Animal Health Centre NEWSLETTER

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Editor:

J.W. Coates

Contributors to this edition of Diagnostic Diary are:

V. Bowes, A. Britton, J. Coates, R. Lewis, D. McIntosh, S. Raverty

From the Director

R. J. Lewis

The Animal Health Branch (AHB) has just completed its business plan for 2000/2001. Although frustrating and time-consuming, it is a rewarding process to review and recognize our capabilities and challenges. The six main goals of the AHB are to:

1. Protect the British Columbia livestock industry from disease outbreaks.
2. Recognize and be able to identify new pathogens of animals and prevent them from becoming significant concerns in British Columbia.
3. Prevent transmission of disease between animals and from animals to man.
4. Ensure safe animal products for British Columbia consumers.
5. Assist veterinarians and producers with the diagnosis of animal disease.
6. Provide support, through agreement with Fisheries, for a fish health and reporting program.

We are fortunate in having a highly qualified staff, good resources, and excellent support. The AHB staff is committed to research projects, on-farm investigations, seminars, diagnostic interventions, and a wide variety of other activities to achieve these goals.

Over the last month, we have been actively involved with the Ministry of Environment, Lands, and Parks (MELP) in investigating the cause of death of a large number of California Big Horn Sheep in the southern Okanagan. One of the major herds in that area has decreased from approximately 250 animals to less than 70 over the course of a few months. Rigorous field investigation by MELP biologists has resulted in several submissions to the AHC for diagnostic testing. Undoubtedly, a wide variety of factors have combined to result in this large scale loss of animals but the most important would appear to be infectious

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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/anhlth/ahc

From the Director *(Cont'd)*

disease resulting in pneumonia. Several different agents have been isolated and we will continue to work with MELP biologists to further identify the various factors and try to control and prevent further losses over the next several years.

At the end of March, Mr. Jay Kim, a long-serving laboratory scientist in the Virology section of the AHC, retired. Jay has been instrumental in assisting with the implementation of PCR testing as well as in the development and improvement of virology testing procedures over the last 25+ years. He will be very hard to replace but we wish him all the best in his retirement. Dr. Jeremy Greenfield, Head of Microbiology, will be retiring at the end of June. Jeremy is the longest-serving staff member in the Animal Health Branch, having just completed 35 years of service. It is difficult to imagine the AHC without Jeremy and, although he keeps saying he is going to leave, none of us is yet convinced! His very valuable experience and encyclopedic knowledge of all things microbiological cannot be replaced, so he has set us a very difficult task indeed.

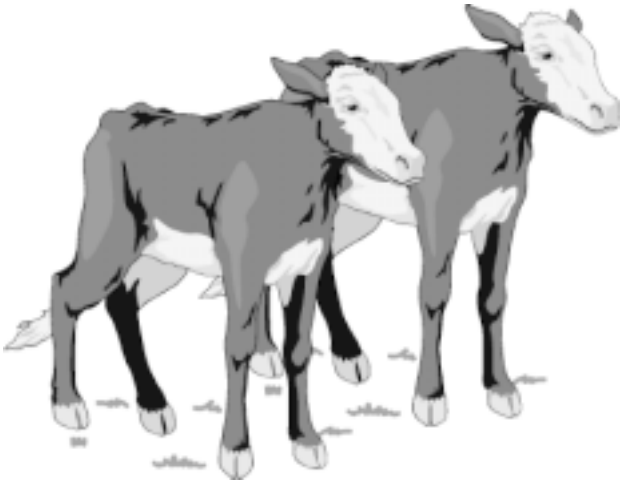
We look forward to receiving good comments and advice from our AHC Advisory Council meeting in late April. As always, we encourage our readers with concerns or questions to contact us at their convenience.

RJL.

Bloat in hand-fed calves:

D W McIntosh

Each year we see calves that die from abomasal bloat. Recently, two separate dairy farms experienced this problem with the loss of a few calves. Abomasal bloat occurs in hand-fed calves on milk replacer. It is seen most frequently when calves ingest large amounts of warm milk replacer at infrequent intervals. This sudden over-filling of the abomasum allows gas-producing bacteria to proliferate with the subsequent release of large amounts of gas that cannot escape fast enough from the abomasum. The abomasum becomes distended within an hour after milk ingestion and death, due to asphyxiation and acute heart failure, occurs within a few minutes.



Monitoring the temperature of the milk replacer and insuring calves are fed at regular intervals will help to prevent the occurrence of this condition.

"He who has health has hope,
And he who has hope, has everything."

Anon

Congenital epitheliogenesis imperfecta in Simmental and Aberdeen Angus, Hereford-cross neonates:

– S Raverty, D McIntosh and J Coates, AHC

Over the course of the last 7 years, congenital anomalies have accounted for 0.2 to 0.02% of the annual bovine diagnoses at the Animal Health Centre (AHC); most common anomalies are cleft palate, limb deformities, umbilical hernia, and various cardiovascular (heart) malformations.

A 3 day-old purebred Simmental calf from a herd of 200 cows and 5 bulls, was presented with a history of normal birth but with a lack of skin over the distal extremities of all four limbs. This was the second calf to present with similar signs in the herd within the last 3 months. Over the course of the last 23 years of farming, there had been no other significant diseases in the herd. These neonates were the first recognized cases of an apparent congenital malformation. There had been no recent introduction of livestock into the herd.

The calf presented in good flesh. Around the circumference of the mid-forelimbs and the hocks of the hindlimbs, there was a discrete, albeit irregular, demarcation between normal and abnormal areas. Distally the limbs featured complete absence of epidermis (skin) although there were occasional islands of skin with more coarse hair, that contained enlarged follicles. Throughout the affected areas, subcutaneous blood vessels were prominent.

