May 2010

Volume 5 Issue 2

The NCVDLS REPORT

Accredited by the American Association of Veterinary Laboratory Diagnosticians

Veterinary News and Information From North Carolina's Diagnostic Laboratories

North Carolina Department of Agriculture and Consumer Services
Steve Troxler, Commissioner

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Holiday Closings...

July 5, 2010

September 6, 2010

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. Tim McComb

Message from the Director

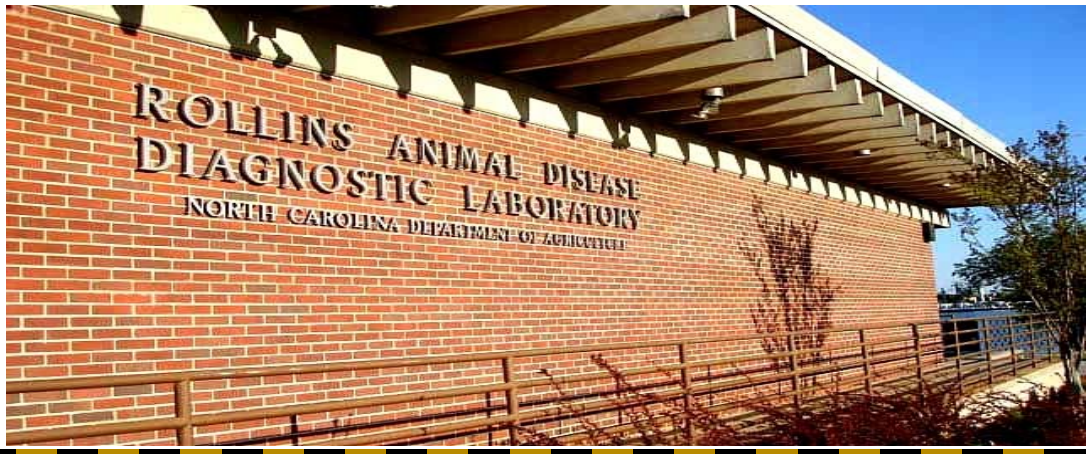
The North Carolina Veterinary Diagnostic Laboratory (NCVDLS) will be participating in the National Animal Health Laboratory Network's (NAHLN) 12 State negative cohort study for foot and mouth disease (FMD). The primary objective of this study, which began on May 1, is to validate molecular diagnostic assays (rRT PCR tests) for FMD. The study will also help the NAHLN to better assess and improve laboratory procedures and processes for sample selection, testing, and result communication; identify information technology needs; assess and implement current notification/communication protocols for foreign animal diseases; and recommend improvements for the overall laboratory surveillance component of future surveillance systems involving diseases in NAHLN Laboratories. NCVDLS' role in the study will be to test samples from swine and small ruminants.
The Rollins Laboratory will soon be renovating its necropsy suite. This will include resurfacing and painting floors and installation of stainless steel cabinetry. There is the potential that the lab will not be able to accept animals for necropsy for one or two days during this time. We are in the process of scheduling contractors and hope to coordinate the work over a long holiday weekend to lessen the impact on necropsy submissions. Client notification will begin after the contractor arrangements become finalized.

The short legislative session convened on May 13 in Raleigh with the General Assembly focusing heavily on the State budget. The Veterinary Division has given up thousands of dollars in operating expenses over the past several years. Any further cuts will severely impact laboratory operations and our ability to provide important diagnostic services to clients. The Department has made several recommendations to the General Assembly in lieu of further budget cuts and these include closure of a satellite laboratory and the elimination of several vacant positions.

As part of our State Government's Green (fiscally and environmentally sustainable) policy, we have been mandated to go paperless and reduce the amount of mail that we generate. This would include many of our laboratory reports. We are encouraging clients to obtain lab results online, if at all possible.

In closing, I am sad to announce that a friend and colleague, Dr. Kimberly Townsend, a veterinary diagnostician at our Northwestern Laboratory, passed away in March after a courageous battle with cancer. She leaves behind two young daughters. Not only was Dr. Townsend an excellent veterinarian but she was genuinely one of the nicest people I have ever met. A flowering dogwood tree was planted on the grounds of the Elkin Laboratory to memorialize her life.

Karen W. Post DVM, MS



Client Corner

NEW TEST SERVICE AVAILABLE Beginning May 26

Effective May 26, 2010, the NCVDLS will begin fee-based testing for Bovine Viral Diarrhea Virus screening by IDEXX BVDV Antigen Capture ELISA. This test is particularly well suited for screening of bulls prior to semen collection at AI centers, show calves, replacement heifers for cow calf herds, stocker calves of unknown BVDV status, and newborn heifer calves in cow calf herds growing their own replacement heifers. Due to the presence of maternal antibodies, serum from calves 3 months of age or older is needed for testing.

The test will be performed on Wednesday of each week. Results will be reported on either the day of the test or the following day. Results can be emailed directly to clients.

Suitable samples for analysis are serum and fresh ear notch. A fresh ear notch should be placed in a clean tube with closure (such as a 5 ml red top serum collection tube) and refrigerated until and during shipment to the laboratory.

The fee for this service is $5 per serum or ear notch for NC customers and $7 for customers outside of NC. Please contact either Gene Erickson or Kim Bennett at 919-733-3986 to coordinate test requests or if you need further information.

Feature Article

There were several notable cases involving bacterial infection with Clostridial organisms. Presented here are a collection of these cases.

Multifactorial Bacterial Enteritis in a Foal

Background: In 2006 all the foals (7) that were born on the farm in this case developed diarrhea within the first 3 to 5 days of life. All but one recovered with treatment which consisted of Probiocin and sulfamethoxazole/trimethoprim (SMZ/TMP). In 2007 and 2008 numerous foals at this farm developed diarrhea at 3 to 5 days of age. Most died in spite of antimicrobial therapy which usually consisted of ceftiofur (Naxcel) and metronidazole. (From 2007 and 2008 two necropsies were performed on foals at WADDL. One was diagnosed as having bacterial enteritis with sepsis (*E. coli*) and one was diagnosed with "neonatal diarrhea and dehydration" of undetermined etiology with the degree of post mortem change cited as a factor impeding optimal evaluation. During this same period 4 fecal samples from diarrheic foals were processed. All samples (from necropsy as well as fecal samples) were negative for *Clostridium difficile* toxins, *Salmonella* spp., and rotavirus. One fecal sample yielded a 1+ growth of *Clostridium perfrnigens* which was not typed. All the animals had been treated with antimicrobials prior to sampling. In 2009 the mares were moved to a different premise for foaling and there were no foals that became ill. The owner had planned to implement this same management change for the 2010 foaling but the birth of the foal in this case report occurred prior to the mares being moved.

