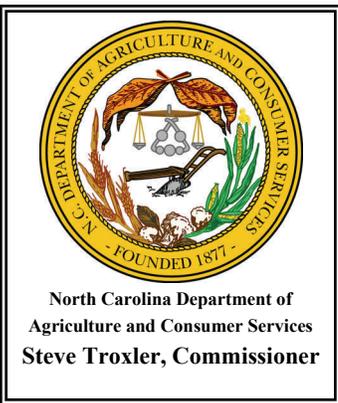


The NCVDLS REPORT



Accredited by the American Association of Veterinary Laboratory Diagnosticians

Veterinary News and Information From North Carolina's Diagnostic Laboratories



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Message from the Director

Welcome to the Spring edition of the NCVDLS Report. Spring is a season of new birth and new beginnings and we are experiencing similar events here in the NCVDLS. NC-LIMS, our previous laboratory information management system (LIMS), is being retired. The new product, USALIMS from Computer Aid Inc., is designed to provide better tracking of specimens throughout our labs, to support improved formatting of client test reports, to expedite distribution of client reports and to enhance client billing services. Simultaneously, we are changing many of our intra-laboratory processes. Some of the new LIMS highlights are: improved user friendliness of reports, clear identification of any outstanding laboratory tests, the ability to rapidly invoice following case completion, and the ability to automatically distribute reports via e-mail or fax. Please note that with the e-mail option, a link to your report will be e-mailed to the address we have on file. The routine mailing of reports is being discouraged based on legislative mandates for "going paperless" and because mailed reports may be delayed by several days due to processing through the State Mail Service Center.

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Holiday Closings...

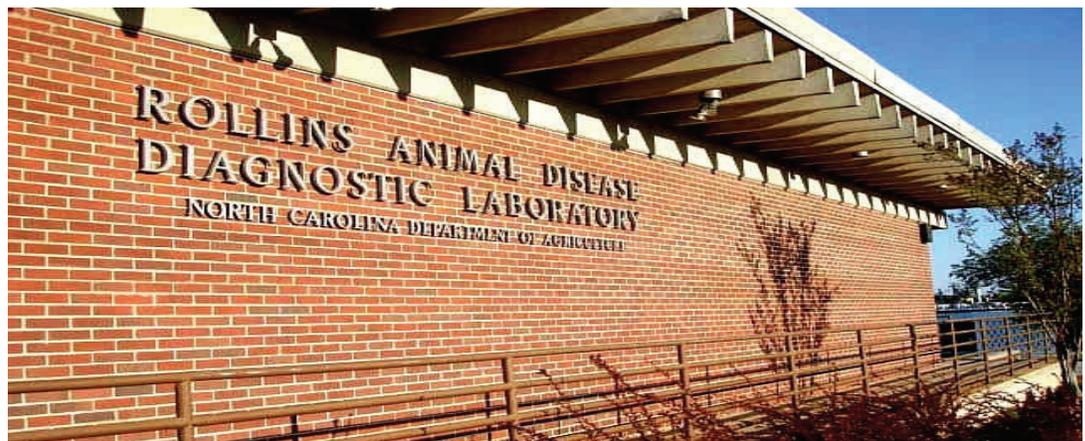
- July 4, 2011
- September 5, 2011
- November 11, 2011

Our laboratories will be closed on the above listed days.

Please e-mail NCVDL@ncmail.net with any comments and/or suggestions concerning The NCVDLS Report
Editor - Dr. David Drum

Although we had initially anticipated "going live" with USALIMS on May 2, 2011 the date has been pushed back to mid July to help ensure that all glitches have been resolved in the system. Letters were mailed to our clients in mid-April asking them to update their account information and designate their preference as to the method of distribution of laboratory reports. If you have not already done so, please contact Ms. Tamara Seago at 919-733-3986 in the Rollins Laboratory Business Office to update your account information. This will ensure that clients will receive laboratory reports in the new format.

With the implementation of our new LIMS will come a nominal fee increase for some services that currently have associated fees and fees will be applied to services that were previously free. The specifics of these changes will be communicated to you



Message from the Director, *continued*

directly. Additionally, a fee schedule and updated Userguide will be uploaded to our website shortly. (<http://www.ncagr.gov/vet/ncvdl/>) It should be noted that fee increases will not be applied to necropsy or routine histopathology services.

As always, client feedback on these changes is welcome and highly appreciated.

Respectfully,

Karen W. Post DVM, MS

Feature Article

Equine Herpes Virus (EHV-1) and Equine Herpes Virus Myeloencephalopathy (EHM)

David J. V. Drum DVM, MS

Western Animal Disease Diagnostic Laboratory

Arden, North Carolina

(This article was compiled from the information contained in a series of recent press releases concerning EHV-1)

News of an outbreak of Equine Herpes Virus (EHV-1) and Equine Herpes Virus Myeloencephalopathy (EHM) occurred following the recent National Cutting Horse Association's (NCHA) Western National Championship held in Ogden, Utah from April 29 to May 8, 2011. In the weeks that followed, cases of equine herpesvirus-1 (EHV-1) started to occur across the western United States and Canada. By the last week of May 2011, 75 cases had been reported in Arizona, California, Colorado, Idaho, New Mexico, Nevada, Oregon, Utah and Washington. In separate cases during the month of April 2011, The New Jersey Department of Agriculture quarantined a horse farm in Monmouth County, NJ after six horses contracted the neurologic form of Equine Herpes Virus-1 (EHV-1). Additionally during April 2011, The Cornell University College of Veterinary Medicine's Equine Hospital was placed under a voluntary quarantine after the confirmation of two cases of the neurologic form of equine herpesvirus (EHV-1).