In addition, there was focally extensive erosion of the tongue. The upper incisors were crowded and medially rotated. The entire length of the ears were folded over one another, and the openings of the ears were obscured. There was moderate shortening of the lower jaw (brachygnathia inferior).

Microscopic examination of the animal's skin, plenum and tongue disclosed similar processes, with extensive ulceration and inflammation.The defects were overlaid by a thick margin of degenerate inflammatory infiltrate, together with variable amounts of dense, eosinophilic necrotic debris and scattered, superficial accumulations of extracellular, bacterial cocci and rod bacilli. There were no other significant lesions. Aerobic culture of the skin yielded light to moderate growth of *Staphylococcus aureus*. Trace mineral analysis proved within normal limits, and serology for BVD was negative.

Within 1 month of this original submission, tissues from an Aberdeen Angus x Hereford neonate, necropsied by a veterinary clinician in another region of British Columbia, were presented with similar lesions involving the distal extremities.

Based on the gross and microscopic lesions in both animals, a diagnosis was made of **congenital bovine epitheliogenesis imperfecta**. This condition is a rare, inherited (autosomal recessive) disease reported in a number of cattle breeds including Holstein, Jersey, Shorthorn, Dutch Black Pied, Swedish Red Pied, German Yellow Pied, Hereford, Ayrshire, Angus, Jersey, Brown Swiss, and Sahiwal. In addition to cattle, this condition has been diagnosed in horses, dogs, cats, pigs and sheep, although in these species its heritable nature has not been fully established.

In the first case, samples of pericardium were harvested at the time of post mortem and forwarded to Dr Schmutz, Department of Animal Sciences, University of Saskatchewan for attempted *in vitro* fibroblast isolation and culture. Unfortunately, appropriate samples could not be grown due to bacterial contamination. However, DNA from this animal was extracted and is archived at the University.

Currently, there is no genetic test available to determine the carrier status of animals. Preventive measures are limited to selective replacement of breeding stock, including bulls and females.

Toxoplasma gondii abortion in sheep:

–A Britton (AHC), J Coates (AHC), A Runnells (Benson View Vet Hosp, Nanaimo, BC)

Nine ewes in an 18-head flock of 50% Dorpor/Kataden sheep aborted over a 4 week period, commencing at 15 weeks of gestation. The ewes were all in good condition. Three fetuses were presented for necropsy. Placental tissue from the specimens exhibited tan/red discolouration of the buttons, or cotyledons. One fetus exhibited increased length of the lower jaw (mandibular prognathism); the other two fetuses were normal in appearance.

Microscopically, all had inflammation of the placenta, in which *Toxoplasma* sp. organisms were observed. The fetuses demonstrated severe, multifocal, inflammation of the brain tissue, together with pneumonia and inflammation of the heart. Laboratory polymerase chain reaction (PCR) analysis of the placenta was positive for ***Toxoplasma gondii***. Blood serum was taken from five ewes following abortion, and all had elevated *Toxoplasma* antibody titers.

Toxoplasma gondii is a well documented cause of abortion in many species, including sheep and goats, as well as humans. Cats, the definitive host of *T. gondii*, are the main source of infection for domestic animals via fecal contamination of feed and pasture. Naïve cats contract *Toxoplasma sp* when they eat infected intermediate hosts, such as rodents. The organisms complete their life cycle in the feline intestine and cats will shed oocysts in their feces for approximately 2 weeks. Once immunity develops, shedding stops. Oocysts are extremely resilient and can survive for more than a year in the environment. In sheep, exposure to *T. gondii* usually does not result in clinical disease. However, if a previously uninfected ewe is pregnant at the time of exposure, abortion can occur. Dams infected late in pregnancy may deliver stillborn or weak lambs.

World-wide serological studies demonstrate that 20% of sheep and one-third of the human population are sero-positve for *T gondii*. While fecal contamination of feed with toxoplasma oocytes is thought to be the main source of infection for domestic animals, it should be noted that humans can also become infected by eating poorly cooked meat containing encysted toxoplasma bradyzoites (the same life stage of *T. gondii* that infects the cat). Since toxoplasma bradyzoites may be present in placenta and fetal tissues, all tissues and fluids from aborting ewes should be handled and removed with caution to prevent human exposure, especially in pregnant women. Although transmission between sheep as a result of abortion is generally believed not to occur, similar precautionary measures would be advisable for sheep as well.

"What I wish here to emphasize is that a correct knowledge of the disease of man caused by worms, and all that is connected with them, is the more difficult to attain the more parasites of animals are ignored".

– Arthur Loos, 1911; from the book *Parasites in Human Tissues*, by TC Oriheland L Ash; published by American Society of Clinical Pathologists Press, 1995.

Coxiella abortion in a dairy goat:

J Coates

A near-term aborted fetus and placenta were received for necropsy. The most remarkable observation on gross post mortem was the accumulation of copious quantities of a thick, creamy-white to carmel-colored placental exudate, combined with severe placental thickening. White felt-like inflammatory deposits were present within the placental membranes. Microscopically, numerous microcolonies of basophilic organisms consistent with the rickettsial organism ***Coxiella burnetii*** were observed within placental chorionic cells. The diagnosis was confirmed by the PCR technique.

Coxiella burnetii abortion in sheep and goats (it may also occur in cattle) tends to occur in late gestation, and weak lambs and kids may be born during an outbreak. The aborted fetus may be well preserved or decomposed (Jubb, Kennedy and Palmer, *Pathology of Domestic Animals*, vol 3, 1993).

For the field practitioner or producer, the most significant observation when faced with coxiella abortions in goats or other animals is the markedly thickened and leathery appearance of the placenta between cotyledons, and the frequent presence, as mentioned earlier, of copious quantities of off-white or carmel-brown placental exudate.