History: In late March of 2010 the owner noticed blood in the feces of a 3 day old foal. A sample was collected at that time and submitted for bacterial culture. In less than 24 hours the foal was dead.

Necropsy: A necropsy examination was performed within a few hours of death on the carcass of a 45 kg., 3 day old Quarter Horse colt showing mild post mortem change. Sunken eyes and inelastic skin were indicative of dehydration. A one meter section of the ileo-jejunum was dark reddish-black to purple. The color change was abrupt and well demarcated. (See photo.) The wall of that gut segment was thickened and its luminal content consisted of a dark reddish-black sanguineous fluid having no particular odor. The mesenteric vessels of the infracted bowel segment were distended. Proximally the jejunum was moderately dilated with a red-brown liquid. Distally the cecum and ventral colon were filled with a dark reddish black fluid having a slight necrotic odor. There was no grossly visible reactivity of the cecocolic serosal or mucosal surfaces.

Figure 1: Note the well demarcated color change.

Histopathology: Intestine; enteritis, necrohemorrhagic, severe, regionally extensive, acute, with bacilli.

Microbiology: Fecal sample; 3+ growth of *Clostridium perfringens* which based upon molecular genotyping was Type A enterotoxigenic (possessed the gene for enterotoxin production). Aerobic cultures yielded a 3+ growth of *Actinobacillus equuli* subsp. *haemolyticus* and non-

hemolytic *E. coli*. The *E. coli* isolate was negative for all virulence factors (Stx1, Stx2, eae, Sta, Stb, LT, CNFI, and CNFII) when genotyped by PCR. No salmonellae were isolated. **Electron microscopy:** Feces were negative for virus particles upon direct examination by negative stain. Small sample volume precluded testing for *Clostridium difficile* toxins. **Necropsy samples:** 2+ growth of *Clostridium perfringens* was isolated from the small intestine. Genotyping revealed that it was Type A enterotoxigenic and also produced Beta 2 Toxin. Aerobic cultures of small and large intestine yielded 2+ growth of *Actinobacillus equuli* subsp. *haemolyticus*. No *Salmonella* or *Yersinia* species were isolated. *Clostridium difficile* toxins A/B were detected by immunoassay in small and large intestinal contents. *Lawsonia* PCR tests performed on feces and intestinal contents were negative.

Discussion: *Clostridium perfringens* enterotoxin has been detected in the feces of approximately 25% of foals with clinical diarrhea, according to a recent study. Additionally, Type A isolates producing the beta-2 toxin have also been implicated in cases of equine enterocolitis. *Clostridium difficile* is an important as a cause of both diarrhea and fatal necrotizing enterocolitis in foals. Affected foals are usually neonates and typically display severe hemorrhagic necrotizing enterocolitis with colic, weakness, profuse watery diarrhea, and dehydration. These organisms, coupled with the isolation of *Actinobacillus equuli* subsp. *haemolyticus* could account for the clinical disease in this foal. (Disease due to *Actinobacillus equuli* in foals may manifest as diarrhea, followed by meningitis, pneumonia, purulent nephritis, or septic polyarthritis.)

Healthy foals that get good colostrum can still get Clostridial infections in the first week of life. Typical clinical signs are severe bloody diarrhea with sepsis and shock. The organism is picked up from the environment and may manifest as an outbreak if the organism is abundant and toxigenic. Some success has been reported with prophylactic metronidazole treatment of foals immediately following birth.

Follow up: All pregnant mares from the farm were moved to another premise some 20 miles distant within 2 days of the death of the foal in this report and, to date, all subsequent foalings and postnatal foal health have been unremarkable.

Dr. Oliver

C. perfringens enteritis in a Quarter horse

An **American quarter horse filly** died at less than 10 hours of age. The foaling had not been observed but the filly was found weak and was not nursing. Necropsy examination revealed the small intestine was thin walled, dilated with gas and yellow liquid contents and there were multiple ecchymotic hemorrhages on the intestinal walls. Histopathology revealed a minimal to mild neutrophilic enteritis with occasional bacilli colonies. *Clostridium perfringens* was isolated from the small intestine and PCR genotyping revealed it as Type A and beta -2 toxigenic. *Clostridium perfringens* is a common cause of bloody diarrhea in neonatal foals. It is rapidly progressive disease often with a fatal outcome. *C. perfringens* is often present in the feces of neonatal foals and may be the first bacterium to isolate the intestinal tract. The presence of this bacterium does not always correlate with disease and it is still not known what factors need to occur for enterocolitis to develop. One study found that foals born on dirt, sand, or gravel floor or in a stall were more likely to develop the disease. *Clostridium perfringens* is commonly isolated from the environment and outbreaks on a farm can occur presumably when foaling areas are heavily contaminated with this bacterium.

Dr. Haugland

Blackleg in Cattle

A 5 month old **Limousine steer** was found dead. The day before the calf had appeared normal. Another 5 month calf was found dead one week earlier. There was a 15x15 cm focus in the quadriceps muscle of the right leg that was black and dry and had the odor of rancid butter. There was also a 1 cm thick layer of fibrin and edema between the pericardial sac and epicardium. Multifocal to coalescing black foci were within the myocardium. Severe, neutrophilic myocarditis and epicarditis with emphysema and large bacilli and neutrophilic myositis with emphysema and large bacilli were present. Clostridial fluorescent antibody (FA) on the skeletal muscle was positive for *Clostridium chauvoei* (blackleg disease).

Dr. Haugland

Clostridium difficile Associated Colitis in a 16 Year Old Saddlebred Gelding

A 16 year old 454 kg **American Saddlebred** gelding was transported to an equine medical and surgical referral hospital with a history of colic of 48 hours duration with progressing intensity despite medical treatment. Physical findings at presentation included an elevated heart rate, a "toxic line" noted on the oral mucous membranes, tight bands of colon palpable per rectum, rectal temperature of 102.2, and a packed cell volume of 50%. After the intravenous administration of 20 liters of fluid the packed cell volume was 46%, the heart rate was 64

Feature Article continued

BPM, and the horse was "becoming more toxic". The patient was humanely euthanized due to the rapid deterioration of condition and lack of favorable response to therapeutic intervention.

