Animal Health Centre NEWSLETTER

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CONTENTS

In this issue:

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From the Director

RJ Lewis:

Helping to Protect the Livestock Industry from Animal Diseases (Foreign and Otherwise)

In the last newsletter, I discussed foot-and-mouth disease and our preparations, should we ever be faced with such a challenge. Since that time, we have updated our Foreign Animal Disease Eradication Support plan, subsequent to several meetings with provincial and federal emergency planners, the Canadian Food Inspection Agency (CFIA), and other provincial government representatives. In addition, Dr. John Coates describes in this newsletter a ten-day workshop on foreign animal held at the new National Centre for Foreign Animal Diseases (NCFAD) in Winnipeg. Dr. De With also discusses her experiences with foot-and-mouth disease in the United Kingdom. In addition, I have just returned from a two-day government/industry forum on foot-and-mouth disease in Ottawa. Provincial animal health representatives, the CFIA, and the leaders of livestock industry commodity groups heard speakers from the United Kingdom and the new Director of the NCFAD, among several others. The importance of this meeting was emphasized by the opening comments of the federal Minister of Agriculture, Mr Lyle Vanclief; the introduction by the president of the CFIA, Mr. Ron Doering; and the remarks and attendance by the Chief Veterinary Officer for Canada, Dr. Brian Evans.

Although we should never consider ourselves to be fully prepared, the ongoing severe problems due to foot-and-mouth disease in the United Kingdom has certainly increased our awareness and improved our preparations to better deal with a foreign animal disease in British Columbia and Canada.

An important meeting on Foreign Animal Disease Emergency Management and Animal Health Zones will take place in Abbotsford on November 22, 2001. The meeting is sponsored by the CFIA and the Canadian Animal Health Coalition and is supported by the Animal

Continued Page 2

TOLL FREE NUMBER AND WEB SITE: The Animal Health Centre toll-free number is: 1 800 661-9903.
This Newsletter, and other information from the AHC, can also be found on the Ministry's Web site:
http://www.agf.gov.bc.ca/croplive/anhlth/ahc

From the Director

(Continued from page 1)

Health and Animal Industry Branches of the British Columbia Ministry of Agriculture, Food and Fisheries. A notice will be sent to all industry representatives shortly. Livestock producers are strongly encouraged to attend.

The recent tragic events in the USA have made us all more aware of the possible use of biological agents as terrorist weapons. While public health and animal health authorities have enhanced surveillance activities, veterinarians and animal owners must also be aware of this potential. The Animal Health Branch will be represented at a two-day conference in Victoria in late October dealing specifically with bioterrorism.

This summer we were pleased to host the annual meeting of the Canadian Association of Veterinary Pathologists in Abbotsford and attendees from across the country and the USA took advantage of the opportunity to visit the Animal Health Centre. We were reminded again of how fortunate we are to have such a well-designed facility and the ability to offer a wide variety of tests to the livestock industry. The keynote speakers at this year's meeting provided information on the transmissible spongiform encephalopathies (or TSE's) such as mad cow disease and a new understanding of some important horse diseases.

As a member of the Accreditation Committee of the American Association of Veterinary Laboratory Diagnosticians (AAVLD), I will have the opportunity later this month to take part in evaluating, for accreditation, a large university diagnostic facility in the United States. The evaluation of veterinary diagnostic capabilities in other facilities will undoubtedly provide good ideas to improve upon the work that we provide at the Animal Health Centre. By reviewing the materials and preparing for this upcoming site visit, I hope to help us to become better prepared for our accreditation to be renewed in 2003.

On behalf of the Animal Health Branch, thank you for your continuing support. We welcome your telephone calls and electronic mail. Please address to me any concerns or ideas you may have on the services we provide at:

Ron.Lewis@gems3.gov.bc.ca I may also be reached by telephone at 604 556-3038

British Columbia government veterinarians participate in UK Foot and Mouth Disease Control:

Nancy de With, DVM, MSc,
Veterinary Epidemiologist
Health Management &
Regulatory Section,
Animal Health Branch

Three veterinarians from the Health Management and Regulatory Section of the Animal Health Branch travelled to Britain recently to assist the United Kingdom Ministry of Fisheries and Food (UK MFF) in the diagnosis and control of foot-and-mouth disease (FMD).