Prior to 2003, the U.S. reports of neurologic EHV-1 outbreaks in the United States were sporadic, with typically none to few outbreaks identified annually. Nationally, reports of neurologic EHV-1 have increased in recent years; this may be attributable to a strain of EHV-1 with a mutation that encodes for a particularly robust replicase enzyme. The result of this mutation is that the virus can reproduce rapidly with a predilection for nervous tissue, therefore the viremia occurs earlier, it reaches a higher peak, and it lasts longer. While all strains of EHV-1 can cause respiratory disease and abortion, only a subset of EHV-1 strains can cause neurologic disease. The United States is not the only region or country that has recognized the issue regarding neurologic EHV-1 cases; other countries have also seen an increase in the number and virulence of reported neurologic EHV-1 outbreaks.

While this recent outbreak has caught the attention of the horse owners across the country, this outbreak will not be the last. It is always best to keep a cool head and learn the facts before reacting to the news of an outbreak of any disease.

Understanding how EHV-1 is transmitted between horses is key to understanding how to slow or stop disease outbreaks. Aerosol transmission is the most common route of EHV-1 transmission from horse to horse through inhalation of droplets from coughing and sneezing. EHV-1 can be transferred via aerosol transmission over short distances, but unlike

Feature Article

equine influenza--which can travel for several hundred yards through the air--a separation distance of a hundred yards or more would likely be sufficient to decrease EHV-1 virus transmission. When faced with an outbreak that spreads through horse-to-horse contact it is important to limit horse movement. Keeping horses at home and keeping them from meeting other horses they're not already in contact with can be key to keeping your horse healthy and stopping the spread of the disease. Shedding by the respiratory route typically lasts for 7-10 days, but can persist much longer. When faced with a case of EHV-1 abortion aborted fetuses, fetal membranes and/or fluids are significant sources of infection. Also infected foals are highly contagious and can transmit infection to other horses. EHV-1 virus can stay viable in the environment for several weeks or longer. Also be aware that the virus can move from horse to horse through surface contamination of equipment, feeding tools, grooming supplies, trailers, or gates. For these reasons, thorough cleaning and disinfection of all horse trailers and equipment that returned from the event where exposure to EHV-1 may have occurred is highly recommended. Cleaning and disinfection of barns, individual stalls, feeders, waterers, buckets, and other equipment should be performed at the end of the quarantine period for all confirmed EHV-1 and EHM cases. Careful consideration when handling barn supplies, tack, and other stable supplies around exposed and nonexposed horses can reduce the risk of transmission.

After infection by any route, incubation period for EHV-1 may be as short as 24 hours, but is typically 4-6 days and can be longer. EHV abortion can occur from two weeks to several months following infection with the virus.

Fever is usually the first sign of illness and can occur within a day of getting infected, but when the infection goes on to cause neurologic disease, signs of neurologic disease usually don't develop for at least seven or eight days, and sometimes up to 14 days after the horse becomes infected. The recommended quarantine period for horse that have been exposed (horse that have been exposed at a show or an event, or on farms that have had confirmed cases) should be a minimum of two weeks to account for the incubation period of cases that aren't apparent yet. However, when dealing with a farm that actually has EHV-1 infected horses, this quarantine period probably needs to be longer - at least three weeks and in some instances four weeks. As a rule, this quarantine is the period begins after the last fever is detected on the farm.

Equine herpesvirus-1 is a very common virus to find in horses. The vast majority of horses are exposed early in life and become lifelong latently infected (showing no signs of illness). The virus generally manifests as a respiratory infection and occasionally causes abortions in pregnant mares. The neurologic form of the virus, however, is an unusual outcome from the infection. The bright light is when a current outbreak ends, the horses that were exposed and infected during the outbreak are unlikely to represent a threat to other horses in the future.

As stated before, usually the first sign of illness is fever. A horse with a fever with no obvious explanation, should raise concerns. Early detection of a fever could lead to an early diagnosis that will allow treatment to commence promptly. As with most equine ailments, early treatment generally leads to a more favorable prognosis. Additional signs of EHV-1 infection include coughing and nasal discharge. Neonatal foals infected in utero are usually abnormal from birth with weakness, jaundice, respiratory distress and occasional CNS signs. Death of the infected neonatal foal commonly occurs within 3 days. In older foals, nasal discharge is the most common sign of illness. When the virus causes an abortion, it typically occurs during late pregnancy (8+ months); occasionally as early as 4 months. Usually there are no warning signs of impending abortion. For veterinarians who choose to necropsy aborted foals, a list of recommended tissues to submit is on the NCDA website. The signs usually associated with the neurologic form of EHV-1 include incoordination of the hind (and occasionally fore) limbs, urine retention, urine dribbling, and recumbency. Neurologic signs may be preceded by a fever and respiratory signs.

Owners might be wondering if a EHV-1 vaccination would be advisable during an outbreak. The answer is not just a simple yes or no. As far as the respiratory and the abortion form of the disease is concerned, most experts feel vaccination is very helpful, although it certainly doesn't prevent individual animals from succumbing to infection. At the present time, none of the vaccines currently available can claim absolute protection against the horse developing neurological disease. It is known that some of the more potent EHV-1 vaccines can have a very significant effect on reducing the shedding of the virus. A decrease in shedding EHV-1 will lead inevitably to a decrease in the number of horses on a premise being exposed to the virus. In terms of preventing infection, vaccination is still a recommended activity. But in terms of vaccinating already exposed horses, it is still an open question. When an owner is deciding whether or not to booster their horse, it would be recommended they consult with their regular veterinarian. Your veterinarian can help you sort through the pros and cons of EHV-1 vaccination and help the owner decide what vaccination plan would work best for their particular situation.