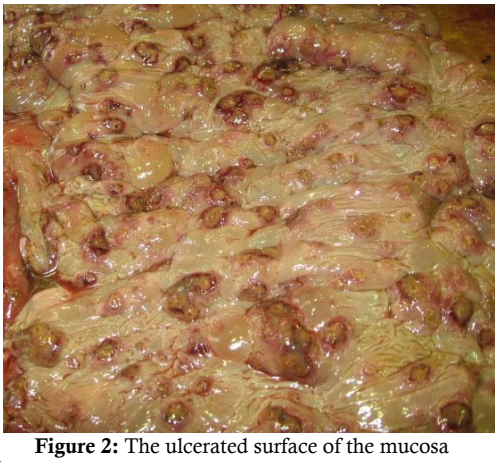


Figure 2: The ulcerated surface of the mucosa

Necropsy: Significant gross post mortem tissue alterations were confined to the cecum and colon where thousands of round, raised, focal, reddish-purple "button" ulcerations with pale, somewhat punctuate centers were distributed over the mucosal surface from the cecum to the level of the transverse colon. (See photos.) The individual ulcers ranged from 3 mm to 1 cm in diameter with many coalescing and covered with a loosely adherent gray-brown plaque. The cecocolic mucosa and submucosa were markedly edematous. Cecocolic content consisted of a dark green liquid of copious volume and having no distinct odor.

Histopathology: Colon and cecum; colitis, diffuse, severe, acute necrosuppurative, with fibrinonecrotic pseudomembrane.

Bacteriology: *Clostridium difficile* toxin assay – Large Intestine: Positive for *Clostridium difficile* toxins A and B by enzyme immunoassay (EIA).

The number one identified risk factor in the acquisition of C. difficile associated disease (CDAD) is alteration of bowel flora following antimicrobial therapy. It is not known whether or not this horse received antimicrobial therapy prior to admittance to the referral practice.

Dr. Oliver

Short Cuts

Companion Animal

Canine

A 3-year-old male neutered **Yorkshire Terrier** had a 5-month history of intermittent diarrhea and weight loss. Fecal examination, Giardia ELISA and abdominal radiographs and ultrasonography were unremarkable. The pet was intermittently treated with antibiotics, an absorbent anti-diarrheal demulcent, probiotics, steroids and a bland diet; clinical signs resolved initially but tended to recur after a few days of treatment. Bloodwork revealed neutrophilia, lymphopenia, and hyperlipasemia. The dog eventually developed bloody diarrhea and was euthanized. The canine was current on vaccinations and heartworm preventive. Gross abnormalities identified included thin body condition (BCS 2/5), moderate subcutaneous edema of the ventral cervical, thoracic and abdominal regions, generalized muscle atrophy, and a diffusely thickened and corrugated colonic and rectal mucosa with mild random mucosal hemorrhage and ulceration. Histopathologic examination revealed marked multifocal to coalescing granulomatous transmural colitis and serositis with necrosis and histiocytic intracytoplasmic yeast consistent with *Histoplasma capsulatum*. Similar inflammatory cells and intracytoplasmic yeast were identified in the lymph node, spleen, liver and lung. The chronic intermittent diarrhea resulted from **colonic histoplasmosis.** The fungi were easily seen on hemotoxylin and eosin staining; however in cases in which the fungi are less apparent, PAS or GMS stains should be performed for identification. *H. capsulatum* is predominantly seen in the Mississippi, Missouri, and Ohio River areas, lives in the soil, and thrives in moist and humid environments. It is present in particularly high concentrations where birds and bats congregate due to the high nitrogen concentration in their feces. The organism is not contagious from animal to animal and there is no sex predilection. It affects animals of all ages, however animals under the age of four are most commonly affected with severe disease. Exposure of young animals with immature immune systems and exposure to large amounts of the fungus are the most common predisposing factors. There is no effective vaccine against *Histoplasma*; disease is best prevented by avoiding contaminated soil. As with other systemic mycoses, humans are susceptible to the disease if exposed to the mycelial stage.

Cote, Etienne. Clinical Veterinary Advisor. St. Louis, MO: Mosby, 2007

Lieb, Michael and William Monroe. Practical Small Animal Internal Medicine. Philadelphia, PA: Saunders, 1997.

Dr. Wade-Caesar

Companion Animal, continued

A litter of **6 week old puppies** and their dam were rescued from an animal shelter by the owners. Four of the puppies died over a 9 day period, after showing signs of loose stool the previous week. The third puppy that died had a cough, went into respiratory distress and died. The fourth puppy that died was clinically normal in the morning, died 2 hours later and was then submitted to the Rollins Laboratory for necropsy. On necropsy examination there was a focally extensive white region in the epicardium of both the left and right ventricles at the apex of the heart. The lungs were diffusely dark red, meaty to rubbery in consistency with moderate to severe pulmonary edema and occasional petechiae on the pleural surface**.** Histopathology findings included severe, multifocal and coalescent, lymphohistiocytic, subacute to chronic myocarditis. Acute, moderate to severe alveolar hemorrhage and histiocytosis were present in the lung tissue. Parvovirus was detected in the heart by immunohistochemistry. The puppy was diagnosed with **Parvovirus myocarditis and left heart failure**. Parvovirus can cause myocarditis when puppies are infected in utero or if they are infected when they are younger than 8 weeks of age. Although we most often see parvoviral enteritis we also see cases of myocarditis caused by parvovirus as in this case. Regarding suspect cases of parvovirus enteritis, it is important for practitioners to remember that false negative ELISA test results can be obtained as the period of fecal shedding is brief. Testing in the early stages prior to fecal shedding or in the late stages of disease can also result in a false negative result. Also vaccine failure does occur, most often due to interference of maternal antibody with the vaccine. Other causes of vaccine failure include improper care and handling of the vaccine and failure to complete or delayed completion of the series of vaccines needed for proper immunization. Vaccine failure is most commonly encountered when owners and breeders vaccinate their own animals.

