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CONTENTS

News from the Chief Veterinarian	1
Neospora update: the dog as definitive host	2
Molecular Diagnostics (PCR) offered at the Abbotsford A.H.C.	3
Toxicology news: new tests available	4
Fungal (mycotic) abortion in a herd of beef cows:	4
Winter dysentery in cattle: a brief review	5
Allergic or hypersensitivity pneumonitis and copper deficiency in a herd of bison	7
Acute liver necrosis and hemoglobinuria in a Jersey cow	8
Nitrate poisoning as a cause of abortions	9
Nervous coccidiosis: what is it?	10
Pine needle abortion in cattle: a brief review:	10
Severe parasitism in immature Cygnet swans	11
Mycoplasmosis (Mycoplasma gallisepticum) in American robins:	11
Lenticular cataracts in net-pen reared Atlantic Salmon (Salmo salar):	12
Juvenile glomerulopathy in a Kerry blue terrier	13
Eliminating rabies in raccoons	14
On Ferrets	15
The world of ants	16

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From The Chief Veterinarian

R. J. Lewis

At the beginning of a new year, we plan for the future but also reflect upon what has been achieved in the past. There were several noteworthy events from the Animal Health Centre perspective in 1998, a few examples of which follow.

As mentioned in the last issue of this newsletter, during 1998 we had a significant disease problem related to infection of backyard poultry flocks on Vancouver Island. **Salmonella pullorum** infection was diagnosed in late October of 1997 and immediately reported to the Canadian Food Inspection Agency (CFIA) since this is a federally reportable disease. In cooperation with the poultry industry and the Ministry of Agriculture & Food (MAF), the CFIA embarked on the difficult logistical task of first finding all the backyard flocks on Vancouver Island and the major southern Gulf Islands, then testing all these birds, and quarantining entire flocks in which reactors were present. The disease was finally eradicated in late October of 1998; 79,142 birds were tested from 49,279 flocks. The Animal Health Centre necropsied and cultured 523 reactor birds from 256 submissions resulting in 27 positive flocks being depopulated. A great deal of resources, human and monetary, by both levels of government and industry contributed to the success of this eradication effort. Due to our combined efforts, Canada retained its pullorum-free status, thus providing a significant export advantage to our producers.

Fowl pox infection was diagnosed for the first time in commercial poultry in late 1997 and 1998. The infection caused fairly high mortality initially and depressed egg production in layers. Following the first recognition of the disease, the presentation of infected birds changed from sudden death resulting from occlusion of the upper airways ('wet' form) to roughened scabby like lesions on the non-feathered portions of the head ('dry' form); on some occasions, both the 'wet' and 'dry' forms occurred in the same flocks. The infection has spread from the initial area (near the Abbotsford airport) to more outlying sites in the Fraser Valley. Although the first presentation occurred in layers, it is



Continued Page Two...

TOLL FREE NUMBER AND WEB SITE: Please note that the Animal Health Centre has a toll free number: 1-800-661-9903. Keep this in mind, if calling long distance. This Newsletter, and other information from the AHC, may also be found on the Internet at our web site: <http://www/agf.gov.bc.ca/croplive/anhlt/ahc>

From the Chief Veterinarian continued...

now also being diagnosed in broiler breeders. Widespread movement of cages and personnel between poultry farms undoubtedly accounts for much of the spread. Several farms are now vaccinating for the disease.

As recounted in this Newsletter regularly, *Neospora sp.* is an important cause of bovine abortion, particularly in dairy cattle. By 1996, 24% of bovine abortion submissions to the AHC were attributed to *Neospora sp.* infection. By 1997, the percentage had dropped to 19% and, in 1998, it decreased further to 16%. The reason for the decline is unclear. Is it due to fewer submissions from farms in which *Neospora* has been previously diagnosed? Or, perhaps improved feed management and increased culling of positive cows has had an impact on reducing infection?

During 1998 we were also actively involved in several different practical research projects. Each laboratory section has contributed to these efforts, often involving a great deal of extra effort by staff. These projects were completed for producer groups, commodity organizations, private companies, universities, and other ministries. The results of such projects have increased knowledge in disease prevention and control and added to nutritional information. The very sophisticated laboratory services provided by the AHC are fully utilized to improve animal health and welfare, primarily in diagnostic services, as well as special projects. If you think we could be of help in practical research proposals and wish to discuss further, please call me at your convenience.

We continue to offer more polymerase chain reaction (PCR) tests and have also added radioimmunoassay testing of blood samples for Vitamins D and B12. Further information on these new tests follows. If there should be additional tests that you require, let us know. From the Animal Health Centre staff, please accept our best wishes for a very happy and prosperous 1999!

Neospora update: the dog as definitive host



"*Neospora caninum*... is a protozoan that infects domestic and wild canids, ruminants, and horses...Neosporosis is economically important in cattle, and is the most commonly diagnosed cause of bovine abortion in California, the Netherlands, and New Zealand....

...Dogs are a definitive host of *N. caninum*. After consumption of tissue cysts in infected mouse carcasses, three of four dogs shed oocysts resembling those of *T gondii* and *Hammondia sp.* ...

