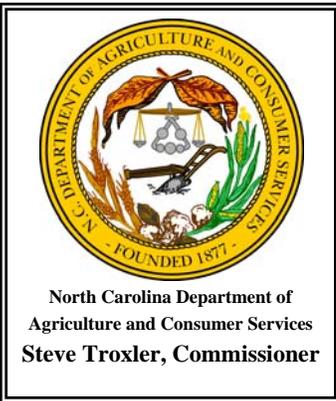


The NCVDLS REPORT



Veterinary News and Information From North Carolina's Diagnostic Laboratories



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Holiday Closings...

- May 25, 2009
- July 3, 2009
- September 7, 2009

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

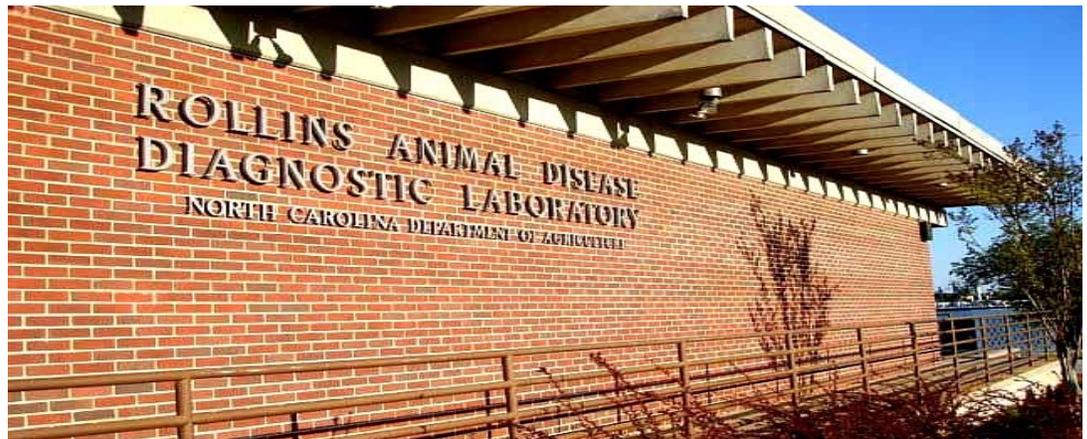
Message from the Director

At this writing, the General Assembly is in town hammering out the details of our next state budget. How much of an impact the slashing and burning will have on our laboratories in the next fiscal year remains to be seen. All state agencies are currently facing budget cuts and we must revert 9% of our appropriated funding by June 30, 2009. Although nothing has been finalized at this point, one of the options we have submitted to the Office of State Budget and Management, as part of our required reductions, is to increase existing fees and impose new fees for services. The most significant change will be assessing charges on specimens from food animal production species. If adopted by the General Assembly, these fees would become effective on July 1, 2009. To date, we still have no official word whether these fees have been adopted, however, as a measure of preparation, we have either mailed or faxed letters to our clients explaining these changes. This client letter appears in this newsletter (see pages 16 & 17) and is available on our website.

I am very pleased to announce that our new Assistant Laboratory Director, Dr. Richard Mock, officially reported to work on April 16. He served for 27 years as Section Head of Virology for the Texas Veterinary Medical Diagnostic Laboratory in Amarillo and brings a wealth of experience to the position. His primary duties will involve daily oversight of the activities of the Rollins Laboratory. Be sure to read his biography later in this newsletter.

While the Canadian Food Inspection Agency has indicated that it found H1N1 human flu virus in a swine herd in Alberta, there is still no hard evidence that this virus exists in US swine herds. Due to the expanding outbreak, the NCVDLS has developed testing algorithms that would allow us to respond to potential needs of our swine industry to perform surveillance testing. These would include screening nasal swabs for the presence of Type A influenza virus, sub-typing the virus in positive swabs by multiplex PCR assay, and performing cell cultures on PCR compatible samples. Be sure to read the information provided by Dr. Gene Erickson, our resident national expert on animal influenza viruses, which also appears in this newsletter.

During the next few months, NCVDLS hopes to begin polling clients for ways we can



Message from the Director, *continued*

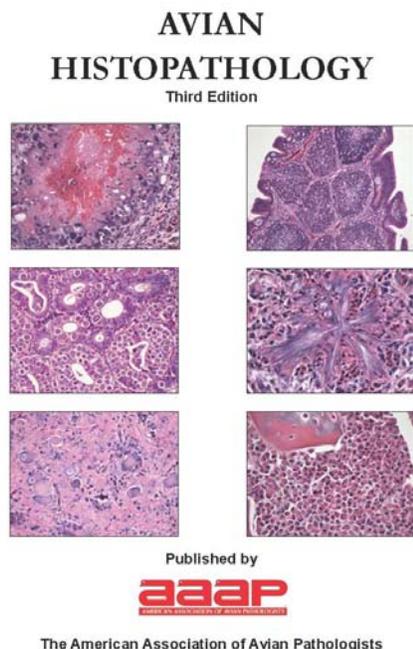
serve them better. Surveys are in the development stages and will be distributed. We hope our clients will now begin thinking of new services we could provide or ways to improve existing services.

Sincerely,

Karen W. Post DVM, MS

Director of Laboratories

Client Corner



In January 2009, the third edition of *Avian Histopathology* was released by the American Association of Avian Pathologists. The 14 chapters of the book are organized by body system and contain more images than preceding editions, for the first time in color. The editor of the third edition is Dr. Oscar Fletcher, faculty at NCSU College of Veterinary Medicine, and the Associate editor is the NCVDL Avian Pathologist Dr. Tahseen Aziz. In addition of being the associate editor, Dr. Aziz is the first author, a coauthor, or a contributor of 12 of the 14 chapters of the book. *Avian Histopathology*, 3rd edition is available through the American Association of Avian Pathologists website at <http://www.aaap.info/mc/page.do?sitePageId=50267>.

Currently under review by the General Assembly is a proposal to increase the fees for services provided by the diagnostic laboratory system. This proposal is a result of the current economic climate and the impact it has had on the state budget. The last changes to laboratory fees occurred in 2005. Internal efforts to reduce spending have included eliminating travel expenditures and eliminating vacant positions within the laboratory system. A detailed outline of the proposed fee increases is included in the back of this newsletter (see pages 16 & 17) and can be found through a link on the website www.ncvdl.com.

Novel Type A Influenza H1N1 of Man and Pig
Dr. Gene Erickson

Well, the concern for mass human mortality has subsided in this country with the recovery of 1,638 people in the US and the death of one pregnant woman with other underlying medical issues. In addition, there was one Mexican infant that died while his family was visiting others in Texas. There have been some really large clusters of transmission, such as the New York private school that resulted in infection of 50 students, all of whom recovered. Mexico City, the epicenter of the pandemic spread of this virus, closed all government offices and businesses for several days, which appears to have dramatically slowed the transmission rates in that country, currently at 1112 confirmed cases with 42 deaths. There is a great deal of controversy on when the virus began circulating in the human population of Mexico, and genetic analyses recently cited in the

CLIENT CORNER, CONTINUED

media indicate that novel type A influenza H1N1 has been circulating in Mexico for the last 8 months. Thus, the famous La Gloria boy that recovered is unlikely to be the index case for Mexico.

The Canadian hog farm, like most of the US cases was directly linked to travel to Mexico. A carpenter returned from holiday in Mexico and shortly thereafter worked on a 220 sow farrow-to-finish unit, transmitting infection to the swine herd. Most of the disease was observed in the grower pigs, but we do not have any details as of yet on other endemic diseases in the herd, which is located in the province of Alberta, not far from the US border. See this link for details of Canada's report of the outbreak to the OIE:

http://www.oie.int/wahis/public.php?page=single_report&pop=1&reportid=8065

As of May 8, Canada has indicated they will market recovered pigs as part of the normal food chain, which had been quarantined with a stop movement order shortly after the outbreak was diagnosed in late April. The government indicates they will only market virus negative pigs. This will be an interesting process, since influenza is a notoriously difficult virus to remove from a continuous flow, farrow-to-finish farm.

Please refer to the multiple links posted below for more information:

The PAHO site will be important to follow the human spread of this virus during the winter season of South America: http://new.paho.org/hq/index.php?option=com_content&task=view&id=1321&Itemid=569

The CDC's Morbidity and Mortality Weekly report updates:

http://www.cdc.gov/mmwr/preview/mmwrhtml/mm5817a1.htm?s_cid=mm5817a1_e

CDC's state map distribution of positive cases and information on the ongoing US outbreak: <http://www.cdc.gov/h1n1flu/>
WHO's daily updates on the spread of the virus across the world:

http://www.who.int/csr/don/2009_05_07a/en/index.html

And, importantly, the OIE's reaffirmation that processed pork from the infected Canadian farm will be safe to eat: http://oie.int/eng/press/en_090507_bis.htm

The NC Pork Council's information on novel type A H1N1:

<http://www.ncpork.org/pages/news/H1N1flu.3things.jsp>

NCDA&CS Advisory to Pork Producers to enhance biosecurity to prevent infection of NC herds: <http://www.ncagr.com/paffairs/release/2009/bio.htm>

Update to Rendering Policies

The FDA has modified the deadline for compliance with new regulations for rendering specified risk materials (see original article in December 2008 newsletter under Client Corner section). The changes will take effect on April 27, 2009; however the FDA will not enforce penalties until October of 2009, allowing time for companies to develop a plan to come into compliance with the regulation. The NCVDLs has been working with contracted rendering companies to be in full compliance at the time the law took effect, April 27, 2009. Updated standard operating procedures (SOP's) are currently in place at all laboratory sites.

Feature Article

Abomasal diseases in calves

By Dr. Peter Moisan

Abomasal disease of cattle is a significant problem that is compartmentalized to the various age groups of animals. In other words, though each age group tends to develop an age-specific set of abomasal problems, no one age of cattle is immune from various diseases of the bovine fourth stomach. The function and morphology of the abomasum closely resemble those of the monogastric animal stomach; however, as food must traverse the proximal 3 bovine stomachs, the abomasum and its internal environment are subject to changes in chemical and physical content of the material received from those stomachs. As a result, the lesions that we see so frequently in the abomasum usually differ markedly in histomorphological appearance and etiology from those of simple-stomach mammals.