McCaw, DL and Hoskins, JD Canine Viral Enteritis. In: Infectious Diseases of the Dog and Cat. 3rd edition, Greene, CE, pp. 63-70. Elsevier Inc., St. Louis, MO 2006.

Greene, CE, Schultz, RD Immunoprophylaxis. In: Infectious Diseases of the Dog and Cat. 3rd edition, Greene, CE, pp.1088-1090. Elsevier Inc., St. Louis, MO, 2006.

Dr. Robinson

A 10 year old 8.6 kg. neutered male **Shih-Tzu** canine was presented to the attending veterinarian with primary complaints of weight loss, lethargy, and anorexia. On physical examination, the dog was febrile (104.3 degrees). Clinical pathology abnormalities included marginally elevated WBC and serum ALKP, globulin, and total protein. An initial improvement in the patient's condition after beginning antimicrobial therapy was followed within 48 hours by an abrupt return of clinical signs and pyrexia (T = 104.2). The ventral diaphragm was not clearly distinguishable in lateral view thoraco-abdominal radiographs taken at that time. The patient was humanely euthanized due to poor prognosis and deteriorating condition. On Necropsy examination, the pericardium contained 18 to 20 ml of an opaque reddish brown fluid of medium viscosity. Myriad whitish-beige nodules were distributed on and throughout the heart. Most of the nodules were 1 to 3 mm in diameter with areas of coalescence particularly in the right and left atria as well as along the coronary vascular structures of the ventricular epicardium (Fig. 1). Thoracic and hepatopancreatic lymph nodes were red and turgid. Histopathology revealed severe, diffuse, chronic active **pyogranulomatous myocarditis**. Special stains of the heart were negative for parasites and fungi. Aerobic bacterial cultures of pericardial fluid, thoracic lymph node, and liver isolated **Beta-hemolytic *Escherichia coli***. The heart lesions are likely the result of hematogenous seeding of *E. coli* bacteria from another inflammatory site. The primary site of infection was not evident.

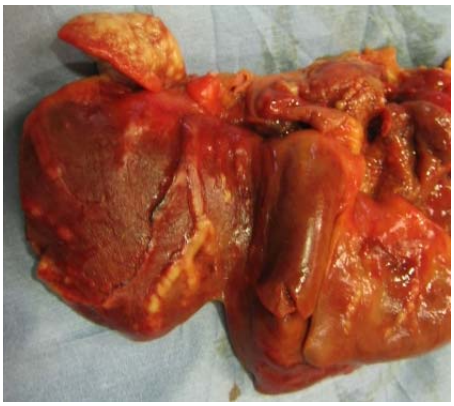


Figure 1: White nodules coalesce along the coronary arteries.

Dr. Oliver

A stray 2 month old **mixed breed female dog** was found April 10 on a farm in the southeastern area of Warren County, NC. She was in very poor body condition and was covered with ticks and fleas. A fecal floatation on 4/10 was positive for hookworm and roundworms. The puppy was dewormed with Drontal. The puppy was rechecked on 4/26 and coccidian oocysts were found in the feces. The puppy was treated with Drontal and Albon. Another fecal floatation was done on 5/14 and trematode ova were discovered. A fecal sample was collected on 5/19 and Dr. Flowers from NCSU-CVM confirmed the ova were ***Paragonimus kellicotti***. All fecal exams were performed by Antech Diagnostics and the technique was centrifugation using zinc sulfate. The puppy was eating well and acting healthy. A physical exam on 5/19 was within normal limits. During the evening of 5/19 the puppy ate well but then became depressed and would not play fetch. She would run only a few steps, then lie down and cough. By 7:30 pm the puppy had very labored breathing and was taken to an emergency hospital. At presentation the puppy was open mouth breathing and was cyanotic. The puppy died a short time later.

Figure 2: *Paragonimus kellicotti* ova

Companion Animal, continued

Necropsy examination revealed a pneumothorax. The lungs were mottled pink and dark pink and were partially collapsed. There were multiple 1-3 mm roughly circular gray foci on the pleura of the left lung. There was a 1.5 cm wide and 2 cm deep tan to brown soft to firm multilobulated cyst in the left caudal lobe. The cyst walls were gray and approximately 1-2 mm thick. The cystic cavities were clear and air-filled and were divided into several chambers. The right caudal lobe and the accessory lobe each contained a similar single cyst. Four 5-6 mm long tan fleshy adult flukes were collected from the cysts.

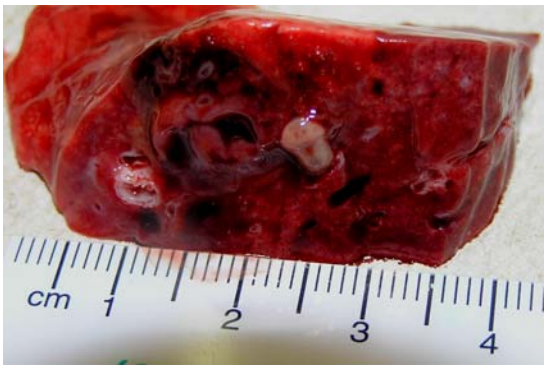


Figure 3: Cut section of lung with exposed cyst

Pneumothorax is a rare complication of lung fluke infections. Pneumothorax is believed to be the result from the rupture of subpleural lesions through the visceral pleura and into the thoracic cavity. The cystic lesions have a direct communication with a bronchial structure and the resultant bronchopleural communication could cause pneumothorax. *Paragonimus kellicotti* is a trematode that infects the lungs in a variety of carnivores most frequently in the Southeastern and Midwestern regions of the US. *Paragonimus* ova are shed in the feces of the final host (carnivores). Miracidia develop within the eggs in 2-3 weeks. Miracidia leave the eggs and infect a small aquatic snail, the first intermediate host. Within the snail, the miracidia multiply and produce cercaria. After 11-12 weeks, the cercaria leave the snail and penetrate the second intermediate host, a crayfish, crab, or shrimp. Cercaria reach the heart of the intermediate host and develop into metacercaria, the infective stage of *Paragonimus kellicotti*. Infection of the final host occurs with ingestion of an infected crayfish, crab or shrimp. The metacercaria excyst in the stomach or duodenum and migrate in the peritoneal cavity to the diaphragm. They penetrate the diaphragm and reach the lungs of the final host where they form cystic cavities in the parenchyma. Within the cyst the fluke matures to an adult and produces eggs. These eggs leave the cyst through a bronchial communication. They are coughed up and swallowed and appear in the feces of the final host 30-49 days after infection. Clinical signs are not always present but when they are chronic cough is most common. Sudden respiratory distress is also reported. The presence of *Paragonimus kellicotti* is detected by the presence of ova in the feces or tracheal wash. The ova are characterized by a single operculum and are large in size (82-106 um x 50-67 um). The characteristic findings on thoracic radiograph of dogs with paragonimiasis are multiloboculated, thin-walled cystic structures. The lesions are usually solitary, but occasionally a cluster of the thin-walled structures may be found in a single lung lobe.

