



EQUINE HEALTH UPDATE

FOR HORSE OWNERS AND VETERINARIANS

Cervical Vertebral Stenotic Myelopathy (CVSM; WOBBLER SYNDROME)

By Ashley Payne, DVM Student (Class of 2021)
Co-authored by Dr. Sandy Taylor, Large Animal Internal Medicine

A neurological condition that may leave your horse with an abnormal gait that might be described as “wobbling” is cervical vertebral stenotic myelopathy (CVSM), also known as Wobbler Syndrome. CVSM is one of the most common causes of neurologic disease in sport horses. Neurologic signs of this disease are caused by vertebral compression of the spinal cord as it passes through the horse’s neck. Horses have 7 cervical (neck) vertebrae that protect the spinal cord as it passes through the spinal canal. (Thoracic, lumbar, and sacral vertebrae protect the spinal cord as it passes along the horse’s back but are not affected by CVSM.) CVSM compression occurs primarily at the joints where vertebral edges can pinch the spinal cord. Any joint space can be affected, with the joint nomenclature indicating which vertebrae are involved (e.g. “C3-C4” indicates the joint formed by the 3rd and 4th cervical vertebrae).

The effects of this disease can range from mild to very severe, depending on the degree of spinal cord compression. Signs are due to the horse’s inability to sense their limbs in space and/or move their limbs appropriately, since the nerve connection between their limbs and brain is damaged.



Typical clinical signs include incoordination that affects all four limbs, with the hind limbs often affected more severely. Mild cases might simply include subtle gait inconsistencies and difficulty changing leads, whereas severe cases might involve spontaneous falling or difficulty standing. Horses with CVSM might demonstrate toe dragging, stumbling, and standing in an abnormal position (**Figure 1**).

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Figure 1. Abnormal stance in a horse with CVSM.

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CVSM; Wobbler Syndrome *(continued from cover)*

There are two types of CVSM. The most common is seen in young horses (6 months to 3 years of age) and is a syndrome of “developmental orthopedic disease.” This syndrome is associated with predisposition to other developmental orthopedic diseases including osteochondrosis, inflammation of growth plates, and joint swelling. Compression of the spinal cord might occur consistently and is then considered “static,” or it might occur only with certain neck movements (“dynamic”). Risk factors for this disease include rapid growth, a high plane of nutrition, and genetics (i.e. breed). The second type of CVSM is due to arthritis and is typically seen in older horses (> 15 years of age). Compression of the spinal cord is typically “static” in these cases because thickened bone secondary to arthritis typically impinges on the spinal cord regardless of neck position; however, neck motion might make signs worse. With both types of CVSM, more than one of the cervical intervertebral joints can be affected.

A complete neurologic examination is the first step in obtaining a diagnosis. Other diseases that can cause similar clinical signs and should be ruled out include equine protozoal myeloencephalitis (EPM), equine degenerative myeloencephalopathy (EDM), and other causes of cervical spinal cord compression (e.g. abscess, tumor). Routine blood work, such as a complete blood count (CBC) and biochemical analysis, are normal in horses with CVSM. Cerebrospinal fluid (CSF) is typically normal but elevated white blood cells might be present in severe cases. Neck radiographs (X-rays) are necessary to determine if cervical vertebral malformations, malalignments, or other bony abnormalities are present; this can be done standing with sedation. In some cases, radiographs might not detect a subtle but consequential lesion; in those cases, a myelogram is necessary. This involves injecting a contrast agent into the space surrounding the spinal cord, after which radiographs are taken to highlight the area that is affected (**Figure 2**). Myelograms must be performed under general anesthesia. Computerized tomography (CT) scans can also show the location and severity of compressive lesions or malformations but can only be performed in small horses and/or the upper part of the neck due to current size limitations of the scanner.

