Animal Health Centre NEWSLETTER

BRITISH COLUMBIA
Ministry of Agriculture, Food and Fisheries

Diagnostic Diary Vol. 10, No. 2. *The Animal Health Centre of British Columbia: a diagnostic laboratory accredited by the American Association of Veterinary Laboratory Diagnosticians.* **September, 2000**

CONTENTS

From the Director 1
Infectious laryngotracheitis (ILT) in chickens .. 3
Summary of pig submissions to the AHC 3
Equine congenital cutaneous papilloma 4
H somnus pneumonia in a nursing beef calf . 5
Pasteurella trehalosi in California Bighorn sheep 6
Toxoplasmosis in a recently imported lemur . 7
Short cuts from the Post Mortem Room 8
Pulmonary botryomycosis in a capybara 10
Botulism in dairy cattle linked to a dead cat 11
Isolation of *Giardia* from a Llama and a sheep 11
Excerpts from the University of Guleph 12
Origins: the roots of toxicology 13
The oncologist's debt to the chicken 13
A child's perspective on a veterinary laboratory 14
Focus on staff 15
Art and reality 16
The Beaver people 16
Microbiologist employment opportunity 16

Editor:

J.W. Coates

Contributors to this edition of Diagnostic Diary are: J. Waddington, H. Schwantje, S. Raverty, R. Lewis, J. Coates.

Mailing Address:

B.C. Ministry of Agriculture, Food and Fisheries
Animal Health Centre,
1767 Angus Campbell Road
Abbotsford, BC V3G 2M3

From the Director

R.J. Lewis

Most people are aware of how expensive it is to provide diagnostic testing facilities whether for animals or people. Animal owners in British Columbia are fortunate in that the Ministry of Agriculture, Food and Fisheries recognizes the value of such testing and put considerable resources into building a new AHC laboratory facility in 1995. The ministry has committed both human and dollar resources into providing a top quality veterinary laboratory facility that is honoured to be only the second veterinary diagnostic laboratory in Canada accredited as a full service laboratory by the American Association of Veterinary Laboratory Diagnosticians. The costs for providing this service are highly subsidized by the ministry since it is essential that we monitor for diseases of concern in animals as well as people. Specimen submissions help provide information on the relative health and well-being of animals in British Columbia and also allow us to develop information relating to diseases that may be transmissible to man. This information is used to assist in directing research activities into animal diseases and provides a database for diseases or nutritional problems that may be inhibiting animal production or welfare.

Animal owners benefit by having direct access to testing capabilities that will alert them to problems in their animals against which they can take action once a specific diagnosis has been made. Although diagnostic expenses are highly subsidized, the costs of testing have continued to increase significantly since the last fee schedule update in September 1995. The absolute increase in supplies costs and the relatively low value of the Canadian versus the American dollar has meant increased costs since most reagents and supplies originate in the United States. We have also been notified that we can no longer provide a GST-exempt accession fee.

As a result, it was necessary to revise our fee schedule effective October 02, 2000. Over the last several months, we have consulted

Continued Page 2

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

with our major livestock commodity groups and compared our charges with those in diagnostic laboratories in other provinces. Although there are increases for many tests, we have kept these to the minimum and also are offering some volume discounts. A copy of the new fee schedule has been faxed and/or mailed out to all our regular clients including veterinary clinics, feed representatives, major commodity groups, etc. The new fee schedule is available on our web site.

Fee increases are never popular but I believe our prices are fair, reflect some measure of our actual costs, and fall in line with those charged in other jurisdictions. The moderate price increases will allow us to continue to develop new, more accurate, and sensitive tests. If you have not received the new fee schedule or do not have access to our web site, please let us know and we will be pleased to send it to you. Should you have any questions or comments, please call and I would be pleased to discuss.

Staff Update – Veterinary Pathologist

We are very pleased to welcome Dr. Ann Britton to the AHC permanent staff. Ann has been working for us part time under contract for some time and we have finally been able to convince her to work with us as a regular employee. Many of you will have seen Ann's name as the pathologist in charge of some of your cases last spring or may have had the opportunity to speak with her.

Ann received her Doctor of Veterinary Medicine (DVM) degree from the Ontario Veterinary College (OVC) at the University of Guelph in 1981, following which she spent one year in a mixed animal practice in southern Ontario. She returned to the OVC for five years of formal pathology training, receiving a MSc in Pathology and embarking upon her PhD in pathology. After moving to southern British Columbia, Ann completed her PhD at the University of British Columbia in Reproductive Pathology in 1991. She has spent several years as a diagnostic pathologist at a private veterinary pathology laboratory.

Dr. Britton is enthusiastic, highly motivated, has a well-developed sense of humour, and brings extensive diagnostic skills and experience to the Animal Health Centre. We are very fortunate and pleased to have Ann return to us on a regular basis as a part time veterinary pathologist.

Staff Update – Veterinary Epidemiologist

Please join with us in welcoming Dr. Nancy de With to the Animal Health Branch. Nancy graduated with her DVM from the Western College of Veterinary Medicine at the University of Saskatchewan in 1995. She has also had experience in both small and mixed animal practices in Saskatchewan and British Columbia.

In 1999, Nancy received her Master of Science degree in Veterinary Epidemiology, also from the University of Saskatchwan. Her most recent responsibility was filling a temporary appointment as a disease surveillance veterinarian with the Saskatchewan Department of Agriculture in Regina. Many of the tasks that she so successfully performed there will be of immediate benefit to our Animal Health Branch programs.

We have had the pleasure of working with Nancy on some projects while she was doing graduate work and look forward to her being an important part of the Animal Health Branch. Dr. de With will be working on a regular half-time basis under the supervision of Dr. Merv Wetzstein in our Health Management and Regulatory section.