Figure 4: Cyst recovered from the lung

Dr. Haugland

A 13 year old female **Rhodesian Ridgeback** dog was presented to the laboratory for post mortem examination. The provided history stated the dog had a history of being "off" for a couple weeks with coughing. The dog later developed panting, was uncomfortable and seemed restless. The following morning the dog presented to the referring veterinarian with a fever, and decreased lung sounds. On post mortem examination the dog was in good body condition (6/9) and well hydrated. The thoracic cavity was filled with at least 100 ml of slightly cloudy pink tinged fluid. The specific gravity of fluid was measured at 1.017 with a total protein less than 3 g/100 mL.. A 5 cm by 7 cm by 8 cm round, firm mass was present in the atrial septum. The mass was variable in color on cross section (white to bright red to dark red to brown). There were small areas of cavitation within the mass. There was a multifocal distribution of firm 2-4 mm wide purple colored masses within the lung tissue. Variable sized dark purple lesions were seen on cross sectioning of the liver. The pancreas was off white in color with distinct areas of hemorrhage and estimated to be four times its normal size. Both adrenal glands were enlarged (approximately twice the normal size). Multiple lymph nodes associated with the main stem bronchi and the liver were dark brown in color. The heart based tumor was identified as a **thyroid carcinoma** with metastasis to the lungs. Additionally there was a nodular hyperplasia of the adrenal glands and a multifocal hematoma in the liver. Ectopic thyroid tumors comprise around 12% of all heart base tumors. Approximately 80% of thyroid carcinomas metastasize to organs such as lungs, lymph nodes and liver.

Dr. Drum

Feline

An 8 year old female domestic medium haired cat was presented for post mortem examination. The provided history stated three months prior the cat developed a **change in gait** which was suspected to be due to lumbar spinal pain. The cat was treated with Metacam and the signs resolved. Five days prior to presentation for necropsy, the cat had jumped from a kitchen counter and immediately started to have signs of pain again. On post mortem examination the cat had a body condition score of 7/9 and was of normal hydration. There was

Companion Animal, continued

over 120 ml of blood in the abdominal cavity. The heart weighed 13.9 grams. There was approximately 2 ml of blood tinged effusion in the pericardial sac. The lungs were spongy on palpation and almost white in color. The liver was a uniform green to brown color. The left kidney was only 25 % the size of the right kidney. Both kidneys were pale tan in color. There was massive engorgement of the spleen (20 cm in length and weighed 92.49 grams). The spleen was meaty on palpation and purple to pink in color with two dark purple colored foci (2 cm in width) on the tail of the spleen. The abdominal hemorrhage was originating from a tear in the spleen over one of the splenic foci. Cytology of impression smears from the spleen revealed a large nucleated cell population and moderate blood contamination. The majority (85%) of the cells were composed of Mast cells (metachromatic granules and moderate amounts of cytoplasm and round central nuclei). Histopathology confirmed the diagnosis of a **Splenic Mast cell tumor** with metastasis to the liver. Also a severe, multifocal interstitial nephritis with fibrosis was responsible for the renal asymmetry.

Dr. Drum

An outdoor only 1 to 2 year old cat was found laying in a box one morning by the owner. Approximately 12 hours later the cat was found dead. This was the fourth cat to die on the premises in the last 3 weeks. The other cats were just found dead. The cat was presented for necropsy at the Rollins Laboratory. On **gross examination** the mucous membranes were pale pink. Approximately 1 to 2ml of serous fluid was in the thoracic cavity. The lungs were pink to slightly icteric and mildly rubbery in consistency. Moderate to large amounts of fluid exuded from the airways on cut surface. Mild splenomegaly was present as the spleen was congested and slightly meaty in consistency. The cortex of the kidneys was yellow on cut surface and multifocal petechiae were in the renal cortex**. Histological examination** revealed the presence of intravascular intrahistiocytic protozoal schizonts in the sections of brain, heart, lung, liver, pancreas, kidney, spleen, and bone marrow. The cat was diagnosed with **Cytauxzoonosis** which is caused by the protozoal organism known as *Cytauxzoon felis*. The natural reservoir host in North America is the Bobcat. Ticks are thought to be the natural vector that transmits the protozoa to the domestic cats.

Greene, CE, Meinkoth, J., Kocan, AA Cytauxzoonosis. In: Infectious Diseases of the Dog and Cat. 3rd edition,Greene, CE, pp.716-722. Elsevier Inc., St. Louis, MO, 2006.