Treatment options for this disease consist of medical management as well as surgical correction. Anti-inflammatory medications might decrease swelling around the spinal cord, and in young horses with developmental orthopedic disease, limiting exercise in addition to reducing fat and carbohydrate intake might help slow the development of CVSM. However, medical management alone is unlikely to lead to long-term improvement. In horses with cervical arthritis, corticosteroids and hyaluronate can be injected into the intervertebral joints to decrease inflammation, but improvement is often transient, and repeat injections are required. The best candidates for surgical treatment are young horses with only one or two

joints affected, mild to moderate clinical signs, and dynamic spinal cord compression. Surgical treatment involves fusing the affected vertebrae to decrease neck motion and subsequent spinal cord impingement (**Figure 3**). Clinical improvement in neurologic signs is expected in approximately 80% of horses that undergo surgical stabilization, with approximately 60% of horses returning to athletic function.

To summarize, CVSM can occur in young horses as part of developmental orthopedic disease and in older horses with neck arthritis. Horses with CVSM typically show signs of incoordination that affect all four limbs, and diagnostic testing often includes cervical radiographs, CSF analysis (spinal tap) and myelography. Conservative treatment is often unrewarding, although cervical steroid injections can improve symptoms in horses with arthritis. Surgical stabilization should be considered in horses with CVSM. Finally, it is critical that a veterinarian be called immediately if you notice incoordination in your horse! Do not ride your horse if you suspect neurologic disease and be very careful handling your horse; they might not have as much control of their legs as they should, which can result in injury to you, others or even your horse. The best course of action is to keep your horse in a stall until a veterinarian can examine him/her.



Figure 2. Myelography showing compression at C4-C5 in a horse with CVSM.

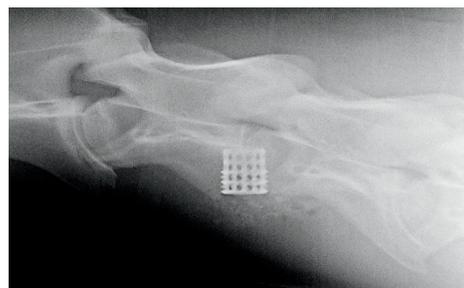


Figure 3. Surgical stabilization (fusion) of the cervical vertebrae with a stainless steel Bagby basket.

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News & Notes

NEW RESIDENTS



DR. STEFANIE HANSEN

is originally from Denmark. She received her Doctor of Veterinary Medicine degree from the University of Copenhagen in 2018. Following graduation, she went on to complete an internship at Rood and Riddle Equine hospital in Lexington, Kentucky. After completion of her internship, Dr. Hansen continued on as an intern at Donnington Grove Equine Hospital in England. Prior to joining the team at Purdue this September, Dr. Hansen spent three months working as an equine ambulatory veterinarian back home in Denmark. Her main areas of interest are equine orthopedics, upper airway, and gastrointestinal disease. However, Dr. Hansen is excited to learn from the wide variety of cases seen by the Large Animal Surgery service at Purdue.



DR. CARLA OLAVE

was raised in Valdivia in the south of Chile. She earned her Doctor of Veterinary Medicine degree from the Universidad Austral of Chile and continued on at Austral to complete an internship in equine medicine and surgery. Following the internship, Dr. Olave received a Master of Science degree at the same university during which time she developed an interest in studying Equine Asthma. This led her to join Dr. Couetil and his team at Purdue in 2017 for a PhD focused on studying Equine Asthma in racehorses. Dr. Olave is excited to continue to be a part of the Purdue family as a first-year Large Animal Internal Medicine resident. Her main areas of interest are neonatology, respiratory disease, and equine sports medicine.