...Neosporosis abortion outbreaks ...may occur in cattle herds fed mixed rations if the feed is contaminated. Farmers should consider taking steps to limit opportunities for dogs to defaecate in feed intended for breeding cattle or goats. In many instances, this could be accomplished by fencing around open-faced buildings used to store feed, uncovered silage piles, and hay."

– from the article by McAllister M., Dubey JP, Lindsay DS, Jolley WR, Wills RA, McGuire AM. *Dogs are definitive hosts of Neospora caninum*. Int J Parasit 1998;28: 1473 – 1478. Ed.'s note: these studies, headed by Dr. McAllister, indicate that the domestic dog is the final, or definitive, host of the protozoal parasite *Neospora caninum*, the organism that has caused so many abortions in British Columbia dairy cattle. As mentioned by McAllister et al., the organism has also been identified in wild canids. Simple, practical preventive measures to avoid exposing cattle to this agent that may be present in dog feces are described in the above condensed version.

In addition to the above comments on bovine *Neospora* protozoal abortion, there is now news that a commercial vaccine will soon be available for distribution to producers from an American manufacturer. Stay tuned. Ed.

Molecular Diagnostics (PCR) Offered at the Abbotsford A.H.C.:

J. Robinson

The following list is the most recent of infectious agents currently available for testing via PCR (polymerase chain reaction test) at the AHC, grouped according to avian/mammalian/fish origin:

Swine:

Actinobacillus pleuropneumoniae, leptospira (multivalent), *Mycoplasma hyopneumoniae*, porcine reproductive and respiratory syndrome (PRRS), swine influenza, swine parvovirus, Transmissible gastroenteritis (TGE), swine dysentery (*Serpulina hyodysenteriae*), swine circovirus (postweaning multisystemic syndrome).

Cattle (bovids, wild and domestic):

BVD virus types 1 and 2, *Clostridium perfringens* toxin, Infectious bovine rhinotracheitis (IBR), leptospira (multivalent), Malignant Catarrhal Fever (both MCF-Wildebeeste assoc. and Sheep assoc. strains), *Mycobacterium bovis* (bovine TB), *Neospora caninum*, *Mycobacterium paratuberculosis* (Johne's disease).

Horses (equids):

Equine herpes 1 and 4, equine influenza, equine viral arteritis (EIA).

Sheep:

Dichelobacter nodosus (sheep footrot), *Mycobacterium paratuberculosis*.

Fur-bearing:

mink parvovirus (Aleutian disease), leptospira (multivalent).

Avian (domestic, wild, exotic):

Avian influenza (AI), avian polyoma virus, infectious bronchitis, infectious laryngotracheitis (ILT), *Chlamydia psittaci*, *Mycoplasma gallisepticum* (MG), *Mycoplasma synoviae* (MS), *Mycoplasma iowae* (MI), *Mycobacterium avium*.

Canids – dogs (domestic and wild):

K9 distemper, K9 parvovirus.

Felids – cats (domestic and wild):

feline parvovirus (feline distemper), herpes virus (Consensus- "Universal")

Salmonids:

piscirickettsia (salmonids), viral hemorrhagic septicemia (VHS) of salmonids, *Renibacterium salmonarum*, Infectious Hematopoietic Necrosis (IHN) of salmonids.

Note:

For a particular species, please look under the appropriate category, e.g., for MCF in bison, refer to cattle. A new addition to our PCR repertoire, listed above, includes identification of **swine circovirus**, and differentiation between circovirus type I (considered non-pathogenic) and type II, the pathogenic strain.

Toxicology news: new tests available

R. Puls



1. **Vitamin D:** we are now able to determine vitamin (vit) D levels as 25-OH-D₃, in serum.

Sample required: 0.5 ml clear, non-hemolyzed serum.

Cost of test: \$15.00/sample

Comment: This test is run by radio-immunoassay and has already identified some interesting problems in poultry flocks. Confinement-housed livestock, animals fed silage rather than sun-cured hay, and those individuals approaching the end of winter in northerly latitudes may be susceptible to vitamin D deficiency.

2. **Vitamin B12 and folates:** these two vitamins are analysed simultaneously using the same equipment as the vitamin D test.

Sample required: 0.5 ml clear non-hemolyzed serum.

Cost of test: \$5.00/sample.

Comment: little is known about the cobalt status of B.C. livestock. Vitamin B12 levels are the best measure of cobalt status. Foliates are required for B12 metabolism. Recent reports indicate cattle may encounter marginal B12 deficiencies at parturition.

3. **Ionophore quantification:** we can now quantitate levels of monensin (Rumensin or Coban), salinomycin (Coxistac or Posistac) and narasin (Monteban or Maxiban) in feeds.

Sample required: 100 g of feed (we cannot detect ionophores in animal tissues).

Cost of test: \$20.00/sample

Web site notice: our site has recently been updated. A list of tests and descriptions done in the Toxicology section at the AHC may be found at:

<http://www.agf.gov.bc.ca/croplive/anhlt/ahc/toxlist.htm>

Fungal (mycotic) abortion in a herd of beef cows:

J. Coates

A herd of 75 beef cows aborted in mid- to late gestation. An aborted fetus was submitted for examination, complete with a portion of retained placenta. On gross examination, the placenta showed enlarged, edematous and necrotic cotyledons, with prominent cupping. Random areas of placenta between cotyledons were often thickened or leathery, and there were areas of adventitial placentation.

