Wyoming State Veterinary Laboratory Newsletter – July 2008

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MESSAGE FROM THE DIRECTOR

For those of you who were able to attend, I hope you found this summer's WVMA meeting informative, interesting, and/or otherwise enjoyable. It is sometimes difficult for those of us on the diagnostic and research sides of veterinary medicine to put these presentations in the correct perspective for practitioners - a balance between practical practice-oriented information and more discipline-related diagnostic nuts and bolts. I hope we were successful. I would like to thank all those who participated on the program including departmental and College of Agriculture faculty and graduate students. Special thanks to the external speakers: Drs. Stacey Anderson (WDH), Charlie Stoltenow (NDSU), John Lawrence (IDEXX), Clayton Kelling (UN), Walt Cook (WLSB) and Hana van Campen (CSU); District 43 State Representative Dan Zwonitzer, and Rob Orchard (Wyoming livestock producer).

***Personnel Issues**:* People are the most important component of WSVL. Working with you, the veterinary practitioners, our faculty and staff provide a valuable service to Wyoming. People are a recurrent theme for this Newsletter. Mentioned in April was the hire of Jonathan Fox who will round out our cadre of pathologists. Our parasitology candidate has also accepted. Dr. Chaoqun Yao received an MD degree from Tongji University (China) and a PhD in Veterinary Parasitology from the College of Veterinary Medicine, University of Georgia. Drs. Yao and Fox will begin in August.

We are beginning two new searches that will give us a full complement of faculty for the Department of Veterinary Sciences. We have already received several applications for the epidemiology position. We are just getting started with the search to replace virologist Dr. Nicky Bratanich who resigned to return to Argentina last March.

***New Building Addition**:* The WSVL and University of Wyoming are again entering into a partnership with the State to design and hopefully receive funding for a new addition to the current facilities that will include a biosafety level-3 necropsy and laboratories. Needs for a BSL-3 facility, increasing building security, ensuring safety of laboratory testing, and providing additional space for increased efficiency of laboratory operations

are critical. We hope that we would have your continued support in this endeavor.

Best wishes,
Don Montgomery

INTERESTING CASES FROM WSVL AND OTHER TIDBITS

Have You Seen…?

Holstein cattle with signs suggestive of unusually rapid onset brisket disease? If you have, we're interested in getting material. We have had several such cases, all with a similar history. There are Holsteins <1 year of age held at an elevation of >5,000 feet. Affected cattle have a history of rapid clinical onset (1 day – 2 weeks) and a fatal outcome. Clinical signs are distended jugular veins, with or without a pulse, lethargy and recumbency. We have yet to get a whole carcass, but have received fresh whole pluck and samples of liver from affected cattle. There is marked right ventricular wall hypertrophy with dilation of the right ventricle and right atrium. A striking feature is dilation of the pulmonary trunk.

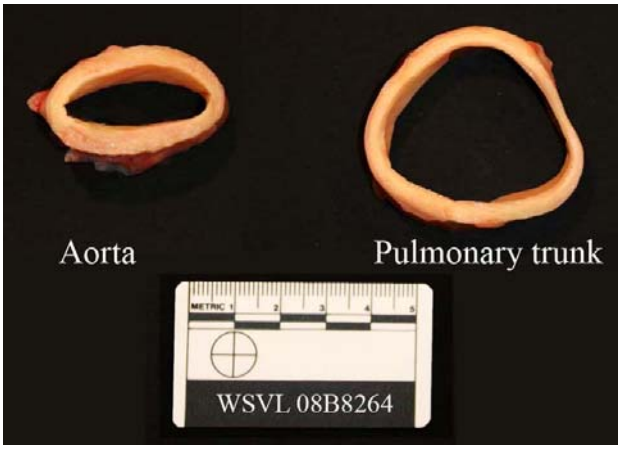

Fig 1. *Markedly dilated pulmonary trunk on right, compared to cross-section of intra-thoracic aorta. This is from a 7-month old Holstein heifer that developed a rapidly progressive (2-day) syndrome of right-sided heart failure.*

Acute respiratory distress in alpacas? Several laboratories around the US have received submissions of carcasses and lung samples from what alpaca owners call "the snots" or "acute respiratory distress syndrome". As far as I know, nothing has been published in the veterinary literature about it. I recently looked at ultrathin sections of alpaca lungs in a search for viral particles, since there are aspects of the syndrome that suggest it is viral in origin.