Ascending Placentitis in Mares

By Michaela Helm, DVM Student (Class of 2021)
Edited by Dr. Jennifer Koziol, Theriogenology

Placentitis is one of the most common causes of pregnancy loss in mares. It occurs primarily during the last trimester of gestation and accounts for about 1/3 of all abortions and perinatal deaths in horses. Placentitis in the mare is usually categorized into one of two categories, ascending or nocardiform, with ascending placentitis being the most common type. Ascending placentitis is a result of microorganisms entering through the caudal reproductive tract, breaching the cervical barrier, spreading to the uterus, and subsequently infecting the placenta. Bacteria commonly implicated in infection include *Streptococcus equi* subsp. *zooepidemicus*, *E. coli*, *Staphylococcus aureus*, *Klebsiella* sp. and *Pseudomonas aeruginosa*. Middle-aged to aged mares that have had multiple foals are at the greatest risk for developing ascending placentitis. Other risk factors include poor perineal conformation and anatomic defects of the caudal reproductive tract such as pneumo-vaginitis or cervical fibrosis.

Clinical Signs

Outward clinical signs of ascending placentitis are often subtle or absent causing the infection to go undiagnosed. The most common clinical signs of placentitis include premature udder development and purulent vulvar discharge. Vulvar discharge is often the first sign of infection but is often undetected if the mare's perineal region is not inspected daily. Not all mares affected with fulminant placentitis present with vulvar discharge or premature udder development. Systemic health is rarely compromised in mares with placentitis. Blood counts, serum chemistry values, and blood lactate usually fall within normal ranges. Due to the inconsistent nature of clinical signs, routine evaluation beginning around the seventh month of gestation is recommended for mares that have previously experienced placentitis or are at higher risk. It is also important to evaluate perineal conformation and correct any anatomic defects prior to breeding.

Transrectal palpation and ultrasonographic examination

Transrectal ultrasonography is a very useful tool to evaluate placental integrity at the cervical star in late gestation, fetal activity, and fetal fluid character. Fetal orbit diameter can also be measured to estimate fetal age if breeding history is questionable. The caudal aspect of the allantochorion at the level of the cervical star is the most frequently affected area in the mares with placentitis because of the ascending route of infection. A thorough examination of this area

with ultrasonography is vital to diagnosing placentitis. In normal pregnant mares, the combined uterine and placental (chorioallantoic) unit is measured. Values of the combined uterine placental thickness (CTUP) for mares with normal pregnancies have been established.

Gestation Day	CTUP (mm)
271-300	<8
301-330	<10
>330	<12

Mares with placental infection or inflammation will have an increase in CTUP measurement or separation of the membranes with purulent material. CTUP can be easily identified by transrectal ultrasound. Once within the rectum, the transducer is advanced until you are just cranial to the cervical-placental junction. The transducer is then moved laterally until the large uterine vessel is visible at the ventral aspect of the uterine body. The CTUP is measured between the uterine vessel and allantoic fluid. A minimum of 3 measurements should be obtained and averaged.

Transabdominal ultrasonographic examination of the reproductive tract

Transabdominal ultrasonography is an excellent tool for evaluating fetal well-being, fetal fluid character, and some areas of the placenta. Fetal health is assessed by measuring fetal heart rate and activity. An examination is best achieved with a 2.5 or 3.5 MHz transducer with a depth setting of 20-30 cm. All four quadrants of the abdomen should be systematically evaluated.

The character of fetal fluids can be evaluated with transabdominal ultrasound. Ideally, an ultrasonographic examination of pregnancy is performed in the quiet mare and not immediately after transport or exercise. The allantoic fluid is generally anechoic (black) to slightly echogenic in late-term mares. Echogenic particles (snowfall appearance) can be noted after maternal or fetal activity but should resolve over time. The amniotic fluid is mild to moderately echogenic (light grey) in the normal pregnancy. Fluids that are highly echogenic and/or consistently have floating flocculent material may indicate infection. Mares with ascending infections usually have increased echogenicity of the amniotic compartment

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Progressive Ethmoidal Hematoma

By Katelyn Wilcox, DVM Student (Class of 2021) – Edited by Dr. Tim Lescun, Large Animal Surgery

Progressive ethmoidal hematoma (PEH) is a disease in which a hematoma (localized bleeding into a tissue), forms in the ethmoid turbinate region. The ethmoid turbinate is a combination of fine bone projections covered in a soft tissue lining that is located at the very back of the nasal passages (**Figure 1**). The hematoma behaves like a tumor due to its progressive and expansile nature, however it is not neoplastic (cancerous). The cause of PEH is unknown but a theory is that it is due to chronic bleeding of the ethmoidal submucosa. Horses that have PEH are usually middle-aged geldings and it is more commonly seen in Arabians, Thoroughbreds, Quarter Horses and Warmbloods. However, there hasn't been a significant correlation between breed and prevalence.