Dr. Robinson

An 8 to 9 year old domestic shorthair male neutered indoor/outdoor cat was presented to the Rollins Laboratory for necropsy. The cat had a one day history of a decreased appetite which progressed to complete anorexia the following day. On the third day the cat had a seizure and died. **Gross necropsy examination findings** included diffusely, reddened lungs with moderate amounts of red tinted fluid oozing from the airways on cut surface. One approximately 1.0 cm diameter nodule was in the right caudal lung lobe. Occasional pinpoint white to yellow foci were in the capsular surface and cut surface of the liver. Numerous multifocal approximately 2 to 3 mm diameter white to light purple nodules were in the cut surface and capsular surface of the spleen. An approximately 0.5cm x 3.0cm x 4.0cm strip of the distal ileum was thickened with the corresponding mucosa characterized as irregular, roughened, and mottled yellow and red. Two to three more round to oval similar thickened patches were scattered in the mucosa of the jejunum. The mesenteric lymph nodes were moderately enlarged. **Histopathology findings** included: 1.Regionally extensive, severe, acute, necrosuppurative enteritis. 2. Regionally extensive and coalescent, severe, acute, necrosuppurative splenitis and lymphadenitis. 3. Multifocal and random, moderate, acute, necrotizing hepatitis. 4. Multifocal, moderate to severe, acute, necrotizing bronchopneumonia. Special **bacterial cultures** were performed on the liver and spleen. Initial screening tests of the bacterial isolate were consistent with *Francisella tularensis*. DFA, slide agglutination and PCR tests were performed by the NC Laboratory of Public Health and the bacterial isolate was confirmed to be *Francisella tularensis* by all three confirmatory tests. The cat was diagnosed with **Tularemia. Tularemia** is caused by the bacterium *Francisella tularensis*. This bacterium can infect many species of wild and domestic mammals as well as fish, amphibians, reptiles and birds. Rodents and rabbits are important reservoir hosts in nature. The bacterium can be spread through the bite of ticks carrying the bacterium. Other arthropods such as mosquitoes may also be important in the transmission of the disease. Predation by domestic animals is an important risk factor for those animals developing this disease. The cat in this

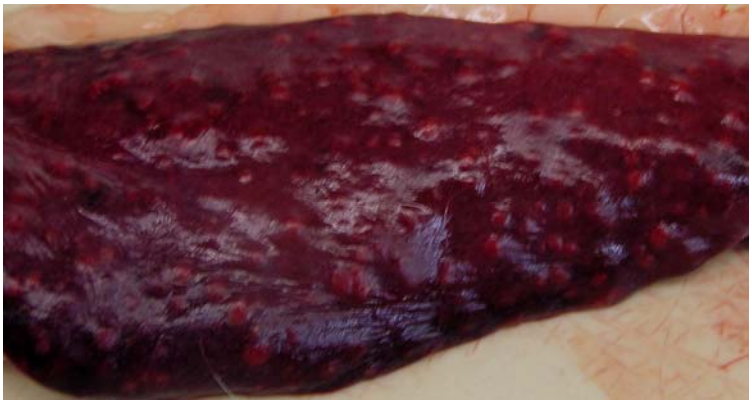


Figure 5: Nodular surface of the spleen

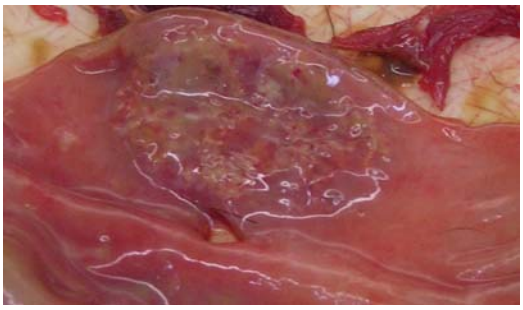


Figure 6: Irregular mucosa of the distal ileum

case had previously been seen ingesting portions of rabbits and rodents and that is the likely source of infection in this cat. The owner had been utilizing reputable veterinary products for tick-control on her cats. Tularemia has zoonotic potential and causes plague-like lesions in humans. Most humans acquire the disease through the bites of arthropods carrying the bacterium. Veterinarians, hunters, trappers, farmers and laboratory workers have an increased risk of acquiring this disease due to the increased risk of exposure to infected animals. Tularemia is a reportable disease in North Carolina.

Songer, JG, Post, KW, The Genus Francisella: In Veterinary Microbiology Bacterial and Fungal Agents of Animal Disease. pp. 208-213. Elsevier Inc., St. Louis, MO, 2005.

Dr. Robinson

LIVESTOCK

Cattle

An **Angus cow** with an estimated body weight of 500 kg was found dead in the pasture. No previous clinical signs were witnessed by the farmer. The vaccinated herd was fed fescue hay, grain and a mineral supplement. The necropsy examination was unremarkable with exception of a rumen pH that measured 5 units. Histologic examination revealed a mild to moderate multifocal suppurative rumenitis consistent with **rumen acidosis**. This condition is a consequence of feeding high grain diets to ruminant animals that are adapted to digest and metabolize predominantly forage diets. This case is highlighted as a reminder that rumen pH can significantly increase after death, particularly when a long period of time lapses between death and necropsy. This cow was not presented for necropsy until at least 12 hours post-death; therefore the antemortem rumen pH value was likely 3.5 to 4.

Dr. Wade-Caesar

A **6 week old calf** was presented to the Rollins Diagnostic Laboratory for necropsy. The owner saw this calf walking one morning and found it dead a short time later the same morning. The calf had a month long history of a swollen, chin, muzzle, gums and bleeding from the gums. The veterinarian medicated the calf but the lesions in the mouth did not resolve. The calf had scours that were treated by the owner. The scours improved but the calf continued to have loose stool. On gross examination the calf was in mild to moderate dehydration status. Multifocal proliferative tan approximately 1.0cm diameter plaques were on the surface of the caudal aspect and rostral edges of the tongue. Similar dark red proliferative material was on the gingiva of the upper and lower dental arches bilaterally. This material surrounded the premolars and incisors. The proliferative tissue was also on the hard and soft palate, edges of the lips and on the muzzle. The muzzle was slightly swollen. The dorsal region of the caudal lung lobe was dark red and meaty in consistency (involving only 5 to 10% of the total lung area). Multifocal similar 0.5 cm to 1.0 cm round proliferative pink, tan masses were attached to the mucosa of the reticulum, rumen, and edge of the omasal mucosa. On **histopathology examination** there was moderate, chronic, proliferative and necrotizing, regionally extensive, cheilitis, glossitis, and rumenitis with bacteria and intracytoplasmic inclusion bodies. Regionally extensive, severe, acute suppurative, bronchopneumonia and pustular dermatitis of the ear were also present. Arcanobacterium pyogenes was isolated from the lung in routine aerobic culture. 150 EPG of trichostrongyle-type ova were observed in the Mcmaster's chamber examination. Strongyloides were also observed in the feces. Electron microscopy confirmed the presence of poxvirus in affected oral tissue (the etiology of the inclusion bodies). This calf was diagnosed with **Papular Stomatitis**. Papular stomatitis is caused by Bovine Papular Stomatitis Virus. This virus usually produces papular and sometimes erosive lesions on the muzzle and buccal mucous membranes of cattle younger than 20 months of age. The lesions usually heal in 3 to 5 weeks. This virus does not usually cause the death of the animal. Occasionally calves can have more severely and widely distributed involvement where lesions are present in the posterior oral cavity, esophagus and gastric mucosa as in this calf. Widely distributed lesions of Papular Stomatitis in calves may be secondary to another disease,