Most affected alpacas have upper respiratory signs and, as the name suggests, a mucoid or muco-purulent nasal discharge. Cases have been seen in the Pacific Northwest and in Idaho, Montana, Wisconsin, Colorado, California and New England. Most animals recover uneventfully. A small fraction develops a progressive form of the disease, terminating in death. These tend to be pregnant animals, often in late gestation.

If you see a respiratory disease going through a group of alpacas, keep this ill-defined syndrome in mind. It may represent more than one infectious disease, since alpacas are susceptible to both bovine and equine infectious agents, as well their own (camelid) infectious agents. The cause is still not defined. Samples that should be submitted to the WSVL are a full range of fresh and fixed tissues, particularly lung. If this affects an entire herd and there are animals that recover, it will be helpful to have acute and convalescent serum.

Dr. Donal O'Toole
Pathologist, WSVL

Canine Dysautonomia-again. And again…

In 1991 Wyoming was the first state in which canine dysautonomia was recognized clinically in North America. A quick search of cases in the laboratory's database since 2000 indicates we confirmed the disease in dogs in Torrington, Laramie, Cheyenne, Douglas, Sheridan and Buffalo. The latest was in Gillette. It involved a 1-year old rat terrier that whelped recently. She was presented with a history of vomiting for 2 days. She was treated previously for eclampsia, apparently successfully. An abdominal exploratory revealed that the bitch had no intestinal motility and gut contents were milked into her colon. A barium swallow was performed and megaesophagus was diagnosed. The bitch inhaled the barium material and developed foreign body pneumonia. Other findings were weight loss, reduced anal tone, and reduced tear production. Due to the poor prognosis, the bitch was euthanized and her carcass submitted to the WSVL. At necropsy the bitch had a dilated esophagus that was ulcerated due to reflux esophagitis. There was foreign material (barium) in major airways and lungs, and severe inhalation pneumonia. Typical histological changes were found in autonomic ganglia and brain stem. These changes consisted of degeneration progressing to neuronal necrosis and loss.

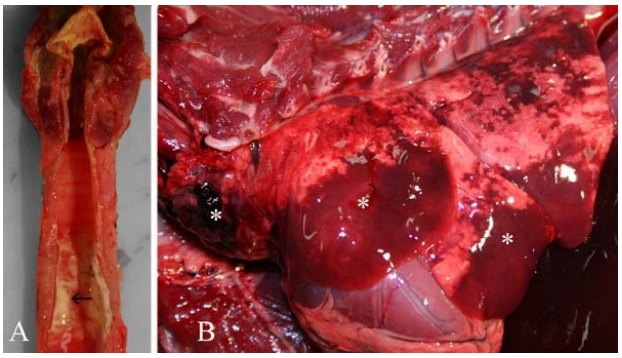

Fig 2. Inhalation pneumonia is a common complication of dysautonomia in dogs. A. Foreign body material in trachea (black arrowhead). B. Consolidation of ventral lobes of the lungs (white stars).

In spite of several large scale epidemiological studies by veterinary research groups at Missouri and Kansas, we don't have a handle on what causes this syndrome or how to avoid it. Dogs from rural environments are overrepresented in case series. Cases occur in every month, with a peak in February -March. Vomiting and regurgitation, as in this case, are the most common clinical signs. Other signs are diarrhea, anorexia, depression, weight loss, dysuria, mydriasis with absent pupillary light reflexes, xerostomia, decreased tear production, decreased or absent anal tone, and elevated nictitating membranes. We get a steady procession of cases from some areas (esp. Torrington) and are not sure if this means particular veterinarians are adept at recognizing the syndrome, or if tends to occur in geographic clusters in the state.