Clinical signs can start as a mild recurrent nose bleeds (epistaxis) or chronic nasal discharge. Some of the more subtle signs include decreased airflow from the nose, respiratory sounds, poor performance and dysphagia (difficulty swallowing). The typical presentation of PEH begins with seeing nasal discharge followed by intermittent epistaxis. If the hematoma is big enough, it can also obstruct the airway and cause respiratory distress.

Diagnosis is made with imaging such as endoscopy, radiograph (x-ray), computed tomography (CT) and MRI. Endoscopic examination of the nasal passage is the definitive way of diagnosing PEH as it allows direct visualization of the lesions. On endoscopy, the ethmoidal hematoma can appear as a smooth-walled and raised mass or it could be ulcerated and actively bleeding. The color is typically green/yellow/purple/red showing the various stages of blood breakdown within the hematoma (**Figure 2**). Radiographs are helpful in determining the location and size of the hematoma but are not useful in definitively diagnosing PEH. That is because hematomas show up as the same opacity as soft tissue structures such as abscess, cyst and other tumors. Unlike other growths, PEH rarely causes distortion of the bones of the face. Radiographs can also be limiting in that it can be difficult to differentiate specific structures and know the exact details of where the hematoma originates from and into where it extends. This is when CT or MRI are preferred, if surgery is pursued, to better plan for the surgery, especially in cases of a large hematoma. However, like radiographs, the hematoma also appears as a soft tissue mass on CT and MRI. This is why a nasal endoscopy is the better way to diagnose a PEH.



There are various ways of managing PEH. The first technique is removal of the hematoma by performing surgery through a frontonasal bone flap to access the sinuses. This can be done under general anesthesia or with standing sedation and local anesthetic. Another option is laser ablation of the hematoma (**Figure 3**), but the hematoma must be small enough for this with the recommended size being <5 cm in diameter. Another option is very risky because it involves injecting formalin into the hematoma, which could risk the formalin getting into the brain, eyes or other parts of the head and cause necrosis that then leads to blindness or death. There was a case report of a horse developing severe neurologic signs after formalin was injected into the PEH and so an owner should be aware of these risks if selecting this method of treatment.

Even with treatment, the hematomas often come back within a year or two, with a recurrence rate of almost 50%. Therefore, follow up endoscopy is recommended to monitor for recurrence. When a small hematoma is recognized early through regular monitoring, the treatment can be less costly and provide a better outcome for the horse in terms of treatment morbidity. Because hematomas can come back after treatment, owners may become frustrated with the repeated treatments and the cost involved. The prognosis can be good if the horse is monitored closely for recurrence, but the prognosis is guarded for a complete cure without recurrence from a single treatment.

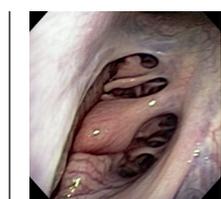


Figure 1. Normal ethmoidal turbinates viewed endoscopically



Figure 2. Ethmoidal hematoma viewed endoscopically



Figure 3. Ethmoidal hematoma post-laser ablation

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EQUINE WELLNESS FORUM

COLLEGE OF VETERINARY MEDICINE

The 2021 Virtual Equine Wellness Forum will take place on February 6, 2021 with subsequent sessions every Wednesday through March 3rd. The Equine Wellness Forum was designed to educate horse owners and equine industry professionals about current horse health issues, ranging from basic preventive health care and husbandry topics to state-of-the-art medical advancements. Connect with colleagues and learn the latest developments in equine medicine from experienced veterinarians! For more information, go to <https://vet.purdue.edu/ce/files/documents/20201209EWF%20Program.pdf> or contact Andrea Brown at ahbrown@purdue.edu or (765) 494-0611.