RJL

Infectious laryngotracheitis (ILT) in chickens:

Infectious larynogotracheitis (ILT) has recently been diagnosed in broilers and young laying hens, in the Abbotsford area of the Fraser Valley. Earlier in the year, a case had been diagnosed in a broiler breeder flock in the B.C. Interior. As many as seven Fraser Valley farms have been diagnosed recently with the disease, which is caused by an avian herpesvirus. Clinically, individual birds show signs of acute respiratory distress, as the trachea (windpipe) is partially plugged with inflammatory exudate induced by the ILT virus. A locally severe erosive and necrotizing reaction in the tracheal and laryngeal mucous membranes is often seen, together with hemorrhage.

The use of aerosolized spray vaccine in the immunization process of poultry for ILT is not recommended, as response to vaccination by this method is uneven at best; in addition, there is the risk of air or wind-transported vaccine virus causing illness in nearby flocks of naïve birds that have never been vaccinated, particularly broiler flocks. The eye droplet method of vaccination, although more time consuming, is a much superior means of assuring safe exposure to the vaccine virus.

In addition, owners should be aware that ILT-vaccinated birds introduced into a group of non-vaccinated, immunologically naïve birds may still carry the virus in a latent form, which may later cause flock disease.

Flock owners should be aware of the need for heightened biosecurity measures when ILT becomes a problem on neighbouring farms.

Disease summary of pig submissions to the Animal Health Centre for 1999:

SA Raverty and J Waddington, AHC

Over the last five years, there has been a progressive decline in the number of pig submissions to the AHC. This is attributed in part to a transition of submission costs

(previously borne by feed companies) to individual producers, as well as the precipitous decline in hog market prices over the last few years. In 1995, 235 pigs were submitted (4.5% of all submissions), whereas, in 1999 there were 168 (3.6%) submissions. Animals of all stages of production were presented, with a bias to those animals from production facilities in the Fraser Valley, and to pig populations exhibiting increased morbidity or mortality.

Usually, 1 to 2 diagnoses were identified per case. Individual case diagnoses comprised either a single animal with multiple diagnoses, or multiple pigs within a group with a single diagnosis. With the exception of fetal diagnoses (represented by a preponderance of idiopathic or unknown-cause abortions), the overwhelming diagnoses in case submissions reflected either an infectious or inflammatory process.

Pathologic diagnoses of suckling piglets less than three weeks of age involved primarily the gastrointestinal tract (11 cases) with *Escherichia coli K88*, nonhemolytic *E coli*, and rotavirus comprising most of the detected pathogens. Respiratory disease was recognized in two cases, musculoskeletal in two, followed by generalized septicemia identified in a single case.

In weaner pigs (3 - 8 wks) most diagnoses involved the respiratory system (20) and consisted of *B* hemolytic *Streptoccocus, Pasteurella multocida* and *Haemophilus parasuis*. There were 8 diagnoses of septicemia primarily due to *Streptococcus suis,* with fewer cases of gastroenteritis (2), encephalitis (2), and dermatitis (1).

Continued Page 8

Disease summary of pig submissions *(Continued from page 3)*

In grower pigs (8-14 wks) there were 13 diagnoses of septicemia, consisting primarily of *Actinobacillus suis* (7 cases), *Pasteurella multocida* (2), and miscellaneous bacteria (1). There were also 10 cases of pneumonia, associated with *Actinobacillus suis* (4), *P multocida* (1) and *Arcanobacterium pyogenes* (1); mixed miscellaneous organisms were isolated in 4 cases. A single case of enteric salmonellosis was diagnosed by culture.

In finisher animals (15 - 21 wks) there were nine cases of enteritis, from which there were four isolates of *Salmonella spp*. bacteria. There were also five cases of pneumonia and three diagnoses of meningitis attributed to *S. suis*.

Fewer overall adult accessions were recorded. Most case diagnoses consisted of pneumonia, nonspecific gastroenteritis, and musculoskeletal lesions. A single case of *Actinobacillus suis* septicemia was identified.

One of the most significant diseases processes identified in this review is septicemia or pneumonia due to *Actinobacillus suis*. With the exception of 1998, there have been increased isolations of this organism since 1996. This bacterial pathogen is not only an emerging pathogen within the Fraser Valley, but also across Canada. Based on this review (with the exception of fetuses), infection with *A. suis* spans all age groups, with the greatest number of isolates in grower-aged animals. The disease is generally characterized by sudden death of healthy pigs via either septicemia or pneumonia. Resistance pattern of this organism to antibiotics also appears to be expanding, and clinically the condition is difficult to treat.

Ed's note: please see additional comments in this issue of Diagnostic Diary reprinted from the September/ 2000 U of Guelph Newsletter, which also discusses the increased prevalence of A. suis in swine disease.

Equine congenital cutaneous papilloma and other lesions in a nonviable foal:

J. Coates (AHC) & J Jackson (Kamloops Veterinary Clinic)

Tissues were received from a nine day-old non-viable purebred Quarterhorse foal that died following a short illness. On microscopic examination, severe goiter (thyroid hyperplasia) was diagnosed on sections of thyroid tissue and, within the heart muscle, there were areas of acute degeneration and mineralization consistent with a nutritional cardiomyopathy. No leg or joint abnormalities were observed, although mandibular prognathism (lengthening of the bottom jaw or mandible relative to the upper jaw), was observed.

The goiter observed in this neonatal filly was considered secondary to iodine deficiency within the pregnant mare's diet. In some instances, although dietary iodine may be adequate in pregnant mares, sources of nitrate within the feed and/or water may impair normal uptake and utilization of the element, resulting in a secondary goiter and inadequate thyroxine hormone production. Multiple metabolic deficiencies affecting different organ systems, linked directly to iodine deficiency, soon develop within the fetus and are apparent in those animals that survive to birth.

In this particular case, the degenerative heart changes were thought to be linked to a primary vitamin E and/or selenium deficiency. In summary, the nutritional status of the pregnant mare was evidently deficient in several areas. Interpretation of the filly's original selenium or vitamin E status was not possible, as the animal had been treated with injectable vitamin E/selenium within a few days of birth.

The most interesting area of investigation however, centered on the presence at birth of obvious raised skin lesions on the lips, nostrils, eyelids, over the rib cage area, and down the legs. Interestingly, these lesions were limited primarily to the right side of the body with the exception of the head, where lesions were on the face, including the ears.