Few dogs recover from dysautonomia. Inhalation pneumonia, as in this dog, is a common fatal complication. It is not uncommon for an exploratory laparotomy to be done in search of foreign bodies, as done here. Harkin et al's article, cited below, has a good review of clinical signs of the disease.

If any of you have noticed any patterns that might provide a clue about the cause of this fatal disease, we'd be most interested in hearing about it

Wise LA, Lappin MR: 1991, A syndrome resembling feline dysautonomia (Key-Gaskell syndrome) in a dog. J Am Vet Med Assoc 198:2103–2106.

Harkin KR, Andrews GA, Nietfeld JC: 2002, Dysautonomia in dogs: 65 cases (1993-2000): 2002, J Am Vet Med Assoc. 220:633-639

Dr. Donal O'Toole
Pathologist, WSVL

Idiopathic Mucosal Colitis – a form of inflammatory bowel disease – in a cat

A 9-year-old domestic shorthair had a history of "chronic diarrhea for the past 5 years. A colonic biopsy was obtained and submitted to WSVL.

Microscopically, there was marked diffuse thinning of the mucosa with attenuation and patchy loss of mucosal glandular architecture. Normal architecture of the lamina propria in areas of glandular loss was replaced by granulation tissue. A leukocytic infiltrate was prominent and varied from neutrophils in the superficial propria to more laminar accumulations of lymphocytes in the more basilar mucosa and submucosa. A hemolytic strain of *E. coli* was cultured.

Idiopathic mucosal colitis is the colonic counterpart of inflammatory bowel disease. Lesions can vary from hyperplastic to atrophic changes in the mucosa and the character of the leukocytic response can differ. Culture of the hemolytic strain of *E. coli* in this case is of undetermined relevance. Culture of this organism may reflect secondary alterations in the normal microflora of the large bowel. The prognosis is guarded based on the chronicity, scarring, and atrophic nature of these changes.

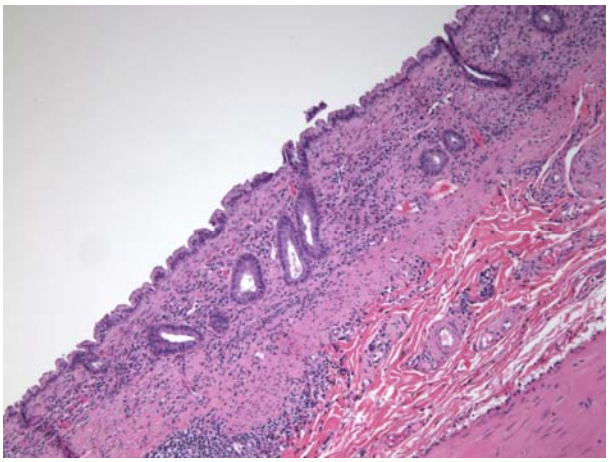

Fig 3. Atrophic colitis in a cat. There is marked thinning of the mucosa associated with patchy loss of mucosal glands, inflammation, and formation of granulation tissue in the lamina propria.

Dr. Ken Mills
Bacteriologist, WSVL
Dr. Don Montgomery
Pathologist, WSVL

Necrotizing Sialometaplasia in Dogs

Necrotizing sialometaplasia is a rare, enigmatic disorder affecting dogs and humans. The basic lesion is diffuse coagulative (ischemic) necrosis with enlargement of usually the submandibular or minor salivary glands in dogs and humans, respectively. In concert with necrosis, florid intraductal squamous metaplasia and hyperplasia can mimic squamous cell or mucoepidermoid carcinoma.

