Med Assoc 1997;211:1261–1267.
5. Dubey JP, Saville WJ, Lindsay DS, Stich RW, Stanek JF, Speer CA, et al. Completion of the life cycle of Sarcocystis neuroma. J Parasitol 2000;86:1276–1280.
6. Chang YF, Novosol V, McDonough SP, Chang CF, Jacobson RH, Divers T, et al. Motor neuron disease. Vet Pathol 2000;37:68–76.
7. Tanabe M, Kelly R, de Lahunta A, Duffy MS, Wade SE, Divers TJ. Verminous encephalitis in a horse produced by nematodes in the family prostrongylidae. Vet Pathol 2007;44:119–122.
8. Charlton KM, Mitchell D, Girard A, Corner AH. Meningoencephalomyelitis in horses associated with equine herpesvirus 1 infection. Vet Pathol 1976;13(1):59–68.
9. Jackson TA, Osburn BI, Cordy DR, Kendrick JW. Equine herpesvirus 1 infection of horses: studies on the experimentally induced neurologic disease. Am J Vet Res 1977;38:709–719.
10. Pursell AR, Sangster LT, Byars TD, Divers TJ, Cole J Jr. Neurologic disease induced by equine herpesvirus 1. J Am Vet Med Assoc 1979; 175:473–474.
11. Patel JR, Heldens J. Equine herpesvirus 1 (EHV-1) and 4 (EHV-4)-epidemiology, disease, and immunoprophylaxis: a brief review. Vet J 2005;170:14–23.
12. Allen GP, Breathnach CC. Quantification by real-time PCR of the magnitude and duration of leucocyte-associated viraemia in horses infected with neuropathogenic vs. non-neuropathogenic strains of EHV-1. Equine Vet J 2006;38:252–257.
13. Goehring LS, van Winden SC, van Maanen C. Sloet van Oldruitenborgh-Oosterbaan MM. Equine herpesvirus type 1-associated myeloencephalopathy in The Netherlands: a four-year retrospective study (1999-2003). J Vet Intern Med 2006;20:601–607.
14. Goodman LB, Wagner B, Flaminio MJ, Sussman KH, Metzger SM, Holland R, et al. Comparison of the efficacy of inactivated combination and modified-live virus vaccines against challenge infection with neuropathogenic equine herpesvirus type 1 (EHV-1). Vaccine 2006; 24:3636–3645.
15. Cummings JF, de Lahunta A, George C, Fuhrer L, Valentine BA, Cooper BJ, et al. Equine motor neuron disease: a preliminary report. Cornell Vet 1990;80:357–379.

16. Divers TJ, de Lahunta A, Hintz HF, Riis RC, Jackson CA, Mohammed HO. Equine motor neuron disease. Equine Vet Educ 2001;13:63–67.
17. Mohammed HO, Cummings JF, Divers TJ, de la Rua-Domenech R, de Lahunta A. Epidemiology of equine motor neuron disease. Vet Res 1994;25:275–278.
18. de la Rua-Domenech R, Mohammed HO, Cummings JF, Divers TJ, de Lahunta A, Summers BA. Association between plasma vitamin E concentration and the risk of equine motor neuron disease. Vet J 1997;154:203–213.
19. Dubey JP, Davis SW, Speer CA, Bowman DD, de Lahunta A, et al. *Sarcocystis neuroma* n. sp. (Protozoa: Apicocomplexa), the etiologic agent of equine protozoal myeloencephalitis. J Parasitol 1991;77: 212–218.
20. Dubey JP, Lindsay DS, Saville WJ, Reed SM, Granstrom DE, Speer CA. A review of Sarcocystis neuroma and equine protozoal myeloencephalitis (EPM). Vet Parasitol 2001;95:89–131.
21. Duarte PC, Ebel ED, Traub-Dargatz J, Wilson WD, Conrad PA, Gardner IA. Indirect fluorescent antibody testing of cerebrospinal fluid for diagnosis of equine protozoal myeloencephalitis. Am J Vet Res 2006;67:869–876.
22. Mackay RJ, Tanhauser ST, Gillis KD, Mayhew IG, Kennedy TJ. Effect of intermittent oral administration of ponazuril on experimental Sarcocystis neuroma infection of horses. Am J Vet Res 2008;69:396–402.
23. Furr M, McKenzie H, Saville WJ, Dubey JP, Reed SM, Davis W. Prophylactic administration of ponazuril reduces clinical signs and delays seroconversion in horses challenged with Sarcocystis neuroma. J Parasitol 2006;92:637–643.
24. Berdorger W. Discovery of the Lyme disease spirochete and its relation to tick vectors. Yale J Biol Med 1984;57:515–520.
25. Hahn CN, Mayhew IG, Whitwell KE, Smith KC, Carey D, Carter SD. A possible case of Lyme borreliosis in a horse in the UK. Equine Vet J 1996;28:84–88.
26. Divers TJ. Lyme disease. In: Sellon D, Long M, eds. Equine infectious diseases. Philadelphia: WB Saunders; 2007:310–312.
27. Chang YF, Ku YW, Chang CF, Chang CD, McDonough SP, Divers T, et al. Antibiotic treatment of experimentally Borrelia burgdoferi-infected ponies. Vet Microbiol 2005;107:285–294.
28. Van Biervilet J, de Lahunta A, Ennulat Ed, Oglesbee M, Summers B. Acquired cervical scoliosis in six horses associated with dorsal grey column chronic myelitis. Equine Vet J 2004;36:86–92.
29. Allen GP, Bolin DC, Bryant U, Carter CN, Giles RC, Harrison LR, et al. Prevalence of latent, neuropathogenic equine herpesvirus-1 in the Thoroughbred broodmare population of central Kentucky. Equine Vet J 2008;40:105–110.
30. Divers TJ, Cummings JE, de Lahunta A, Hintz HE, Mohammed HO. Evaluation of the risk of motor neuron disease in horses fed a diet low in vitamin E and high in copper and iron. Am J Vet Res 2006;67:120–126.
31. Fortier LA, Motta T, Greewald RA, Divers TJ, Mayr KG. Minocycline and doxycycline suppress IL-1 and MMP-13-induced catabolism to a greater extent in synoviocytes than articular. In press.