The abomasal lesions that we commonly see are arbitrarily divided into 4 categories for convenience: bloat, ulcer disorders, abomasitis (infectious), and indigestion:

I. Abomasal Bloat

Simple abomasal bloat is a common finding. In most instances the gas that accumulates in the abomasum passes distally into the intestinal tract where it is passed with the feces (abomasal gas cannot be passed in the proximal direction in any appreciable volume over a short time). However, with accumulations of abomasal gas, especially when ileus is involved, there is a tendency for the gas to cause discomfort to the calf with grinding of the teeth (bruxism), rolling, and other signs of colic. In these instances, the abomasal gas will cause noticeable distension of the abdomen. This can lead to torsion of the abomasum with rapid necrosis of the abomasal wall and death unless immediate surgical intervention occurs. Another sequel, similar in its pathophysiology to rumen bloat of older animals, is cardiovascular compromise secondary to the tremendous distension of the abomasal viscus and pressure on the heart and lungs.

An etiology for abomasal gas and subsequent bloat is often not apparent. Simple ileus with overpopulation of bacteria from the gastrointestinal tract is thought to be the cause of some cases. Special circumstances include the invasion of the abomasum by specific pathogens (see below, Abomasitis). One of the more common causes of abomasal bloat in submissions to the NCVDL is *Sarcina vitullorum* overgrowth.¹ This organism, which is a soil- and rumen-dwelling bacterium, is an anaerobe that populates the calf abomasum and may grow to sufficient numbers to generate abomasal bloat in a young calf. The affected calves are usually dairy calves, 1-14 days-of-age and often have a vague or brief history of enteritis or diarrhea that may involve ileus. Antibiotics are thought, in some cases, to cleanse the system of resident bacterial populations and allow the growth of *Sarcina* species, which are prolific gas producers. The combination of antibiotic use and ileus may allow the production of a fatal amount of gas (carbon dioxide) by these agents. Culture of *Sarcina* species is possible but only under experimental conditions, as these bacteria are anaerobic and fairly fastidious in their growth requirements. Affected animals have a markedly distended abomasum and die after a short course of abomasal bloat. Necropsy lesions include hyperemia of the abomasal mucosa with emphysema or gas bubbles in the mucosa and submucosa. Many cases have a ruptured abomasum as well, which is a particularly common finding a few hours after death.

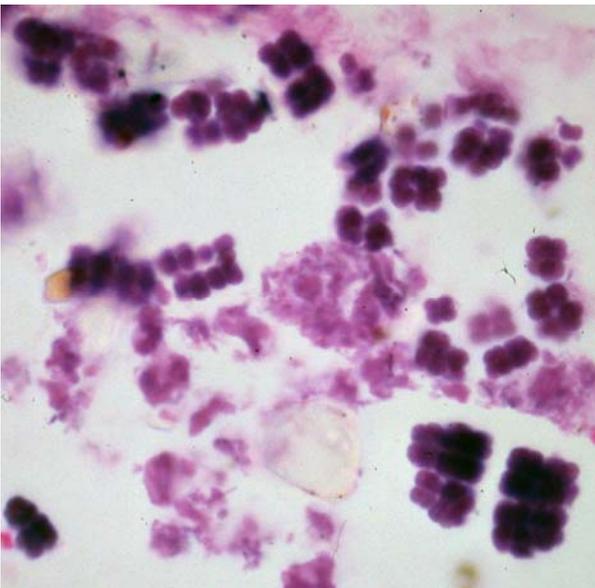


Figure 1: *Sarcina* in situ.

The histological appearance is pathognomonic for *Sarcina* overgrowth. Organisms form octets and larger aggregates of large cocci, usually located over the abomasal mucosal surface (figure 1). Invasion of the tissues is not seen in this condition.

II. Abomasal Ulcers

Ulcers are common in the abomasum of all ages of cattle. The etiology, when determined, varies widely. In this article, I

Feature Article continued

will concentrate on the ulcers seen in young calves. In dairy calves, the ulcers and abomasitis that we encounter are usually associated with inadequate milk replacer diets (see below under Indigestion). In beef calves, which are usually 6-8 weeks-of-age, these abomasal ulcers tend to be in individual calves and the etiology is seldom apparent.² Clinical signs are lacking in most cases (in beef calves), and the animals are found dead. Necropsy examination usually reveals a calf in good body condition with a perforating abomasal ulcer and localized or generalized peritonitis (figure 2). Though hairballs are often seen in the abomasum at necropsy, these are seldom thought to be a main factor in the etiology. Suspected causes, which vary by region, include epitheliotrophic viruses, such as IBR or BVD, rough abrasive feeds, lack of antioxidants (Vitamin E and selenium deficiencies have been implicated), and copper deficiency. Copper is required for collagen biosynthesis and a deficiency may cause structural weakness of the abomasal tissues as well as other connective tissues in the body.



Figure 2: Gross photo of perforating abomasal ulcer.

III. Clostridial and mycotic abomasitis

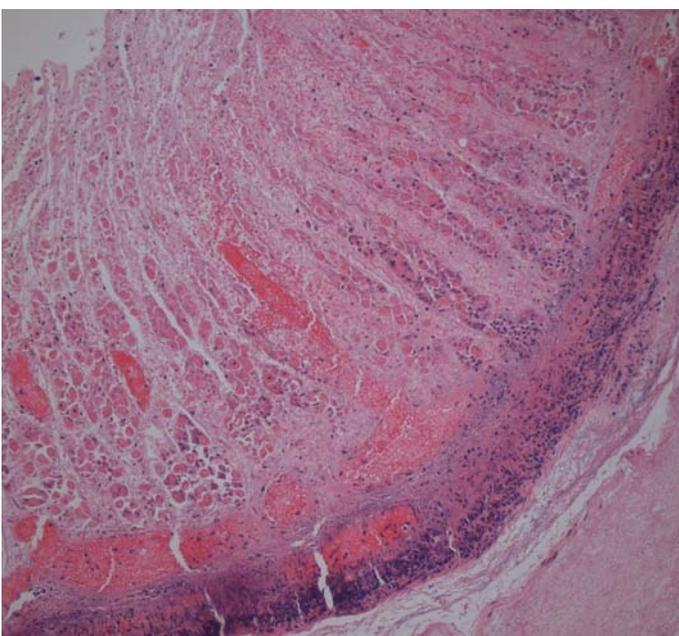


Figure 3: *Clostridium septicum* in necrotizing abomasitis case.

The clostridial abomasal diseases of calves include a select few agents that are economically important, particularly in very young calves. Enterotoxemia caused by *Clostridium perfringens* Type D is considered a disease of older, weaned calves, usually on heavy grain feeding, and deserves separate consideration. *Clostridium septicum*, *Clostridium perfringens* Type A, and possibly some other species of clostridia are responsible for the invasive clostridial abomasal diseases of calves. *Clostridium septicum*, the agent of braxy of sheep, is occasionally isolated from cases of necrotizing abomasitis in calves (See Short Cuts article in this issue). These organisms (*Cl. perfringens* and *Cl. septicum*) are gram positive and, when viewed microscopically, are responsible for marked necrosis and damage to the abomasal mucosa and submucosa (figure 3). Gastric rupture is often seen with these infections as well.⁵

Mycotic abomasitis is a common finding in debilitated or immune deficient calves (particularly those with failure of passive transfer). The agents associated with fungal abomasitis include

various Zygomycetes (such as *Rhizopus*, *Mucor*, *Mortierella*, and others) or *Aspergillus* species. *Candida* species (most often *Candida albicans*) are occasionally seen as well in our submissions. The reasons for invasion of these agents into the abomasum (and other organs of the gastrointestinal tract, most commonly rumen, reticulum, and omasum, but also intestine and liver) are largely unknown, however, mycotic abomasitis is most often seen in animals fed poor quality milk replacer (see below, under Indigestion) or those with ileus. The filamentous fungi show tropism toward vascular invasion (figure 4). With *Candida* infection, the hyphae, pseudohyphae, or budding forms (occasionally all present within the lesions of the same animal) are located within the mucosa and submucosa, and most often within large accumulations of necrotic debris and fibrin vascular thrombi (figure 5). It is thought that these organisms thrive in the presence of tissue debris, a diminished mucosal immune cell population, and residual

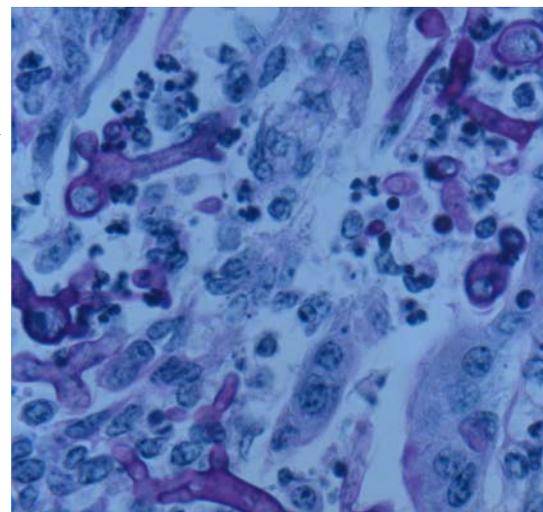


Figure 4: Zygomycete abomasitis.