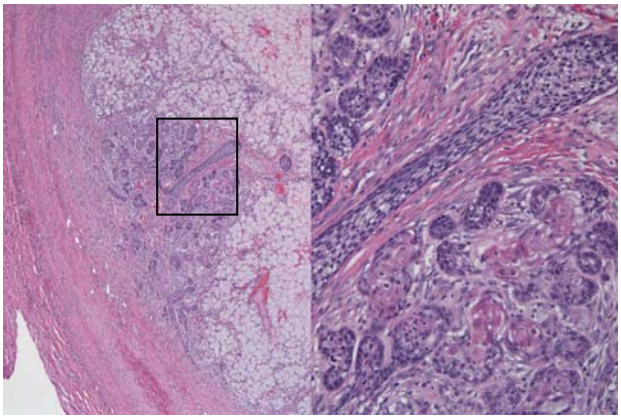

Fig 4. Salivary gland from a dog with necrotizing sialometaplasia. A lower magnification (left) shows the extensive coagulative necrosis (pale areas) with focal intraductal squamous metaplasia and fibrous thickening of the capsule. The higher magnification shows more detail of the intraductal squamous metaplasia mimicking a carcinomatous proliferation.

The cause of this condition is unknown. Most authors favor the view that the lesions are due to trauma with resultant vascular damage leading to edema and necrosis of salivary glandular tissue. Most reported cases in dogs affected small breeds, commonly terriers. Recently, an encapsulated mass was removed from the left side of the jaw of a 1-year-old Pekinese dog. The mass had been present for approximately one month and had not responded to previous antibiotic therapy. A fine needle aspirate was not diagnostic. Microscopic examination revealed a lesion typical of necrotizing sialometaplasia. Apparently, from the cited reference, surgical removal of the affected gland does not result in meaningful clinical improvement. Anticonvulsant medication has, however, been effective.

Brooks DG, et al: Canine necrotizing sialometaplasia: a case report and review of the literature. JAAHA 31:21-25, 1995.

Dr. Don Montgomery
Pathologist, WSVL

Fatal Potomac Horse Fever

A 6-year-old quarter horse gelding was presented to the submitting veterinarian with a somewhat convoluted history. The gelding was a “rodeo” horse that had drunk water from a local river during a stop. Apparently there was a history of trauma and mild diarrhea was noted on the day the illness started. The horse died despite treatment and postmortem samples were submitted to WSVL. The only notable lesions were in the large bowel. Patchy areas of superficial mucosal necrosis with surface-oriented bacterial overgrowth were bordered by a mild infiltrate of neutrophils. In other areas of the lamina propria as well as submucosa leukocytic infiltrates were mainly lymphocytes and plasma cells. Based on negative cultures for *Salmonella,* PCR testing for *Neorickettsia (*formerly *Ehrlichia) risticii,* the cause of Potomac horse fever, was conducted and results were positive (nucleic acid detected).

The lesions of Potomac horse fever are largely non-diagnostic. Organisms can be observed with silver stains in the apical cytoplasm of colonic glandular epithelial cells but results are often difficult to interpret. PCR testing is more definitive. In this case, the cause of the colitis was most likely *Neorickettsia risticii* based on the compatible but non-diagnostic lesions and positive PCR results.

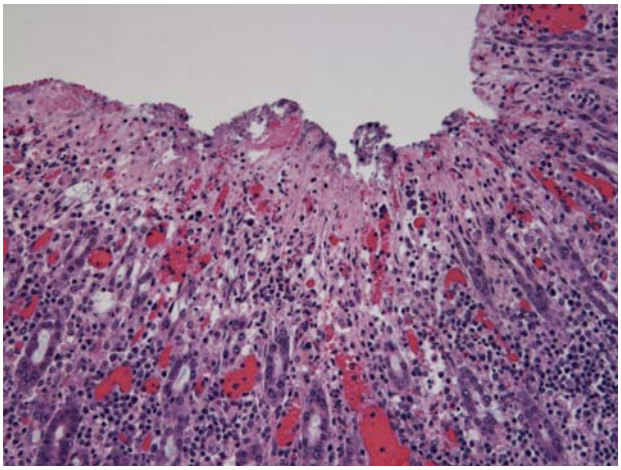

Fig 5. Colon from a horse. Lesions consist of superficial mucosal necrosis bordered by a mild neutrophilic infiltrate. Inflammation in the remainder of the lamina propria consists mainly of lymphocytes and plasma cells. Bacterial overgrowth is present on the necrotic surface suggesting secondary infection.