C burnetii abortion in goats has been reported previously in *Diagnostic Diary* (July/97), when another case was diagnosed in a BC goatherd. The actual incidence of this organism in domestic animals in BC, via serological or other tests, is not known. The agent is zoonotic; that is, the disease may be transmitted from animals to humans. Care should always be taken by owners and by veterinarians when handling aborted fetuses and placental tissues from aborted sheep, and those of goats, in particular. Other major causes of abortions in small ruminants in BC that are also zoonotic are toxoplasma and chlamydia.

In humans, infection with *C burnetii* is called Q (Query) fever, usually an influenza or flu-like condition that can result in inflammation of the heart valves (endocarditis), or an interstitial pneumonia. Infection is commonly spread by inhalation of the agent in aerosols. In Nova Scotia, parturient cats are a source of Q fever in humans; in one investigation in Nova Scotia cats, 24% were serologically positive for the organism. In NS, still-born kittens have been found to be an excellent potential source of this zoonotic organism. In another investigation in eastern Canada, snowshoe hares were frequently observed to harbor the organism. Raccoons are thought to readily transport *Coxiella burnetii* into an urban environment, where humans may be exposed to the agent.

In humans, sheep and goats are the principal domestic livestock reservoirs of the disease. *C burnetii* is present in the milk of infected cattle, sheep, or goats. Pasteurization destroys the organism (Radostits et al, *Veterinary Medicine*, 1994), but there is a risk to those who may drink raw milk, especially raw goats'milk. In this case, both the goat owner and the veterinarian who worked on this case were advised immediately of the laboratory results. Out of interest, blood testing of individuals working on the goat farm, and of the veterinarian, is planned.

– comparative medicine comments on Q fever, and incidence of the agent in N.S. cats, were taken primarily from lectures given by Dr. Thomas Marrie, Dalhousie University, at the 1995 annual conference of the Canadian Assoc. of Veterinary Pathologists, at Charlottetown, PEI.

Tapeworm cysts in lamb muscle tissue:

J Coates

Veterinary inspection of several lambs at time of slaughter had revealed numerous small white nodules dispersed throughout skeletal muscle. Microscopically, these fibrous lesions contained walled-off tapeworm larvae consistent with the larval form (***Cysticercus ovis***) of the canid nematode ***Taenia ovis.***

T. ovis is a tapeworm commonly found in dogs and wild carnivores throughout the world, which has, as its larval or intermediate form, a cysticercus (*Cysticercus ovis*) which develops in the heart and skeletal muscle of sheep and goats. Transfer of the very resistant eggs from the tapeworm segements in canid feces to lambs or sheep is often enhanced by the contamination of open water by sewage, or by use of sewage as fertilizer in fields. It also occurs directly by contamination of animal feeds or pastures with canid feces.

As in this case, infection of lamb or sheep carcasses with the intermediate form of this parasite results in condemnation at slaughter, which can be a costly loss to producers. Sheep or goat owners should have their dogs dewormed regularly with an appropriate agent to prevent or eliminate the presence of the adult *Taenia ovis* tapeworms within dogs' intestinal tracts. Contamination of sheep pastures or feeds by dog feces should be avoided.

Copper toxicity in llamas:

A Britton (AHC) &
J Coates (AHC)

Two llamas, 9 - 11 months of age, were presented to the AHC following acute onset of weakness, recumbency and death. The animals were in good body condition. Internally, both llamas exhibited pale, mottled, slightly firm livers with occult blood in the urine. One llama had marked pleural and mediastinal hemorrhage, marked blood accumulations within the chest cavity, and hemorrhage in the proximal portion of the small intestine (duodenum). The second llama had a small area of stomach mucosal erosion, but no evidence of generalized bleeding. Microscopically, both llamas exhibited massive liver damage with bile duct hyperplasia and early portal fibrosis. Liver and kidney copper levels were in the toxic range at 254 & 347 ppm and 160 & 150 ppm (wet weight), respectively. Blood serum taken antemortem from one of the llamas revealed serum copper at 8 ppm, which is in the toxic range.

Copper toxicity is most commonly reported in sheep, a species which avidly bioaccumulates copper in hepatocytes when copper levels in the feed are elevated. Initially, the accumulating copper causes no apparent damage to liver cells. However, upon exceeding its storage capacity, individual hepatocytes die and liver regeneration is stimulated. At this stage, serum liver enzymes rise. When replacement of liver cells (hepatocytes) can no longer keep pace with cell loss, plasma copper levels increase and compounds the hepatic necrosis. Further elevation of plasma copper leads to a hemolytic crisis and subsequent death. Although llamas experience massive hepatic necrosis and release of copper into circulation, the hemolytic crisis as observed in sheep is less likely to occur [(1)]. Affected llamas exhibit poor clotting of blood and subsequent internal hemorrhage, which may reflect reduced vitamin K production due to liver damage. Overall, llamas are believed to be less sensitive to copper toxicity than sheep, but more sensitive than cattle.

The llamas submitted to AHC were from an established herd fed local grass hay, 16% dairy ration, and free choice mineral mix. The animals were from a group of three juvenile males fed the dairy ration free choice. The remainder of the herd received

Copper toxicity in llamas: *(Cont'd)*

about 1 kg of the ration daily. Feed analysis revealed 43 ppm copper in the dairy ration, which falls within the recommended guidelines for supplementation of cattle rations. However, cattle have a higher requirement for copper than do llamas. Although there is no record of their feed consumption, it is probable that the juvenile llamas ate more than 1 kg of the free choice cattle ration daily, and consequently were exposed to excessive copper. Chronic feeding of cattle rations to llamas has been reported to cause sporadic copper toxicity. A similar case was reported in a U.S. zoo, where four llamas died over a 2 year period[(2)].