Microscopically, the lesions were consistent with cutaneous papilloma, characterized by delicate "cauliflower" areas of epidermal proliferation. The epidermal layer of the skin was of variable thickness, and there were frequent, prominent accummulations of superficial keratin (orthokeratotic hyperkeratosis); underlying dermal or connective tissue proliferation was minimal.

Congenital cutaneous papillomatosis occurs rarely in domestic animals, and is mentioned only briefly if at all, in most standard texts. In the equine species, one of the most recent reports discusses 5 separate cases of equine congenital papillomatosis [1]. Evidently there has been little research on the papilloma virus of horses or small animals but, in cattle, 6 different bovine papilloma (DNA) viruses have been identified[2]. In horses, papillomas (warts) are commonly seen in horses less than 3 years of age[3], but can be seen at any age. Spontaneous recovery is usual, but the proliferative skin lesions may persist for 5-6 months[2].

Electron microscopy failed to yield direct visual evidence of virus in the skin specimens. In addition, wax blocks of tissue from this case were sent to the Prairie Diagnostic Centre at Saskatoon, for identification of bovine/equine papilloma virus, via immunohistochemistry. These results were also negative. Equine cutaneous papilloma is presumed caused by a host-specific DNA-containing papovavirus[4]. The congenital form shows no breed or sex predisposition; skin lesions may persist for 1-9 months, and then regress[4].

(1). Valli VE, Lumsden JH. Equine congenital cutaneous papillomatosis: a report of 5 cases. *Equine Veterinary Journal*, 1981; 13: 59-61.

(2). Radostits OM, Gay CC, Blood DC, Hinchcliff KW. *Veterinary Medicine,* 9th ed; 2000: 1244-1247. WB Saunders Co, New York

(3). Reed SM, Bayly WM. *Equine Internal Medicine*, 1st ed 1998: 551-552. WB Saunders Co, Philadelphia, Toronto.

(4). Rooney JR, Roberston JL. *Equine Pathology*, 1st ed 1996: 306. Iowa State Univ Press.

Hemophilus somnus pneumonia in a calf:

J Coates (AHC) & J Jackson (Kamloops Veterinary Clinic)

Fresh and formalinized specimens were submitted to the laboratory from a crossbred, month-old nursing crossbred beef calf. The calf was one of three that had recently died while at pasture, from a cowherd of 200 animals. Treatment had been attempted by the owner but the animal failed to respond and died the following day. Pneumonia had been diagnosed by the practitioner on gross necropsy, and ***Haemophilus somnus*** was suspected as a possible cause.

Microscopic examination of lung tissue revealed a **severe, subacute, fibrinosuppurative bronchopneumonia**. *H. somnus* was cultured from the lung in heavy growth. No viral agents were identified or isolated.

According to Radostits et al (1), the pneumonic form of disease caused by *H somnus* is being seen with increasing frequency, although detailed lesions have seldom been clearly described in the literature. The pneumonic form is described in feedlot calves; isolating the organism from a month-old calf as seen in this case is less commonly reported. The calf was nursing its mother at pasture, at the time of its illness and death.

Continued Page 6

Hemophilus somnus pneumonia in a calf *(Continued from page 5)*

The range of clinical findings associated with *Haemophilus somnus* infection in cattle has changed remarkably in the last two decades. Historically, thromboembolic meningoencephalitis (i.e., blood-borne bacteria localizing within brain vessels) was the major form of the disease, while today this syndrome is seen less frequently; other expressions of the condition including pneumonia are eye lesions (opthalmitis); ear infection (otitis); and inflammation of the heart muscle (myocarditis).

Methods of transmission and portal of entry of *H somnus* are unclear. Evidently a feature of this organism is its persistence at mucosal sites within the body in both subclinical and diseased animals. The organism can be isolated from the respiratory and reproductive tracts of normal animals[1]. In this case, it is presumed that the organism was circulating within the cowherd, and that the calf likely acquired the organism from its mother.

Satisfactory control measures are not available, because the pathogenesis and epidemiology of the disease are poorly understood. During an outbreak, constant surveillance to detect early clinical cases, combined with early treatment, are the most economical and effective means of control[1]. Vaccines in various forms (usually as killed bacterins) have been available to veterinarians and producers for prevention of *H somnus* infection and disease for some time, but their effectiveness is still uncertain[1].

(1). Radostits.O M, Gay CC, Blood DC, Hinchcliff KW. *Veterinary Medicine,* 9 th edition; 2000: 895-901.

Pasteurella trehalosi in California Bighorn sheep:

H Schwantje

Pasteurella trehalosi was recently cultured from the lungs of a free-ranging male **California Bighorn sheep** *(Ovis c.californiana)*. In the past year there has been a die-off of these animals in the south Okanagan valley of British Columbia. Chronic-active bacterial bronchopneumonia, combined with moderate to heavy lungworm infestation, has been a consistent finding. Numerous lungworms (*Protostrongylus* spp), both larvae and adults, were observed in alveoli as well as in bronchioles, suggesting infection with *Pr. stilesi* and *Pr. rushi*. ***Pasteurella multocida*** has been the predominant organism identified, although other bacteria such as *Arcanobacterium pyogenes* (formerly *Actinomyces pyogenes*) have also been cultured from the lungs of these animals. *Mycoplasma spp* have been identified in lung tissues, via PCR, as well as viruses (PI3 virus).

Although *P. trehalosi* has been positively identified in only one of the specimens, its isolation is significant, and represents the first time that a *P. hemolytica*-type organism has been cultured from pneumonic lung in this recent bighorn sheep die-off. The organism is a strain of *Pasteurella* bacteria that may still be referred to as ***Pasteurella hemolytica biotype T***. A number of different *P trehalosi* biotypes have been identified in bighorns; all likely have the potential to cause or contribute to disease, depending upon the immune status of the animal. Researchers are still attempting to understand the disease processes that occur during a die-off, where even supposedly less pathogenic biotypes may become more virulent. In addition to *P trehalosi, Pasteurella hemolytica* was identified from a nasal culture swab taken from a ewe that had died several months earlier in the same general region; however, the biotype was not determined.