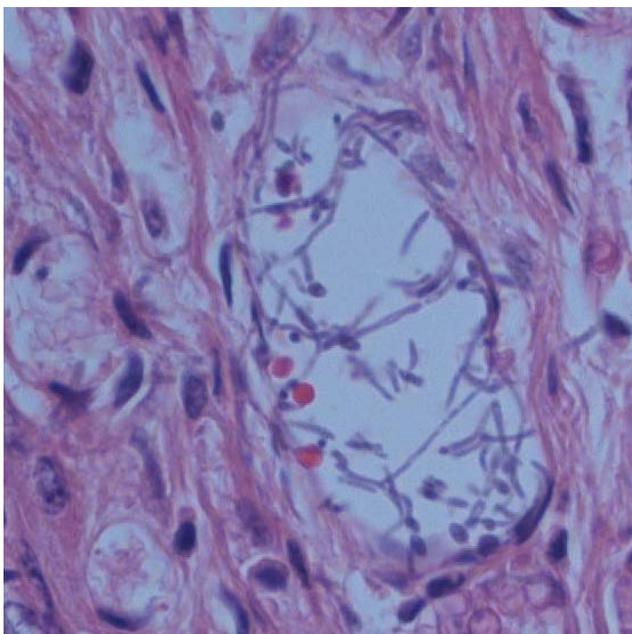
Feature Article continued

Figure 5: *Candida* abomasitis.

tend to proliferate in the rumen and other forestomachs when this milk material pools, with secondary inflammation and occasional opportunistic infection by these agents as a consequence. In the most severe cases, death results from infectious disease of the abomasum and other stomachs.⁴ Sepsis can also be seen following breach of the mucosal barrier by these opportunistic agents. Necropsy usually reveals a white or brown growth over the abomasal and other gastric mucosa (figure 6). Bacterial and mycotic or candidal abomasitis is confirmed by histopathology. The emaciation and indigestion caused by poor quality milk replacers or failure of closure of the esophageal groove are usually ameliorated by a return to suckling or weaning to solid feed.

proteinaceous ingesta, such as fermented milk or milk replacer.³

IV. Indigestion

Though indigestion in calves is a common event, it is often overlooked until it results in starvation or debility of the animal. In this context, we will refer to young calves that are on milk replacer diets that may be considered the so-called “ruminal drinkers”. These are usually dairy replacement calves (occasionally beef calves, depending on circumstances) that have a failure or partial failure of closure of the esophageal groove. When drinking from a pail, and not suckling, the esophageal groove fails to close, and instead of rapid shunting of milk replacer or other fluids from the oral cavity to the abomasum, some fluid is pooled in the rumen. This pooling also occurs when calves drink cold milk or milk replacer, as the decreased temperature tends to depress closure of the esophageal groove. Esophageal groove closure is probably also hindered during the ileus that accompanies enteric disease.

Various bacteria, *Candida* species, and other fungi

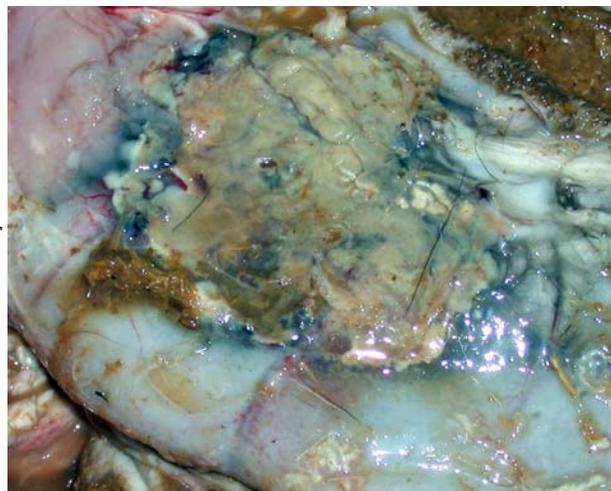


Figure 6: Exudate over the abomasal mucosa from a calf affected by ruminal drinking.

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- 3: Jensen HE, Basse A, Aalback B. Mycosis in the stomach compartments of cattle. *Acta Vet Scand.* 1989; 30:409-423
- 4: Radostitis OM, Gay CC, Hinchcliff KW, Constable PD. Diseases of the alimentary tract – II. In *Veterinary Medicine: A textbook of the diseases of cattle, horses, sheep, pigs, and goats.* 10th edition, 2007; pp293-382
- 5: Songer GJ, Miskimins DW. Clostridial abomasitis in calves: case report and review of the literature. *Anaerobe.* 2005; 11:290-294

Short Cuts

COMPANION ANIMAL

Feline

A 7 year old female black and white **domestic shorthaired cat** was presented to the laboratory for post mortem examination. The history included anorexia of two days duration and hypothermia. The cat's condition progressed to becoming semi-comatose. Its vaccination history was unknown. On post mortem examination the cat was very dehydrated (over nine percent). There was thick, bilateral nasal discharge with a foul odor present. The medial left lung lobe was rubbery on palpation and the gastric wall was thickened. Histological lesions included a ulcerative gastritis, moderate to severe pulmonary arterial hypertrophy and intimal proliferation, with focal bronchiolitis, and chronic, multifocal myocardial fibrosis. While the cause of death was attributed to cardiopulmonary failure due to chronic hypertension, the interesting finding in this case was the isolation of a **Group B *Salmonella* sp.** from the lung. This was the likely cause of the thick upper respiratory discharge with a foul odor.

Dr. David Drum

A 3 year old pregnant female **Ragdoll cat** was found dead one evening when the owner returned from work. The cat was due to deliver her kittens in 6 days. The cat had last been seen alive by the owner that same morning and had appeared healthy at that time. On gross necropsy examination mucous membranes were pale pink. The liver was diffusely yellow, but did not float in formalin. The left kidney was missing. The right kidney was present but significantly enlarged. A few petechiae were on the capsular surface of the spleen. The left horn of the uterus and left ovary were missing and the body of the uterus and right horn of the uterus were twist 540 degrees clockwise. The right horn contained 2 kittens. Each kitten had two kidneys. Severe diffuse endometrial and myometrial congestion, hemorrhage and necrosis were seen on histological examination of the uterus. There was no histological evidence of metritis or sepsis. Diagnostically significant bacteria were not isolated from this cat either. The queen was diagnosed with **Uterine torsion, Uterine horn and Ovary agenesis, and Renal agenesis.** The cause of death in this case was likely hypovolemia and shock as a result of the uterine torsion. Uterine torsion is rare in cats. When it occurs it is usually in late gestation. This cat may have been predisposed to develop a uterine torsion because of the missing left uterine horn and ovary. The right kidney was enlarged (hypertrophic) and compensating for the agenesis of the left kidney.

1. Young, James, D., Hillis, George P., and McKibbin, Marie L. Uterine torsion in a cat. Canadian Veterinary Journal 1991; 32: 479.

Dr. Stacey Robinson

Canine

According to the history provided, a 7-month-old intact female **Beagle** canine was euthanized due to chronic anemia and leukopenia for 2 months duration. Intermittent vomiting and anorexia were witnessed. The parvovirus ELISA was negative within the first few weeks of illness (the pet had received the full distemper-parvovirus vaccination series). The owner thought the pet may have ingested oleander plant from the yard; therefore supportive therapy was provided. At recheck, the pet responded to treatment; yet the anemia persisted. Tick serology was negative and the pet was treated with Clavamox and doxycycline. Within the following 2 weeks, the anemia completely resolved. One week later, the pet was submitted to the veterinarian for vomiting, diarrhea, vocalization, polyuria and polydipsia. On necropsy, the perineal region was stained with mild amount of blood. The stomach contained about 3-5 ml partially digested bile-tinged food and the colonic surface was lightly covered with bloody mucus. Histopathologic examination revealed diffuse crypt necrosis and degeneration with villous atrophy, blunting and fusion, and lymphofollicular atrophy of the small intestine; and erythroid and myeloid hypoplasia of the bone marrow. The gastrointestinal signs identified prior to death were a result of **canine parvoviral enteritis.** This case was particularly interesting because the history (vaccine history, chronicity of clinical signs) was not consistent with or suggestive of parvovirus infection. Although the puppy received the appropriate distemper-parvovirus vaccination series, it is likely that

COMPANION ANIMAL, CONTINUED

the chronic history of anemia and leukopenia resulted in decreased immunity, which predisposed this puppy to developing the viral infection. The exact cause of the anemia and leukopenia remained unidentified; however the bone marrow findings supported the history.

Dr. Mahogany Wade

Visceral leishmaniasis was diagnosed in a 3 year old **Coton de Tulear** canine submitted to the Western Animal Disease Diagnostic Laboratory (WADDL) in late march of 2009. The dog had a history of gradual deterioration in condition over at least a two month period. Abnormalities noted included loss of hair, reduction of food intake, weakness with markedly reduced energy level, severe anemia, and later, profuse bleeding from the nose.

At necropsy the body of the 3.6 kg., intact male, champagne Coton de Tulear was in poor nutritional condition with body fat stores depleted and skeletal muscle mass markedly reduced with bony prominences visible along the ribs, scapula, and pelvis. The hair coat was notably thinned out on the flanks but no overt alterations of the skin were evident. Generalized tissue pallor was striking as a result of markedly reduced cadaverous blood volume. Tartar accumulation on the teeth was significant. Wet and heavy lung lobes had a rubbery texture. The spleen was reddish-brown and compact while various lymph nodes throughout the body were reddish-brown and slightly enlarged. A light reddish-brown bone marrow was of a semi solid texture.