The sporadic occurrence of copper toxicity in llamas fed cattle rations may reflect increased sensitivity of some individuals, such as juveniles, to marginally high copper levels. However, it seems likely that there may be additional factors involved. Low total molybdenum in the feed, creating a high copper:molybdenum ratio (greater than 10:1), may lead to a relative increase in copper bioavailablility since molybdenum reduces copper uptake. Zinc also interferes with copper uptake and zinc supplements fed to llamas may be protective, although no studies have addressed this possibility to date. The opposite is true of monensin. In sheep, monensin enhances uptake and hepatic storage of copper. The effect of monensin supplemented cattle feed in llamas is unknown, however based on studies in sheep, this feed should be avoided.

Similar to other livestock, rations should be formulated for llamas to meet species-specific daily nutrient requirements. If there is concern regarding the copper status of llamas fed a cattle ration, feed and water analysis plus serum chemistry for hepatic enzyme levels would be useful in detecting an impending problem. Steps could then be taken to reduce any further exposure.

References:

(1). Mullaney TP, Slanker MR, Fitzgerald SD, Elliott MW, Braselton WE, Main, KE. Copper Toxicosis in Llamas. *39th Annual Meeting of the AAVLD*, 1996: pg. 36

(2). Junge RE, Thornburg L. Copper Poisoning in Four Llamas. *JAVMA*, 1989; 195:987-989

Hydromyelia and syrinx formation in a 14 month-old llama:

- S Raverty & A Britton, AHC

A 14 month-old female llama born with a congenitally kinked tail was recently submitted to the AHC for necropsy. Last May, at 7 months of age, the animal was clinically evaluated for progressive urinary then rectal incontinence, with hind-limb paralysis and loss of sensation over the dorsal aspect of the hooves. Because of deteriorating condition and no response to supportive care, the animal was electively euthanized in early December, 1999.

External examination disclosed a few lice throughout the wool. There was a moderate amount of stable froth within the windpipe (trachea) and bronchi, and the lungs diffusely exhibited marked congestion and edema. There was an abrupt left lateral deviation of the vertebral column distal to the lumbosacral junction. There was marked expansion of the spinal cord at the level of thoracic vertebrae 7 – 8, with disruption of the grey and white matter. The parenchyma appeared pale grey-white, glistening, and clotted.

The affected thoracic spinal cord revealed moderate to marked enlargement of the central canal with compression and lateral displacement of the adjoining grey and

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Hydromyelia and syrinx formation in a 14 month-old llama: *(Cont'd)*

white matter. Moderately deep within the white matter, large cavitations were observed ecccentrically within the lateral aspect of cord segments 7 and 8. These cavitations were frequently bound by scattered aggregates of glial cells and macrophages, that contained myelin. The margins of the spinal cord defect were bordered by irregular fringes of torn tissue. Upon microscopic examination, there was no histologic indication of congenital hydrocephalus.

The spinal cord lesions were consistent with a hydromyelia with syrinx (fistula) formation. Hydromyelia is defined as dilatation of the central canal of the spinal cord, with an abnormal accumulation of fluid. The proposed pathogenesis includes physical disruption of the cord tissue secondary to increased cerebrospinal fluid pressure, with rupture of the dilated central canal and tracking of fluid into the adjoining parenchyma.

Due to the inflammatory infiltrate in this case, the syringomyelia (a fluid filled cavity in the spinal cord) was considered a secondary process and likely a sequela to spinal cord trauma, impaired vascular flow, inflammation, or some other entity. The lesion was sufficiently severe to account for the antemortem, clinical signs.

In domestic animals, this condition is most commonly encountered in young dogs. It primarily affects the cervical spinal cord region, with variable involvement of the thoracic and lumbar regions. This is thought to be the first reported case of this condition in llamas, and investigations are underway to further characterize the nature of this process.

Lead poisoning in Trumpeter swans:

V Bowes

Over the past several years there has been a consistent number of submissions during the winter months of wild Trumpeter Swans with a simple clinical history of being found dead. On gross examination, these birds are in good body condition and have adequate fat stores. The only gross lesion is marked distension of the esophagus with recently ingested feed, primarily swamp grasses and a few crustaceans. This lesion, in the absence of other gross findings, is highly suspicious of lead toxicity. Lead is a neurotoxin which affects the muscle tone of the esophagus, thus preventing ingested food being moved along the digestive tract. Careful screening of gizzard contents in all cases revealed a variable number (1-8) of partially eroded lead shot, which would have been ingested along with the normal small pebbles necessary for the birds' digestion.

The Trumpeter Swan, with its long neck, is able to reach areas of pond bottoms that are out of reach to other, smaller waterfowl. These pond deposits of lead shot, accumulated over years of waterfowl hunting, lie inert within pond bottoms. However, once ingested, the acid in the bird's stomach begins to dissolve the pellet, and lead is thus absorbed into the bloodstream.

Although the finding of lead shot in gizzards is highly suggestive of lead toxicity, it is only confirmed by finding elevated levels in the liver and kidney. If poisoned birds are found early enough, supportive care and the administration of chelating agents to bind the lead in the bloodstream may be sufficient to see the bird through to recovery. Radiographic evidence of lead shot in the gizzard and surgical removal of the pellets may also be necessary.

Despite the ban on the use of lead shot in the industry, pond bottoms will continue to harbor this potential threat to wild waterfowl for years to come.