Pneumonia is commonly observed in bighorn sheep die-offs. *P. trehalosi* is recognized as causing both acute and chronic forms, alone or in combination with other bacteria, viruses, and various levels of lungworm infection. While *P trehalosi* has been isolated in some bighorn sheep die-offs, it is important to note that it can also

be cultured from the tonsils of healthy bighorns. Although important in terms of an individual animal's death, this and other infectious organisms isolated from dead bighorns are only one aspect of the disease process; there are many factors that influence the health of these animals long before they develop pneumonia. In this respect, the pneumonia complex of bighorn sheep resembles the shipping fever complex of domestic cattle – a complex of contributing stresses that includes infectious agents, body condition and nutritional status, as well as a host of environmental influences.

Toxoplasmosis in a juvenile lemur:

S Raverty (AHC) & D Huff (Huff Animal Hospital, Tsawwassen, BC)

One of two juvenile pet lemurs, recently imported from Ontario, was presented for clinical examination following an acute onset of lethargy and weakness. Examination disclosed an enlarged, fluctuant abdomen, and (x-ray) radiography revealed a markedly distended, gas-inflated, small intestine. Clinical pathology featured an inflammatory leukogram. The lemur died during the latter stages of the examination.

At necropsy, the lemur was in moderate body condition with generalized jaundice (yellow discoloration of the eyelids and membranous lining of the oral cavity). There was marked accummulation of edema fluid within subcutaneous tissue of the ventral abdomen. Approximately 500 mL of tan-yellow fluid, which readily clotted on exposure to air, was observed within the abdominal cavity *per se*. The small intestine was diffusely inflated with gas, and small portions of pale yellow, tenacious food material adherend to the mucosa. A prominent mesenteric lymph node featured abundant inflammatory necrotic debris on its cut surface. The liver was moderately enlarged, with numerous small, discrete necrotic areas observed throughout its parenchyma. Variable amounts of proteinaceous fibrin strands overlaid the diaphragmatic aspect of the liver.

Random areas of acute heart muscle degeneration and necrosis were observed microscopically, together with variable numbers of a mixed inflammatory cell populations (lymphocytes and histiocytes, with fewer plasma cells and neutrophils). Small clusters of extracellular and intracellular protozoal bradyzoites (4 – 6 um in diameter) were seen widely dispersed throughout the heart muscle and, more rarely, interspersed within necrotic foci. Immunohistochemistry tests were positive for toxoplasmal antigen within the areas of heart injury. Blood serology detected elevated antibody titers of 1:256 for *Toxoplasma gondii* (consistent with acute exposure) in this lemur. Follow-up serology of the second lemur involved in this case was declined by the owner.

The cause of death was attributed to fulminating, multisystemic, toxoplasmosis.

Toxoplasma gondii are ubiquitous, obligate, intracellular coccidial parasites. The cat and related felids are the definitive hosts, with generalized systemic involvement recognized in a number of animals, including humans. Infection in this animal was likely due to exposure to contaminated feed. Infection in herbivores (e.g., sheep or goats) is generally acquired by consumption of feed contaminated with toxoplasmal oocysts, whereas in carnivores (e.g., dogs and related canids) it is through ingestion of meat containing bradyzoites within cysts. Although *T. gondii* is a zoonotic organism (i.e., capable of transmission from animals to man), risk of transmission of this parasite from the lemurs to the owner was not considered significant. Morphologically, principal diagnostic differentials for the protozoa observed within areas of injured heart muscle would include *T. gondii, Neospora caninum, Sarcosporidia spp, Isospora spp, Leishmania donovani* and *Trypansoma cruzi*.

Short cuts from the Post Mortem Room:

Horses of every size and breed, or submitted portions thereof, are a continual observation at the AHC. They frequently present unique and, at times, dramatic lesions. A case of **equine congenital cutaneous papillomatosis** has been described earlier in this newsletter. In another case, a massive slab **fracture of the occipital bone of the skull with secondary severe hemorrhage** was observed in a mature horse that inadvertently rolled or fell backwards after rearing at the end of a lunging rope. The fracture line ran horizonatally and anteriorly through both occipital condyles, then extending into the basilar portion of the occipital bone (the base of the cranial cavity that supports the brain). The occipital fracture and separation line was a centimetre wide; massive hemorrhage surrounded the brain stem and cortex, and extended into the underlying soft pharynx. The animal died about an hour following the injury.

❖ ❖ ❖ ❖ ❖

A **meningeal melanoma tumor** was diagnosed within the cranial cavity of a dappled-white to grey thoroughbred gelding, approximately 13 years of age. The horse had been showing unusual and vague nervous signs referable to the brain or cranial nerves for over a year, which had been gradually progressing in severity. The tumor mass, 3-4 cm in diameter, was located immediately posterior to the cerebellum, overlying the dorsal aspect of the medulla; the latter appeared moderately flattened, or compressed. Melanoma cells were also observed advancing along the perineural sheaths of some of the cranial nerves ventral to the medulla, and a lymph node adjacent to the right guttural pouch was also involved. No tumors were observed on the perineal area of the animal.

❖ ❖ ❖ ❖ ❖

Severe **copper deficiency** was detected at necropsy of a mature llama, with a liver copper level of 3.0 ppm (wet weight). Adequate copper levels in mature llamas are recorded in the literature at 25 – 100 ppm (Puls RE: *Mineral Levels in Animal Health* 1994).

❖ ❖ ❖ ❖ ❖

Canine distemper was diagnosed in a seven week-old unvaccinated pup from Surrey. The animal was euthanized after showing progressively severe evidence of brain irritation, characterized by sporadic convulsions. Microscopically, the animal had acute vacuolation and necrosis of neurons, most evident within the medulla; swelling (edema) of the brain tissue was also present, together with a mild increase in brain tissue cellularity (gliosis). Viral inclusions bodies were not readily detected within brain tissue, although the odd structure resembling an inclusion body was seen elsewhere, most notably within the mucosal cells of the glandular stomach. The animal also had diffuse bronchointerstitial pneumonia, characteristic of distemper virus. Canine distemper virus was identified via the polymerase chain reaction test procedure (PCR), which identifies viral DNA.