We have already seen a couple cases of PHF this year and expect to see more during the rest of the summer and early fall. The WSVL offers a PCR for this agent and we prefer to receive both EDTA blood and feces. We do

PCR on both samples but only charge for one assay as a way to insure we aren't missing any cases. The Wikipedia web site below has some decent information on PHF that would be good to review if it has been a while since you have dealt with this disease.
http://en.wikipedia.org/wiki/Potomac_Horse_Fever

Dr. Ken Mills
Bacteriologist, WSVL
Dr. Don Montgomery
Pathologist, WSVL

Dogs, sick livers, and copper

An 8-year-old castrated male Labrador retriever was presented with a complaint of vomiting and anorexia. Physical examination and radiographic studies were unremarkable, but a clinical pathology screen revealed high total bilirubin (2.5 mg/dL; reference: 0.1 – 0.3 mg/dL) and high ALT and SCOT (1117 U/L and 222 U/L respectively. An exploratory laparotomy revealed abnormal caudal lobes of the liver; the whole liver was "discolored" but not yellow. Histologically, there was moderate subacute portal-periportal hepatitis with abundant stainable copper in hepatocytes and macrophages, and a moderate amount of stainable iron in Kupffer cells and macrophages.

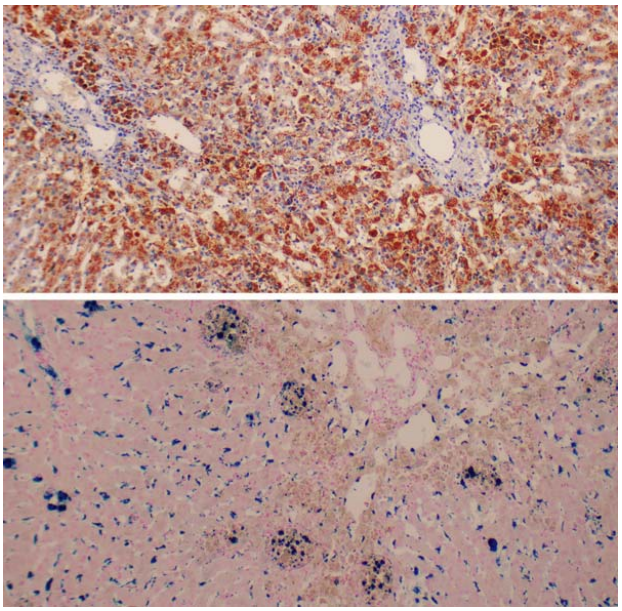

Fig 6. 8-year-old Labrador retriever with hepatitis and accumulation of copper (dark pigment; upper image) and iron (dark pigment; lower image) in liver. Chemical analysis confirmed the presence of excessive copper and iron.

Chemical analysis of the fixed liver confirmed abnormal concentrations of copper (1691 ppm; reference range: 30-100 ppm) and iron (1591 ppm; reference range:100-300).

There is always some concern when analyzing fixed tissues that elevated metal concentrations result from fixative contamination rather than from the tissue itself. In this case, however, the fixative contained negligible copper and iron. The owner was concerned that the dog was poisoned by something in his environment. Acute poisoning of the magnitude suggested by these tissue concentrations would have manifested as severe hemorrhagic gastroenteritis and acute hepatic necrosis, rather than the subacute lesions seen in this dog. Poisoning was therefore unlikely. The tentative diagnosis was subacute portal-periportal hepatitis with associated copper and iron accumulation.

Hepatic copper accumulation can result from increased copper uptake, defective copper metabolism, altered biliary excretion of copper, or secondary to chronic hepatitis. Chronic copper accumulation resulting in chronic hepatitis is a well-described genetic disease in Bedlington terriers and in certain other breeds, notably West Highland White Terriers, Skye Terriers, and Dalmatians. Hepatic copper is typically high (>2,000 ppm) and accumulation typically begins in centrilobular areas.