Pulmonary cryptococcosis in a stranded Dall's porpoise (*Phocoenoides dalli*):

– S Raverty, AHC; C Stephen, Center for Coastal Health, Nanaimo, BC; J Watson, Malaspina University-College, Nanaimo, BC; G Ellis, Department of Fisheries and Oceans, Nanaimo, BC

Dall's porpoises *(Phocoenoides dalli)* have been reported throughout the eastern rim of the Pacific ocean, as far north as the Pribilof Islands of Alaska, and south to San Francisco, California. They are considered among the most abundant and frequently encountered cetacean species off the British Columbia coast. As part of an ongoing collaboration with the Department of Fisheries and Oceans, Nanaimo, to monitor the cause of death of these animals, a post mortem examination was performed on a recently stranded porpoise. Representative tissues were submitted to the Animal Health Centre.

The porpoise was an adult male recovered from Gabriola Pass, British Columbia. Gross evaluation indicated that the animal was in good flesh. There was a large volume (1.0 - 1.5 L) of dark red fluid within the abdominal cavity. The glandular compartment of the stomach featured multiple erosions and occasional hemorrhagic ulcers; the distal intestine contained digested blood, attributed to terminal shock. No ingested food or feces were noted within the gastrointestinal tract. The most salient lesions were confined to the thoracic cavity; the right lung was enlarged and consolidated, with numerous firm nodules on cut surface. There was approximately 0.5 L of blood-tinged fluid within the chest. The mediastinal lymph nodes were prominent and pale. A small number of adhesions were apparent between the visceral pleura and the external surface of the heart (epicardium).

Microscopic examination of lung tissue revealed massive accumulations of extracellular, pleomorphic, thin walled, 4-6 um diameter, oval to spherical, uninucleate yeast organisms. Individual yeast cells had a prominent, 2-4 um-wide, clear to slightly amphophilic capsule, and finely stippled to homogeneous, basophilic cytoplasm. The yeast organisms were interspersed within abundant amounts of pale, mucinous material and scattered inflammatory cells.

Based on the yeast morphology, a diagnosis of **pulmonary cryptococcosis** was made. These organisms are found world-wide within nature, and are particularly abundant in areas contaminated by pigeon feces. This yeast is considered noncontagious, with infection generally acquired through inhalation or ingestion. Although infection with this pathogen has been reported previously in a 7 year-old, captive Atlantic bottlenose dolphin (*Tursiops truncatus*) and a female Pacific white-sided dolphin (*Lagenorhynchus obliquidens)*, this incident is believed to be the first observed in a wild cetacean, and represents the sentinel case in a Dall's porpoise.

The lack of inflammatory infiltrate in this animal suggests a reduced immune response. Further studies in an attempt to resolve the pathogenesis of this condition are underway. Close evaluation of submitted tissues failed to reveal any underlying or predisposing conditions.

Proposed British Columbia Marine Mammal Stranding Network

In joint effort with the Department of Fisheries and Oceans, Vancouver Public Aquarium, the British Columbia Society for the Prevention of Cruelty to Animals and other agencies, the Animal Health Centre, British Columbia Ministry of Agriculture, Food and Fisheries is working to revitalize interest in establishing a coastwide BC Marine Mammal Stranding Network. For those clinicians along the BC coastline interested in participating in the strandings of live and dead marine mammals, please contact Dr Stephen Raverty, Animal Health Centre, Abbotsford, BC, at 1-800-661-9903 during regular office hours, or by e mail at Stephen.Raverty@gems3.gov.bc.ca . Additional details are to follow.

Short cuts from the post mortem room:

J Coates

As mentioned elswhere in this newsletter, ***Coxiella burnetii*** rickettsial abortion was diagnosed in a group of dairy goats. Recently, ***Toxoplasma gondii*** protozoal abortion, and **chlamydial** bacterial abortion, have also been diagnosed in sheep. Small ruminant disease plays an important part in the daily submissions to the AHC. Interestingly, all 3 of these infectious causes of small ruminant abortion are potentially transmissible to humans. That is, the organisms are **zoonotic**. When carrying out obstetrical procedures in these animals, it is best to keep this in mind. Keep the remains of any aborted fetal tissue including placenta away from the family farm cats, or dogs, too.

If you desire an excellent reference text on diseases transmissible between animals and humans (zoonoses) in your home or in your veterinary clinic, the book ***Zoonoses and Communicable Disease Common to Man and Animals*** is highly recommended, with P.C. Acha and B Szyfries as editors. The text was published as a 2nd edition in 1989 by the Pan American Health Organization of the United Nations' World Health Organization (WHO) located in Washington, DC. The book can be purchased or ordered from the Bookstore at the University of Saskatchewan, Saskatoon, or from the UBC Bookstore. (*There may now be a 3rd edition. Ed.*)

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Fecal samples and rectal biopsies were received from 4 mature Holstein dairy cows. Despite a good appetite, the animals were demonstrating signs of gradual weakness, wasting, and persistent diarrhea. Acid-fast stains on the fecal specimens were all negative. One of the 4 biopsies was positive for **mycobacteria**, using acid-fast stains on rectal tissue. ELISA tests on blood sera from all 4 animals was positive for **Johne's disease**; PCR testing on fecal samples from all 4 animals was also positive. In addition, culture of the organism was also positive in all 4 specimens.

The battery of tests used to diagnose Johne's disease in this case complemented each other, although the number of positive cases detected by direct acid-fast stains on fecal smears and rectal biopsies was disappointing. It does demonstrate, however, the value of approaching the diagnosis of this condition by several angles. Traditionally, the standard ELISA test utilized by the AHC's Monitoring Laboratory is considered somewhat insensitive as a blood serum antibody test for Johne's disease, but nevertheless highly specific. That is, results showing positive are almost invariably positive, with a 99% accuracy rate. **PCR testing of fecal samples** in this case corresponded exactly with the serological and bacterial culture results.