Feature Article continued

When faced with screening a potentially EHV-1 exposed or ill horse, there are a number of diagnostic tools your veterinarian can use. These tests include Virus isolation, PCR and Serology.

When trying to use serology, it is important to remember that due to the inevitable widespread EHV-1 exposure of the general horse population and routine vaccination of the general equine population, serologic testing on a single sample is uninformative. Serologic testing which demonstrates a 4-fold or greater increase in serum antibody titer between acute and convalescent samples collected 7–21 days apart provides presumptive evidence of EHV-1 infection, as long as there was no EHV vaccination during this time period. The serum neutralization (SN) test, also called the virus neutralization (VN) test is most commonly used. The acute sample should be collected as soon as horses are placed under observation, or at the earliest onset of clinical signs. In the midst of an outbreak, detection of rising virus-neutralizing antibodies in paired serum samples can be used to screen for horses that were exposed to the virus. A proportion of both affected and unaffected in-contact horses may seroconvert, providing indirect evidence that EHV-1 is the etiologic agent. It is also important to remember that neutralizing antibodies do not distinguish between EHV-1 and EHV-4 infections.

As initially stated, keeping abreast to the current information is key during periods of disease outbreak. The following are additional sources of information on EHV-1.

USDA-APHIS website

EHV information sheets, color brochures, historical information, and a review of disease mitigation strategies are available on the USDA-APHIS website:

<http://www.aphis.usda.gov/vs/nahss/equine/ehv/>

AAEP website:

General EHV resources through the American Association of Equine Practitioners:

http://www.aaep.org/ehv_resources.htm

Neurologic Disease Guidelines:

http://www.aaep.org/pdfs/control_guidelines/Neurologic%20Disease%20Guidelines.pdf

Equine Herpes Virus:

<http://www.aaep.org/images/files/EquineHerpesvirusGuidelines051711.pdf>

Biosecurity Guidelines:

http://www.aaep.org/pdfs/control_guidelines/Biosecurity_instructions%201.pdf

Biosecurity Instructions for Caretakers - English & Spanish:

http://www.aaep.org/pdfs/control_guidelines/Instructions%20to%20grooms.pdf

National Cutting Horse Association (NCHA) website:

For history of the current outbreak and additional outreach materials:

<http://www.nchacutting.com/>

ACVIM Consensus Statement on EHV-1:

<http://onlinelibrary.wiley.com/doi/10.1111/j.1939-1676.2009.0304.x/pdf>

Short Cuts

COMPANION ANIMAL

Canine

Optic nerve meningioma in a dog

An 8-year-old male neutered Schnauzer dog was presented to the local practitioner in January with obvious pain in the left eye. Palliative treatment relieved the clinical signs until April, when buphthalmos was identified. Surgical enucleation revealed a caudal orbital mass that was approximately 1cm in diameter, pressing on the globe. The mass was in the region of the optic nerve, which was not identified in the gross specimen.

Following formalin fixation of the globe and attached mass, histological examination revealed that the mass was a meningotheelial meningioma. The mass was comprised of epithelioid cells that were poorly demarcated and displayed mild atypia (Figures 1,2). The optic nerve was nearly totally effaced by the mass of cells.

There are numerous subdivisions of meningioma by type of histological appearance. The cells are mesenchymal in origin and tend to arise from the arachnoid layer of the meninges, and are usually also attached to the dura mater. Though malignant by location, meningiomas are seldom invasive. The invasive meningiomas are more commonly seen in extracranial locations in the dog and cat. Retrobulbar meningiomas, such as the case with this patient, are quite rare. They may exhibit slightly higher incidence of invasiveness than those of the cranial cavity.

Figure 1

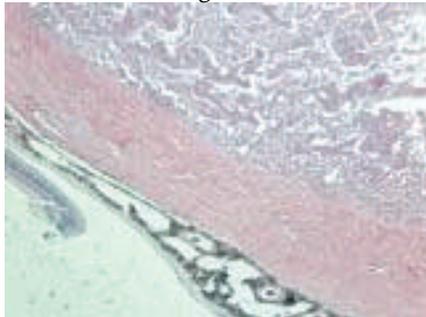
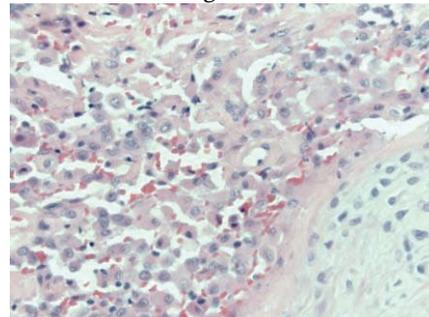


Figure 2



References:

1. Jain D et al. Intraorbital meningiomas: a pathologic review using current World Health Organization criteria. Arch Pathol Lab Med. 2010, 134:766-770.
2. Montoliu P et al. Histological and immunohistochemical study of 30 cases of canine meningioma. J Comp Pathol. 2006, 135:200-207.
3. Summers BA et al. Tumors of the central nervous system. In: Veterinary Neutopathology. Ed. Mosby-Year Book, Inc. St Louis, Missouri. 1995; 355-362.

