TEMPOROHYOID OSTEOARTHRPATHY

Temporohyoid osteoarthropathy is a relatively common neurologic disorder in the horse. Since 2000, we have examined 26 horses with this condition. The etiology of this arthritic disease is unknown and although previous otitis interna/media is generally blamed there is little evidence to support this hypothesis. Only a small number of cases have active infection (mostly gram positive cocci and one case Aspergillus) of the inner ear area. The disease is most common in middle age horses, youngest that we have examined being 4 years of age, and approximately 40% have bilateral disease, although the clinical signs are almost always unilateral. This may very well be an osteoarthritic disease in the horse with certain risk factors such as cribbing. The signs are variable with some horses having behavioral signs associated with being ridden or while eating; these are likely signs of pain. Acute onset of neurologic signs are believed to occur when the fused T-H joint fractures. Fractures may be associated with sudden movements of the head and have sometimes been reported in horses following passage of a nasogastric or after dental procedures. Neurologic signs are those associated with cranial nerve VII and/or VIII dysfunction. Diagnosis is made by clinical signs, endoscopy of the guttural pouch, radiographs or computed tomography. CSF can be inflammatory in approximately 25% of cases and in a rare case, septic (consider Staphylococcus aureus). Treatment goals are directed towards decreasing inflammation and infection, if present, and preventing complications, of which exposure keratitis due to facial nerve paralysis is perhaps the greatest complication. Approximately 50% of the horses return to use with medical treatment. Unfortunately, we are not able to predict clinical response at the time of the initial diagnosis! Some horses may require many months for nerve function to return. We have routinely recommended a ceratohyoidectomy for both patient comfort and to prevent further fractures at the fused T-H joint(s).

CNS TRAUMA

Trauma to the CNS continues to be a major cause of morbidity and mortality in horses. Spinal Cord trauma is more common than brain trauma in race horses and performance horses mostly due to falling and injury to the cervical vertebrae. Horses experiencing acute brain trauma with either recumbency of several hours or those with fractures involving the basilar bones were associated with a poor prognosis. Specialized imaging may be helpful in these cases and in horses with other brain disorders. Horses with abnormal mentation due to brain disease are likely to have abnormal CT findings, (odds of having abnormal results for CT imaging of the head in a horse with head trauma and abnormal mentation was 30 times the odds for a similar horse without abnormal mentation). Basic treatments for head trauma include maintenance of oxygenation, perfusion to the brain, decreasing edema formation, stopping hemorrhage, controlling seizures, inflammation and oxidative damage in addition to repair of displaced fractures and prevention of secondary infection. Treatment of spinal cord trauma is similar but surgery and/or immobilization may be more important aspects of cervical cord trauma.
**BOTULISM**

Botulism in horses seems to have an increase in incidence associated with the more common practice of feeding “round bales” of hay. Diffuse neuromuscular weakness occurs in all cases with dysphagia being present in most cases of type B botulism which is most common in the Northeast U.S. If a dead animal is in the feed type C may occur. In the adult horse the disease is caused by ingesting the preformed toxin in decaying forage and rarely is it caused by wound contamination. Botulism in 3-8 week old foals due to ingestion of the organism and sporulation in the bowel lumen, “shaker foal syndrome” is common in those areas that also observe type B botulism in adults. Antitoxin should be administered early in the course of the disease, feeding and fluids should be administered via nasogastric tube and antibiotics (not aminoglycosides, procaine penicillin or tetracycline) administered as treatment for aspiration pneumonia. Adult horses that are recumbent and cannot rise have an extremely low prognosis.

**EQUINE PROTOZOAL MYELITIS**

I think we would all agree that equine protozoal myelitis (EPM) has been for the North and South American equine practitioner a difficult and contentious disease. Our ability to properly diagnose the disease has not been good and many questions remain regarding proper treatment. Only with an understanding of epidemiology of the disease, diagnostic tests, pharmacokinetics and pharmacodynamics of the anti/protozoal medications and from clinical experience can we make the most educated recommendations for treatment and prevention of the disease.