Adventitial placentation is a compensatory attempt by the fetus to promote new placental villus formation for nutrient exchange, where there has been loss of function or necrosis of normal cotyledonary tissue.

Microscopically, placental tissue was severely inflamed (placentitis), with frequent areas of tissue necrosis and many inflammatory cells. There was also vasculitis (inflammation) of larger placental arterial vessels. Special stains revealed random, septate fungal hyphae throughout the damaged placental tissue. Fungal culture of fetal tissues yielded positive growth for *Aspergillus* sp from the placenta, lung, and stomach contents.

Continued Page Five...

Fungal (mycotic) abortion in a herd of beef cows: (Continued...)

In cattle, fungal (mycotic) abortion arises in herds exposed to mouldy feed. The animals inhale clouds of fungal spore-laden dust thrown into the air as the food source (such as hay) is disturbed or torn open. These inhaled fungal spores later find their way to the pregnant uterus via the cow's bloodstream. Weeks later, abortion occurs.

As the inflammatory reaction progresses within the placenta, adventitial placentation frequently occurs within the intercotyledonary stroma to compensate for increasing loss of functional tissue. Eventually even this effort fails, and abortion results. Occasionally, skin lesions may be seen on the aborted fetus. Microscopically, there may also be evidence of a fungal pneumonia within fetal lung, tissue, and rarely, brain infection.

Mouldy feed or hay should never be fed to pregnant cattle. Invariably, abortions can be anticipated if this practice continues for long. If mouldy portions of hay are limited, they should be removed from the remaining feed supply.

Winter dysentery in cattle: a brief review

J. Coates

Over the last few months, bovine submissions from all areas of the province have been tagged by the practitioners with the clinical term "winter dysentery". Affected herds usually have a history of sudden outbreaks of multiple cases of bloody diarrhea (dysentery) in mature animals. Within three or so days an entire herd may show varying signs of acute diarrhea, with feces often dark brown, dark green, or black (melena). Fecal matter is often streaked with blood or mucus, and has a foul odor. Course of the illness is 1 to 4 days or so. Most animals recover, although in some instances individual animals, severely affected, have died.

Traditionally, bovine coronavirus is associated with these outbreaks of dysentery in adult cattle (although Koch's postulates have yet to be fulfilled), since coronaviruses are often detected in fecal specimens via electron microscopy. An earlier association with *Campylobacter jejuni* has been refuted by subsequent studies (Thomson's Special Veterinary Pathology, 2nd ed., 1995). Evidently, cattle that have had the disease cannot be reinfected for several years due to an acquired immunity.

Practitioners should keep in mind that the spiral colon is the target organ for so-called coronaviral winter dysentery, and sample specimens of spiral colon tissue should always be submitted along with other tissues whenever sending material to the laboratory for examination. The cecum tends to be unaffected. On gross exam, the spiral colon and distal colon are empty or contain minimal fluid, while the mucosa is moist and shiny, and streaked with linear areas of hemorrhage, giving the lesion a "zebra striping" effect. There are no obvious fibrinous deposits on the mucosal deposits. Microscopically, the colon lesions consist of focal crypt epithelial damage and necrosis of cells within the mucosa (Thomson's Special Vet Path, 1995). Other necropsy changes are nonspecific.

Continued Page Six...

Winter dysentery in cattle: a brief review (Continued...)

Recently, a Fraser Valley dairy herd was affected with a similar syndrome, except that many of the animals were passing variable quantities of frank blood, rather than a true dysentery. Fecal flotations for coccidia were negative as were multiple cultures for *Salmonella sp.* and BVD virus. About a hundred mature animals were affected to varying degrees; 2 died. One of these was submitted to the AHC for necropsy.

On gross examination, the most significant finding was frank hemorrhage within the spiral colon; the colonic mucosa was smooth and edematous. Microscopically, there was a colitis with crypt necrosis, a moderate to prominent mucosal inflammatory infiltrate, and prominent dilation and congestion of superficial mucosal vessels. There had been ongoing bleeding from these superficial mucosal capillaries into the colon. In addition, numerous large intranuclear inclusion bodies suggestive of adenovirus were seen within kidney tubule epithelium, although they were not detected within the colonic mucosa per se. On electron microscopy, adenoviral-like particles were observed within gut contents, and in at least one other tissue. Virus tissue culture was negative, although some bovine adenoviruses are notoriously difficult to grow on cell culture..

Samples from several affected animals that recovered revealed no evidence of viral or bacterial pathogens, or parasites. Coccidial oocyst numbers were negative or low on all fecal flotations, and, on the single specimen necropsied, no coccidia were seen microscopically within affected intestinal sections.

In considering similar disease syndromes in other species, practitioners may recall the disease called "hemorrhagic enteritis" of young turkeys, in which the birds bleed to death rapidly from injured mucosal capillaries caused by an avian adenovirus. Other lesions are also present in turkeys with adenoviral hemorrhagic enteritis that indicate a systemic viremia.