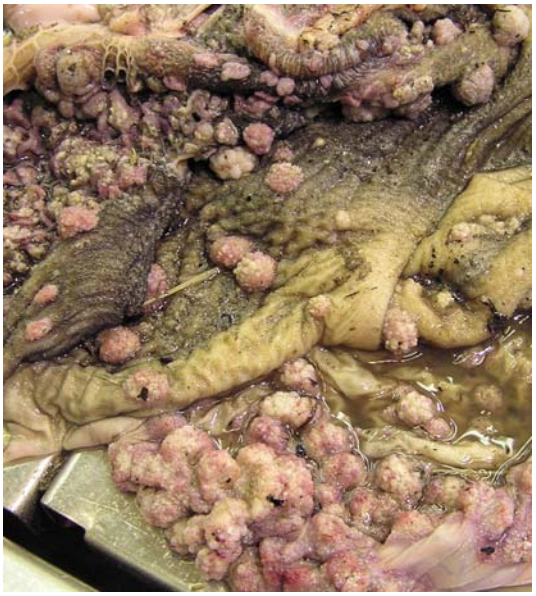


Figure 4: Papular masses located in the rumen and reticulum

LIVESTOCK, CONTINUED

an indication that the calf is immunosuppressed, or secondary to a dietary deficiency or low-level toxicosis. The calf was negative for BVD by virus isolation. This virus has zoonotic potential as it has been associated with skin lesions in people.

Kahrs, Robert. F. Papular Stomatitis. In: Viral Diseases of Cattle, 2nd edition. pp. 179-184. Iowa State University Press, Ames, IA, 2001.

Dr. Robinson

Equine

A 5 year old **miniature donkey** jack had a 3-4 day history of shifting weight on all 4 legs. The owner thought the excessively long hooves were causing pain. The donkey also started vocalizing differently and then bit the dog and a person. According to the owner the donkey was normally very tame and friendly. The next day the owner saw the donkey rolling on his back and then roll onto his side and die. Necropsy examination revealed extremely long hoof walls with severe separation of the hoof wall from the lamina of all 4 feet. Moderate subcutaneous edema of the polls, base of ears, both shoulders, and along the back was present. The internal body temperature 2 hours after death was elevated and measured greater than 106 F (the upper limit of the thermometer). The liver was enlarged and light tan. No other significant gross lesions were present. The preliminary diagnosis at necropsy was hepatic lipidosis secondary to the severe foot problems although the hepatic lipidosis did not appear severe enough to cause death and it did not explain the fever. Rabies testing was requested because a person had been bitten and a definitive cause of death was not found at necropsy. The brain was **positive for rabies** virus by FA at the NC Public Health Lab. The positive rabies result was surprising because the donkey did not have obvious neurological clinical signs. This was an unusual presentation of rabies in which neurological clinical signs were more like behavior changes, which could have been attributed to severe lameness. However, at necropsy the donkey was febrile indicating systemic disease and a cause of death was not found thus further

Careful questioning of the owner and recognizing that a definitive cause of death was not found at necropsy was important in this case to get the correct diagnosis and protect public health. The person bitten by the donkey was given post exposure rabies vaccinations and the other animals on the farm including a commercial dairy goat operation were carefully examined and observed.

Dr. Haugland

Small Ruminant/Camelid

An 11 year old **male neutered llama** was presented to the Rollins laboratory for necropsy with a history of swelling of the nose and muzzle that occurred 7 months prior to death. The llama was treated with amino acids and plasma. Six months after the onset of clinical signs the llama developed ascites. Bloodwork indicated that the llama had hypoalbuminemia, hypoglobinemia, and hypoproteinemia. LDH, CK and Creatinine were elevated. The veterinarian performed an abdominocentesis and obtained clear, cell-free fluid. The llama improved initially after the abdominocentesis but over the following 3 weeks the edema and ascites returned and worsened. The llama also had some muscle wasting and weightloss. The llama became uncomfortable, assumed a cushed position, stopped eating and became weak. The owner elected for euthanasia. On **gross necropsy** examination there was severe subcutaneous edema under the chin, ventral abdomen and in the distal extremities. The abdomen contained approximately 3 L of serous fluid. The cortex of the kidneys was granular on cut surface. The mucosa of C3 was roughened and thickened. Multifocal white pinpoint foci were in the pancreas and in the adjacent abdominal fat. **Histopathology findings** included: 1. Moderate to severe, diffuse, chronic glomerular amyloidosis with tubular ectasia and proteinuria in the kidney. 2. Subacute, severe, diffuse, dermal and subcutaneous edema in the haired skin and subcutis. 3. Moderate acute to subacute, multifocal, necrosuppurative pancreatitis with regionally extensive necrotzing steatitis. 4. Subacute, moderate to severe, diffuse submucosal edema in C3. The llama was diagnosed with **Renal amyloidosis with tubular ectasia and proteinuria and Pancreatitis with necrotizing steatitis.** This llama had features consistent with the nephrotic syndrome (hypoalbuminemia and generalized edema), which usually occurs secondary to glomerular disease. A brief search of the literature did not reveal any reports of renal amyloidosis in camelids. Pancreatitis has been reported in one alpaca and in a llama.

Hamir, AN, Habecker, PL,Tillman, C.: Pancreatic necrosis in a llama. Vet Rec (1998) 142: 644-645.
Anderson, DE, Constable PD, Yvorchuk, KE, et al.: Hyperlipemia and ketonuria in an alpaca and a llama. J. Vet Intern Med. (1994) 8 (3): 207-11. (abstract).

Dr. Robinson

A 3 year old female **Nubian goat** was presented to the Western Animal Disease Diagnostic Laboratory for post mortem examination. The provided history stated the goat was euthanized because it had been recumbent since giving birth ten days prior. The goat still had a good appetite and its offspring were doing well. Treatment had included injects of antibiotics and corticosteroids. On post mortem examination the goat had a body condition score of 3-3.5/5 and was of normal hydration. The brain was negative for fluorescence under ultravio-

LIVESTOCK, CONTINUED

let light, but the brain was unusually soft. The lungs were spongy to rubbery on palpation. Histopathology revealed multifocal, moderate neuronal necrosis and cavitation with **metastrongyle adults** in examined brain tissue and a mild **eosinophilic pneumonia** with numerous nematode adults. The lesions in the brain were consistent with a *Paralephstrongylus tenuis* infection while the lung lesions were consistent a *Muellarius capillarus* infection.