❖ ❖ ❖ ❖ ❖

Subacute, severe, **suppurative bronchopneumonia** was diagnosed in a litter of eight week-old **Whippet pups** that had a clinical history of ongoing respiratory distress. Heavy growth of ***Bordatella bronchiseptica*** was identified on bacterial culture. There was no evidence of viral involvement. Culture and PCR tests for canine distemper virus were negative.

Hemolytic E. coli O149:K88 was isolated in heavy growth from a group of five week-old weanling pigs that were dying acutely. On post mortem, intestinal contents were a reddish color suggestive of blood; the mucosa of the small intestine was deeply congested to hemorrhagic, and delicate threads of fibrin adhered to the intestinal mucosa or within the intestinal lumen contents. A large number of pigs were at risk in the group, and acute losses had already occurred.

❖ ❖ ❖ ❖ ❖

Acute fatal **embolic pneumonia** was diagnosed grossly and microscopically in a 22 month-old Holstein milking cow that was unexpectedly found dead in its stall one morning, prior to milking. There had been no history of illness. Necropsy revealed a large liver abscess, located near a portal vein. It is thought that bacteria entered the systemic circulation of the animal following erosion of a venous vessel wall by bacteria, resulting in a showering of the lung with myriads of bacterial organisms. Culture of the abscess and lung tissue revealed a mixed population of organisms including nonhemolytic *E coli*, *Corynebacterium sp* (not *pyogenes*), alpha *Streptococcus sp*, *Pasteurella sp*, and *Proteus* sp.

❖ ❖ ❖ ❖ ❖

A perforated **abomasal (stomach) ulcer** was diagnosed in a 6 week-old female Holstein calf, that died after a brief illness. The discharge of abomasal contents into the abdominal cavity of the animal rapidly led to shock, toxemia, peritonitis, and death. The specific or definitive cause(s) of abomasal ulcer in calves is still unknown[1], but there is an association with changes in feed or feed management. This calf was receiving calf starter pellets at the time.

Other findings were a significant amount of wood shavings within the animal's stomach compartents, together with normal milk curd. The wood shavings may have been acting as a local irritant to the delicate mucosal lining of this neonatal animal's glandular stomach (abomasum). A suppurative bronchpneumonia was also observed, from which nonhemolytic *E. coli* was cultured in heavy growth.

Thyroid hyperplasia, or goiter was also present. The broad-reaching effects of the thyroid hormone, thyroxine, on an animal's well-being, including metabolic influences leading to activation of intracellular mitochondrial energy metabolism and the promotion of intracellular new protein synthesis, are well known[1].

Abomasal ulcers in calves have several clinical expressions. The incidence of abomasal ulcers in milk-fed vealer calves is higher when the animals have access to roughage than when roughage is not provided. Calves are also susceptible to abomasal ulcers when they are changing from a diet of low dry matter content (milk or milk replacer) to one of a higher dry matter content (grass, hay, grain). The type of roughage to which calves are exposed may also be a factor. Pellets produced from corn silage were associated with more lesions than pellets produced from barley straw or alfalfa hay[1].

Abomasal ulcers also occur in **young suckling beef calves** at pasture, often between three-twelve weeks of age, and their pathogenesis is similarly not fully understood. Perforating ulcers may be seen in conjunction with the presence of stomach or abomasal hairballs (so-called trichobezoars), but whether hairballs initiate ulcers or develop later, is not known[1]. The significance of hairballs in initiating abomasal ulcer is doubtful, since hairballs are seen in calves that die from reasons other than those related to the abomasum.

In suckling beef calves with perforated ulcers there is no sex predilection, nor evidence of breed predisposition. The bacterial agents *Clostridium perfringens* type A,

Continued Page 10

Short cuts from the Post Mortem Room *(Continued from page 9)*

Helicobacter pylori, or *Campylobacter spp,* do not appear to be directly involved in ulcer formation. This writer has often investigated the liver copper levels in suckling beef calves with perforated abomasal ulcers submitted to the AHC for necropsy. Some animals have liver copper levels that are low and others do not. To date, there does not seem to be a distinct relationship.

(1). Radostits OM, et al: *Veterinary Medicine*, 9th ed, 2000: 335 - 339

❖ ❖ ❖ ❖ ❖

Canary pox virus was identified in a two year-old canary that demonstrated markedly proliferative and inflammatory lesions over one eyelid and adjacent skin tissue of the head. Numerous large viral inclusion bodies were readily seen within the affected tissue microscopically. Poxvirus was identified via electron microscopy. The submitted bird also had evidence of systemic pox infection (viremia), characterized by extensive tissue injury and cell necrosis within the spleen and liver.

While several birds in the aviary were developing lesions over various parts of the body including the head, others developed blister-like lesions on the distal scaly portions of the legs and claws. The number of affected birds was increasing. Following diagnosis, the remaining healthy birds were immediately immunized by the owner with a live canary pox vaccine.

Canary poxvirus should be considered enzootic in most flocks of canaries, and birds of any age are susceptible. Canaries can develop the skin, diphtheritic, or viremic form of the disease. The viremic form is commonly observed. Canaries may also develop a chronic form of avian pox characterized by wart-like lesions on the eyelids and beak, or a diphtheritic form characterized by lesions in the windpipe (trachea), and oral cavity. The poxvirus that infects canaries is the most lethal member of the pox family, and most birds with the viremic form of the disease die. Mortality rates in a flock vary from 20-100%, depending on virus strain. Canary pox infections can be prevented by using a commercially-available attenuated live vaccine that provides temporary immunity. (B.W. Ritchie. *Avian Viruses: Function and Control*. 1995: 302 – 304. Wingers Publishing Inc.)