The diagnosis of EPM is based upon knowledge of: prevalence of the disease in a geographic area, the most common clinical signs, risk factors for the disease, awareness of sensitivity and specificity of diagnostic tests, in addition to ruling out other disorders and response to treatment. The disease should only exist in horses exposed to opossums which are in an area from lower Maine to southern Florida on the east coast. Racing and show horses are at greatest risk. There is no proven breed predisposition, although Standardbreds were reportedly more common in 2 studies. The disease incidence peaks at 4-6 horse years of age and rarely affects horses less than 1 year of age. Infection and clinical diagnosis is less common in the winter months in the more northern states. Adverse events and shipping within last 60 days appear to be additional risk factors. The clinical signs include ataxia in 95% of cases, muscle atrophy in approximately 40% of cases, cranial nerve deficits in less than 12% of cases, seizure in 6% and some mild change in mentation in many cases. The ataxia is mostly tetra ataxia (70%), hind limb ataxia 20% with a very low percentage having coccygeal signs. Ataxia and/or atrophy is asymmetrical in approximately 69% of cases. Cranial nerve dysfunction is asymmetrical in virtually all of the cases with frequency of lesions being greatest in cranial nerve 7 followed by 5, 8, 12, 9, 3 in that order. The most common and confusing differential for EPM is cervical compressive myelopathy (CCM). An understanding of risk factors, clinical signs and diagnostic tests for CCM (see below) are helpful in distinguishing EPM from CCM. There is no highly sensitive and specific serologic test for EPM. There is a fairly close correlation between a high IFA and a positive (not weak positive) western blot test. There is some correlation between a high IFA and likelihood of EPM but the exact correlation is not known and reports on likelihood
% of disease may sometimes be a little misleading in my opinion. The IFA does not have a high sensitivity, but has a modest specificity (13% or more of non-EPM neurologic cases having titers \( \geq 320 \)) and sometimes a prolonged turnaround time for results if both \( S. neurona \) and \( Neospora hughesi \) are tested. The WB for \( S. neurona \) has a sensitivity of 87-92% on serum but low specificity. Testing of CSF will improve WB specificity approximately 30% above serum specificity. The UC Davis IFA test offers an advantage of also testing for \( Neospora hughesi \) but infection with this protozoan agent is rare in horses in the east (1% in one Kentucky study). The “new” SAG 2 and 3/4 ELISA assay should improve specificity of testing but only if CSF is tested. SAG 2, 3 and 4 are generally expressed in the merozoite and sporozoite stages. An ELISA based upon the \( Sarcocystis neurona \) surface antigen 1,5,6 (SAG-1,5,6) is commercially available, but of 14 \( S. neurona \) isolates tested, 7 did not express the SAG-1 antigen. An updated SAG-1,5,6 stall side test is now available but there is minimal scientific data on the sensitivity and specificity of the assay. In my opinion, until a more accurate test becomes available, we should continue to combine historical information, clinical impression (including response to therapy) with either WB, IFA, or preferably ELISA 2,3-4 testing of at least serum and ideally CSF, in addition to response to treatment for diagnosing EPM.

The only U.S. Food and Drug Administration approved and available drugs for treating EPM are Ponazuril (Marquis®), Diclazuril (Protazil®), ReBalance® (pyrimethamine /sulfadiazine, PRN Pharmacal). Both ponazuril and diclazuril are triazine anti-protozoal agents effective in-vitro and in-vivo against a large number of apicomplexa organisms. These drugs have a long T ½ requiring several days to reach maximum serum and CSF concentration, both are static drugs and have low CSF to serum ratio (1:20). In vitro \( S. neurona \) is inhibited (but not killed) by 0.1 – 1 µg/ml of ponazuril and with current approved dosing of Marquis®, CSF concentrations are 0.16 µg/ml after 7 days of therapy. Although Marquis® is a very safe drug, my concern is that we do not give enough of the drug. The same might well be true for Protazil® although one study suggested \( S. neurona \) may be more sensitive to diclazuril. An inadequate dose of ponazuril may explain some poor responses and an estimated relapse rate of 9%. Alternatives to triazine treatments include the addition of pyrimethamine, a cidal anti-protozoal drug with better CSF to serum ratio (>25%) but lower bioavailability and higher toxicity and decoquinate, a 4-hydroxyquinoline, that is not Food and Drug Administration approved. Treatment with Th1 immune modulators could be “risky” as necropsy studies of EPM cases suggest there is a relative suppression of Th2 cytokines in those horses. Prognosis is somewhat related to: severity of the clinical disease, early and aggressive treatment and of course having the correct diagnosis. Prevention should focus on decreasing exposure to opossum feces (closed feed containers, protected hay storage). In endemic areas intermittent treatment during high risk periods are used. Racing and western performance horses have been found to be at increased risk of EPM. Intermittent treatment of at risk horses has been evaluated as a means of preventing CNS infection with \( S. neurona \). Administration of ponazuril every 7 days or diclazuril daily or every 3 days may have some efficacy in preventing disease. This time frame corresponds with recent research that investigated the kinetics of \( S. neurona \) invasion in the horse. A non-Food and Drug Administration approved treatment has been promoted for the past 2 years. This product is a combination of decoquinate (a coccidiostat) and levamisole (an anti-parasitical and immune modulating drug). The treatment course is 10 days and the drug is compounded. Decoquinate does appear to be highly effective against \( S. neurona \) in cell culture studies. To my knowledge
there is no published information on bioavailability of the drug in the horse and there is only a single (PubMed) article published on its use in horses.