As part of a third contingent of Canadian veterinarians, I traveled to the UK in April and May of this year. There were six of us in the third contingent, the other three members all being from the federal Canadian Food Inspection Agency (CFIA) Programs area.

All six of us were sent to the same UK MAFF office, located in the city of Carlisle, in the county of Cumbria. We made farm visits in the county to inspect and examine livestock for evidence of foot-and-mouth disease. More than 50% of the FMD cases have occurred in Cumbria. The disease has devastated farmers in the area.

Our duties were of three types: i) Surveillance, ii) Tracing, or iii) Report Cases. So-called "clean" veterinarians (i.e. those who had not been exposed to FMD, or whose last exposure occurred at least 72 hours previously) were sent on surveillance and tracing visits, as these activities were the lowest in risk with respect to detecting or observing FMD virus. "Dirty" veterinarians (i.e. those who had been on an infected premise within the past 72 hours) were allocated the report cases, which were more likely to be FMD.

During my last week in Carlisle, I worked in the Epidemiology Section. The work in Cumbria consisted of field epidemiology, as most data analysis was done at the UK MAFF office in London. My duties included the visiting of infected premises following a diagnosis of FMD. I questioned the farmer (and his/her family) on the movements of people, vehicles, equipment, and animals on and off the farm during the incubation period of the disease, prior to the actual occurrence of clinical signs.

We all had strict protocols to follow prior to, and upon return to Canada, so that we did not bring virus back with us. These procedures included cleaning all clothing and disinfecting footwear, and refraining from farm visits in Canada for 14 days following our return.

Our work in the United Kingdom was valuable. We gained experience and acquired practical skills and knowledge on disease outbreak management to further enhance our emergency preparedness and response to any Foreign Animal Disease outbreak that might occur in British Columbia.

Guardians of the Gate:

J Coates,
Veterinary Pathologist,
AHC

This past February, I was privileged to attend an intensive 10-day course at Canada's National Centre for Foreign Animal Disease, at Winnipeg. The course was unique, since we received actual clinical, 'hands-on' experience with a selected number of these foreign animal disease agents, in addition to the usual assortment of lectures and film portrayals. The diseases studied were thus lifted from passive textbook essays into real life situations. Clinical histories that were presented with each case simulated situations that would be anticipated in real life. Observing these exotic diseases in live animals and later at necropsy was at times unpleasant, but the knowledge gained was very great and will not be soon forgotten. Animals in the trials were treated humanely, and not allowed to suffer unduly. All clinical trials were monitored and approved by the Canadian Council on Animal Care.

Diseases studied (not all were represented by clinical cases) covered all corners of the globe. With modern transportation capabilities, many of these infectious agents are a present threat to the well-being of Canadian agriculture, and to the economy of Canada. Diseases studied included pseudorabies, foot and mouth disease, African swine fever, classical swine fever, rinderpest, BSE (mad cow disease), and highly pathogenic avian influenza, to name only a few. Some agents, like rabies, bovine tuberculosis, and West Nile virus, are already present in this country and require our constant vigilance. Public health aspects of a number of foreign animal diseases were also discussed, as was the topic of bio-terrorism.

As veterinarians, we have a major responsibility in being aware of and recognizing these diseases. None of us is exempt in this responsibility. Small animal practitioners have as much to contribute as their large-animal or avian-practitioner cousins. Part of our responsibility is educating an increasingly urban society in the importance of

Continued Page 4

Guardians of the Gate *(Continued from page 3)*:

maintaining and nurturing a veterinary diagnostic infrastructure that is capable of detecting the presence of these agents.

Approximately 28 individuals attended the course. Participants – all veterinarians - came from every region of Canada. Several colleagues from the United States and the United Nations, contributing as lecturers or as demonstrators, shared their personal knowledge and experience on many of the foreign animal diseases studied. The programme was sponsored by the Canadian Food Inspection Agency (CFIA). I am grateful to CFIA, and to my employer, the British Columbia Ministry of Agriculture, Food and Fisheries, for enabling me to attend the course.

Working with our colleagues practicing in the field on the one hand, and Canada's federal veterinarians and laboratories on the other, our provincial diagnostic laboratory at Abbotsford has an integral role to play in maintaining animal health in this province, and in the nation. No less a role is expected of an informed citizenry. In truth, we are all Guardians of the Gate.