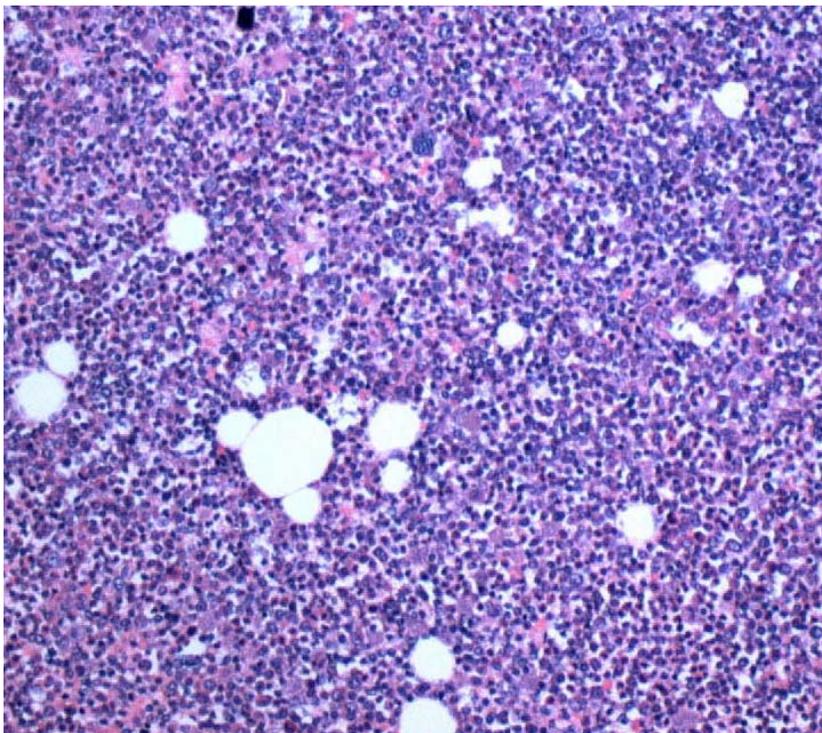


Figure 1. Bone marrow, leishmaniasis. Granulomatous inflammation with intrahistiocytic protozoa. HE stain .

Significant histomorphologic alterations were described as:

1. Spleen, lymph node, and bone marrow; splenitis, lymphadenitis, myelitis, granulomatous, chronic, diffuse, with intrahistiocytic protozoa and hemosiderosis.
 2. Heart; myocarditis, mild, multifocal, granulomatous, chronic, with intrahistiocytic protozoa.
 3. Kidney; interstitial and membranoproliferative glomerulonephritis, severe, diffuse, chronic, with glomerular senescence and sclerosis, proteinuria, fibrosis, and intrahistiocytic protozoa.
 4. Liver; hepatitis, chronic, mild to moderate, diffuse, granulomatous, with intrahistiocytic protozoa and hemosiderosis.
 5. Stomach; gastritis, subacute, severe, regionally extensive (middle lamina propria) with mineralization.
 6. Lung; alveolitis, necrotizing, diffuse, acute, severe, with mineralization and alveolar edema.
- Gingiva; gingivitis, necroulcerative, regionally extensive, chronic active, severe, with bacteria.

Peter Moisan, DVM, Dipl ACVP, Dipl ABVP,
Food Animal Specialties – Rollins laboratory

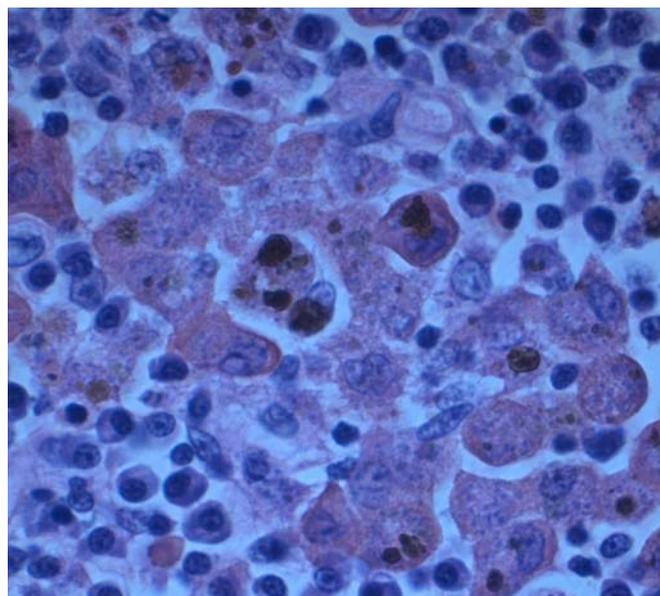


Figure 2. Lymph node; leishmaniasis. Granulomatous inflammation with intrahistiocytic protozoa. HE stain.

COMPANION ANIMAL, CONTINUED

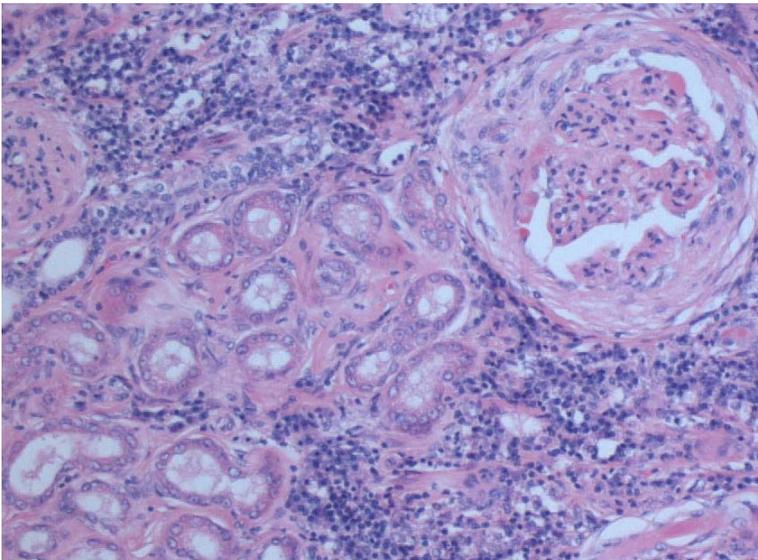


Figure 3. Kidney; leishmaniasis. Interstitial and membranoproliferative glomerulonephritis, severe, diffuse, chronic, with glomerular senescence and sclerosis, proteinuria, fibrosis, and intrahistiocytic protozoa. HE stain.

Changes in the gingiva, lung, and stomach were primarily the result of severely compromised renal function.

Marked erythrocyte turnover with hemosiderosis was responsible for the reddish-brown staining of the spleen, bone marrow, and lymph nodes. (See Figures 1, 2, and 3)

Leishmania is a genus of trypanosome protozoa, and is the parasite responsible for the disease leishmaniasis. Visceral leishmaniasis is a chronic, severe, disease of humans, dogs, and certain rodents characterized by clinical signs and lesions consistent with those encountered in this case. Infection in dogs is prevalent in Central and South America, the Middle East, and in the Mediterranean region. (The disease has recently been reported to be endemic in Foxhounds in North America.) Isolated cases are diagnosed around the world in animals that have visited endemic areas. The parasites are transmitted by the bite of several species of phlebotomine sandflies which are found throughout the world's inter-tropical and temperate regions. The incubation period is highly variable with a range of three months to several years. The most reliable diagnostic test for canine leishmaniasis is the direct observation of the parasite in bone marrow or lymph node sam-

ples. The NCVDLs has diagnosed only 3 cases of canine leishmaniasis in the preceding 10 years. Interestingly, in each of the three cases the dog had spent a significant amount of time in the Mediterranean region. The Coton de Tulear in this case was imported from southern Spain at the age of 8 months.

Author's Note: Analysis of information, in part from the National Climatic Data Center in Asheville, N.C., indicates that – associated with global climate change – plants and animals have shifted their ranges by about six kilometers per decade toward the poles during the past quarter of a century. Spring events, such as blooming, frog breeding and migrant bird arrivals, have advanced 2.3 days per decade. Tropical pathogens are moving up in latitude and striking species not adapted to deal with them. (Scientific American, March 2009, pp. 78 – 79.) With North Carolina's vibrant tourism industry, rapidly growing population, and relatively temperate climate it is quite likely (if not assured) that veterinarians will encounter more of these so called "tropical" pathogens.

Dr. Richard Oliver

An 8 year old spayed female **Golden Retriever** presented to the veterinary hospital with weakness and a 1 day history of an inability to lift her tail. The only abnormal blood chemistry result was hypercalcemia (14 mg/dl). The dog was treated with IV fluids and antibiotics but she continued to weaken and stopped eating. Seven days later abnormal blood values included; azotemia, slightly elevated ALKP, hypoproteinemia, hypoalbuminemia, hypokalemia, anemia (HCT 25.1%), thrombocytopenia (29,000), and the calcium was still elevated. An abdominal ultrasound was performed 2 days later and the findings included a mildly enlarged left adrenal gland, 1-2 mm calculi in the bladder, and the spleen was diffusely enlarged with several ill-defined hypoechoic areas. The largest hypoechoic area was aspirated. The dog died early the next morning. Necropsy gross findings included a hemoabdomen (300-400 ml) and a diffusely enlarged and turgid spleen with nodules and 2 tears in the capsules (one tear was located over a nodule). The blood vessels leading to and from the spleen were turgid and firm. A 1x1 cm nodule was found in the left enlarged adrenal gland. The heart was pale and there were multiple foci in the myocardium. A 3x2x2 cm firm white mass was found at the base of the heart between the pulmonary trunk and the ascending aorta. The pulpy nucleus of the intervertebral disc of L6-L7 was mineralized and prolapsed dorsally into the spinal canal. Histopathology revealed an adrenal cortical adenoma, vascular thrombi of the spleen, periacinar liver necrosis, myocardial necrosis and fibrosis, chemodectoma, and erythrophagocytosis. The **adrenal cortical adenoma** may be the most important lesion. Typically this tumor is not functional but it will cause hypercortisolism in 10-15% of cases. Hypercortisolism can cause increased levels of procoagulation factors II, V, VII, IX, X, XII, and fibrinogen and can decrease antithrombin levels. Thrombin-antithrombin complexes, a marker of subclinical thrombosis, can be significantly increased in dogs with hypercorti-

COMPANION ANIMAL, CONTINUED

solism (R. Jacoby, et al). Therefore this dog could have had elevated levels of cortisol resulting in a hypercoagulability state with subsequent vascular thrombi of the spleen. The chronic active myocardial fibrosis may have been the result of thrombi in the myocardium. Vascular thrombi may have caused erythrocyte shearing and thus upregulated erythrocyte removal (erythrophagocytosis) by the liver and spleen. The vascular thrombi, infarcts, and erythrophagocytosis within the spleen caused the diffusely enlarged and turgid spleen. The turgidity of the spleen likely made the capsule more friable and it either ruptured on its own or secondary to penetration during the needle aspiration. Also severe hypoxia of the liver developed near the end and this could be contributed to vascular thrombi, anemia due to erythrophagocytosis, and the bleeding from the spleen. Anemia, vascular thrombi, and myocardial dysfunction were likely the cause of death. The chemodectoma was an incidental finding. The partial tail paralysis secondary to intervertebral disc disease was an unrelated clinical sign.