Dr. Pete Moisan

Following a splenectomy, a 6-year-old male neutered mixed breed dog died. Almost a year and a half prior to death, pelvic hemangiopericytoma was diagnosed and radiation therapy was performed. Two months prior to death, a sarcoma was diagnosed on a lip mass and pulmonary metastases were identified. Received for necropsy was the dog and formalin-fixed mass from the spleen and pancreas. Gross exam findings included white mucous membranes, 200 ml hemoabdomen, missing spleen and pancreas, multifocal white firm nodular well-circumscribed masses ranging from 0.5 cm to 5 x 4 cm throughout the pulmonary parenchyma, a 1 cm mass similar to that described in the lungs in the left ventricular myocardium near the apex, approximately ten 0.5 to 1 cm white, glistening, relatively soft spherical masses throughout the liver, and moderate lymphadenopathy of the sternal lymph nodes. **Metastatic sarcoma most suggestive of hemangiopericytoma** was identified histologi-

COMPANION ANIMAL, CONTINUED

cally in the spleen, heart, lung, liver, and tissue labelled pancreas. The cause of death in this canine is due to severe blood loss; however the metastatic lesions are quite interesting because hemangiopericytomas infrequently metastasize. These tumors are malignant, have a high incidence of recurrence and tend to be locally aggressive and invasive. Furthermore, these neoplasms are usually found in the skin and subcutis of dogs.

Dr. Mahogany Wade

An 8 year old male English Springer Spaniel dog was presented for necropsy with a 2 to 3 day history of walking slowly and general lethargy. The dog had a recent decrease in appetite and vomitus was found in the dog's pen when the dog was found dead. The owner had noticed that the dog had orange-colored feces that were otherwise of normal consistency. On gross necropsy examination the mucous membranes, concave surface of the ear pinnae, sclera, subcutaneous fat, internal abdominal and thoracic fat, lungs, kidneys and the greater vessels of the heart were diffusely icteric. The urinary bladder contained approximately 80 ml of red urine. Mild splenomegaly was present. Orange, formed stool was in the colon. Histopathology findings included erythrophagocytosis with hemosiderosis in the spleen and liver. Bile casts with tubular epithelial hemoglobin imbibition were in the kidney and erythroid hyperplasia was present in the bone marrow. The diagnosis was: **Autoimmune Hemolytic Anemia**. Autoimmune hemolytic anemia or immune-mediated hemolytic anemia can be primary (idiopathic where no underlying cause is found) or secondary to some other agent such as bacteria (*Ehrlichia*), fungi, parasites (*Babesia* and *Dirofilaria immitis*), certain drugs and toxins. Based on the lack of other etiologies detected this dog most likely had primary (idiopathic) autoimmune hemolytic anemia. According to the veterinary literature English Springer Spaniels are predisposed to developing primary autoimmune hemolytic anemia.

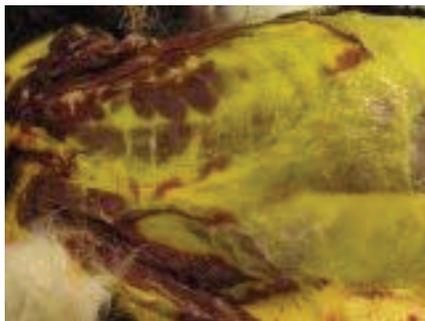


Figure 1: Diffuse icterus of subcutaneous fat.

Figure 2: Diffuse icterus of omental and mesenteric fat.

Reference:

Shaw N, Harrell K. IMHA: Diagnosis and Treating a Complex Disease. *Veterinary Medicine*. 2008; 103:660-670.

Dr. Stacy Robinson

Antifreeze intoxication is a common occurrence in North Carolina pets. Though most often diagnosed in dogs, feline intoxication is also frequently seen. Most cases appear to be from accidental intoxication, though occasional malicious poisonings are suspected as well. Animals tend to consume ethylene glycol due to the taste, which is intensely sweet, similar to propylene glycol (propylene glycol is used as a safe alternative to ethylene glycol in some radiator fluid formulations). An amount of 6.6ml of antifreeze per kilogram is the lethal dose for a dog and 1.5ml per kilogram for a cat.

Clinical signs in dogs and cats usually include staggering and lethargy, similar to "drunkenness" and, indeed, the metabolism of ethylene glycol is by the hepatic enzyme alcohol dehydrogenase. The onset of clinical signs is 12-24 hours in a cat and 36-72 hours in a dog. At that time, acute renal failure develops, and polyuria is seen. The urine is isosthenuric at that time, consistent with acute renal disease. Creatinine and BUN levels are markedly elevated. Few other causes of acute renal failure

COMPANION ANIMAL, CONTINUED

in dogs and cats will result in such rapid elevation of products of azotemia.

Gross necropsy results are usually fairly non-specific. The history of antifreeze ingestion is usually suspected by the owner and/or presenting veterinarian. In some cases, intoxication is suspected but the agent is unknown. Dehydration of the patient is the most common finding at the time of necropsy of a pet intoxicated with ethylene glycol. Microscopic findings are diagnostic for antifreeze intoxication. There are intratubular crystals within the renal tubules. These are most concentrated in the renal corticomedullary junction. The crystals are often referred to as shaped like “sheaves of wheat” and are prism-shaped to acicular and multiple within tubules. Though often difficult to visualize with the light microscope (figure 1), polarization of the sections reveals the crystals located within tubules (figures 2 and 3). Kidney scrapings from cut surfaces of the renal cortex will also reveal the polarizing crystals.

Ethylene glycol is itself non-toxic until metabolism in the liver. The product is absorbed rapidly from the intestine and excreted unchanged by the kidney. Small amounts of ethylene glycol are metabolized to glycoaldehyde in the liver. This is metabolized to glyoxylate and then to oxalate as the final product. It is the glycoaldehyde and glyoxylate that are most toxic to the kidneys, causing disruption of tubule cell membranes by exhausting membrane ATP stores. The oxalate crystals accumulate in the renal tubules and are the cause of positive polarity.