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Bioterrorism's threat to the poultry industry:

Dated September 17, 2001 (courtesy of Joshua Lipsky):

The following is a commentary written by Charles Beard, vice-president of research and technology for the U.S. Poultry & Egg Association:

"I have hesitated to write this editorial for concerns that will soon become obvious to you but it is now time to address the issue. I haven't wanted to discuss the risks of bioterrorism to the poultry industry because I didn't want to be accused of giving our enemies any ideas. The events this week in New York and Washington, D.C., clearly show that they already have all the ideas they need and are not waiting for anyone to tell them how to conduct their attacks.

The U.S. Department of Agriculture has just released an alert on the subject of foreign animal diseases in the aftermath of the two terrorist attacks. The intentional introduction of infectious diseases into our food animals is clearly a concern of many officials who have the responsibility to protect our food supply. There is discussion about increased disease surveillance to rapidly detect and identify any diseases that might appear in our animals. Without outlining how it can be done, just trust me when I say that a bioterrorist animal disease attack can be accomplished cheaply, quickly and efficiently.

What the poultry industry can do to counter the use of disease introduction in bioterrorism is to implement known steps of biosecurity. When properly done, biosecurity measures may not prevent the attack but will help confine any disease introductions to the premise where they were introduced. Hopefully, that will allow for the proper identification and control/eradication of the disease before it can spread beyond that site into the rest of the complex. While everyone involved in the industry must participate in such a tightening of biosecurity, growers and farm managers have the most significant roles. Poultry houses need to be located behind locked gates and fences, house doors should be locked, feed delivery drivers should not enter the houses and neither should anyone else if they don't have a need to be in there. That includes neighbors, friends and relatives.

Company representatives, equipment repairmen and others who go from farm to farm should clean up, change or decontaminate footwear and wear clean, disposable outer clothing. A disinfectant foot bath at every house door that is kept fresh and clean is a good idea.

If the industry can keep any disease introductions - be they accidental or malicious - confined to the initial premise, we can minimize bioterrorism effects against poultry. If we don't do that, we could have a catastrophe on our hands. This is one place where we are not helpless.

Only we can fix this lax biosecurity problem, not the USDA, and not the Department of Defense. Only the industry players who make the business of poultry production happen can take the next step. Now is the time to get your operation in shape before it is too late."

Poultry news:

V Bowes

i. The ever-present challenge of *E. coli*:

Infections with the bacteria *E. coli* in poultry can develop at all ages with a variety of presentations depending on route of infection, the presence of other initiating pathogens and the immune status of the bird. In chicks less than one week of age it is seen as infections of the yolk sac (yolk sacculitis) and navels (omphalitis) due to either hatching out contaminated eggs or early on-farm exposure. In growing chickens it is seen as air sacculitis or pneumonia in birds with poor ventilation or co-infection with respiratory viruses such as Infectious Bronchitis Virus (IBV) or Newcastle Disease Virus (NDV), as slaughter condemnations for cellulitis due to localized infections of skin scratches, and as colisepticemia/pericarditis when the bacteria become systemic in the bloodstream. Colisepticemia in breeder fowl must be distinguished from acute fowl cholera caused by *Pasteurella multocida*, which is also seen occasionally and now must be reported to the provincial veterinarian to satisfy the export requirements of Russia and China.

Over the past few years the turkey isolates of *E. coli* have demonstrated an increasing profile of antibiotic resistance, initially to neomycin and more recently to enrofloxacin. Fortunately, most of the isolates to date have been cultured from the intestinal tracts of healthy birds but it is just a matter of time before antibiotic resistant pathogenic strains are recovered.

The *E. coli* organism is present as a normal inhabitant of the intestinal tract and environment, so preventing colibacillosis is related to managing the factors that allow *E. coli* to cause disease.

ii. Infectious Laryngotracheitis (ILT):

Last year's outbreak of ILT in the Fraser Valley has subsided, most likely due to the effectiveness of modified vaccination programs and heightened biosecurity. An epidemiological study by Dr. Nancy DeWith (BCMAFF) was undertaken in an effort to determine specific factors leading up to the outbreak. A survey was mailed to poultry producers and there was a return rate of approximately 62%. From the preliminary data analyzed, from 10 positive farms and 29 controls, the only significant difference between the affected and unaffected farms was farm size (# barns, # birds), such that a larger farm was more likely to have a case of ILT than a smaller farm. Other factors thought to contribute to the spread of virus are inadequate on-farm biosecurity and inappropriate litter management such as the spread of untreated manure on nearby berry farms.