Keeping in mind that only one animal was actually necropsied, a bovine adenovirus may have been responsible for a portion of this clinical syndrome of hemorrhagic colitis. Recent reports in the literature document bovine adenovirus as a cause of fatal hemorrhagic colitis in mature cattle, as well as in calves (see reference below). In this case, all remaining animals gradually improved. Adenovirus is seen occasionally at this laboratory as a cause of systemic disease in calves (sometimes associated with non-cpe BVD virus), but has rarely been associated with systemic disease in adult cattle.

When attempting to determine the specific cause of herd outbreaks of so-called winter dysentery, adenoviral colitis should be considered as a possible differential for bovine coronavirus. Other differential diagnoses to consider are *Salmonella typhimurium* or (less likely) *Yersinia pseudotuberculosis* infection; more peripheral possibilities are coccidiosis, or even grain indigestion (albeit not a true dysentery).

(Reference: *Bovine adenovirus type 10 identified in fatal cases of adenovirus-associated enteric disease in cattle by in situ hybridization*. Jour Clin Micro 1996; 1270 - 1274, by JA Smith et al).

Allergic or hypersensitivity pneumonitis and copper deficiency in a herd of bison:



Allergic or hypersensitivity pneumonitis (acute allergic extrinsic alveolitis) was diagnosed in a group of bison, in which several had died. Fresh and fixed tissues were submitted by the veterinary practitioner for study.

Microscopically, all lung samples revealed a diffuse, severe, interstitial pneumonia, with hyaline membrane formation, eosinophilic and lymphomononuclear peribronchial and interstitial infiltrates, occasional obstructive bronchiolitis, early alveolar septal fibrosis, and intra-alveolar giant cell formation.

Bacterial cultures for pathogens were negative. Viral tissue cultures were also negative, as were fluorescent antibody (FA) tests for BRSV, IBR, and PI3 viruses. PCR tests for BVD and malignant catarrhal fever (sheep-associated) viruses were also negative.

The animals had been in a large corralled area for months, with no change in pasture. They were fed grass hay and haylage, some of which was observed to be mouldy.

On the basis of the clinical history, microscopic examinations, and laboratory studies, the respiratory disease in these animals was diagnosed as an allergic or hypersensitivity pneumonitis. Removal of the offending mouldy feed was urgently recommended. In addition, there is always the concern that pregnant animals exposed to mouldy feed may abort at a later date.

In addition to the concern with an allergic respiratory disease caused by mouldy feed, copper levels in the livers from some of the animals were very low, in the range of 3.0 ppm (using levels recorded for cattle as a guide, adequate liver levels in bison range from 25 - 100 ppm). Analysis of the various dry hay and haylage rations indicated a marked elevation of dietary molybdenum (Mo) over copper (Cu), with a ratio of Mo:Cu in the haylage of approximately 4:1. In addition, the livers were also severely deficient in selenium (Se).

Ideally, the Cu:Mo dietary ratio should be greater than 6:1. Dietary Mo of these animals was elevated, averaging 21 ppm per feed sample. High dietary Mo levels interfere with normal absorption of Cu from the diet and contribute to secondarily-induced copper deficiency. In addition, dietary Mo levels greater than 10 ppm. are claimed to be capable of causing toxicity, regardless of Cu levels (Puls R, *Mineral Levels in Animal Health*, 1994).

A review of the existing levels of dietary supplementation with copper and selenium was strongly recommended, together with a review of other trace minerals and vitamins. Aside from any negative influence of dietary Mo on copper utilization within the gastrointestinal tract, and considering the very low Se measured in liver tissue, it is likely that the diet of these animals was very deficient in both these elements.

Later discussions with the veterinary practitioner involved in this case indicated that some of the acute deaths reported after the initial episode may have been directly due to severe copper deficiency ("falling disease"). It is not known, however, if any of these later cases may have had microscopic evidence of an allergic pneumonitis, as observed in the earlier specimens submitted to the AHC.

Acute liver necrosis and hemoglobinuria in a Jersey cow:

J. Coates

A 7 year-old nonpregnant Jersey cow was necropsied after a 2-day illness. The principal clinical observations noted by the veterinary practitioner had been recumbency, passage of blood-stained stools (melena), bloody or dark urine, elevated temperature, and loss of appetite.

The animal was in very good body condition and fat stores were excessive. Although a Channel Island breed, fatty tissues were strongly yellow in appearance suggestive of jaundice. There were extensive paint-brush type hemorrhages seen on all major tissues, most notably the heart muscle, the mesenteries supporting the intestinal loops, and the abdominal lining membrane (peritoneum). A grapefruit-sized area of caseous necrosis was observed in the liver, and the adjacent gall bladder was severely edematous (swollen). Kidneys were a dark color, and a small quantity of brown urine was detected in a contracted bladder. Melena was found throughout the intestinal tract.

Microscopically, the most significant findings were severe necrosis of the liver; the presence of blood-stained granular fluid within kidney tubules suggestive of hemoglobin (hemoglobinuria); and extensive mucosal bleeding throughout the intestine and colon. Within the liver, special stains revealed numerous beaded and filamentous organisms consistent with *Fusobacterium necrophorum*. In addition, large gram positive rods were also seen; these had subterminal spores that produced prominent lateral swelling of the bacillus walls. These organisms were most consistent with *Cl. novyi*, or *Cl. hemolyticum*. *Cl. hemolyticum*, the suspected causative agent in this case, was not detected on culture from fresh liver specimens; however, it is a fastidious organism, and difficult to grow. No other pathogens were detected within the intestine or other tissues on bacterial culture. Viral studies were negative.