Ascending Placentitis *(continued from page 4)*

before the allantoic compartment. In contrast, mares with nocardioform placentitis, the amniotic fluid tends to remain normal, but the allantoic fluid becomes highly echogenic. Transabdominal ultrasound may also show placental separation and purulent material at the base of the horn when nocardioform placentitis is diagnosed.

Hormonal Assays and Biomarkers

Acute-phase proteins such as serum amyloid A (SAA) have been shown to increase in the face of placentitis. Serial SAA may be used to follow treatment success in mares with placentitis as SAA should lower following the initiation of treatment.

Treatment Strategies

Therapies for treating mares with placentitis should be selected for efficacy, pharmacokinetics of drugs in pregnant mares, and client compliance for administration. Although bacterial infections are the inciting cause, the subsequent inflammation and rise in prostaglandin are the reason for premature delivery of the foal. Treatment strategies are aimed at addressing infection, inflammation, and uterine contractility. Progestin in the form of altrenogest (i.e. Regumate) is a mainstay of therapy as it quiets the uterus combating contractility. Multiple antibiotics have been shown to pass through the placenta and into the uterine fluids, however, you must consider long

term use and client compliance in administration. Firocoxib, phenylbutazone, flunixin meglumine, and pentoxifylline have all been incorporated in protocols for their inflammatory properties.

Delayed delivery and improved foal survival are important endpoints when treating mares with placentitis. Foal viability following treatment with several drug combinations has been assessed in mares with induced placentitis. When a combination of progestins (altrenogest), TMS, and pentoxifylline treatment were used in one study, 10/12 mares delivered viable foals. In some cases, you may initially choose an aggressive treatment plan consisting of intravenously administered drugs for a short period (2-3 weeks) and continue long term treatment with orally administered drugs. The ultimate goal of placentitis treatment is to prolong pregnancy as long as possible to allow for appropriate fetal maturation.

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Got Mold?

EQUINE LEUKOENCEPHALOMALACIA

By Julia O'Rourke, DVM Student (Class of 2021) – Edited by Dr. Steve Hooser, Toxicology and Dr. Stacy H. Tinkler, Large Animal Internal Medicine

Equine leukoencephalomalacia (LEM), also known as “moldy corn disease,” is a serious, life-threatening condition that affects horses, ponies, donkeys, and mules. The causative agent of LEM is a group of mycotoxins referred to as fumonisins, of which B1 is the most prevalent. Fumonisin is produced from the metabolism of *Fusarium verticillioides* (formerly *F. moniliforme*), a fungus that grows on corn and cereal grains. Its distribution is worldwide and is primarily seasonal in occurrence. In North America, most cases are sporadically seen throughout late fall and into early spring although feeding of moldy corn can result in cases throughout the year. A dry growing season followed by a wet harvest has been found to be associated with the growth of *Fusarium spp.* and disease outbreaks.

Presentation and clinical signs

All equids are very sensitive to fumonisin toxicosis, however older horses may be more susceptible to disease compared to younger horses. Clinical signs associated with feeding of fumonisin-contaminated moldy corn usually become evident within 14 to 21 days of daily ingestion. The onset of clinical signs is acute, and death most often occurs 2 to 3 days thereafter. In some cases, there is no forewarning and sudden death may be the only clinical sign.

Fumonisin B1 is neurotoxic and results in central nervous system signs associated with leukoencephalomalacia. “Encephalomalacia” means softening of the brain tissue and is due to hemorrhage or inflammation. “Leuko” means white and leukoencephalomalacia therefore refers to softening of the white matter of the brain. It is considered one of the most serious types of brain injury and can affect specific parts of the brain or can be more widespread leading to complete dysfunction.