Canadian Cattle Identification Programme: the role of the Animal Health Centre:

S Raverty (AHC)

With recent emergence of zoonotic diseases (disease transmissible from animals to humans) such as Bovine Spongiform Encephalopathy ("mad-cow" disease), and epizootics of economically important conditions such as foot and mouth disease (FMD), there has been considerable interest and effort in establishing a cattle identification scheme in Canada to facilitate trace-back of individual animals to the herd of origin. This programme will expedite identification of originating herds, so that resources may be focused at the source location, thereby limiting spread of specific diseases. The culmination of this effort is the Canadian Cattle Identification Program (CCIP), which was initiated January 1, 2001.

As many livestock producers and veterinarians are aware, all cattle leaving a herd of origin as of July 1, 2001, including those animals destined to the Animal Health Center (AHC), must be ear tagged. When cattle are presented to the AHC for post mortem examination, the tag number of each animal will be recorded and monthly reports compiled and forwarded to the Canadian Cattle Identification Agency in Calgary, Alberta, for entry into their database. For those animals which are not tagged, or where tags have been lost, the name and address of the farm will be recorded and forwarded to the CCIA. In those cases where the producer or veterinarian conducts a post mortem in the field and only tissues are sent to the lab, the identification number should be forwarded by the field practitioner or owner.

Between July 1, 2001 and July 1, 2002, enforcement of this tagging policy by the Canadian Food Inspection Agency (CFIA) consists of extension and notification. For any additional details regarding this program in regards to the AHC, please call during regular office hours at 1-800-661-9903, or check the CCIA website at www.cattle.ca/CCIA/faq.htm

Influenza A virus isolated from two foals with pneumonia:

Ann Britton and John Robinson (AHC)

In April and May of 2001, two foals were presented to the Animal Health Centre for necropsy. The first foal was a seven day-old Standardbred with a three-day history of respiratory disease unresponsive to treatment. Other horses on the premises, including the foal's dam, were unaffected. The second foal was a six week-old Clydesdale with a similar history of progressive respiratory disease unresponsive to treatment. The other horses on the premises were also affected with respiratory disease, which was characterized by a fever, cough, and runny nose in two mares and a severe pneumonia in one stallion. The mares and stallion recovered.

At necropsy, the foals were in good body condition. The major changes in both foals were restricted to the lungs, which were rubbery, congested, and edematous. The second foal also exhibited pus in the airways of the left lung. Both cases were diagnosed as interstitial pneumonia of probable viral etiology. The bacterial bronchopneumonia of the second foal was attributed to the presence of *Streptococcus equi zooepidemicus*, which is a common bacterial pathogen of the equine lung. Both of the foals were positive for Influenza virus. Although the Influenza virus was demonstrated to be type A, it did not react with antibody for Equine Influenza A1 or A2, the common antigens in equine flu vaccines. Serum samples taken from the affected adults on the Clydesdale farm exhibited low titres to Equine Influenza A1 and A2 but significant titres to the Influenza A virus isolated from

both the Clydesdale foal and the unrelated Standardbred foal. This suggests that all of the Clydesdale horses were suffering from disease caused by the Influenza A virus which was isolated.

Influenza virus is a common cause of respiratory disease in horses, which is usually restricted to the upper respiratory tract. Signs include depression, fever, cough and runny nose. Death can occasionally result from secondary bacterial bronchopneumonia as a result of damaged respiratory clearance mechanisms. The disease usually presents as outbreaks in susceptible horses following exposure to individuals that are shedding virus. These individuals may exhibit signs of respiratory disease, but at other times can be clinically unaffected.

Vaccinated horses may become infected without developing clinical disease and can act as a reservoir of infection for other horses. Vaccination is not always completely protective. However, vaccinated horses tend to be less severely affected by the disease. Natural immunity lasts from 1 to 2 years whereas immunity induced by vaccines is much less effective, and boosters should be given every 3 to 4 months in horses at risk.

Transmission of the virus occurs by direct contact and by aerosols as a result of the severe coughing characteristic of the infection. Transmission by virus on solid objects may also occur. The incubation period is 2 to 3 days, so outbreaks occur quickly after exposure to an infected animal. Foals can contract the disease, although maternal antibody is usually protective for at least the first month.