Figure 1

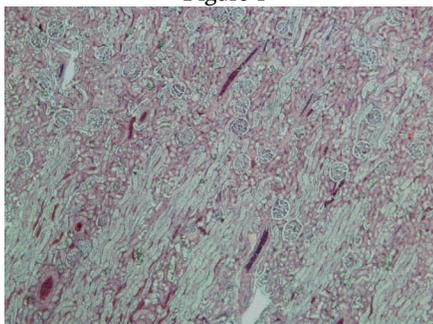


Figure 2

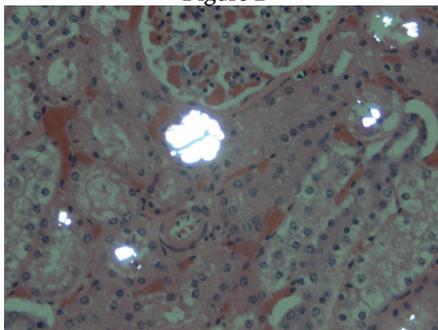


Figure 3



- References:1. Maxie MG, et al. Urinary system. In: Jubb, Kennedy, and Palmer's Pathology of domestic animals, vol. 2. Elsevier Publishing, Edinburgh, UK. 2007:470-472.
2. Thrall MA,, et al. Clinicopathologic findings in dogs and cats with ethylene glycol intoxication. J Am Vet Med Assoc. 1984; 184:37-41.

Dr. Peter Moisan

LIVESTOCK

Cattle

An approximately 120-150 day gestation Angus fetus was submitted to the laboratory. The owner reported finding one other 25lb fetus that was fully formed but not haired in the field the day before. Placenta was not available for examination. Autolysis was severe. Subcutaneous edema was noted throughout the fetus. The brain was hyperemic and semi-gelatinous. The heart was pale and the lungs were firm on palpation, nonaerated and fluid congested. Approximately 15mls of hemorrhagic fluid filled the thorax. The liver was enlarged and pale tan. Perirenal hemorrhage and edema surrounded the kidneys. The abdomen contained approximately 25mls of red watery fluid. The stomach contained approximately .5ml of red cloudy liquid. The CSF was hemorrhagic. No other abnormalities were identified on gross exam. Histopathology findings included encephalitis that was characterized as multifocal, necrotizing, mild, and acute to subacute and myocarditis that was characterized as multifocal, necrotizing, moderate to severe, acute to subacute. Immunohistochemistry for *Neospora caninum* was requested on both the brain and heart. *Neospora caninum* was detected in both fetal tissues. The protozoan parasite *Neospora caninum* has become increasingly recognized as an important cause of reproductive failure in dairy and beef cattle. Wild or domestic canids are the natural definitive host for *Neospora caninum*. Infected animals may shed large numbers of oocysts in their feces, which may then be ingested by intermediate hosts such as cattle. Sheep, goats, horses, deer, and other animals are also suitable intermediate hosts. There is no direct transmission between cows; however, the parasite can be maintained by vertical transmission of the organism from the dam to the fetus in utero. At birth, congenitally infected calves may have neurologic signs, be underweight, unable to rise, or have no clinical signs. Congenitally infected calves have a higher rate of abortion, particularly during their first pregnancy, and a high rate of vertical transmission to their offspring. *Neospora* abortion typically occurs in mid gestation and may occur throughout the year. Abortion storms may occur with multiple abortions over a period of 1-2 months. The aborted fetuses are typically autolyzed and usually do not have characteristic gross lesions. A vaccine is available.

Dr. Kim Hagans

Between June and August of 2010 nine calves out of a group of 41 died. The owner attributed the deaths of the first two calves to choke as they were found with their heads caught in a wire fence. In the month of July three calves were found dead or recumbent and died shortly afterwards. During the last week of August four calves were found in a state of recumbency with 3 of them having indications of hind limb fracture. Each was humanely euthanized and transported to the Western Animal Disease Diagnostic Laboratory.

The calves were Holstein or Holstein cross steers of 5 to 8 months of age with body weights varying from 137 to 227 kilograms. Calf # 24 a Holstein cross steer had sustained multiple rib fractures which were in various stages of healing. The proximal shaft of his right femur had a comminuted fracture and the greater trochanter of the femur was avulsed. Extensive muscle necrosis with hemorrhage and seroma formation was associated with the femoral fracture. Calf # 25 a Holstein steer was reported to have been in a "dog sitting" posture on the evening prior to being euthanized. Two separate compression fractures of lumbar vertebral bodies (L2 and L6) along with hemorrhage into the spinal canal were demonstrated at necropsy.

LIVESTOCK, CONTINUED



Calf # 26 a Holstein calf had multiple rib fractures in various stages of healing and with midshaft fractures of both the right and left femurs. Calf #31 a Holstein cross steer had multiple rib fractures in various stages of repair, a comminuted proximal shaft fracture of the left femur and a compound, comminuted fracture of the right tibia with extensive tissue necrosis. Note: We were able to cut the ribs of each steer with minimal resistance and the cortices of the long bones, e.g. femur, were thinned.