Pulmonary botryomycosis in an adult capybara (*Hydrochoerus hydrochaeris*)

S Raverty

The capybara (*Hydrochoerus hydrochaeris*), the world's largest known rodent, is a semi-aquatic rodent from South America. The animal may attain lengths up to 100-135 cm, heights of 56-60 cm, and a weight of 35-60 kg. The animal is indigenous to South America, in the region east of the Andes Mountains, and is primarily terrestrial. In the wild, the life span of these animals ranges from 8-10 years, and in captivity up to 12 years.

A capybara presented to the AHC for post mortem examination was severely emaciated, with bilaterally symmetric hair loss throughout its dorsal thoracolumbar or back area. There were multifocal to coalescing tan-yellow to red nodules observed within the lung, immediately below and elevating the lung's pleural membrane, as well as extending deeply into underlying tissue. The nodules bulged on cut surface, and were occasionally gritty on sectioning.

The nodules consisted of varying numbers and concentrations of small, spherical to lobulated granules which centrally contained poorly delineated, gram negative bacillus bacteria, ensheathed by peripherally radiating, brightly eosinophilic (Splendore-Hoeppli) material. These so-called botyromycotic granules were

circumscribed by mild to moderate accumulations of degenerate and viable neutrophils, with fewer macrophages, epithelioid cells, Langhan's type multinucleate giant cells, and sparse amounts of dense, eosinophilic necrotic debris. Death was attributed to botryomycosis of the lung and subsequent emaciation.

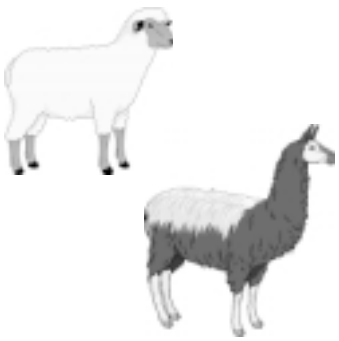
In humans, domestic livestock, and companion animals, botryomycosis is a chronic, sporadic bacterial infection usually localized to the skin and subcutaneous tissue, and more rarely within internal viscera. The condition is characterized microscopically by the formation of eosinophilic (red-staining) granules similar to those of actinomycosis and actinomycotic mycetoma, but can be differentiated by routine culture and microscopic examination. The most commonly isolated bacteria are *Staphylococcus aureus, Escherichia coli,* and *Streptococcus spp*, with *Proteus spp,* and *Pseudomonas aeruginosa* less frequently identified. In this case, heavy growth of *Pseudomonas spp* was obtained from the lung and adjacent mediastinal lymph nodes. The pathogenesis of this condition has not been fully resolved, but likely reflects an imbalance between the host animal's immune response and bacterial virulence factors.

Type C botulism in dairy cattle linked to a dead cat:

" Four hundred twenty-seven of 441 adult Holstein dairy cattle from a 1,200 –cow dairy died over a one-week period during early spring 1998. Affected animals were from four lactation pens, one of which included the bullstring. Signs included weakness, recembency, watery diarrhea, and death. Eighty animals from the pens were dead approximately eight hours after the first ill cows were noted. Affected cows would collapse on stimulation and extend all 4 limbs with moderate rigidity… The animals had been fed a load of total mixed ration that included a bale of rotten hay containing a dead cat. No common toxicants were identified, and pathologic examination revealed no consistent lesions. Testing of tissue from the cat carcass found in the feed sample using mouse protection bioassay identified the presence of type C botulinum toxin. Samples of feed, tissue from affected animals, cat tissue from feed, as well as milk and blood serum were also tested using an enzyme-linked immunosorbent assay (ELISA) specific for type C *C botulinum*. Two samples of rumen contents were tested and found to be positive for botulism by ELISA, and 1 of 3 liver samples had a weak positive finding. No botulinum toxin was found in milk or sera using the ELISA."

Abstract taken from the article by Galey FD, Terra R, et al.: *Type C botulism in dairy cattle from feed contaminated with a dead cat. J Vet Diag Invest* 2000; 12: 204 - 209.

Isolation of *Giardia* from a llama and a sheep:



"…*Giardia* cysts were detected in feces of a domestic llama (*Lama glama*) and in feces of lambs (*Ovis aries*) from Wisconsin, USA. All animals examined were immature, and they had recent histories of poor condition and passing unformed or semiformed, pale stools. *Giardia* cysts from both host species were excysted *in vitro,* and the trophozoites were cultivated… Furthermore, *Giardia* cysts from both sources were shown to produce infection in Mongolian gerbils (*Meriones unquiculatus*). The finding of *Giardia* in the llama represents a new host recorded for this parasite. Also, this is the first report of *Giardia*-infected sheep in the Western Hemisphere…"

Kiorpes, Kirkpatrick CE, Bowman DD. *Can J Vet Res* 1987; 51: 277 – 280.

Excerpts from the University of Guelph's Laboratory Services newsletter

(vol 4, no.3, Sept. 2000):

(*Ed.'s note: we reprint this material from the most recent issue of the Ontario Animal Health Laboratory Services newsletter, with thanks to our Ontario colleagues, and especially to Dr. G Maxie, editor.)*

***Clostridium difficile* enterocolitis in animals:** "…*Clostridium difficil*e is a strictly anaerobic, gram-positive to gram-variable, spore-forming bacterium that is becoming increasingly recognized as a cause of gastrointestinal disease in humans, horses, and pigs… *C difficil*e is a highly diverse organism, with more than 400 unique types, and has several virulence factors. Exotoxins A and B are the most significant factors, and bacterial production of exotoxins is correlated with pathogenicity of individual strains of *C difficil*e. .. *C difficil*e is currently the most commonly diagnosed cause of colitis in adult horses and foals at the Ontario Veterinary College, accounting for 20 – 25% of cases… *C difficil*e has been identified in association with diarrhea in **foals** as young as one day of age. Clinical signs can vary from mild diarrhea to severe hemorrhagic, necrotizing enterocolitis, and the small intestine is more consistently affected in young animals than in manure horses… The lack of an established intestinal microflora may make foals more susceptible to colonization by *C difficil*e, although human infants frequently have the organism in feces but rarely develop disease…

Documented reports of *C difficil*e infection in **pigs** are sparse, but are restricted to young pigs. Most significant is evidence of diarrhea and increased mortality in neonatal pigs between 1 and 7 days of age, although one report describes disease in 8 week-old weaners…."