We are as yet uncertain of the significance of this virus. This may represent a new strain of Equine Influenza A as a result of antigenic drift, The fact that in-contact horses on one farm were unaffected by respiratory disease suggests that this may not be an entirely new virus in the lower mainland, and that there may be general immunity to the agent in the horse population at large. Individual susceptibility may have played the most important role in the development of these cases as, to our knowledge, there were no reports of severe respiratory disease associated with the Cloverdale rodeo other than the Clydesdales reported here.

Serological studies of the prevalence of this virus in the lower mainland are planned and will be reported in future issues of the newsletter. As always, proper precautions such as vaccination, adequate ventilation, good hygiene, and isolation of new introductions should be observed to prevent the development of acute respiratory disease in horses.

Equine degenerative myelopathy in a two year-old thoroughbred:

S Raverty and
D McIntosh

A two year-old thoroughbred colt was recently presented to the Animal Health Centre with a 30-day history of progressive ataxia and weakness. The animal had originally been evaluated May 11, with a follow-up examination on May 15, 2001. The colt was mildly depressed, cranial nerve examination was unremarkable, and there was no indication of muscle asymmetry or atrophy. Neurological examination disclosed severe proprioceptive deficits, paralysis affecting all four limbs and mild, unilateral reduction in the range of motion of the neck.

When walking in-hand, the horse was unable to maintain a straight line, its movement characterized by exaggerated swinging of the front legs, and abnormal foot placement of the hindlimbs. This incoordination was exaggerated when the horse was walking with its head elevated, or while turning clockwise. The animal was also readily unbalanced by the "sway test" while walking. Knuckling of the hind fetlocks and stumbling was noted when the animal was walked on soft ground.

Equine degenerative myelopathy *(Continued from page 7)*

Ante-mortem hematology and clinical chemistry disclosed an inflammatory profile with a stress-related reduction in lymphocytes (lymphopenia). The animal was dehydrated, azotemic (elevated blood urea nitrogen) with low electrolytes, and an increased AST (aspartate aminotransferase). Cerebrospinal fluid was aspirated and appeared normal; there was a weak positive titer to the equine protozoan *Sarcocystis neurona,* and no apparent increase in protein levels. Based on increased titers and active neurologic disease, equine protozoal myelitis (EPM) was considered a prime differential. The clinical status of the horse rapidly deteriorated. The animal developed excessive secretion of saliva, and was euthanized, May 30, 2001.

On necropsy at the AHC, the colt presented in good flesh. Involving the ventral half of the right caudal lung lobe there was a moderate aspiration pneumonia. There was slight opacity of the meninges of the brain noted along the ventral aspect of the medulla and caudal cerebellum. Otherwise, there were no other apparent lesions.

Histopathology confirmed the aspiration pneumonia. In addition, profound degeneration and necrosis of ventral motor neurons was revealed at multiple levels throughout the entire length of the spinal cord.

Despite the proprioceptive abnormalities and weakly positive *S neurona* titers, a diagnosis of equine degenerative myelopathy (EDM) was made, based on the histologic findings. This sporadic condition is an acquired neuromuscular disease of young horses which typically present with incoordination and progressively deteriorate over time. The pathogenesis of this condition is related to insufficient vitamin E consumption; in adult horses with this deficiency a similar, yet distinct entity, equine motor neuron disease (EMND) may result. If diagnosed early in the progression of the disease in young horses, favorable responses have been reported with megadoses of vitamin E.

Short cuts from the post mortem room:

J Coates

Severe coccidiosis was diagnosed histopathologically in submitted sections of small intestine taken by the practitioner at necropsy from a nursing 3 month-old pastured beef calf. The veterinarian suspected coccidiosis, and on necropsy diagnosed hemorrhagic enteritis. Random calves in the group of 300 animals had diarrhea, which at times appeared dark or blood-stained. Calves would cease nursing and become weakened, and finally recumbent. According to the practitioner, the calves had been restrained together in pens with their mothers just prior to release on pasture. Close confinement may have enhanced spread of the fecal-borne coccidial organisms among these young animals.