This case of **multiple pathological fractures** was attributed to a **macro mineral imbalance** when follow up history revealed that the group had been put on a lot with a diet consisting almost exclusively of corn gluten earlier that summer. Corn gluten is a good source of both protein and energy but there are some precautions to note in its utilization as a feed source. It has a high phosphorous concentration while being nearly devoid of calcium which can lead to problems related to bones and bone growth (animals will metabolize bone calcium). Urinary calculi (the formation of hard masses of mineral salts in the kidney or urinary bladder) may also be manifested. Corn gluten also tends to have an elevated sulfur concentration. A specific disorder associated with excess sulfur is polioencephalomalacia. Because of these risk factors corn gluten feed should be limited to 50% or less of total dry matter intake for beef cattle, with many nutritionist recommending even lower limits.

Dr. Richard Oliver

LIVESTOCK, CONTINUED

Equine

According to the history provided a 60-90day gestation equine fetus with placenta was found in a group of 6 mares. The owner was uncertain which mare aborted the fetus. All of the mares vaccinations were current and the herd was fed grain and alfalfa hay. The horses are dewormed regularly. No other abortions were reported. The submitted 60-90 day gestation 0.67kg male Standardbred fetus measured 19cm in length from crown to rump and was encased in placenta. The brain was liquefied. Subcutaneous edema was identified along the carcass. All the tissues were pale. No other abnormalities were identified on gross examination. Histopathologic examination revealed a placentitis that was characterized as histiocytic, mild, diffuse with mineralization. *Klebsiella pneumoniae* was identified on cultures of the fetal fluids, liver and placenta. Bacteria gain entrance to the reproductive tract and travel to the uterus, causing infection of the fetal membranes, resulting in abortion. Retention of the placenta is often a sequel to bacterial abortion as is infection of the uterus (endometritis and/or metritis).

Dr. Kim Hagans

An 11 year old hunter-jumper Thoroughbred gelding had an acute onset of limited neck movement, which was treated as a musculoskeletal injury. Later inappetance developed and 17 days later the horse was ataxic with decreased proprioception in the rear. It had lost weight, and was unwilling to flex or move his neck. Radiographs of the neck revealed degenerative arthritis of the articular facets from C4-C7. No CSF tap was performed. Treatment consisted of Triamcinolone and Amikacin intra-articular injections of the affected articular facets and oral NSAIDS. Two weeks after treatment started, neck pain had increased and neurological status had significantly deteriorated and the horse was eventually euthanized 43 days after the initial onset of neck pain. The articular facets of C5-C7 were examined at necropsy and only minimal changes of the articular cartilage were present, which were not considered sufficient enough to cause the significant clinical signs. The CSF was yellow and contained many 1-3 mm wide air bubbles around the cervical and cranial thoracic spinal cord. After formalin fixation of the brain a 2x2x1 cm focus of malacia and multiple yellow gelatinous foci within the right internal capsule (see picture 1) and a similar 2.5x2.5x0.5 cm focus in the corona radita of the left sidewere grossly evident. Histopathology revealed severe granulomatous meningoencephalitis and severe granulomatous meningitis of the spinal cord. Numerous yeast forms consistent with *Cryptococcus sp.* were found within the brain lesions (see picture 2 – GMS stain – organisms stain purple). *Cryptococcus neoformans* was isolated from the spinal cord. *Cryptococcus neoformans* is a yeast-like fungus that resides in soil, bird feces, and on some plants. The common route of infection is via inhalation. This infection is rare in horses and most common in cats. Cases of cryptococcal granulomas in the nasal cavities, cryptococcal pneumonia, abortion, and meningitis have been reported in equids but the upper and lower respiratory lesions are most common. It is unknown how the fungus gets into the nervous system but it possibly gains entry from the nasal cavity via the cribiform plate or hematogenously via pneumonia. Since this horse did not have any lung granulomas or systemic disease, the cribiform plate is more likely the route of entry into the nervous system. This disease is not considered contagious from animal to animal but there could be a common source of the fungus in the environment, possibly putting other horses at risk.



Picture 1



Picture 2

Dr. Jennifer Haugland

LIVESTOCK, CONTINUED

Caprine

A 1.5 year-old Nubian goat is found dead after a 2-3 week period of weight loss, lethargy, and anorexia. The doe had been treated with antibiotics, vitamins, and was dewormed with Fenbendazole without any improvement. At birth, the doe was diagnosed with a significant heart murmur, as a palpable thrill over chest was noticeable according to the owner. The thrill became less noticeable as the goat aged, but the goat did not thrive or grow like similar aged goats in the herd. On necropsy, the goat was in good body condition (BCS 4/9), was mildly dehydrated (5-7%), and had minimal tissue autolysis. The heart was slightly rounded, and the pulmonary trunk was distended. The right ventricular walls were thickened, and there was a significant amount of pale yellow to white fibrotic material within the walls of the proximal pulmonary trunk at the level of and including the pulmonary valve distally for 3-4cm. Approximately 75-80% of the lumen of the pulmonary trunk/artery was occluded by this material. There was a dark red focal lesion on the epicardium over the left ventricular wall which measured 0.3cm X 0.5 cm. The kidneys were pale yellow to tan in color, and were somewhat soft to touch. The left kidney had a focal lesion on the capsular surface, pale white to yellow in color, measuring 0.6cm X 0.3cm, and extended into the renal cortex on cut surface. The right kidney had a similar focal lesion in the renal cortex. Histopathology on the pulmonary artery showed chronically active severe arteritis with bacterial colonies present. The heart had regionally extensive severe fibrosis with suppuration and bacterial colonies. The kidneys both had severe cortical infarcts with hemorrhage, fibrosis, and bacterial colonies present. *Arcanobacterium pyogenes* was cultured in the heart, and the heart valve. The changes seen grossly and on histopathology are consistent with a **severe vegetative valvular endocarditis**. The chronic valvular disease in this goat greatly increased the risk for the development of endocarditis.