…Preliminary studies suggest this organism may be an important cause of **canine** diarrhea. *C difficil*e was also implicated as the cause of severe diarrhea in two cats from the same household…"

…the sensitivity, cost-effectiveness, and relative speed of the toxin ELISA test make this assay a practical diagnostic method in conjunction with appropriate history and clinical signs.."

— *Ontario AHL Newsletter*, September 2000: De Lay J, Archambault M, and Weese JS: *Clostridium difficile enterocolitis in animals. Ed's note: an ELISA test for identification of C difficle toxin in intestinal content samples is also available at the AHC's Animal Health Monitoring Laboratory. Please contact L Curley, supervisor of the AHML.*

❖ ❖ ❖ ❖ ❖

PRRSV and *Actinobacillus suis* interaction: "…The interaction of respiratory disease virus (PRRSV) and other pathogenic disease agents within the same animal population has been known to enhance the clinical manifestations of these agents. **In this report, we suggest a possible link between PRRSV and Actinobacillus suis infections.**

…*Actinobacillus suis*, once considered an opportunistic pathogen, is believed to be a common commensal organism in conventionally reared swine, with these herds showing only sporadic disease, generally associated with stress or trauma. **Outbreaks of clinical disease due to *A suis* occur more frequently in minimal-disease or high-health status herds, suggesting that a lack of immunity in these pigs may be important in disease expression**. A review of submissions to the AHL from 1997 and 1998 revealed that 35% of *Actinobacillus suis* submissions were from grow/finisher pigs… With sudden unexpected deaths being the most common history…"

— Josephson G, Charbonneau G, Archambeault M, Carman S, Cai H. *PRRSV and Actinobacillus suis interaction*. Ontario AHL Newsletter, September 2000.

The roots of toxicology: an etymology approach:

"The words "toxic" and "toxicology" have Greek origins. From early antiquity they have been connected with substances that cause death, and the meaning of the two words derived has prevailed throughout the ages. Their etymology brings us back to the ancient world, where hunters daubed their bows, named "toxa" in Greek, with poisonous substances. This is where use of the words "toxic" in English and "toxique" in French or other languages derives from.

The words come from the ancient Greek "toxon" which means bow. The word "toxon" derives from the Mycenean word "to-koso", which might have been borrowed from the Skythic language (+tax'sa) if we accept that they were famous for their bows…

Looking back to the 4th century BC, Aristotle (1) seems the first to speak about a medicine that was called "toxic" by Celts. The medicine brought on death easily and quickly when hunting. It was used when the hunters wanted to offer the dead animal to a god or to prevent an animal's rotting.

Strabo (2) mentioned in his work, *Geographica*, that in the Iviriki area (today's Albania and Yugoslavia), the local population of Ivires used a special substance called "toxicon" to bring on death. It was a botanical mixture that resembles the "selinon" (celery) plant and is an analgetic…

Orivasios (5) clarified that toxical medicine is an ointment the ancients daubed their bows with. He mentioned that if someone drinks it, he might not escape death. As an antidote he instructs drinking decoction of gonglyidos (a plant) seed or milk of prinos (also a plant), and eating the root of the pentafillon plant mixed with goat's blood, or cydonia apples…"

— From the article by Ramoutsakis IA, Ramoutsakis YA, et al: The roots of toxicology: an etymology approach. *Vet Hum Toxicol* 2000; 42: 111.

The oncologist's debt to the chicken:

(Ed's note: an oncologist is an individual who studies tumors)

"Our understanding of avian tumour virology has laid the groundwork for much basic cancer research. Most outstanding has been the delineation of oncogenes*. These were first discovered in avian leukosis and sarcoma viruses, but are of fundamental importance in most types of non-viral cancer too, both in animals and humans. Avian tumour viruses gave us the tools to to probe complex signalling pathways in the cell that go awry in cancer. Avian retroviruses have also aided medical research in other ways: HIV infection is treated by anti-retroviral drugs directed against enzymes, such as reverse transcriptase, first discovered in chicken viruses… Marek's disease virus (MDV) has served as a model transforming lymphotropic herpesvirus for human lymphomas and lymphoproliferative disease caused by Epstein-Barr virus. Unravelling a novel human herpesvirus causing Kaposi's sarcoma also owes a debt to the chicken, not only to MDV, but also to the choriollantoic membrane assay as a model for tumour angiogenesis… Once again, oncologists are indebted to the chicken!"

(*an oncogene is a gene capable under certain conditions of causing the initial and continuing conversion of normal cells into cancer cells. The term may be used to denote such a gene in a viral genome or a cellular gene derived from alteration of a proto-oncogene…)

— From the abstract and review article by Robin A Weiss, Institute of Cancer Research, Chester Beatty Laboratories, London, England. Published in *Avian Pathology*; 1998: 27: S8-S15.

A Child's Perspective on the Animal Health Centre:

While going over papers in his office recently, Dr. Jeremy Greenfield, the AHC's senior microbiologist, discovered a letter received by the Laboratory in 1972 from a group of grades six to seven elementary school children had been shown through the old veterinary laboratory on Gladwin Road, in Abbotsford. The note below (replete with innovative spelling and grammar) is a composite of letters written by the schoolchildren. In the words of Dr Greenfield, written at that time, "our intent is not to laugh at the students who were alert, interested and inquiring, but to laugh with them. It is also us who must appear to be eccentric characters, performing curious tasks".