A group at Tufts University recently reported chronic hepatitis with copper accumulation in the Labrador breed. Of 24 dogs studied, most were older (median of 9.3 years) and presented with reduced appetite, vomiting, lethargy and weight loss. Typical lesions were mild to moderate and subacute to chronic. Most dogs had modest accumulations of copper without an obvious zonal distribution in most. Median survival time was one year, but 11 of 24 dogs died within two months of diagnosis. The role of copper in affected Labradors, as in the chronic hepatitis syndrome of Doberman pinschers, merits additional investigation. Copper can both trigger hepatic disease, and accumulate in livers with preexisting inflammation. According to the Cody veterinarian who treated the Labrador reported here, the dog was treated with antibiotics and appeared to respond well.

Shih JL, Keating JH, Freeman, LM, Webster CRL: 2007, Chronic hepatitis in Labrador retrievers: clinical presentation and prognostic factors. J Vet Med Int Med 21: 33 – 39.

Drs. Merl Raisbeck & Donal O'Toole
Toxicologist, WSVL
Pathologist, WSVL

FROM THE WYOMING DEPARTMENT OF HEALTH

Temporary Unavailability of Human Rabies Pre-exposure Vaccination

In June of 2007, the Sanofi Pasteur production facility in France where the IMOVAX Rabies Vaccine is made was closed due to renovations that were required by FDA and French regulatory agencies. It is expected that the Sanofi Pasteur facility will not be operational until mid-to-late 2009. In May of 2008, it became apparent that Novartis, the maker of RabAvert, the only other rabies vaccine available for humans, would be unable to keep up with the demand for the vaccine. Rationing of the remaining vaccine supplies at both companies for post-exposure use only was instituted.

Recently, the Centers for Disease Control and Prevention and the National Association of State Public Health Veterinarians reached an agreement with both companies whereby the companies agreed to sell the vaccine for pre-exposure use if the request for the vaccine is approved by the requestor's State Health Department.

Here in Wyoming, anyone needing pre-exposure vaccine can submit a request through the State Public Health Veterinarian. The contact information for the State Public Health Veterinarian is as follows:

Karl Musgrave, DVM, MPH
Office: 307-777-5825
Cell:307-421-8591
E-mail: Karl.Musgrave@health.wyo.gov

FROM THE WYOMING LIVESTOCK BOARD

I'm sure most of you have heard that we have a brucellosis infected herd in Daniel Wyoming. The epidemiology is ongoing, and we have not yet definitively identified the source of the infection, but it looks very likely that it will determined to be infected elk. Dr. Combs, AVIC and I have had several in-depth discussions with the affected producer. At this time, the producer is reluctant to depopulate the herd. He has tentatively set dates to test the herd 5 times over the next 12-13 months. Until the herd has been tested and classified negative under the UMR, the herd will remain under quarantine. This is the only herd in Wyoming that has been identified as infected with brucellosis to date. However, if this herd is not depopulated by August 29, Wyoming will likely lose its brucellosis-free status. We have requested an extension of that deadline from APHIS, but the request was denied. We are appealing that decision.

We have made some progress in preparing for bluetongue virus, should it return to Wyoming this summer or fall. At a meeting held in Casper on July 14 it was unanimously agreed that the best way to combat the disease currently is with the use of insecticides to repel the midges that transmit the virus. The two compounds that are labeled for use in sheep within Wyoming include the Y-Tex PYthon ear tags, which contain an insecticide that is released over time. These tags are generally effective for 5 weeks after the tags are applied. The cost per tag is approximately $1.50/sheep. The suggested tag weighs 9.5 grams, and is not the Y-Tex Magnum PYthon tags that are commonly used on cattle. PYthon tags are readily available at most local feed stores.

The other compound is a product from KMG Company, Permectrin CDS. Although this product is labeled as a pour-on it is best to apply as a spray to the underside of sheep to repel midges. The cost of this product is approximately $0.10/sheep and lasts for approximately 2-3 weeks. This product is not currently approved for spraying on sheep- but we are working with the Wyoming Department of Agriculture and EPA to get that approval.

Dr. Walt Cook
Wyoming State Veterinarian