On the basis of gross and microscopic findings, and other laboratory studies, death in this case was attributed to bacterial toxemia, suspected of being produced by *Cl hemolyticum*. Bacillary hemoglobinuria, or so-called Red water disease, is an acute toxemic disease affecting primarily cattle, but has also been observed in sheep and rarely in dogs.

Liver injury caused by migration of immature liver flukes often provides a suitable microenvironment for germination of spores of *Cl hemolyticum* with subsequent multiplication and toxin synthesis. In this case, however, liver flukes were not detected. Microscopically, bacteria resembling the opportunistic pathogen *F necrophorum* may also be significant, as this agent will cause local liver injury and necrosis, thus providing a suitable microenvironment for proliferation of *Cl hemolyticum* spores.

Cl hemolyticum is a soil-borne organism that may be found naturally in the gastrointestinal tract of cattle. It can survive for long periods in contaminated soil or in bones from carcasses of infected animals. After ingestion, latent spores ultimately become lodged in the liver. As previously mentioned, such a focus of bacterial germination is most often caused by immature liver fluke infection, less often by high nitrate content of the diet, accidental liver injury or puncture, or localized liver infection with organisms such as *F. necrophorum*. In addition to severe local liver necrosis, the beta toxin produced by the proliferating clostridial bacteria causes widespread hemolysis or disintegration of red blood cells, with consequent extensive tissue hemorrhages as seen in this case, together with the phenomenon of hemoglobinuria ("red water disease").

Continued Page Nine...

Acute liver necrosis and hemoglobinuria in a Jersey cow: (Continued...)

Most attempts at treatment of bacillary hemoglobinuria are ineffective, as the disease is usually well advanced by the time of diagnosis. Some cattle in endemic areas may be carriers of the organism. In areas where the disease occurs, vaccination is recommended. In this writer's experience, most cases diagnosed at the AHC have been in cattle from the Fraser Valley. The animal described in this case was the only cow on the farm, although there were a number of sheep; the practitioner has advised the owner that the sheep are also at some risk from disease caused by this clostridial organism.

The pasture involved in this case is very low and wet, and swampy for parts of the year, thus providing ideal habitat for the ongoing survival of *Cl hemolyticum*.

Nitrate poisoning as a cause of abortions:

Periodically, incidents of bovine abortion seen are suspect for nitrate abortion. Inevitably, following standard examination and studies of aborted fetal tissues including placenta, there is no evidence of an infectious etiology to these abortions. Widespread placental degeneration, characterized by extensive secondary mineralization, may at times be seen throughout the placental trophoblast, stromal, and vascular tissue, in suspected cases of nitrate toxicity. This change is a nonspecific process, however, and not unique to abortions caused by nitrate toxicity.

Vitreous humour (eye fluid) taken from these calves may be positive for nitrates, if the sample is taken quickly after abortion, and if fetal post mortem decomposition has not become too advanced. Practitioners are urged to keep this in mind when investigating abortion outbreaks in cattle. An examination of the feed supply of these animals will often indicate a nitrate source such as green feed or oat haylage, etc. Feed samples suspected of containing high levels of nitrates may be analyzed at the AHC.

Diagnosis of nitrate toxicity is usually made in the absence of other factors, infectious or otherwise, that might be linked to the abortions. A comprehensive examination of all fetal tissues, including brain and placenta, is always warranted in these cases, as other factors or agents may also play a role. Low level nitrates may interfere with normal fetal thyroid activity, predisposing the fetus to thyroid hypofunction. Nitrate abortions may occur as a "storm", following a sudden feed change to the offending dietary ingredient. Retained placentas in aborted cows can be anticipated.

When in doubt about the nitrate levels of feed or water sources, send these in for analysis to the AHC. And remember to aspirate eye fluids from calves where you suspect the possibility of nitrate toxicity, as this can also be rapidly appraised for nitrate levels. Eye fluid that proves positive is a useful diagnostic finding, whereas a negative finding on eye fluid does not necessarily rule out the possibility of nitrate toxicity.

In addition, other signs of nitrate toxicity may be occurring in the cowherd as exposure to feed levels rise. These include acute colic, diarrhea, respiratory distress, and even death, as the levels of hemoglobin conversion to methemoglobin increase. Abortions may thus be one of the earlier signs of a lower level of nitrate toxicity, in which oxygen levels to the developing fetus are significantly reduced.

Nervous coccidiosis: what is it?

Veterinary practitioners occasionally come across clinical cases of bovine coccidiosis, usually in weaned cattle in late fall or early winter, that also display clinical evidence of nervous signs. Affected animals are usually recumbent, dehydrated, and unwilling or unable to stand. Frank blood mixed with thick mucus may be seen in their stools; necropsy examination reveals evidence of colonic coccidiosis.

The precise nature or etiology of the nervous signs seen remains somewhat enigmatic. A labile neurotoxin (LNT) in the serum of calves has been identified and characterized, but its real significance with regards to clinical signs is poorly understood (Isler et al).