CERVICAL COMPRESSION MYELOPATHY

Diagnosis of cervical vertebral compressive myelopathy (CVCM) continues to be an area of investigation. Intra-vertebral ratios on plain cervical films has proven to be the screening test of choice for most clinics, but sensitivity and specificity of the test are variable between clinics/radiologist reading the films, and there is poor correlation between the site of the abnormal ratio and the lesion site. Since the site of cord compression is often inter-vertebral rather than intra-vertebral in horses, an additional inter-vertebral measurement has been recommended. The inter-vertebral measurements more accurately reflect specific site(s) of compression and location and number may affect decisions to proceed with a myelogram and possibly surgery. Recently, we have even questioned the accuracy of the 50% reduction in the dorsal myelographic column for diagnosis of CVCM. Accuracy of this criteria seems to vary with vertebral site, C5-C7 being most accurate. Measurement of the reduction in dural diameter at the inter-vertebral sites in comparison to adjacent mid-vertebral body dural diameter might be more accurate for compression in the mid-cervical region. For horses >3 years of age, Levine has found older Warmbloods to be at highest risk of CVCM. Risk factors including breed predisposition and clinical signs for CVCM have recently been reviewed. In addition a large multi-center study looking at the signalment and diagnostic features of CVCM has been recently completed. Horses with CVM were younger (median 2 years) than control horses. Thoroughbred, Warmblood, and Tennessee Walking Horses were significantly over-represented in the CVM group. Horses with a history of racing or English performance were significantly more likely to be affected than those used for Western/pleasure activities. Asymmetric gait deficits (71/166) and cervical hyperesthesia (44/91) were common in CVM. The most frequent necropsy lesions were stenosis and articular process osteophytosis. Agreement between radiography and necropsy was between 65% and 71%. Agreement between myelography and necropsy was between 67% and 78%. The agreement between findings of necropsy and imaging was only moderate, indicating that accurate diagnosis with these techniques can be challenging.

One of the more interesting finding in equine neurology is that horses with cervical facet osteoarthritis may have stumbling gaits without involvment of the spinal cord or at least the myelogram is considered normal (could be lateral compression). A fair percentage of these horses have improved gaits following facet injection with corticosteroids. Cervical pain in horses has become a common diagnosis for an abnormal stiff, stumbling gait and cervical facet injections with corticosteroids have allowed return to function in many horses.

EQUINE HERPESVIRUS-1

Equine herpesvirus-1 (EHV-1) is, for multiple reasons, a problematic infectious disease in the horse. EHV-1 may cause several different clinical syndromes, some of which result in epidemics of disease following the arrival of an infected horse on the premise. The virus can also be maintained in carrier animals, which may then result in either self-disease (most likely) and/or spread at some later point in time. Recent studies have shown that carrier horses given very large and repeated doses of dexamethasone will have viral reactivation at only low levels and no or limited transfer of the virus to in contact horses. This helps explain why we have rarely had
EHV-1 outbreaks or spread in hospital housing although stressed or corticosteroid treated horses are commonplace and undoubtedly some are carriers. Spread of the virus is mostly horse to horse as it’s persistence in the environment is relatively short. Transmission is typically via infected nasal secretions, although fetal membranes and placental fluid from the aborted fetus are also highly infective. After direct contact, the virus first colonizes the nasal mucosa and then replicates in regional lymph nodes by 1-3 days. This is followed by viremia for 3-14 days (depending upon the strain). From day 2 or 3 post-infection to at least day 7, there should be high virus load in nasal secretions. With high virus loads and the appropriate host (mostly older horses with low levels of cytotoxic T lymphocyte response to EHV1) there might be sufficient transmission of EHV-1 from lymphocytes to CNS endothelial cells to cause CNS disease.