BVD-mucosal disease was clinically diagnosed by a veterinary practitioner in a dehydrated, 3 month-old Holstein heifer that had a foul diarrhea and numerous erosions on its muzzle and hard palate. At the time, because of the severity of the muzzle and mouth lesions, the veterinarian had also been concerned about the possibility of foot and mouth disease. The animal was submitted for necropsy at the AHC. In addition to the oral changes reported by the practitioner, inflammation was readily apparent in the small intestine and colon. Microscopically, the intestinal lesions were diagnostic for BVD-mucosal disease, characterized by a severe necrotizing cryptitis. Tissues were confirmed positive for BVD-mucosal disease virus via PCR, and also by tissue culture.

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The herpesvirus of malignant catarrhal fever (MCF) was identified via PCR in brain tissue taken from a white-tailed deer that had been showing clinical signs referable to the central nervous system. Microscopic examination revealed a severe nonuppurative lymphocytic meningoencephalitis, together with severe inflammation of blood vessels (a fibrinoid necrotizing vasculitis). These microscopic findings are characteristic of MCF infection. Brain tissue had been previously found negative for rabies by a federal laboratory.

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Border disease virus (BD virus) was identified in 3 stillborn/nonviable lambs from a flock suffering from mummified fetuses, abortions, stillbirths and nonviable neonates. Two of the term lambs were abnormally small in size. One had bilateral congenital dilatation of kidney tissue (hydronephrosis), together with a complete absence of functional kidney cortical tissue. Another had a virtual absence (aplasia) of the distal lumbar and sacral spinal cord, together with associated vertebral and pelvic anomalies. Other lambs on the farm that had been born alive and later died evidently were weak on their legs, unable to stand, or "shook" when attempting to stand. There were no obvious wool changes seen in the 3 lambs submitted.

It is perhaps noteworthy that during the previous winter the owner had mixed his pregnant ewes on pasture with calves for the first time. It is possible that his sheep acquired Bovine Virus Diarrhea (BVD) virus from the cattle. Persistently infected calves are able to infect sheep with the pestivirus that causes either BVD in cattle, or Border Disease (BD) in sheep. Other infectious agents were not detected. Some nutritional concerns were also identified in this group of animals on the basis of feed analysis, and their potential role in some of the earlier losses was not completely ruled out.

When BD infection is first introduced into a flock that is in the early stages of pregnancy, an outbreak with infertility, abortion, and congenital disease in lambs from ewes of all ages is possible. Subsequently, older ewes in the flock will develop immunity, and the disease will then only occur in introduced animals and maiden ewes (Radostits et al: *Veterinary Medicine,* 9th edition). Blood testing is available at the AHC upon request, to determine if animals within a flock have been exposed to the Border Disease virus.

Since writing this piece, a second case of BD-BVD virus-related abortion has occurred in a flock of sheep. Again, there had been co-mingling of sheep with pastured cattle. Presumably, this pestivirus may migrate either way, within these two separate species.

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Megabacteriosis was diagnosed in a mature passerine Fire Finch that had died fairly suddenly. Very dense masses of rod-shaped, super-large, so-called *megabacteria* (which are probably fungi) were seen within the glandular stomach (proventricular) lumen, and within the proventricular mucosa. A patchy, chronic-active proventriculitis was also present. The megabacterial organism is thought to be an abnormal inhabitant of the proventricular region of the passerine alimentary tract, especially when present in very large numbers. Dehydration and malabsorption follow. Injury to the proventricular mucosa may permit a variety of bacteria to enter the systemic circulation. This bird had a secondary hepatitis. Acid-fast stains on all tissues were negative for avian mycobacteria. No trichomonads were seen within the proventriculus, although these flagellated protozoa are best detected on a very fresh specimen via a direct, wet-mount microscopic examination.

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Continued Page 10

Short cuts from the post mortem room *(Continued from page 9)*

Skeletal myopathy and thyroid hyperplasia were diagnosed in a day-old nonviable foal. The animal also had hypogammaglobulinemia linked to inadequate colostral intake. On later blood serum trace mineral analysis, the aborted mare was deficient in selenium and borderline in serum iodine.

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Bovine adenoviral inclusion-body hepatitis and splenitis was diagnosed in a markedly hypoglammaglobulinemic neonatal calf that had been systemically ill with navel ill and multiple joint infection (fibrinosuppurative polyarthritis). The adenoviral infection was probably opportunistic in this severely ill and immuno-suppressed animal.