No significant pathological lesions have been observed in the brain tissue of mice inoculated with serum from calves with “nervous coccidiosis” (Isler et al). Evidently calves with coccidiosis that are showing nervous signs are often copper deficient (Jubb, Kennedy and Palmer, Path of Domestic Animals, 1993).

- from the article by Catherine Isler et al. *Characteristics of the labile neurotoxin associated with nervous coccidiosis*. Can J Vet Res 1987; 51: 271 - 276.

Pine needle abortion in cattle: a brief review:



Ponderosa pines (*Pinus ponderosa*) are widespread in the southern interior of B.C., and always merit consideration where abortion storms may arise in pregnant cattle.

From a case-history point of view, pine needle abortion implies the animals have had direct access to pine needles, either from trees, fallen branches, or pond water that has become saturated with pine needles from which the aborting substances have been leached. Abortion can apparently follow the consumption of green or dry needles, but ranchers generally think that cows more readily eat needles that have been dried on branches or ingested from fallen trees.

Cattle originating from pine-free areas seem to be more predisposed to pine needle abortion than native cattle. Sudden weather changes, starvation, and changes in feed are other predisposing factors to consider. Sheep may also abort after eating pine needles.

Clinically, aborted cows will frequently have retained placentas, and will progress to become ill with septic metritis; some will die. Abortions usually occur during the last trimester of pregnancy, and can begin as early as 48 hours after ingestion of pine needles. Abortions may continue for 2 weeks after exposure ends. The number of exposed cows aborting evidently varies widely.

Affected cows may have pine needles in their manure. If a necropsy is done on a cow suspected of having eaten pine needles, look for the needles in rumen contents.

Representative aborted fetuses together with portions of placenta should always be submitted to the laboratory for standard necropsy examination, regardless of any suspicions of pine needle abortion, so that infectious agents or other factors can be ruled out, as far as possible. Diagnosis of bovine pine needle abortion is somewhat circumspect, as there are no truly diagnostic lesions on the aborted fetus, either grossly or microscopically. In some instances, calves may be born weak and premature, rather than aborted.

– from *Laboratory Diagnosis of Livestock Abortion* by CA Kirkbride, 3rd ed; and *Experimentally induced pine needle abortion in cattle*; James LF, Cornell Vet 67: 294 - 299.

Severe parasitism in immature Cygnet swans:

Several immature, 3 month-old wild Cygnet swans from a waterfowl park area in the Lower Mainland were submitted for necropsy, after being found dead. Necropsy revealed severe body wasting, with marked loss of body fat stores. The young birds were all similar on post mortem, with areas of severe inflammation and obstructive exudate within the glandular stomach (proventriculus).

Within the severe inflammatory exudate, and extending into the mucosal lining of the stomach, were numerous small roundworms, consistent with *Echinuria uncinata*, a common proventricular parasite of a wide variety of wild waterfowl (Wobeser G, *Diseases of Wild Waterfowl*, 1981). The life cycle of the parasite is indirect, with freshwater crustaceans such as waterfleas (*Daphnia sp*) acting as intermediate hosts. As in the case received at the AHC, the parasites burrow deeply into the proventricular mucosa and submucosa, producing such a severe, nodular-type inflammatory response that actual stomach obstruction may result.

The condition appears to be most severe on heavily utilized shallow ponds. It is observed in waterfowl parks with little or seasonally decreased water flow, in which large populations of the waterflea intermediate hosts develop. With decreased water volume, the stress of crowding adds to increased worm retention in the young birds. The disease has been controlled by maintaining a fast flow of water through the shallow ponds, which reduces the waterflea population.

Mycoplasmosis (*Mycoplasma gallisepticum*) in American robins:

S. Raverty

Six to 7 of 30, 3 week old, American robins (*Turdus migratorius caurinus*) transported from the British Columbia interior to the lower mainland in late July as part of a study to investigate the effects of environmental contamination on immune function, developed stertorous respiration, mucopurulent to serous, nasal discharge and swollen eyes.

A single bird was presented for necropsy. The bird was in poor body condition with bilateral peri-orbital swelling and expansion of the cranial sinuses by abundant, caseonecrotic debris. Similar deposits obliterated the caudal third of the right lung lobe and occluded the entire right, cranial abdominal, air sac.

The cranium was formalin fixed and decalcified, and serial sections of the sinuses were prepared. Microscopic examination disclosed a severe inflammation of the sinuses (sinusitis). Pooled swabs taken from the choanal cleft, as well as conjunctival and infraorbital sinuses, were positive by polymerase chain reaction (PCR) for *Mycoplasma gallisepticum*.

Historically, mycoplasmosis is associated with chronic respiratory disease in chickens and sinusitis in turkeys. However, since 1994, there has been an increased incidence and geographic expansion of avian *Mycoplasma conjunctivitis* (*Mycoplasma gallisepticum*), primarily involving house finches (*Carpodacus mexicanus*). The infection appears to have originated from the mid-Atlantic states and spread southward to Florida, throughout the northeastern seaboard of the United States, and the eastern provinces of Canada. Sporadic cases have been reported in Michigan and more recently, Texas.