Outbreaks of neurologic disease, associated in many situations (83%) with a mutation in the polymerase gene, the mutant virus’s suppression of chemokines and cytokines, and high levels of viremia resulting in CNS vasculitis, and spinal cord disease.

There appears to be an age predilection for EHV1 myelitis. Adult horses > 4 years are the great majority of cases. There are rare reports in yearling and young adults. In one report older mares were more likely to be affected with the neurologic disease. Older horses tend to get higher levels of viremia than younger horses regardless of infection with neurotrophic or “non-neurotropic” strains. Although a minority, cases of neurologic disease are sometimes associated with the “non-neurotropic” strain. The current dogma that a significant percentage of EHM outbreaks are caused by a mutant strain of EHV-1, containing a single genetic substitution G2254 within the gene encoding the viral DNA polymerase may be overly simplistic. Outbreaks are most common at boarding stables, show facilities, and seemingly slightly less common at racetracks.

Is there a sex predisposition? One report suggests it is most common in non-pregnant, competition horses. Kendrick, in his early experimental studies, suggested EHM was more easily produced in pregnant mares. Does tissue factor (TF) activity along with level of viremia play a major role in the CNS pathology? TF is likely highest in the endothelial cells of CNS and placenta! Does prior vaccination increase or decrease the risk of developing EHV-1 neurologic disease? This is unproven and vaccination is strongly encouraged realizing that vaccines offer only partial protection.

Other questions:

Should we perform nasal swabs for PCR and genotyping on all horses with fever of unknown origin? What do we do with positive cases?

How contagious are horses with CNS signs? Moderate but the early febrile horses are highly contagious!

How do we prevent outbreaks at referral hospitals and show centers, etc.? How do we manage horses at a facility that has the virus? Ring quarantine, repeated testing, excellent vaccination are the standard recommendations. There is no definitive proof to my knowledge to suggest that a superior vaccine is available!
What are the indicators for the rather expensive treatment with either Valacyclovir 25 mg/kg PO q8-12h or Ganciclovir (2.5 mg/kg IV q8-12h) in EHV-1 outbreaks?

When, if at all, should steroids, aspirin and/or Clopidogrel be used in EHV-1 CNS disease? I believe there is a time for their use!

Should horses be slung with severe EHV-1 neurologic disease? What is their prognosis? For horses that need to be slung, recovery to functional use is rare but does occur.

What is the best way to manage the bladder and rectum atony which are often seen with EHV-1. Bethanechol, indwelling urinary catheter and rectal evacuation along with antibiotics are the standard of care for those horses with urinary and rectal dysfunction.

**PARELAPHOSTRONGYLUS TENUIS**

_Parelaphostrongylus tenuis_ infection in young horses is a recently reported neurologic disease of horses. In areas of North America that routinely diagnose _P. tenuis_ in small ruminants and camels, infection in the horse should be considered and will be seen! The clinical sign of acute onset scoliosis is nearly diagnostic for the equine disease. Affected horses have been 6 mo-3 years of age. The parasite appears to have a migratory predilection in the horse for the dorsal gray column of the cervical and sometimes thoracic cord. If the lesion extends three or more vertebral segments, acute onset scoliosis with the head deviated away from the lesion side will occur. Hypalgesia to analgesia will be found on the affected (convex) side over the affected segments. There may be very mild ataxia and paresis of the limbs on the affected side. In the few cases we have performed CSF collection and evaluation on, the results were unremarkable, unlike _P. tenius_ in small ruminants and camels. The signs are characteristic enough in horses in an endemic area to make the diagnosis. Initially, there is no pain in manipulation of the neck, but after several weeks, arthritic changes develop. Treatments have included high doses of ivermectin and steroids, but lesions have been so advanced that successful treatment does not seem possible. Some clients have worked with physical therapists to provide exercises and braces in hopes of a return to normalcy, but this has been uniformly unsuccessful.