The earliest signs are predominantly neurologic and can include incoordination, circling, lethargy, anorexia, depression, head pressing, and blindness. These are quickly followed by hyperexcitability, agitation, sweating, and dementia. Recumbency and seizure-like convulsions tend to occur just prior to death. Fumonisin is also toxic to the liver, therefore affected horses may also show signs of liver damage, including inappetence, icterus (jaundice), edema of the head and neck, and petechiae (small bruises or bleeds) of mucous membranes.

Diagnosis

A diagnosis of LEM is primarily made based on clinical signs, physical exam, and a history of exposure to moldy corn confirmed by mycotoxin identification in the feed. Abnormalities observed on a biochemistry panel are nonspecific, but usually involve indications of liver damage, such as increases in serum concentrations of liver enzymes (AST, GGT, LDH, and serum bilirubin concentration). Cerebral spinal fluid analysis may show a nonspecific increase in protein

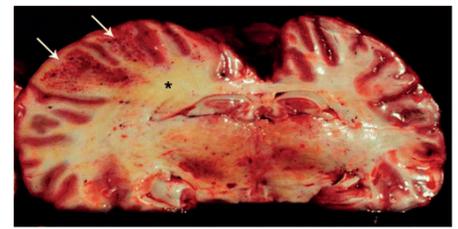


Figure 1. Equine brain affected by LEM. Left cerebral hemisphere is enlarged. There is white matter necrosis evidenced by yellow discoloration (star) and hemorrhage (arrows). Source: <https://journals.sagepub.com/doi/full/10.1177/1040638713504572>

and total nucleated cell counts. Postmortem examination is useful in confirming a diagnosis of LEM. Grossly, focal areas of white matter necrosis are observed throughout one or both halves of the brain (**Figure 1**). Microscopic examination of the cerebral white matter reveals brain liquefactive necrosis, congestion, and bleeding.

There is no way to test for fumonisins in animal tissues, however, tests are available to detect and measure fumonisins in the feed. It has been determined that horses receiving feed which contains more than 10ppm of fumonisin B1 are at risk for developing LEM. When submitting feed samples for analysis, it is important to keep in mind that LEM develops with prolonged exposure to contaminated corn. Therefore, the feed being analyzed should represent what the horse was eating over the last month or more, and not necessarily that which is currently being fed. Current feed may be less likely to contain the source of infection.

Treatment and prevention

Unfortunately, there is no specific medical treatment to reverse the toxic effects of fumonisins and treatment primarily consists of supportive therapy. Any feed that has the potential to be contaminated should be removed from all susceptible horses immediately. Activated charcoal and laxatives may be given to help eliminate toxins from the gastrointestinal tract. Flunixin meglumine, a non-steroidal anti-inflammatory, is administered to help control inflammation. Mannitol or DMSO is used to help treat cerebral edema associated with LEM. Intravenous fluid therapy is needed to maintain hydration. Sedation may be necessary if the patient is neurologic and at risk of injuring itself or others. Owners should be prepared for a poor prognosis. Mortality is approximately 99% once clinical signs of LEM appear. Cases of recovery have been reported, however neurologic deficits often persist.

Prevention is simple... do not feed moldy or damaged corn to horses. While less expensive, corn and cob mix, ground ear corn, and corn screenings are more likely to be contaminated with fumonisins and therefore are not suitable feed for horses. It is important to invest in good quality feedstuffs and ensure proper storage to minimize the risk of mold growth.

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EQUINE HEALTH UPDATE

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The Equine Sports Medicine Center

Purdue's Equine Sports Medicine Center is dedicated to the education and support of Indiana horsemen and veterinarians through the study of the equine athlete. The Center offers comprehensive evaluations designed to diagnose and treat the causes of poor performance, to provide performance and fitness assessments, and to improve the rehabilitation of athletic horses. Other integral goals of the Center are to pioneer leading-edge research in the area of equine sports medicine, to provide the highest level of training to future equine veterinarians, and to offer quality continuing education to Indiana veterinarians and horsemen. For more information visit our website:

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