Continued Page Twelve...

Mycoplasmosis (*Mycoplasma gallisepticum*) in American robins: (Cont)

Free ranging American goldfinches (*Carduelis tristis*) have been the most commonly afflicted birds although horizontal transmission to blue jays (*Cyanocitta cristata*) and anecdotal reports of infection in northern cardinals and wild Downy woodpeckers have been cited. *M. gallisepticum* has also been isolated from wild ducks and geese.

Isolates from wild species have been genetically homologous (based on PCR) and are distinct to those bacteria affecting farmed poultry. The possible transmission to farmed stocks has not been fully resolved, and there is limited zoonotic potential (i.e., transmission to humans).

Seasonal fluctuations in disease occurrence have been reported and is attributed to juvenile dispersal. In this case, it is difficult to resolve whether the birds were sub-clinical carriers prior to transport, or whether the infection was recruited, post introduction. In either case, physiologic stress associated with transport likely contributed significantly to predisposing the birds to infection, and subsequent development of clinical signs.

This disease is not commonly recognized in song birds in B.C., and should additional birds be identified with signs of peri-orbital swelling (conjunctivitis), please contact the Ministry of Environment, local veterinarians, or the Animal Health Centre.

Lenticular cataracts in net-pen reared Atlantic Salmon (*Salmo salar*):

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Excised eyes were submitted from 10, market size, salt water, net pen reared Atlantic salmon (average weigh 2.45 kg) from a population of 10,000 fish which exhibited an increased incidence of cataracts. The overall lesion prevalence at the time of harvest was between 6-16% and varied according to weight classes with reduced growth performance noted in clinically affected fish. The stock was maintained on a poor quality, proprietary feed ration. Histopathology confirmed the grossly noted lenticular cataracts, which in 4-5 of 10 fish were profound and likely would have contributed significantly to impaired vision.

During the last 10-20 years of development within the aquaculture industry along B.C.'s west coast, there have been 2 major episodes of cataract formation recognized in production fish stocks. The first incident, approximately 15 years ago, involved a zinc deficiency in juvenile fish.

In fish, lenticular cataracts have been attributed to dietary deficiencies of tryptophan, thiamine, riboflavin, zinc, vitamin A and vitamin C. Environmental factors have also been incriminated, such as gas supersaturation, possible cold stress, excessive sunlight exposure or ultraviolet radiation. Parasites {Intraocular trematode metacercariae (*Diplostomum* spp, *Tylodelphys* spp) or metacestodes (*Gilquinia squali*)} have been associated with cataracts in fresh and salt water stocks, respectively. Local extension from endophthalmitis or ulcerative keratitis, as well as the effects of toxins such as thioacetamide, crude oil extracts, and aromatic hydrocarbons, have also been associated with lenticular lesions in a number of natural and experimental fish models.

A Scottish investigator has recently reported an association with cataracts affecting farmed Atlantic salmon in Ireland, Norway and Scotland. In contrast to the European study which involved fish immediately prior to smoltification as well as recently introduced into salt water, the west coast case was approaching market size. In both situations, the pathogenesis of the condition likely involved a complex interaction of nutritional and other factors. Review of feed formulation and trace mineral analysis were recommended.

Juvenile glomerulopathy in a Kerry blue terrier:

S. Raverty

A 6 month-old, male Kerry blue terrier with a history of poor weight gain, vomiting, and generalized lethargy, was presented to a local veterinary practitioner for clinical evaluation. Aside from fair body condition, physical examination proved unremarkable.

Clinical chemistries were compatible with possible renal failure. Urine analysis completed at the Animal Health Centre disclosed markedly elevated protein levels.

Due to the progressive decline of this dog (which was refractory to medical intervention), recognized history of renal disease in the family lineage, and poor prognosis for recovery, the animal was humanely euthanized.

Necropsy examination confirmed reduced muscle mass and revealed moderately enlarged, pale yellow grey kidneys with multiple, white, glistening, linear striations emanating from the corticomedullary junction, extending to varying levels of the overlying cortex. Throughout the gastric mucosa, there was mild congestion and hyperemia. No other lesions were apparent.

The most salient microscopic lesions which were limited to the renal cortex and gastric submucosa follows. Throughout the cortex, there was segmental to diffuse glomerular fibrosis with occasional hypercellular tufts, segmental synechiae, crescent formation and obsolescent glomeruli. Multiple foci of periglomerular and interstitial fibrosis were apparent together with mild, medial hypertrophy of arterioles. In multiple sections of gastric mucosa, scattered within the mid to basilar region of the lamina propria, were small to intermediate size, mineralized deposits.

The age, clinical findings, and post mortem lesions were compatible with a congenital and likely heritable glomerulopathy (severe kidney disease). Another, closely related dog was euthanized at approximately 1.5 years of age, in which histopathology had disclosed a chronic glomerulonephritis (kidney inflammation). Familial glomerular disease with attendant glomerulonephritis has been reported in a number of dog breeds including the doberman, samoyed, rottweiler, greyhound, Bernese mountain dog, and soft-coated wheaten terrier.

The precise pathogenesis of this condition has not been fully resolved; however, the clinician has initiated molecular investigation (retained liver has been forward for PCR screening against select gene segments) to further resolve the nature of transmission.