**VIRAL ENCEPHALITIS**

The initial outbreak of WNV in horses (and humans) in North America occurred in 1999 with 25 equine cases. The greatest yearly number of cases (15,257) was in 2002. The outbreak initially spread from the northeastern U.S. to the southeastern U.S., followed by a western spread that eventually involve horses in all of the continental states. The percentage of infected horses that demonstrated clinical signs is unknown but was likely < 10%. Clinical signs of WNV are variable since disease may occur in any area of the central nervous system resulting in either changes in mentation, signs of cranial nerve deficits, ataxia and/or paresis. Fasciculations seemed to be unusually common compared to most other adult equine CNS disorders. Fever is not found in many of the naturally-occurring cases and was not present even in some experimentally infected horses. Diagnosis is by finding a positive capture IgM ELISA and ideally by seroconversion. Mortality rate in clinically affected horses has been variable, but generally <
30%. Clinically diseased horses that have rapid improvement in the first week of illness often appear to have full recovery. Some horses may have clinical improvement but maintain residual CNS deficits. Treatments for clinically diseased horses have consisted mostly of supportive therapy, although hyperimmune WNV plasma is available. Four vaccines are currently approved for WNV vaccination in horses in the U.S. and all appear to be highly efficacious if administered properly. In the eastern region of the U.S. equine eastern encephalomyelitis has been more of a problem than WNV since 2006! EEE virus occurs in natural cycles involving birds and Culiseta melanura in some swampy areas nearly every year in the late summer and fall. There is evidence for the introduction of southern EEEV strains to New York, followed by local amplification, perpetuation, and overwintering. The virus may occasionally escape from enzootic areas by other vectors (Aedes sp. Culex sp. etc.) and in birds to cause unexpected outbreaks of EEE. To my knowledge, most all cases are in horses without proper vaccination!! The vaccines for both WNV and EEE are considered to have excellent protection. Equine rabies, we see it and it can be hard to diagnose on the initial exam!

**PRIMARY HYPERAMMONEMIA**

Primary hyperammonemia is a not uncommon cause of acute and fulminant encephalopathy in horses often associated with mild colic 12-24 hours previously. This disease is caused by marked increase in enteric ammonia production which overwhelms the detoxification capacity of the normal liver (liver enzymes and function are normal). Blindness, dilated pupils, propulsive walking, head pressing are the most common signs. Blood ammonia, lactate and glucose are very high and bicarbonate is low. Treatments include: sedation if needed to control the horse, oral administration of neomycin and/or lactulose to lower ammonia production and crystalloid therapy with added KCL. Horses are generally better in 24 hours or have progressed to coma during that time. Survival rate is approximately 50%.

**NEONATAL ENCEPHALOPATHY**

The pathogenesis of neonatal encephalopathy (NE) is still unproven, but from a clinician’s point of view I believe there are two basic types: (1) the foal that is affected in utero often associated with placentitis or other causes of neonatal stress and appears mentally abnormal at birth; and (2) foals that appear normal for 4 to 30 hours before developing seizures, loss of affinity of the mare, etc. Foals with the first condition sometimes have multiple organ dysfunction, evidence of severe placental compromise including high creatinine, high calcium, and have a guarded prognosis. Foals in the second category are believed to have had focal hemorrhage in the brain at birth causing inflammation 4 to 30 hours later resulting in the neurologic signs. Dystocia may infrequently predispose to this. Treatments for the “dummy foal” are numerous but few, if any, are proven to be efficacious. I prefer the following treatments:

1. Control seizures: Valium (5 to 10 mg) and/or phenobarbital (5 mg/kg IV). If the foal is recumbent try to keep head slightly elevated.
2. Intranasal oxygen therapy: If hypothermic, warm slowly. Keep blood dextrose 80 to 150 mg/dl. Vitamin C intravenously (30 mg/kg) mixed in low maintenance fluids (20 to 40 ml/kg/day) as long as the foal is urinating; do not fluid overload! I also commonly give 5 ml of thiamine in the fluids.
3. Magnesium sulfate: 10 to 20 mg/kg IV three to four times daily. If the foal has hypoventilation, give caffeine (No Doz) 10 mg/kg. “I know there is some controversy about efficacy, but I have seen it work on several foals”.

5. If there is inadequate response to the above treatment, preferably mannitol or possibly DMSO IV are reasonable treatments.

6. Feeding the septic or encephalopathic foal: Feeding should not occur if the foal is hypotensive (based upon clinical examination) or hypothermic. If these are not an issue, the 50 kg foal should be fed 200 to 500 ml every two hours depending upon intestinal motility.

   More recently a concept of aberrant activity of the fetal HPA axis and/or elevated pregnancy concentrations has been reported to be associated with some mild cases of neonatal encephalopathy. Some of these foals may respond to thoracic squeeze (simulating passage through the birth canal) as a tactile stimulant; be careful of fractured ribs. Thus, NMS appears to represent a number of different but related physiologic and pathophysiologic conditions that accompany and soon follow expulsion from the uterus.