Dr. Brad Barlow

In February, 2 goat fetuses were submitted to the Monroe Laboratory. The history stated these were 2 of 8 aborted fetuses from a herd of Boer goats that had been purchased about 2 months earlier. Abortions occurred between 120 and 140 days of gestation and the 2 presented kids were approximately 135 days of gestational age. The fetuses were moderately autolytic. Each was presented with a small portion of placenta. An abortion panel revealed that the placenta was positive for *Chlamydomyphila abortus* (old Genus *Chlamydia*) by ELISA testing. The other tissues were negative for chlamydia.

Histopathology was also performed. Multifocal necrotizing hepatitis and splenitis was identified, though there was substantial autolysis in each tissue, precluding the identification of subtle pathological features. Necrotizing placentitis was identified in sections from the placenta (Figure 1). Vasculitis of the large arterioles of the placental stroma was also pronounced (Figure 2). Punctate organisms, consistent with intracytoplasmic elementary bodies, 0.2-0.3um in diameter were identified in some of the trophoblast cells over placental cotyledons. Neutrophils in varying stages of degeneration were associated with the cotyledonary epithelium as well.

Chlamydomyphila abortus is a common cause of abortion in sheep and goats. The agent is responsible for late term abortions and the condition is commonly referred to as enzootic abortion of ewes. Differential diagnoses in small ruminants include abortions due to *Campylobacter fetus*, *Coxiella burnetii*, and *Toxoplasma gondii*. Each agent causes outbreaks of abortions. Also of significance, each of these organisms is zoonotic, causing various diseases in humans. The gross lesions are usually seen in the placenta, with suppurative inflammation centered on cotyledons and extending into the intercotyledonary spaces. *Toxoplasma gondii* is slightly different in that the gross exudate is confined to the cotyledons and contains mineral. *Campylobacter fetus* causes targetoid pale areas in the fetal liver, as well. Other gross features are similar among aborted animals, and differentiation requires further laboratory testing.

We encourage the submission of aborted fetuses by practitioners and their clients. Of critical significance as well is submission of placenta from aborted fetuses. In the present case, the diagnosis would not have been possible without placenta. We have guidelines about materials to be submitted from these cases on our website for practitioners performing field necropsies of abortion cases.

LIVESTOCK, CONTINUED

Figure 1

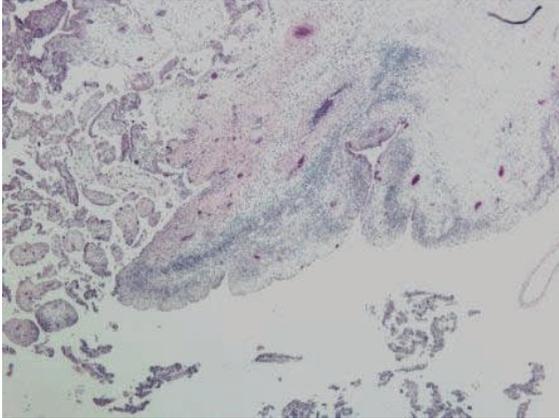
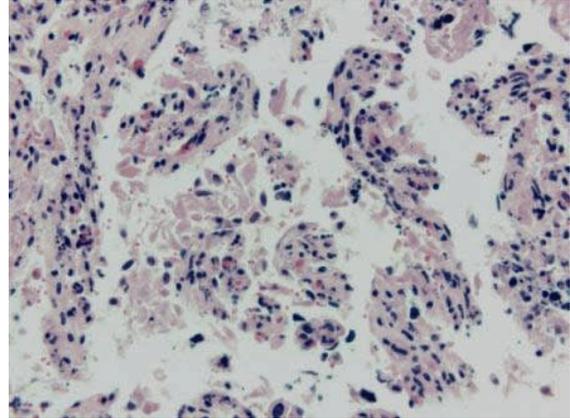


Figure 2



Reference:

1. Gutierrez J, et al. Monitoring clinical outcomes, pathological changes, and shedding of *Chlamydophila abortus* following experimental challenge of periparturient ewes utilizing the natural route of infection. *Vet Microbiol.* 2011; 147:119-126.
2. Stuen S, et al. Treatment and control of chlamydial and rickettsial infections in sheep and goats. *Vet Clin North Am Food Anim Pract.* 2011; 213-233.

Drs. Kim Hagans and Peter Moisan

Ovine

A 5 year old Suffolk ewe died suddenly from hemothorax. The thorax was filled with 4-5 liters of blood, which came from a ruptured large subpleural hematoma of the right caudal lung lobe. Under the hematoma was marked pulmonary hemorrhage, which at the center of the hemorrhagic lung was a trematode. Multiple long, linear, black tracts (flake tracts) and yellow, mineralized nodules were found in the liver. A single 3 cm wide cyst containing 2 trematodes was found in the liver (Figure 1). This trematode based on morphology was likely *Fascioloides magna*. This parasite can cause significant damage in the liver of the accidental hosts and occasionally will be found in the lung.