Jacoby, Robert, et al. 2001. *Biochemical Basis for the Hypercoagulable State Seen in Cushing Syndrome*, Archives of Surgery. V136; 1003-1007, 2001.

Dr. Jennifer Haugland

A 2 to 4 year old male neutered **giant schnauzer** weighing 43.0 kg was found dead in the owner's yard one evening. The dog had last been seen alive at 6:30 am earlier that morning and had appeared healthy at that time. The dog was kept indoors but had access to the backyard through a doggie door. The yard was fenced on all sides with a 6 foot privacy fence. Gross necropsy findings included red mucous membranes, slightly hypertrophied right ventricle, multifocal petechiae and linear hemorrhages in the epicardium of the ventricles, moderate hemorrhage in the thymus, moderate pulmonary edema and 100-150 ml of yellow ingesta admixed with paper towel, pieces of white, thin plastic, cardboard box, a few brown leaf fragments and parts of an orange box from Baker's chocolate. Histopathological findings included moderate to marked, multifocal thymic hemorrhage that may have been the result of a combination of postmortem and agonal changes. After the gross necropsy, it was confirmed by the owner that he found part of a Baker's chocolate box out in the yard. The owner suspected that they had left the pantry door open giving the dog access to an 8oz box of Baker's chocolate. These findings are most consistent with **Chocolate Toxicity**. Theobromine and caffeine are the toxic compounds in chocolate. Baking chocolate contains about 10 times the amount of theobromine as in sweetened chocolates such as milk chocolate. Ingestion of less than 0.2 oz/kg of Baking chocolate is considered potentially lethal to dogs. Based on this lethal dose, the history, Baker's chocolate box in the stomach, and lack of significant histological findings it was determined that this dog likely ingested a toxic dose of chocolate. The toxic compounds in chocolate result in cardiac muscle and central nervous system stimulation. Clinical signs include hyperactivity, restlessness, incoordination, seizures, tachycardia, and arrhythmias. Death occurs within 6 to 24 hours with an acute exposure to theobromine. Liver and gastrointestinal contents can be tested for theobromine and caffeine to help confirm a diagnosis in cases where chocolate package materials are not found in the stomach.

1. Gfeller, Roger W. and Messonnier, Shawn P. Handbook of Small Animal Toxicology and Poisonings. St. Louis, 1998, Mosby.
2. Helseltine, Johanna, Five Common Toxins Ingested by Dogs and Cats. Compendium Continuing Education for Veterinarians 2008; 30 (11): 578-587.

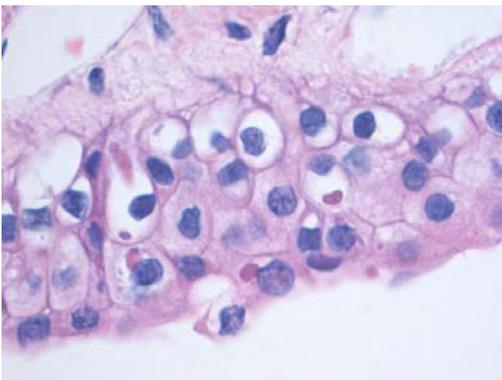


Figure 1: Inclusion bodies present in the renal pelvis

Dr. Stacey Robinson

A four week old **St Bernard puppy** was presented with a four day history of ocular discharge, weakness, seizure-like activity, decreased appetite, and frothing at the mouth. Several littermates had been showing the same progression of signs with varying onset, however none of them had died. This puppy was the most severely affected and was euthanized for the purpose of necropsy examination. Aside from the accumulation of purulent material in the eyes, no gross abnormalities were evident on necropsy. Histological examination of the tissues demonstrated inclusion bodies consistent with **canine distemper virus (CDV)** in the renal pelvis, lungs, and hippocampus. CDV is an RNA virus in the Morbillivirus family that can cause a wide range of clinical signs. These include fever, nasal and ocular discharge, anorexia, vomiting and diarrhea. Affected animals

COMPANION ANIMAL, CONTINUED

may display only a few or all of these signs. The initial infection occurs in the lymphatic tissues and from there it can spread to gastrointestinal, respiratory, urogenital and neurologic systems. The virus more often affects puppies but can affect older dogs as well. In this case, the bitch was recently acquired with an unconfirmed vaccination history, and also became affected shortly after this puppy was submitted.

Dr. Tim McComb

Exotics

An adult male **Mini Rex rabbit** became inappetent and was euthanized the following day. Physical examination revealed hypothermia and an abnormal abdominal palpation. The referring veterinarian suspected liver lobe torsion. On necropsy, the pet was in good body condition, mildly dehydrated and mildly autolyzed. A 360 degree torsion of the right lateral hepatic lobe was identified. The affected lobe was swollen and congested with fibrinous exudate along the surface. Histopathologic examination revealed acute massive necrosis with congestion and a fibrinous and heterophilic peritonitis. This was an interesting case because **hepatic lobe torsion** is uncommon and because the veterinarian made this diagnosis via abdominal palpation only (ancillary diagnostics were not performed).

Dr. Mahogany Wade

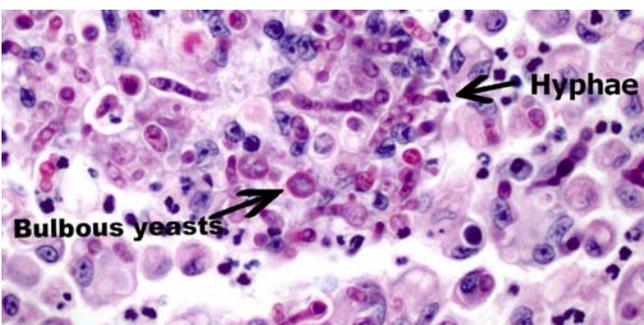
A 3.5 year old male **Sandfire Bearded Dragon** was presented to the laboratory for post mortem examination. The presented history stated the animal was not eating properly, was at the end of brumation but still lethargic and not displaying normal mating signs. The owner was initially concerned about coconut substrate ingestion leading to an intestinal obstruction. The animal had been healthy and had gone through 3 normal brumations. The animal's last feces appeared to be whole blueberries and an undigested cricket. On post mortem examination the liver was yellow-green in color and grossly enlarged. Sections of liver floated when placed in buffered formalin solution. There was scant content in the gastrointestinal tract and there was a 2 cm wide, circular thickening in the wall of the stomach. Histopathology revealed evidence of **lymphoma** in the liver, kidney and wall of the stomach. Additionally, the animals had severe diffuse hepatic lipidosis.

Dr. David Drum

SURGICAL BIOPSY

Recently a biopsy from a 10 year old male **DSH feline** was submitted. The dermal and subcutaneous mass was approximately 3cm in diameter and present for over a month. Histologically the dermis and subcutis are markedly expanded by a large aggregate of epithelioid macrophages and occasional neutrophils and lymphocytes. Within the cytoplasm of nearly all macrophages are numerous septated fungal hyphae and bulbous fungal spore structures.

Dermatophyte pseudomycetoma is an atypical rare form of dermatophytosis that involves the deep dermal and subcutaneous tissues and is typically caused by *Microsporum canis*. This disease is commonly seen in cats. Feline dermatophytic pseudomycetomas occur almost exclusively in Persian cats but may be seen in other longhaired breeds. Hyphae within the deep dermis and subcutis cause the formation of mycetoma-like granulomas.



This infection produces one or more subcutaneous nodules that are often ulcerated and draining. They occur mostly over the dorsal trunk or tail base. These cats may also have a more typical, superficial dermatophyte lesion on other areas of body. Lymphadenopathy is occasionally seen. Dogs are rarely affected with a possible predisposition in Yorkshire Terriers. Horses are rarely affected and the disease is usually caused by *Trichophyton equinum*.

Figure 2: Fungal hyphae and spores present within macrophages

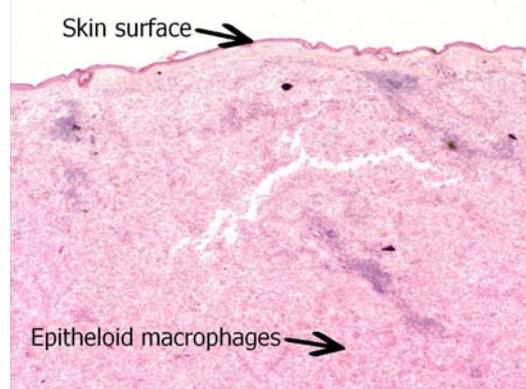


Figure 1: Macrophages expanding dermis and subcutis

Dr. Steve Rushton

SURGICAL BIOPSY, CONTINUED



Figure 1: Canine urinary bladder; botryoid rhabdomyosarcoma, cut surface.

The mass was highly cellular and the cells formed streams that were suspended in a small amount of preexisting fibrovascular stroma and associated with a mild desmoplastic reaction. The cells were spindle and cell borders were indistinct, with brightly eosinophilic homogeneous to finely granular cytoplasm, occasionally containing distinct cross-striations (interpreted as z-bands). There was a large nucleus to cytoplasm ratio. The nuclei were ovoid to round and often bizarre, and contained a coarsely granular and marginated chromatin pattern with prominent single or multiple nucleoli. Multinucleated cells and strap-like cells were common (Figure 2). There were 0-2 mitotic figures identified per high power field.