**NEUROBORRELIOsis**

A wide variety of clinical signs have been attributed to *Borrelia* infection in horses, but cause and effect have been difficult to document in most cases. Because of both the high prevalence of antibodies against *Borrelia* in horses in some regions of the U.S. and the difficulty in proving clinical disease in most cases, Lyme disease is undoubtedly the most controversial equine disease in those areas. The clinical signs most often attributed to equine Lyme disease include stiffness and lameness in more than one limb, muscle tenderness, hyperesthesia, lethargy, and behavioral changes. Unlike human Lyme disease, joint effusion has been minimal in most Lyme-suspect horses. Muscle wasting and pain over the thoracolumbar area have been present in a few horses with high serum titers and some of these horses have had neurologic signs. In one report, two horses were diagnosed with Lyme neuroborreliosis and both had chronic, necrosuppurative-to-nonsuppurative, perivascular-to-diffuse meningeoradiculoneuritis on necropsy examination. Hyperesthesia, lumbar pain and muscle wasting were the initial clinical findings followed by ataxia of all four limbs, facial nerve paralysis and finally head tremors with depression in one horse. On necropsy, spirochetes were identified by Steiner silver impregnation in both cases, predominantly in the affected dura mater of brain and spinal cord. *Borrelia burgdorferi sensu stricto* was identified by polymerase chain reaction, with the highest spirochetal burdens in tissues with inflammation, including the spinal cord, muscle, and joint capsules. In another report, a horse with severe neck stiffness that progressed to ataxia had lymphohistiocytic meningitis and *B. burgdorferi* DNA in the cerebrospinal fluid (CSF). That horse originally responded to doxycycline treatment but relapsed after discontinuing the treatment. Another case was a Thoroughbred hunter that presented for lameness and ataxia and had lymphocytic pleocytosis and was PCR positive for *Borrelia* on CSF analysis. The horse initially responded well to doxycycline but had some deterioration when treatment was discontinued. The author has examined two other suspect Lyme horses with ataxia and severe
lymphocytic infiltration and thickening of the meninges. Based upon these few cases, it may be
that ataxia and lumbar muscle wasting caused by lymphohistiocytic meningitis and
radiculoneuritis, with occasional fasciculations and neck stiffness, are common characteristics of
neuroborreliosis in the horse. CSF would likely show a lymphocytic pleocytosis and, although
uncommon in humans with neuroborreliosis, some horses have CSF that is PCR positive for B.
burgdorferi. The two most frequently used drugs for treatment of Lyme disease in horses are
intravenous (IV) tetracycline and oral (PO) doxycycline or minocycline. Minocycline is
preferred over doxycycline. Minocycline has better oral bioavailability than doxycycline in the
horse and it attains higher concentration in CSF and aqueous fluids because it is more
lymphophilic and less protein bound than doxycycline. Minocycline (4 mg/kg PO q12h) may
eventually replace doxycycline as preferred treatment for Lyme disease in the horse.
Doxycycline protocols as used in human Lyme disease may not be as successful in the horse
because of the low bioavailability of doxycycline in the horse and the likelihood of more long-
standing infections in horses than in humans prior to treatment.

Equine motor neuron disease (EMND) may have decreased in incidence or reporting but
sporadic cases exist. Recent work continues to explore the strong link between anti-oxidant
deficiencies, i.e., tocopherol and pro-oxidant excess in the pathogenesis of the disease.
Deficiencies in vitamin E may cause abnormal permeability in the CNS blood-brain barrier
which may play a role in the pathogenesis of this oxidative disease. Another oxidative disorder
of the equine CNS (neuronal axonal dystrophy or degenerative myelopathy) has been recently
reviewed and we sporadically see cases in young horses some of which must have heritable
components.

GUTTURAL POUCH MYCOSIS

Mycotic disease of the guttural pouch may cause acute onset of neurologic signs (mostly
dysphagia or epistaxis. The close relationship between guttural pouches, cranial nerves, and
sympathetic structures make neurologic abnormalities due to diseases of the guttural pouches
(especially mycosis) possible. Surgical ligation of arterial supply to the diseased site is the
preferred treatment. Medical treatment provides some benefit but if dysphagia is present many
months may be required before there is return of function.

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