" Dear Dr Macdonald and Staff,

Thank you for taking our class through the Veterinary Laboratory, the most thrilling experienced tour I have ever had in my life. I thought it was really interesting. I think that this is very important to the farmer and for the everyday person. I am sure it will help me in my science class… There must be thousands of dollars to put in to make the laboratory worth while. I figured the building and all the equipment must have cost a fortune…

We enjoyed you telling us about Pathology, it was nice knowing about for an hour. If you want to be a Pathologist how many years do you have to go to university?… Those words are sure hard to remember. All I remember is toxicology, which means the study of poison… The chemistry room was big and filled with everything the mad scientist could think of…I liked the Post-Mortem room where you had a lamb. How come lambs have 4 stomachs. I never knew the intestines of a sheep were about 100 ft. long. That really astonished me. That dead cow wasn't the greatest, because of it I never ate my lunch. I hope we will come again. The part I liked best was all the little chickens on the table, that were split open… The calf, the Siamese pigs, the unborn monkey were all so interesting. When I told my mother about the Siamese Piglets she thought it was quite ridiculous, but I just happened to think it was cute…I have a question for you. What do you do with the eyes of the animals after you cut them open?…Of all I liked best the furnace which you put the animals when you're through with them, sure would hate to fall in. But I still don't understand it when you burn the animals and no smell or smoke comes out. I liked the idea of a non-pollution incinerator.

"…In the room with bacteria, I learned that there are millions of germs in one little thing. I thought it was really something to see what caused pimples. I had a boil and I got so bad I got put in iceilation and I couldn't walk on it… I also enjoyed seeing the live rabbits, hamsters, mice and gerbils. I wish I could have kept one there so sweet.

I could talk about the laboratory all day but I haven't any more time. It was all we talked about days after. I don't know if I'd like to work there though because I don't like blood anywhere but on myself. After our trip some of us had butterflies in our stomachs and could hardly eat. But we still enjoyed it. I sure wish we could go again.

"…I hope some day the only cause for animals to die is old age. I wanted to be a veterinarian before, but now I wanted it even more. Thanks for everything…so until the snow falls good-bye".

Yours sincerely, (yours truly, yours for ever, my honour, from)

"N. Students"

P.S. Thanks for telling me what went wrong with my rabbit."

— with thanks to Dr Jeremy Greenfield, who gladly shared this small piece of written laboratory history for all of us to read and enjoy. And no, we haven't kept rabbits, hamsters, gerbils or other laboratory animals in our laboratory for many years. Ed.

Focus on Staff: Wanda Fiessel BSc, MLT

The Animal Health Centre (AHC) is happy to announce that Ms. Wanda Fiessel recently joined the bacteriology section of the laboratory. Wanda's home is southern Saskatchewan. Later, she moved to Regina, where she acquired her Bachelor of Science degree (majoring in both microbiology and biochemistry), as well as a Certificate in Laboratory Technology from the University of Regina, in 1988. Ms. Fiessel received her practical training for general Medical Technology at Pasqua Hospital in Regina, later registering with the Canadian Society of Laboratory Technology (CSLT) in 1989.

Wanda has worked for the Pasqua Hospital Chemistry department, prior to obtaining a position as Senior Bacteriology Technician with Professional Medical Laboratories, of Saskatoon. Later, she worked as Quality Control Specialist at Kalium Potash Corporation, in Belle Plaine.

Wanda served on several committees of the Saskatchewan Society of Medical Laboratory Technology (SSMLT) that were responsible for organizing scientific conferences, laboratory licensing, as well as developing health and safety guidelines for laboratory workers. Ms. Fiessel also served as the Continuing Education representative for the province of Saskatchewan.

Ms Fiessel has also worked at the Saskatchewan Veterinary Laboratory in Regina, where she became Senior Bacteriology Technologist. Although she worked in all departments of the laboratory, her main interests were in research and development of new testing methods in veterinary bacteriology, including studies on *Mycobacterium paratuberculosis*, the cause of Johne's disease. Wanda also became interested in the various strains of coliform organisms responsible for neonatal enteritis, especially in calves and pigs; as well as the daily identification of bacterial organisms significant to domestic livestock, such as *Actinobacillus suis, Streptococcus suis, and Hemophilus parasuis*. In addition to these areas of endeavour, Ms. Fiessel implemented quality control procedures within the Regina laboratory.

Wanda has numerous pastimes and hobbies, particularly soccer, which she has both played and coached.

Wanda's background of experience, coupled with skills honed at the Regina Veterinary Diagnostic Laboratory, bring added depth and knowledge to the Bacteriology section of the Animal Health Centre, and to the laboratory as a whole.

We welcome her to British Columbia, and to our laboratory.

Art and reality:

"…Through art alone are we able to emerge from ourselves, to know what another person sees of a universe which is not the same as our own and of which, without art, the landscapes would remain as unknown to us as those that exist on the moon. Thanks to art, instead of seeing one world only, our own, we see that world multiply itself and we have at our disposal as many worlds as there are original artists, worlds more different one from the other than those which revolve in infinite space, worlds which, centuries after the extinction of the fire from which their light first emanated, whether it is called Rembrandt or Vermeer, send us still each one its special radiance…"

— from *Time Regained: In Search of Lost Time*, vol vi by Marcel Proust (1871 – 1922).

The Beaver people:

"…Late in the fall the house is well plastered with mud, and it is by observing the time of this operation that it is possible to forecast the near approach of the freeze-up.

And it is the contemplation of this diligence and perseverance, this courageous surmounting of all difficulties at no matter what cost in labour, that has, with other considerations, earned the beaver, as far as I am concerned, immunity for all time. I cannot see that my vaunted superiority as a man entitles me to disregard the lesson that he teaches, and profiting thereby, I do not feel that I have any longer the right to destroy the worker or his works performed with such devotion…."

— from Grey Owl's *The Men of the Last Frontier*: 1st published 1931.

Microbiologist employment opportunity:

With the imminent retirement of Dr. Jeremy Greenfield, our veterinary diagnostic laboratory will shortly require an enterprising and enthusiastic DVM/PhD or PhD microbiologist to fill this supervisory position, as Head of the Bacteriology section of the laboratory.

If you are interested in this position, please contact Dr. Ron Lewis, Director of the Animal Health Centre, at 1-604-556-3003. Dr. Lewis's mailing address is on the letterhead of this newsletter.