Rhabdomyosarcoma of the canine urinary bladder is very rare. It is most often seen in female dogs and has a characteristic botryoid (“grape-like”) gross appearance (Figure 1). With this tumor, histological confirmation of the lesion is usually easily accomplished. However, in a few instances, immunohistochemical differentiation is necessary.¹ A very guarded prognosis is warranted with this type of lesion due to the propensity for local and regional metastasis.

Dr. Peter Moisan

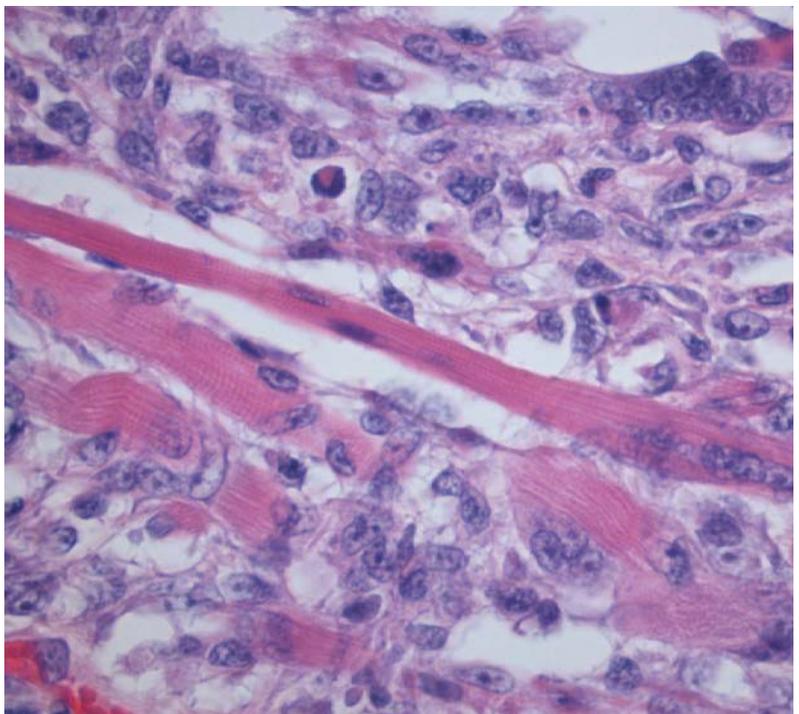
References:

1. Cooper BJ, Valentine BA. Tumors of muscle, in Meuton DJ, editor, *Tumors in Domestic Animals*, 4th edition. pp319-363,2002.

Biopsy material from canine **urinary bladder** was received at the Rollins Diagnostic Laboratory. The history indicated that the dog, a female, 3-year-old, English setter, had developed hematuria and dysuria. As the dog was recently purchased, no earlier, detailed history was available. Cytological evaluation of a urine sample revealed a large amount of cellular debris and crystals of undetermined type. Antibiotic therapy did not successfully resolve the problem and an exploratory laparotomy was performed. The surgery revealed the presence of a transmural mass in the region of the trigone of the urinary bladder, involving both ureters and the serosal surface. The mass was approximately 3x4x4 cm and irregular to cone-shaped in gross appearance, with a multinodular surface. The nodules were also apparent in cut sections of the mass (figure 1). Hematoxylin and eosin stains of 4um sections were made and routine histological evaluation of the lesion was performed.

Within the sections, there was a markedly polypoid, poorly circumscribed and incompletely encapsulated spindle cell mass, most consistent with a **rhabdomyosarcoma**. Neoplastic cells extended from the superficial propria submucosa, elevating the mucosa, to the serosa, which was mostly obliterated, and into the peritoneal space.

Figure 2: Canine urinary bladder; botryoid rhabdomyosarcoma, with strap and multinucleated cells.



LIVESTOCK

Cattle

The Rollins Laboratory received two cases of **rabies with splenomegaly in beef calves** that did not have typical clinical signs. These are in addition to the 2 year Angus bull reported in the [December 2008 newsletter](#). The first calf was 45 days old and was found down at 1 pm. The calf had been acting normal the day before. The owner was able to get the calf to stand but it was ataxic and could not walk far before lying down again. The ataxia progressively worsened during the night and it was also kicking at its abdomen. The calf was found dead the next morning. Necropsy gross findings included moderate hepatomegaly, multiple loops of small intestine with bloody, mucoid contents, marked splenomegaly, decreased amounts of forage in the GI tract and no milk curds in the abomasum. Based on the clinical signs and gross findings, the preliminary diagnoses included *Clostridium perfringens* enterotoxemia and lymphosarcoma. Rabies testing was requested because of the clinical sign of ataxia and the FA test at the NC Public Health Lab was positive. The second calf was 90 days old when it was found down. It was treated with Nuflor, but was found dead the next morning. Three other 60-90 days old calves had died on this farm in the past 30 days. One of those calves had been diagnosed with pneumonia via necropsy about 20 days earlier. Necropsy gross findings included moderate splenomegaly and watery ingesta in the small and large intestine. Preliminary diagnoses included diarrhea and lymphosarcoma. Based on gross findings, the very short duration of the non-specific clinical sign of recumbency and the recent deaths confirmed to be or suggestive of pneumonia rabies testing was not requested initially. However, since pneumonia was not found as expected, the brain was examined histologically and the pathognomonic lesions of rabies were found. Frozen brain was sent to the NC State Laboratory of Public Health and rabies was confirmed by fluorescent antibody testing. The enlarged spleen in both of these calves and the 2 year old bull previously reported is an interesting finding with an unknown etiology that could potentially lead the veterinarian in the wrong direction. These two cases highlight 3 points: 1. the clinical signs of rabies in livestock can be vague, non-specific, and of short duration, 2. if necropsy exam does not reveal an obvious cause of death then the brain should be collected and examined, and 3. all dead livestock should be handled with care to minimize human exposure to the rabies virus.

Drs. Jennifer Haugland and Stacey Robinson

A 3 month old **beef calf** was presented to the Rose Hill Laboratory after showing signs of dyspnea, bloat, and ptyalism. Severe abomasitis with edema of the abomasal wall and a fibrinopurulent peritonitis were observed in gross necropsy (figure 1). Histologic examination of the abomasal wall demonstrated severe necroulcerative abomasitis with bacilli present. A fluorescent antibody test returned positive for *Clostridium septicum*. Braxy is an acute infectious disease characterized by inflammation of the abomasal wall, toxemia and a high mortality rate. *Clostridium septicum*, a soil-born organism, is generally regarded as the causative bacterium. The disease occurs in midwinter with heavy frosts and is typically only observed in weaner and yearling animals. In this case, it caused a primary abomasitis, from ingestion of frozen grass or other feed permitting invasion by *C. septicum* resulting in a fatal toxemia. Commercial vaccines are available to prevent or reduce the severity of disease.

Dr. Carlton Rouse

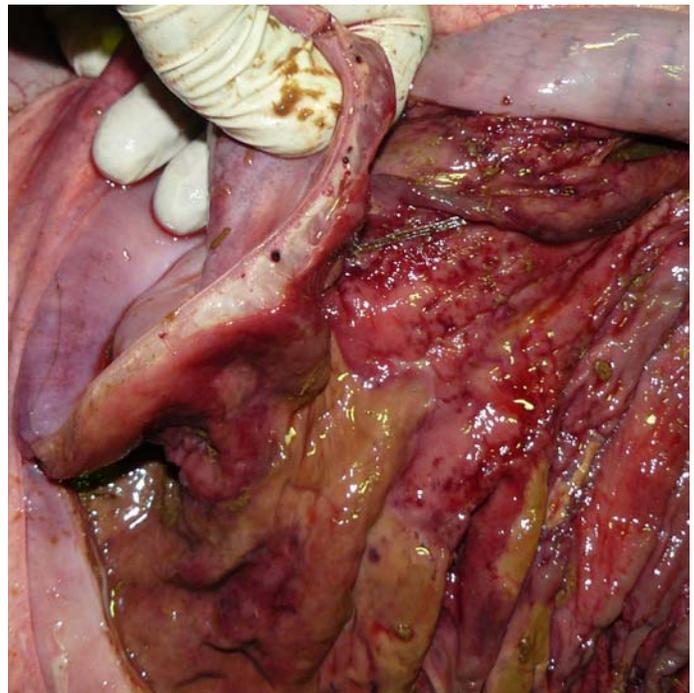


Figure 1: Note the expansion of the abomasal wall and the hemorrhagic appearance of the mucosa

LIVESTOCK, CONTINUED

Small Ruminant

A four month old male **pygmy goat** was presented with a 24 hour history of decreased appetite, depression, weakness, and ataxia. While receiving veterinary attention, the goat became acutely cyanotic, regurgitated, and died. On gross necropsy examination the abdomen contained approximately 500 mL of clear red tinged fluid with a slight musky odor. The urinary bladder and ureters were severely distended but intact. The kidneys were pale and enlarged (measuring approximately 10 cm x 7 cm). The bladder contained many small white stones and on dissection there was severe bruising of the urethra just caudal to the sigmoid flexure of the penis. Male sheep and goats receiving a high grain diet are prone to developing **urinary calculi** as a result of imbalanced calcium to phosphorus ratio from the diet. The urethra narrows at the sigmoid flexure of the penis preventing these calculi from passing and often leading to complete obstruction of the urethra. As urine accumulates the bladder often ruptures, however in this case the bladder remained intact.