* * * * *

Edge of Perception:

I'm a pilgrim on the edge,

On the edge of my perception.

We are travelers at the edge,

We are always at the edge of our perception.

- photographer/artist Scott Mutter.

Eliminating rabies in raccoons:

“ Rabies can no longer be considered a local or even a regional issue. With preventive measures now receiving federal and international attention and support, many states are beginning to recognize a common objective. Without rabies control programs in infected areas of Florida and Ohio, raccoon rabies could spread west to the Rocky Mountains, the next natural barrier. Without treatment in Texas to contain and eliminate coyote and gray fox rabies, that strain of rabies could spread through the extended range of coyotes in the United States and Canada....

Canada has also taken an active interest in vaccination programs. Quebec, Ontario and the Canadian Food Inspection Agency have recently negotiated international agreements, all through Cornell University. In May 1997, an emergency agreement... between the province of Quebec and Cornell brought northwestern Vermont into Cornell’s program in order to stop the advance of rabies into Quebec. Since that time, only two rabid raccoons have been reported in the region, with no further spread of infection.”

From Cornell University’s *Diagnostic Bulletin*, summer 1998, vol 2, No. 3.



On the light side: a farmer was milking his cow one day when a fly flew into the cow’s ear. The fly then passed through the cow and came out in its milk. With resignation the farmer remarked, “in one ear and out the udder”.

On Ferrets:



“The ferret is a native of Africa. The animal was first domesticated in the northern part of that continent by the Egyptians, hundreds of years ago. Long after its first domestication it was taken across the Mediterranean Sea and introduced into Europe, from which it has spread to many parts of the civilized world...

There are two varieties - white and dark or brown. The white variety is called Albino or English...the other dark brown variety is known as the Fitch-ferret...

Ferrets belong to the Mustelidae family. Marten, weasel, European polecat... are close members of the same family, while allied (if not more distant relatives) are otter, badger and skunk. Ferrets have short legs, elongated, muscular and lithe bodies; they are blood thirsty, determined of purpose and relentless; hardy and prolific breeders, rather short lived, habits clean...

It has been domesticated and raised in confinement for a great many years. It is a favorite animal among English farmers, where a few are kept and raised on nearly every farm for the purpose of keeping rats away...”

from *Ferret Facts and Fancies*, by A.R.Harding, published 1943 by A.R. Harding Pub. Co., Columbus, Ohio.

“Animals shall not be measured by man. In a world older and more complete than ours, they move finished and complete, gifted with extensions of the senses we have lost or never attained, living by voices we shall never hear. They are not brethren; they are not underlings; they are other nations, caught with ourselves in the net of life and time, fellow prisoners of the splendor and travail of the earth.

- quote attributed to Henry Beston, courtesy of the *Washington Post*

The world of ants:

“The single most important quality of the ant colony is the existence of the worker caste, which comprises females subservient to the needs of their mother, content to surrender their own reproduction in order to raise sisters and brothers. Their instincts cause them not only to give up having offspring on their own but also to risk their lives on behalf of the colony. Just leaving the nest to search for food is to choose danger over safety...

“Everywhere ants are found, their species have struck a bargain with insects that feed on plants. Aphids, scale insects, mealybugs, treehoppers and the butterflies of lycaenid and riodinid butterflies... give sugary secretions to the ants for food. In return they are protected from enemies. The ants go further, sheltering them with walls of carton or soil, and sometimes they even take them into the nest as virtual members of the colony. This symbiosis, called trophobiosis from the Greek for “nourishing life”, has proved one of the most successful in the history of the land ecosystems. It has contributed greatly to the dominance of both the ants and their wards...

“During their hundred million year history, the ants have pressed to startling extremes of adaptation. Some of the most specialized forms are virtually beyond imagination...

“Bert Holldobler, in the course of research on the predatory ants of the subfamily Ponerinae, began a detailed study of *Odontomachus bauri*... and became fascinated by the blinding speed and force of the jaw closure. So hard is the blow when the tips of the mandibles strike a hard surface that the ant flips itself backward through the air... the movement of jaws is not merely fast: it is the fastest of any anatomical structure ever recorded in the animal kingdom!...

“...If all of humanity were to disappear, the remainder of life would spring back and flourish. The mass extinctions now underway would cease, the damaged ecosystems heal and expand outward. If all the ants somehow disappeared, the effect would be exactly the opposite, and catastrophic. Species extinctions would increase even more over the present rate, and the land ecosystems would shrivel more rapidly as the considerable services provided by these insects were pulled away.

“...Humanity will in fact live on, as will the ants. But humankind’s actions are impoverishing the earth, we are obliterating vast numbers of species and rendering the biosphere a far less beautiful and interesting place for human occupancy. The damage can be fully repaired by evolution only after millions of years, and only then if we let the ecosystems grow back. Meanwhile let us not despise the lowly ants, but honor them. For a while longer at least, they will help to hold the world in balance to our liking, and they will serve as a reminder of what a beautiful place it was when we first arrived.”

– from the book entitled *Journey to the Ants*, by Bert Holldobler and Edward O Wilson, 1994; Belknap Press of Harvard University Press, Cambridge, Massachusetts; and London, England.