Figure 1



Dr. Jennifer Haugland

LIVESTOCK, CONTINUED

Camelid

A full-term 1-day-old female llama had been open mouth breathing since birth and there was no evidence of dystocia or cleft palate. The cria was unable to breathe and nurse simultaneously. The owner administered milk via a bottle, but the milk gathered at the base of the throat and was expelled from the mouth. Since the referring veterinarian was unable to pass a nasal tube, choanal atresia was suspected and the cria was euthanized. Necropsy results revealed patency of both nares via passage of a tube and the water filling test. Other findings included a flat 2 x 1 cm pedunculated edematous mass lined by mucosa located adjacent to the pharyngeal opening of the right and left auditory tube, an elongated soft palate that covered the epiglottis, and mild wrinkling and moderate thickening of the pharyngeal mucosa caudal to the pedunculated masses. Histologic examination revealed neutrophilic pharyngitis (wrinkled pharyngeal region) with edema and mucosal hyperplasia and suppurative pharyngitis (pharyngeal masses) with multifocal ulcers and granulation tissue. The cause dyspnea in this cria was due to an **elongated soft palate**. Choanal atresia was not identified. The pharyngeal masses were an interesting and incidental finding and while they did not create a complete obstruction, they likely contributed to the respiratory issues. Choanal atresia is a congenital condition that occurs when a membrane, bone or combination of both blocks the passage of air between the nasal cavity and larynx. This condition is relatively common in llamas and alpacas. The typical presentation is persistent dyspnea, as these animals are obligate nasal breathers and must be able to breathe through their nose to survive. This is problematic in newborn crias because this condition prevents them from being able to breathe and nurse simultaneously. Inability to perform this function results in choking, dyspnea, and aspiration pneumonia. The cause of choanal atresia is poorly understood and still debated in the camelid community; there is evidence that the condition is inherited. Anatomically, when trying to rule out choanal atresia in llamas/alpacas, it is important to remember the dorsal nasal meatus. If difficulty is encountered when attempting to pass a tube, remember to aim ventrally into the ventral nasal meatus which leads directly to the nasopharynx. If the tube is unable to be passed into the ventral meatus, then choanal atresia should be suspected. This diagnosis can be confirmed by filling the nares with water (water filling test); pooling of the water in the nasal cavity further supports the diagnosis.

Dr. Mahogany Wade

WILDLIFE

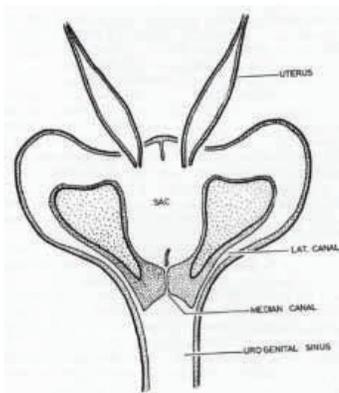


Figure 1

A torsion of the left lateral vagina after a 9 month history of chronic vaginal discharge was diagnosed in a 2 year old Opossum. Chronic pyometra and vaginitis were the source of the 400 ml of fluid in the left lateral vagina, which eventually twisted. Rupture of the vagina and subsequent peritonitis was the cause of death. The opossum is a marsupial and the marsupials have a unique female reproduction tract. There are paired lateral vaginal canals and two uteri (see Figure 1). In addition, this opossum had myeloid leukemia which may explain why the uterine/vaginal infection did not resolve with treatment.

Dr. Jennifer Haugland

WILDLIFE—CONT.

A broad-winged hawk (*Buteo platypterus*) approximately 1 year of age had been at a wildlife rehabilitation facility for about 6 months. A tibial breakage occurred in captivity and the bird was recovering from surgery when she stopped eating and began breathing heavily a day or 2 before dying. Necropsy findings included clouding of the air sacs and approximately 6 or 7 solid to cheesy, dark reddish black masses ranging in size from .5 to 1 centimeter scattered in the thoracic and abdominal air sacs. The lungs were dark red and slightly firm. A small amount of pale tan, thick exudate was noted in the trachea near the bifurcation. Histopathology findings included granulomatous tracheitis and granulomatous airsacculitis, with numerous nematode type ova noted in the air sac sections. Direct microscopic examination of a wet prep sample of air sac material identified numerous ova resembling **respiratory nematodes, likely of the genus *Cyathostoma***.

Respiratory nematodes in broad winged hawks and other raptors have been reported. The adult worms are typically found in the trachea, lungs or air sacs. Infections of this parasite may be direct or indirect by the ingestion of paratenic hosts such as insects or earthworms. Adult worms were not identified in this hawk, likely having been eliminated by recent treatment with Ivermectin.



Reference: Martin Lavoie, et al. Respiratory Nematodiasis in Raptors in Quebec. *Journal of Wildlife Diseases*, 35(2), 1999, pp. 375–380

Dr. Reginald Ridenhour

DEPARTMENTAL NEWS

ROLLINS LABORATORY

Departmental News: Rollins Bacteriology Section welcomes a new medical technologist Laura Tweed, a registered veterinary technologist and an ASCP-certified Medical Technologist.

Rollins Serology section welcomes the addition of Amber Morgan also as a Medical Technologist.

Dr. Mahogany Wade-Caesar and her husband Maurice are the proud first-time parents of a new son, Truth Maurice born on May 21.

ARDEN LABORATORY

On April 7, 2011 Paul Rector was recognized by NCVDLs Director Dr. Karen Post for his 35 years of outstanding service as a Medical Laboratory Assistant (MLA II) at the Western Animal Disease Diagnostic Laboratory (WADDL). Mr. Rector began employment at WADDL on November 5, 1975. On April 18, 2011 a congratulatory lunch was held at the Western Animal Disease Diagnostic Laboratory to acknowledge Mr. Rector's long years of service for the department.



CE ATTENDANCE

On April 1, 2011, Drs. Haugland and Moisan attended the Eastern Fish Health Workshop in Charleston, South Carolina. The topic of the day-long seminar was Histology of fish diseases.

On February 19, 2011 Dr. Richard Oliver lectured on Zoonotic Diseases at the 8th annual Wildlife Rehabilitation Workshop at the WNC Nature Center in Asheville.

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