Dr. Drum

POULTRY

A 9 month old **Black Star male chicken** was presented to the Western Animal Disease Diagnostic laboratory alive for post mortem examination. The provided history stated the bird had been ill for the following month with weakness, lethargy, difficultly breathing and diarrhea. The owner stated that the bird had not been right since birth and was unable to fly like the other birds. On physical examination the bird was weak and seemed disoriented. The bird was in lean body condition. Subcutaneous edema over the thorax and in the groin area was noted. The abdominal cavity was full of yellow colored semi-opaque effusion. Four distinct fluid filled cysts were present in the abdominal cavity. The cysts took up approximately 80 % of the abdominal cavity. The cysts were multicompartmentalized with areas of firm tissue palatable after the cysts were drained. The point of attachment for the cysts was the dorsal abdominal wall adjacent to the kidneys. The liver was brown in color with a military pattern of pale colored raised nodules. Serious atrophy of epicardial fat was grossly evident. The lungs were spongy on palpation with areas of red colored tissue. The cecal tonsils were enlarged and pale in color. On histolopathologic examination of the tissue, lesions in the kidney were consistent with **nephroblastoma**, while lesions in the liver, kidney, spleen, and intestine were consistent with **Lymphoid leukosis**. It is generally considered that nephroblastomas are caused by a retrovirus.

Dr. Drum

A 3 year old **Peacock** with a history of recent weight loss and open mouthed breathing was examined at the Griffin Animal Disease Laboratory. Findings included a subcutaneous, firm, oblong mass in the neck, approximately 3.5 by 6.0 centimeters in size. The encapsulated mass contained pale to dark yellow, caseous material. Pale tan, irregular shaped foci, .5 to 1 centimeters in size, were noted in the liver, and a pale tan mass partially surrounded the gall bladder. Caseous granulomas separated by infiltrates of lymphocytes, plasma cells, macrophages, with several large multinucleated giant cells were noted in histopathology sections, and intralesional acid-fast bacteria morphologically suggestive of Mycobacterium were further identified. The National Veterinary Services Laboratory (NVSL) identified the acid fast bacteria as **Mycobacterium avium**. Avian Mycobacteriosis (Avian Tuberculosis) is typically caused by species of bacteria in the Mycobacterium avium complex (MAC). Avian tuberculosis is an infectious disease with potential for spread to other birds and has been known to occur in humans, especially in individuals with immune compromising disease conditions.

Dr. Ridenour

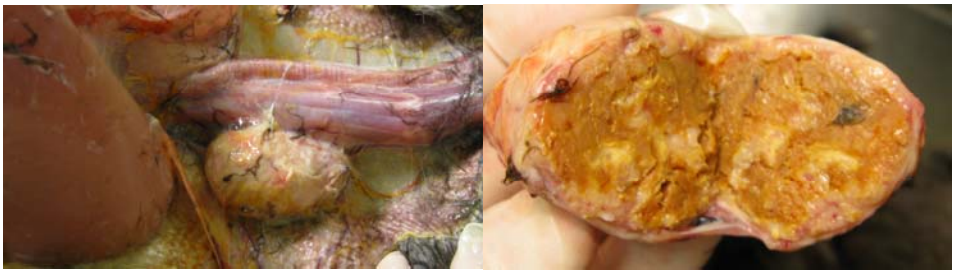


Figure 5: Cervical mass

Departmental News

Monroe Laboratory

Dr. Ridenour gave a presentation on Backyard Poultry diseases to a group at the Union County Agriculture Extension Center in March.

Elkin Laboratory

Dr. Kimberly Lee Townsend age 51, of Hawthorne Rd. passed away Sunday March 7, 2010 at her residence. She was born April 23, 1958 in Forsyth County to John A. and Camilla Galloway Townsend. Ms. Townsend attended NCSU where she graduated in 1980, Magna Cum Laude, with a BS degree in Zoology. She was also in the first graduating class of NCSU Veterinary Medicine, receiving her DVM in 1985. Kim started her veterinary career in publishing of veterinary literature in Kansas City, Mo., and then continued in Veterinary pharmaceuticals in Greensboro, N.C. She then followed up with a year in Basel, Switzerland. She was currently employed with the N.C. dept. of agriculture in Elkin as a veterinary diagnostician , and a member of Elkin First United Methodist Church. She was preceded in death by a brother, John A. Townsend, a sister Sandra Gail Townsend, one nephew Kelley J. Huitt, and a Niece Kippen Huitt.
She is survived by her parents, John A. and Camilla Townsend of Lincolnton; two daughters, Kelly Christian Arnold, and Camilla Hyland Arnold of the home; two sisters Pati Townsend Cooper and husband Michael, of Lincolnton N.C and Cari Townsend Rhodes and husband Jeff, of Fletcher, N.C. ;three nephews and two nieces. Memorials may be made to the American Cancer Society, p.o. box 22718 Oklahoma City, Ok. 73123; or Mountain Valley Hospice and Palliative Care 401 Technology Ln. suite 200, Mt. Airy N.C. 27030

CE attendance

Dr. Mahogany Wade-Caesar attended the Southeastern Veterinary Pathology Conference (SEVPAC) in Tifton, GA on May 15 & 16.

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Assistant Director

Dr. Richard Mock

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Dr. Tahseen Abdul-Aziz

Dr. Peter Moisan

Dr. Steven Rushton

Dr. Alison Tucker

Veterinary Diagnosticians

Dr. Jennifer Haugland

Dr. Stacy Robinson

Dr. Mahogany Wade-Caesar

Veterinary Microbiologists

Dr. Gene Erickson

Dr. Karen Post

Laboratory Section Supervisors

Kim Bennett—Virology

Sandy Murphy—Bacteriology

Mary Horne—Histopathology

Jennifer Pruitt—Serology

Beverly Wood—Molecular Diagnostics

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