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Parvoviral enteritis in domestic cats has been diagnosed in at least two different submissions recently, with intestinal lesions observed microscopically characterized by severe mucosal injury, including crypt gland necrosis and dilation. Confirmation of the presence of the virus usually was accompanied by PCR tests on fecal/gut specimens, and the fluorescent antibody (FA) procedure. Earlier, this disease was diagnosed in a young raccoon from BC's Lower Mainland.

When carrying out necropsies on raccoons, the roundworm of that species is commonly observed, especially if the animal is more than a few months of age. Keep in mind that the raccoon roundworm, ***Baylisascaris procyonis*** is a zoonotic agent, and has been reported as the cause of illness in humans. In humans and other species that are inadvertently exposed to the roundworm eggs by ingestion, developing larvae have a tendency to seek out brain and other neural tissue, in a form of aberrant larval

migration. Young children are especially at risk, as fecal/oral contamination with raccoon scats is much more likely to occur. Brain lesions and containing parasites consistent with migrating larvae (larva migrans) of *Baylisascaris sp* have been diagnosed at the AHC in wild pigeons, Douglas squirrels and in ratites.

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Nutritional cardiomyopathy (white muscle disease) was diagnosed in a group of very **young lambs** from Vancouver Island. Several young lambs had died in the flock, with the animals found very weak, or dead. Microscopically, random areas of heart muscle degeneration and necrosis were observed. Skeletal muscle lesions were not readily detected. Liver selenium levels in these animals were adequate upon measure at 0.5 and 0.6 ppm (adequate levels recorded by Puls at 0.25 ppm to 1.50 ppm), so it is assumed that deficient flock dietary vitamin E levels may have led to the condition. The veterinarian who had submitted the tissue specimens on his client's behalf was notified immediately of these results. Following this submission, a second Island case of nutritional heart disease in lambs secondary to vitamin E-Se deficiency was later received.

It is perhaps noteworthy that in lambs, heart muscle lesions caused by vitamin E-Se deficiency are often found in the right ventricle of the heart, while in calves they tend to be more readily found in the left ventricle. It's always a good idea to submit both heart and skeletal muscle samples when suspicious of nutritional (white) muscle disease.

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A calf was submitted for necropsy from a central B.C. beef cattle operation by the practitioner. When calving began, several cows delivered **stillborns.** According to the owner, dystocia or calving difficulties were not a factor; the animals appeared normal on gross inspection. On necropsy, the submitted calf had a grossly swollen thyroid gland (goiter) that weighed 56.0 gm. Calves with thyroids weighing more than 30.0 grams are suspected of having iodine deficiency[1], and this was confirmed on microscopic examination of thyroid tissue. Stillborn or neonatal goitre is a direct result of inadequate levels of the trace element iodine in the diet of the pregnant cowherd.

1. Smyth et al. Stillbirth/perinatal weak calf syndrome: a study of calves with an abnormal thyroid gland. *Vet Rec* 1996; 139: 11 – 16).

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Salmonella typhimurium has been isolated from vealer calves with fibronous enteritis; the organism has also been identified in cowherds where animals were clinically ill with severe diarrhea, at times with visible shreds of mucosa or fibrin in the expelled feces.

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Mycotic (fungal) abomasitis, or inflammation of the glandular stomach, has been diagnosed periodically in young Holstein calves, most recently in a week-old Holstein vealer calf. This condition may arise where animals are being stomach-tubed extensively with oral electrolytes and nutrients, in an attempt to relieve the dehydration and weakness that is so common in neonatal diarrhea. If treatment with these electrolytic and glucose-based preparations continues for too long, often in conjunction with antibiotics, there is a risk that an abnormal microflora may emerge within the neonatal calf stomach compartments, particularly the rumen and/or abomasum, resulting in fungal proliferation and secondary mucosal injury and inflammation.

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Severe inflammation of the larynx (laryngeal necrobacillosis) was diagnosed in a month-old llama upon receipt of formalinized laryngeal tissue for microscopic examination. Severe ulceration and inflammation of the inner membranous lining of the larynx (the voice-box area) had occurred, with intense inflammatory cell infiltrates. Secondary severe swelling and accumulation of inflammatory exudate had nearly blocked the airway of the very young animal, and it soon died of respiratory distress, despite treatment.

Using a special stain, numerous filamentous Gram-negative bacterial rods were observed throughout the inflamed and ulcerated larnyngeal tissue. Although no fresh tissue was available for bacterial culture, the nature of the laryngeal lesion was consistent with the syndrome of so-called laryngeal necrobacillosis, which is usually associated with the bacterial organism ***Fusobacterium necrophorum***.

This organism is found normally in the intestine of cattle, sheep, swine, and sometimes other species, including humans (Gyles & Thoen: *Pathogenesis of Bacterial Infections in Animals,* 1993). It is likely that llamas and other camelids carry this organism as well.

As seen microscopically in this case, bacteria resembling F *necrophorum* were present in large numbers in the laryngeal lesions. The organism is thought to cause the illness secondary to a prior injury to the delicate laryngeal membranous lining (Radostits et al: *Veterinary Medicine*, 8th ed., 1993), especially in young, immature animals. In cattle, the condition occurs most commonly in animals under 3 months of age, and is often linked to other ongoing disease or stressors in the animal, or an underlying nutritional deficiency (Radostits et al). Undoubtedly, these same predisposing factors would be relevant to llamas and other camelids.

Young suckling animals may innocently chew on stems of straw or clover or other abrasive material, thus inadvertently injuring the inner lining of the larynx, and provide an avenue of entry for the feces-borne organism *F necrophorum*. Providing plenty of clean bedding in confined areas should help to prevent this condition in young, susceptible animals.