Dr. Tim McComb

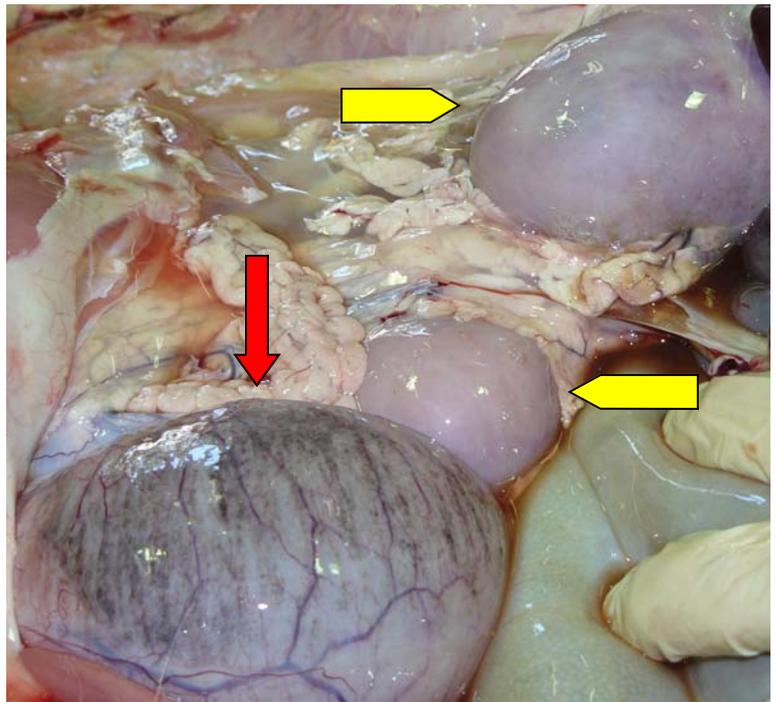


Figure 1: Note the grossly enlarged kidneys (yellow arrows) and the distended urinary bladder (red arrow)

Swine

Two 18 week old mixed breed pigs from the same litter were submitted 4 days apart for necropsy. The litter of pigs had been purchased at 12 weeks of age and were being raised on pasture. All but the first submitted pig were gaining weight and appeared healthy. The first pig was thin and on the day before submission the ears had turned blue and the pig had become weak. The second submitted pig was found dead 4 days later with no prior clinical signs. Both pigs had vegetative proliferations on the mitral valves and the aortic valve of the second pig. In addition, both pigs had severe interlobular pulmonary edema. *Erysipelothrix rhusiopathiae* was isolated from the vegetations of both pigs. *E. rhusiopathiae* is the etiologic agent of erysipelas, which is commonly known as diamond skin disease. This disease is commonly prevented in commercial pigs by vaccination. This bacterium causes septicemia with inflammation and necrosis possible in multiple organs. The bacteria that most commonly cause vegetative valvular endocarditis are *Streptococcus* spp. and *Erysipelothrix* spp. However, vegetative proliferation on the heart valves is the least common lesion seen in erysipelas and it is unusual to see this lesion in two littermates.

Drs. Jennifer Haugland and Stacey Robinson

Equine

An owner recently purchased several horses including 4 **pregnant mares**. All four mares aborted during the same week approximately 2 weeks after their arrival on the new farm. All four mares were within the last 9 days to 5 weeks of gestation. The animals were malnourished at the time of purchase and had only received one rhinopneumonitis (EHV-1) vaccine when they arrived at the new farm. These new horses were intermingled with the owner's other horses which were current on vaccinations. Two of these aborted fetuses were submitted for necropsy at the Rollins Diagnostic Laboratory. On gross necropsy examination fetus 1 had multifocal ecchymotic hemorrhages in the endocardium of the ventricles which extended into the myocardium. Petechial hemorrhages were in the capsular surface of the spleen and kidneys. The lungs were mottled pink and red on cut surface and were mildly rubbery in consistency. A large hematoma was identified in the liver with multifocal petechiae on the capsular surface of the liver. Fetus 2 had a pale tan focus in the epicardium of the left ventricle. The lungs were tan/purple on cut surface and were diffusely atelectic. Occasional 3mm tan foci were in the parenchyma of the liver. Occasional petechiae were in the capsular surface of the spleen. Histopathological findings included bronchiolitis and necrotizing interstitial pneumonia with intranuclear inclusion bodies and necrotizing, multifocal, random hepatitis with intranuclear in-

LIVESTOCK, CONTINUED

clusion bodies in both fetuses. Pooled tissues from fetus 1 and pooled tissues from fetus 2 were both positive for Equine Rhinopneumonitis virus by virus isolation and Fluorescent antibody tissue section technique (FATST). The diagnosis was **Equine rhinopneumonitis virus (EHV-1)** infection. Abortions that occur as a result of this virus usually occur after 7 months gestation. Respiratory signs may be subclinical in infected mares and they may abort without any clinical signs. Fetuses that are close to term may be alive at birth but die shortly thereafter.

1. Smith, Bradford P. Large Animal Internal Medicine. St. Louis, 2002, Mosby.

Dr. Stacey Robinson

Camelid

A 4 year old male **llama** was presented to the laboratory for post mortem examination. The presented history stated the llama was found dead as if it died in its sleep. The animal was let out into a pasture next to a female llama. On post mortem examination, the stomach contained a large volume of watery ingesta. There were over 40 mostly whole Mountain Laurel leaves, often still attached to the stems, among the stomach content. Ingesta was noted to be tightly packed in the sinus cavities and the pharynx. Signs of **mountain laurel toxicity** usually occur within six hours after consumption. Symptoms include incoordination, excessive salivation, vomiting, bloat, weakness, muscular spasms, coma and death. The animals are often found down, unable to stand, with their head weaving from side to side. In more severe cases breathing becomes difficult along with slowing of the heartbeat.

Dr. David Drum

POULTRY

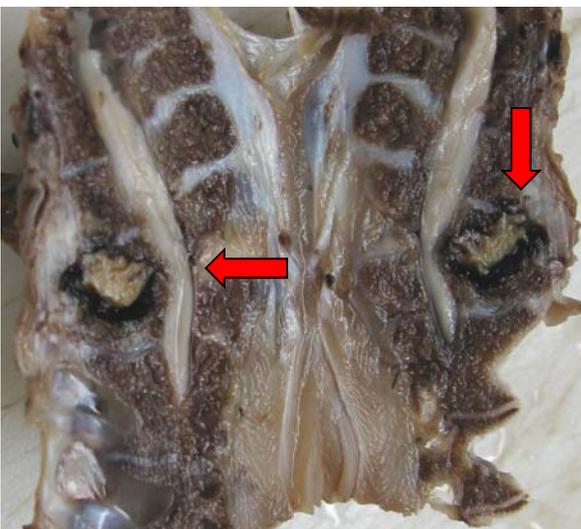


Figure 1: Sagittal section of a T4 vertebral lesion (arrows).

Enterococcus cecorum has been isolated from several recent cases of **vertebral osteomyelitis** (spondylitis) in the area of the articulating (movable) thoracic vertebra in broiler chicken flocks. Male birds 49 and 50 days of age were examined. The affected birds were presented sitting on their hocks with both legs paralyzed and extended forward, resembling spondylolisthesis (kinky back), a noninfectious condition also seen in broiler chickens. Swelling at the level of the articulating thoracic vertebra was visible from the ventral surface of the spine. Longitudinal incisions through the vertebral swellings revealed areas of pale yellow, necrotic material in the body of the articulating vertebra. Vertebral osteomyelitis (spondylitis), necrotizing, subacute, focal and extensive, severe, with sequestered necrotic tissue and intralésional bacterial colonies, and with segmental compression of the spinal cord that caused severe demyelination and axonal loss were found on histopathology. *Enterococcus cecorum* was consistently isolated from the lesions. In an article published in World Poultry in 2007, Dr. Tasheen Aziz, avian pathologist at the Rollins Laboratory, has reported for the first time this lesion in broiler breeders.

Dr. Reg Ridenhour



Steve Troxler
Commissioner

North Carolina Department of Agriculture
and Consumer Services
Veterinary Division
Diagnostic Laboratory System

David T. Marshall, DVM
State Veterinarian

Karen W. Post, DVM, MS
Director of Diagnostic
Laboratory System

March 9, 2009

Dear NCVDL Client,

As you are aware, North Carolina is experiencing a severe budget crisis. All state agencies are facing budget cuts and we have been asked to revert 9% of our appropriated budget by June 30, 2009. Although nothing has been finalized at this point, one of the options we have submitted to the Office of State Budget and Management as part of our required reductions is to increase existing fees and implement new fees. These are in an enclosed updated fee schedule. The most significant change will be the imposition of fees on specimens from food production animal species. If adopted by the General Assembly, these fee changes would become effective on July 1, 2009.

Although we have tried to keep fee increases to a minimum in the past, with our last changes occurring in 2005, these economic times are difficult and the fee changes were only proposed as a last resort. It must be noted, however, that in a comparison of our increased/ new fees with those of other veterinary diagnostic laboratories, NCVDL fees are lower. Internally, we have reduced expenditures, eliminated travel, and eliminated some positions. Reductions for the next fiscal year will at least equal and most likely exceed the 9% reduction target. The only other viable option would be to eliminate certain services, as increased user fees and/or decreased services are the only ways for laboratories to continue to function.

We wanted to share this information with our clients even though these fees are currently only in the proposal stage. A final decision by the General Assembly could be made as early as April or as late as August, 2009. You will be informed of any further developments.

The loyalty of our clients is highly appreciated and we hope the fee changes will not be a burden to you. We would appreciate it if our clients would support us by letting their local legislators know about the importance of our diagnostic laboratories in protecting animal and public health through disease diagnostics and surveillance.