In laryngeal necrobacillosis, a useful diagnostic tool for the practitioner is that affected animals usually demonstrate inspiratory distress (dsypnea), as opposed to the expiratory dyspnea seen in a true bronchopneumonia. In some instances, a roaring noise (stridor) is created as the animal struggles to pass inhaled air through the inflammed, swollen, and partially occluded larynx. Variations of this condition occur; the lesion may be present within an injured tongue, or within the mouth. Laryngeal necrobacillosis will respond to persistent and judicious treatment, provided a veterinarian is called promptly before laryngeal swelling and damage becomes excessive.

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Biting lice were observed within a hair sample taken from a llama, that had been sent in for examination. The animal had extensive scurfiness or dandruff in its haircoat. The veterinary practitioner suspected pediculosis. Microscopically, the morphologic features of the parasites were consistent with ***Damalinia sp***, a genus of biting lice.

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*Dr. Louis Pasteur's motto in life was "*Travailler, travailler toujours.*"*

Neospora update:

"Neosporosis has emerged as a major cause of abortion in cattle in many countries… dogs can serve both as intermediate and definitive hosts… Antibodies to *N. caninum* were demonstrated in the sera of naturally exposed water buffaloes, coyotes, red foxes, and camels suggesting that these hosts are also natural intermediate hosts for *N. caninum*… Tachyzoites and tissue cysts are the stages found in the intermediate hosts and both are intracellular…Tissue cysts have also been found in the peripheral nerves of a horse… and once in an ocular muscle of a congenitally infected foal.

Neospora has also been demonstrated in bovine fetal brains 31 days post-inoculation of the dams with tachyzoites…

Neospora caninum affects both dairy… and beef cattle…It is a major cause of abortion in dairy cattle in the U.S., New Zealand, and the Netherlands… Canada…

Clinical signs have only been reported in individual calves younger than two months of age. Abortion is the only clinical sign observed in adult cows. Cows of any age may abort from 3 months of gestation to term. Most Neospora-induced abortions occur at 5-6 months of gestation. Fetuses may die in utero, be resorbed, mummified, autolyzed, stillborn, born alive but diseased, or born clinically normal but chronically infected. Within herds, abortions may be clustered, sporadic, or epidemic…

Presence of specific antibodies in serum from an aborted cow is only indicative of exposure to *N. caninum*…

Although *N. caninum* is closely related to T. *gondii, Sarcocystis spp* and other apicomplexans, cross-reactivity has not been a major issue in animals experimentally-infected with *N. caninum* and related apicomplexans…

Although *N. caninum* infection can cause lesions in several organs, fetal brain is the most consistently affected tissue…hepatitis is more common in epidemic than sporadic abortions. There are no pathogonomonic gross lesions of neosporisis…

Finding *N. caninum* antibody in fetal serum or precolostral calf serum indicates infection, but a negative result in a fetus is less useful as antibody synthesis in the fetus is dependent on the stage of gestation, level of exposure, and the time between infection and abortion…

Several polymerase chain reaction (PCR) methods have been reported to detect N. caninum DNA (*Ed.'s note: at the Abbotsford AHC, a PCR test is available for diagnostic purposes in suspected cases of neosporosis)*…

Isolation of *Neospora* in cell culture is rarely possible because most organism in bovine fetuses die with autolysis of host cells…

Neospora caninum is efficiently transmitted vertically in cattle, even for several generations… but horizontal transmission seems to be necessary to introduce new infections in the herd. No horizontal cow to cow transmission has been demonstrated. Until the recent discovery of the oocyst, environmental transmission of *N. caninum* to cows was unexplained. Seroepidemiologic data support the role of the dog in the life cycle of *N. caninum*… Although nothing is known at present regarding the frequency of shedding of *N. caninum* oocysts by canids in nature, the resistance of the oocyst, and whether dogs shed oocysts more than once, it is prudent to protect feed and water from contamination with dog feces. Dogs should not be allowed to eat aborted fetuses, fetal membranes, or dead calves. There is no vaccine for preventing Neospora-induced abortion in cattle or to prevent oocyst shedding in dogs. Prevention of transmission of the parasite from dam to fetus has not been demonstrated.

Continued Page 14

Neospora update: *(Cont'd)*

While the culling of seropositive cows as a means of reducing N. caninum infections in a herd has been suggested, it is impractical in high prevalence herds.

A recent serological survey in Japan reported a higher prevalence of N. caninum infection in dogs on dairy farms with abortions… "

– taken with appreciation from the a review article by JP Dubey: Recent advances in Neospora and neosporosis. Veterinary Parasitology 1999: 84; 349 – 367.

The Cow Contract:

" In the simplest of terms, our contact with dairy cattle includes the barter of housing, feeding, and comfort for meat and or milk for food, and hides for clothing. With this contract, we have a moral obligation to routinely evaluate how well we are living up to our end of the deal.

Dairy cattle cannot audit our performance or write a report about how well we honour the contract. Nonetheless, cows show displeasure with their situation: fear, unusual behaviour, injuries, lameness, reproductive failure, metabolic diseases, infectious diseases, or milk production. They are counting on us to read and interpret their report in this format.

Savvy cattle care professionals live up to their end of the bargain, auditing their contributions, and sweetening the deal for cattle in their care. Some, however, are unaware that their actions or inaction, their design or construction of a facility, their choice of a husbandry system, or their management within a system, infringes upon the contract. And in many cases, they are simply unaware that they have broken the contract.

As animal care professionals, our time spent on dairy farms provides an opportunity for observation and assessment - an audit of how well we are doing with our end of the cow contract… our cow contract is self-regulated and self-audited: a moral obligation to deliver care, comfort and food in exchange for milk, meat and other products. Let's continue to strengthen our part."

– by Neil Anderson; from CEPTOR: Animal Health News, vol 7, no. 4, Dec. 1999; the newsletter of Ontario Agriculture, Food and Rural Affairs, Veterinary Science.

Stillbirth/perinatal week calf syndrome and leptospira:

"…Leptospiral infection has been reported in calves which were either stillborn or dead from perinatal weak calf syndrome; 356 such calves were examined for evidence of association between leptospiral infection and macroscopic, histological and microbiological findings, and the parity of the dam. Calves in which leptospiral antigen was detected in the placenta were significantly lighter by an average of 6 to 10 kg than calves with no antigen in the placenta. Calves infected with Leptospira were more likely to be infected by *Actinomyces pyogenes* or *Bacillus* species. No other significant associations were detected. The adrenal gland, lung and placenta were the most useful organs to examine for leptospiral antigen. The placenta was the only antigen-positive tissue in 8.9% of the calves submitted with their placenta."

– abstract from the article *Stillbirth/perinatal weak calf syndrome: a study of calves infected with Leptospira*.": Smyth JA, Fitzpatrick DA, Ellis WA; Veterinary Record 1999: 145, 539-542.

Botulism in poultry:

"In the past six months, we have had reason to suspect botulism, as a causative agent of neurologic signs in broiler flocks in Ontario with increased frequency. Its occurrence in confined broiler flocks has been previously documented, although originally it was seen most commonly in free-ranging and wild birds. The disease is the result of ingestion of preformed *Clostridium botulinum* exotoxin, and in poultry this is most commonly type C toxin. The toxin is produced under anaerobic conditions and at 10 – 47 degrees C. With high doses of toxin, the onset of clinical signs is rapid (hours) but can require several days with lesser doses.

"…Type C organisms readily grow in the gastrointestinal tract of chickens but also in wild birds. As a result, the organism is commonly found in the environment around poultry farms. The main factor enhancing spread of the organism is the resistance of the spores to environmental insult and thus their continued persistence. Botulism is thought to occur typically as a result of ingestion of preformed toxin. Its source can be in carcasses, food or water contamination, maggots, insect larvae (such as beetles) and other invertebrates. Additionally, there is a syndrome known as **toxico-infection** that results from production of the toxin within the chicken's intestinal tract. Toxin production occurs in association with other unknown factors to produce clinical disease.

"…Affected chickens typically show signs of progressive paralysis, with involvement of limbs, neck, and eyelids being most common. Morbidity and mortality may vary depending on the amount of toxin ingested. Unlike other disease conditions, there is an absence of gross and microscopic lesions and this condition often becomes a diagnosis by exclusion…

The standard means of confirming the diagnosis is by mouse bioassay for toxin, preferably the serum or less suitably in crop or gastrointestinal contents from moribund birds…The test is run here in the Bacteriology Unit at the Animal Health Laboratory for a fee of $100…"

– Shelley Newman DVM, DVSc, Diplomate ACVP. Animal Health Laboratory Newsletter, U of Guelph; Dec 1999: vol 3, no 4.

(Ed.'s note: unfortunately, we do not presently have a test for botulinus toxin identification at the Abbotsford Animal Health Centre).

Selenium and the immune response: factors inhibited by selenium deficiency:

- Resistance to microbial and viral infections
- Neutrophil function
- Antibody production
- Proliferation of T and B lymphocytes in response to mitogens
- Cytodestruction mediated by T lymphocytes and natural killer cells.

– from *The Compendium*, Food Animal edition, October 1997: by WS Swecker, Dept. Large Animal Clinical Sciences, Virginia-Maryland Regional College of Veterinary Medicine, Virginia Tech, Blacksburg.

Maternal hypothyroidism and fetal development

(a comparative medicine comment):

"Thyroid deficiency during the latter two thirds of gestation and the first months after delivery can result in mental retardation and sometimes neurologic deficits… During the middle and last trimesters, thyroid hormone is supplied by both the mother and the fetus but mostly by the mother. This is most evident in the fate of infants with sporadic congenital hypothyroidism. Most of these infants are normal at birth, and even among those with no thyroid secretion, umbilical-cord serum thyroxine concentrations at birth are 25 to 50 percent of normal, an indication that transplacental passage of maternal thyroxine has taken place… Most mothers of infants with congenital hypothyroidism have normal thyroid function, and the thyroxine they provide in utero must explain why the infants have so little clinical evidence of hypothyroidism at birth…When dietary iodine intake is very low, both pregnant women and their fetuses have poor thyroid function throughout gestation. The consequences of combined maternal and fetal hypothyroidism for the infants are not only mental retardation but also neurologic defects – spasticity, ataxia, and deaf-mutism – that do not occur in infants with congenital hypothyroidism… Therefore, the beneficial effect of iodine is more likely to be a result of increased maternal thyroid secretion than of increased fetal thyroid secretion… Despite the presumption that hypothyroidism in most pregnant women is caused by chronic autoimmune thyroiditis, which cannot be prevented, the difference among countries suggests another possibility – iodine deficiency, which is preventable…"

– *editorial by R D Utiger, in the* New England Journal of Medicine*, Aug 1999, vol 341, no 8*.

Erratum: fatal cardiomyopathy in a llama:

In the December 1999 issue of Diagnostic Diary, an article presented by Drs. Raverty (AHC) and Jacobson (Reimer Veterinary Clinic, Kelowna) should have been entitled *"Fatal myocardial degeneration and necrosis associated with vitamin E deficiency in an adult Llama (Lama glama)."* The word *fetal* had inadvertently been printed into the article heading, rather than *fatal*. Ed. apologizes to the authors, and to readers, for this error.

" Our wills and fates do so contrary run
That our devices are overthrown,
Our thoughts are ours, their ends none of our own."

– The Player King in Shakespeare's *Hamlet*.