Sincerely,

Karen W. Post DVM, MS

Karen W. Post, DVM, MS, D-ACVM
Director of Laboratories

CC: Dr. David Marshall
Mr. Howard Isley

NCDA & CS/ Veterinary Division
Statutory Authority - GS106-6.1

Fee Title/Description	Current Fee Rate	New Fee Rate
435400014 Necropsy Fee		
Necropsy Fee (Companion Animal: small/large)	\$30.00	\$50/\$75
Livestock Necropsy Fee		\$25.00
Poultry Necropsy Fee (up to 8 birds; \$5.00 per additional bird)		\$15.00
Organ Pluck (Tissue Handling Fee)	\$5.00	no change
435400044 Necropsy Disposal Fee		
Necropsy Disposal Fee - Based on weight of animal at necropsy (1-100 lbs./101-500 lbs./over 500 lbs.)	\$5/ \$15/ \$30	\$15/ \$25/ \$50
435400045 Cytology Fee		
**Cytology Fee - All Species	\$10.00	\$15.00
435400015 Histopathology Fee		
**Surgical Biopsies/Fixed Tissue Mail-Ins - All Species (1-5 slides; \$5.00 for each additional slide)	\$30.00	\$35.00
435400043 Microbiology Fee		
Parasitology (fecal flotation or direct exam)		\$5.00
Fungal Culture		\$10.00
Livestock/Poultry Culture/susceptibility		\$10.00
NPIP Salmonella Culture		\$6.00
Ruminant Mastitis Culture		\$5.00
Bulk Tank Milk Sample Culture		\$15.00
Companion Animal Culture/susceptibility	\$10.00	no change
CEM Cultures	\$25.00	no change
434160 Veterinary Services Fee		
Salmonella arizonae Tube test	\$0.10	no change
Mycoplasma gallisepticum ELISA test	\$0.15	\$0.50
Mycoplasma gallisepticum HI test	\$1.00	no change
Mycoplasma gallisepticum Plate test	\$0.15	\$0.35
Mycoplasma meleagridis ELISA test	\$0.50	no change
Mycoplasma meleagridis HI test	\$1.00	no change
Mycoplasma meleagridis Plate test	\$0.50	no change
Mycoplasma synoviae ELISA test	\$0.50	no change
Mycoplasma synoviae HI test	\$1.00	no change
Mycoplasma synoviae Plate test	\$0.50	no change
Salmonella pullorum-typhoid Tube test	\$0.10	\$0.15
Salmonella pullorum-typhoid Plate test	\$0.10	no change
Salmonella enteritidis Tube test	\$0.08	no change
Salmonella typhimurium Tube test	\$0.08	no change
Equine Infectious Anemia test	\$6.00	no change
Serum Separation	\$1.00	no change
Vacutainer Tubes	\$0.04	no change
Bordetella avium ELISA test		\$1.00
Anaplasmosis cELISA test		\$2.00
Leptospirosis MAT (includes 6 or 7 serovars)		\$7.00
Misc. Poultry Serology (e.g. IBD, APV, AE, REO, CAV)		\$1.25
Misc. Ruminant Serology (e.g. BLV, BTV, CAE, Johne's, Neospora caninum)		\$1.50
Porcine ELISA (e.g. Mycoplasma hyopneumoniae)		\$1.50

**Previously, microbiology, histopathology and cytology fees were assessed on companion animal species only

DEPARTMENTAL NEWS

NCVDLS

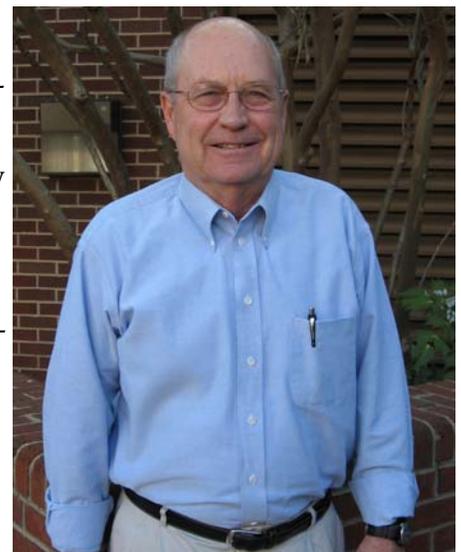
Several NCVDLs employees were honored last month with Service Awards:

Denise Blanchard – Administration/Rollins Laboratory – 5 years
Maurice Burnett – Virology/Rollins Laboratory – 5 years
Chad Menard – Molecular/Rollins Laboratory – 5 years
Bill O’Shields – Serology/Rollins Laboratory – 5 years
Elizabeth Ortiz – Bacteriology/Rollins Laboratory - 5 years
Joey Stephenson – Building Maintenance/Rollins Laboratory - 5 years
Kitty Angier – Bacteriology/Rollins Laboratory - 10 years
Sherry Casey – Administration/Monroe Laboratory - 10 years
Susan Gay – Administration/Rollins Laboratory - 10 years
Dr. Peter Moisan – Diagnostic Pathology/Rollins Laboratory - 10 years
Charlotte Dover – Administration/Rollins Laboratory - 15 years
Dr. Reginald Ridenhour – Veterinary Diagnostician/Monroe Laboratory - 15 years
Bing Tang – Molecular/Rollins Laboratory - 15 years
Dell Weaver – Administration/Rollins Laboratory - 15 years
Brenda Howell – Administration/Elkin Laboratory - 20 years
Cindy Nipper – Histopathology/Rollins Laboratory - 20 years
Dr. Darrell Rector – Veterinary Diagnostician/Elkin Laboratory - 20 years
Earlene Allen – Virology/Rollins Laboratory - 25 years
Sharon Greer-Graham – Virology/Rollins Laboratory - 25 years
Deloys Lee – Administration/Rollins Laboratory - 30 years

ROLLINS LABORATORY

Dr. Richard Mock joined the Rollins Laboratory in April as Assistant Director. He holds degrees from Texas A&M University (BS), North Carolina State University (MS) and the University of Illinois (PhD). After completion of his graduate studies, he was a faculty member of the University of Illinois, College of Veterinary Medicine and Veterinary Diagnostic Laboratory. Dr. Mock comes to the Rollins Laboratory after serving as Virology Section Head for 27 years at the Texas Veterinary Medical Diagnostic Laboratory in Amarillo. He is an active member of the American Association of Veterinary Laboratory Diagnosticians (AAVLD) where he served on several committees, including, Serology, Virology and Membership. For that same organization, he is a past member of the Executive Board and presently serves on the Accreditation Committee.

Elizabeth Ortiz has been promoted to Assistant Supervisor in the Bacteriology section. She has been employed in Bacteriology for 5 years.



Welcome Dr. Mock!

DEPARTMENTAL NEWS

ROLLINS LABORATORY

Dr. Mahogany Wade has been accepted into the Anatomic Pathology Program at North Carolina State University College of Veterinary Medicine. The residency program provides 3 years of advanced training in anatomical pathology. She will officially begin in May 2009.

Dr. Jennifer Haugland and her family welcomed baby Erin into the world on May 4th. Erin was an impressive 9 lb, 12 oz! She was 22 inches long and both Mom and baby are doing great!

Tamara Seago and her family welcomed Addyson Ryleigh Seago on February 19, 2009. She was a healthy 6 lbs 11 oz and measured 19 inches long.



Addyson Ryleigh Seago

ROSE HILL LABORATORY

Dr. Tim McComb and his family welcomed baby Elena into the world on February 21st. Elena was a healthy 7 lb, 6oz, and 21 inches long. She continues to be a healthy and happy addition to the family.



ARDEN LABORATORY

Sandra Gorman passed away at her home on May 12, 2009. Sandra was employed as a microbiologist at the Western Animal Disease Diagnostic Laboratory from May 1977 to July 2003. She was a tremendous asset to the Western Animal Disease Diagnostic Laboratory and a wonderful person that will truly be missed.

CE ATTENDANCE

Dr. Peter Moisan attended the 40th Annual Meeting of the American Association of Swine Veterinarians in Dallas, Texas from March 7-10, 2009.

Directory

Rollins Laboratory - 919-733-3986

Director

[Dr. Karen Post](#)

Assistant Director

[Dr. Richard Mock](#)

Veterinary Pathologists

[Dr. Tahseen Abdul-Aziz](#)

[Dr. Peter Moisan](#)

[Dr. Steven Rushton](#)

[Dr. Alison Tucker](#)

Veterinary Diagnosticians

[Dr. Jennifer Haugland](#)

[Dr. Stacy Robinson](#)

[Dr. Mahogany Wade](#)

Veterinary Microbiologists

[Dr. Gene Erickson](#)

[Dr. Karen Post](#)

Laboratory Section Supervisors

[Beverly Wood—Molecular Diagnostics](#)

[Kim Bennett—Virology](#)

[Sandy Murphy—Bacteriology](#)

[Mary Horne—Histopathology](#)

[Jennifer Pruitt—Serology](#)

Quality Assurance Manager

[Ghazala Jawad](#)

Western Laboratory

PO Box 279 Arden, NC 28704

Phone: (828) 684-8188

Fax: (828) 687-3574

Director

[Dr. Richard Oliver](#)

Veterinary Diagnostician

[Dr. David Drum](#)

Northwestern

Laboratory

1689 N Bridge St Elkin, NC

28621

Phone: (336) 526-2499

Fax: (336) 526-2603

Director

[Dr. Darrell Rector](#)

Veterinary Diagnostician

[Dr. Kim Townsend](#)

Griffin Laboratory

PO Box 2183 Monroe, NC 28111

Phone: (704) 289-6448

Fax: (704) 283-9660

Director

[Dr. Kim Hagans](#)

Veterinary Diagnostician

[Dr. Reg Ridenhour](#)

Rose Hill Laboratory

PO Box 37 Rose Hill, NC 28458

Phone: (910) 289-2635

Fax: (910) 289-2070

Director

[Dr. Carlton Rouse](#)

Veterinary Diagnostician

[Dr. Tim McComb](#)

Diagnostic Laboratory Advisory Committee

Mr. Larry Wooten	N.C. Farm Bureau
Dr. Richard Kirkman	Private Veterinary Practitioner
Dr. Gene Erickson	NCDA&CS Veterinary Diagnostic Laboratory System
Dr. Rick Sharpton	Perdue, Inc
Dr. Shannon Jennings	Nash Johnson Farms
Dr. Leslie Wolf	DHHS- State Public Health Laboratory
Dr. Karen Post	NCDA&CS Veterinary Diagnostic Laboratory System
Dr. Eric Gonder	Goldsboro Milling
Dr. David Marshall	NCDA&CS Veterinary Division
Dr. Randy Jones	Livestock Veterinary Services
Dr. Jennifer Haugland	NCDA&CS Veterinary Diagnostic Laboratory System
Dr. Betsy Sigmon	Creature Comforts Animal Hospital