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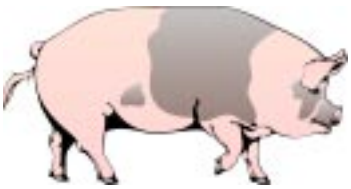

Selenium toxicosis was diagnosed in a group of feeder pigs following ingestion of whey that had been supplemented with additional selenium in an attempt to control aggression. The animals became recumbent and, although mentally alert, could not stand. One animal was able to assume a dog-sitting posture if assisted, but hind-limb paralysis was still present.

Microscopically, there was severe degeneration of the central spinal cord grey matter within the cervical and lumbar areas of all 3 animals examined (poliomyelomalacia, severe, with extensive gitter cell proliferation, within the ventral motor horns).

Liver selenium levels in earlier pigs from the same group were recorded at 3.24 and 4.09 ppm (wet weight), respectively. Normal, adequate levels of liver Se in pigs are listed from 0.40 to 1.20 ppm (Puls: *Mineral Levels in Animal Health,* 1994). The recumbent animals had not ingested any selenium-enriched whey for 3 or more days. Although the liver selenium levels in this latter group of animals were found within normal limits, it is thought that the damage done to spinal cord tissue was nevertheless a sequel to earlier, repeated, high-dietary selenium exposure. There were no additional losses of this nature following removal of selenium from the whey.

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Coxiella burnetii (Q fever) and small ruminant abortion:

J Coates

In recent years during the spring kidding and lambing period, abortion caused by the infectious agent *Coxiella burnetii* has been diagnosed at the AHC. *C burnetii* is a gram-negative, coccobacillus organism which, in the past, has often been described as a rickettsia (1*).* The agent is transmissible to humans. It has world-wide occurrence, and is found in many wild and domestic animals. The organism is highly infectious; under experimental conditions, only one organism is believed necessary to produce infection. In humans, where it may have several forms of expression, it is referred to as Q (Query) Fever. In a recent investigation of human Q fever cases in Newfoundland, an association with goat abortions was recognized. Interestingly, in a survey of Nova Scotians for antibody against *C. burnetii*, the rate of seropositivity was highest among veterinarians – 49% of those tested had antibodies (1).

Abortion is the principal expression of the disease that is of concern to livestock owners: at the AHC, the disease is most commonly diagnosed as a cause of abortion within dairy goat operations, although it is capable of infecting any ruminant species,

including sheep and cattle. Most infected animals will actually have a persistent, relatively asymptomatic infection.

There are usually significant and characteristic changes seen within the placenta of an aborted goat, which an astute owner or practitioner may observe. The most significant of these is the presence of a thick, copious, caramel-colored to chalky-white exudate that overlies the placental tissue, which in turn is markedly thickened and opaque within the normally translucent intercotyledonary areas (i.e., between the so-called cotyledons, or "buttons"). This characteristic exudate and markedly thickened placenta are almost always present, and should alert the examiner to the likelihood of *Coxiella burnetii* abortion. Aborting animals should be isolated from the herd or flock.

Microscopic placental changes in Coxiella abortion: microscopy usually reveals myriads of intracellular *Coxiella* organisms within the trophoblast cells of the placental tissue. At the AHC, the identification of the organism in aborted tissues is confirmed by the polymerase chain reaction (PCR) test.

Pasteurization eliminates the organism from milk: questions received at this laboratory from concerned clients or veterinarians usually relate to the efficacy of pasteurization in eliminating the organism from milk, as well as the possibility of the organism spreading to other animals or to human attendants. The organism in most circumstances is destroyed by pasteurization (2). The real concern is for individuals who consume raw milk. Q Fever is just one more reason why it is inadvisable to consume milk or milk products that have not been pasteurized.

Raw milk consumption increases human risk to zoonotics: in addition to *C burnetii* (the causative agent of human Q fever), there are other infectious agents that may be present in non-pasteurized milk which are a risk to humans. These include brucellosis, leptospirosis, various coliform (e.g. *E coli*) organisms, as well as bovine tuberculosis. All are destroyed by pasteurization. Incidentally, pasteurization was originally developed by the French scientist Louis Pasteur as a means of controlling microbial growth in fermenting wines. The technique was later used in milk processing, as its value in eliminating harmful organisms became apparent. Milk pasteurization was one of the most significant public health tools developed in the 19th century and resulted in a marked reduction in the incidence of human tuberculosis, especially in children.

Transmission of C burnetii: body fluids such as urine, feces, milk, placental discharges fluids and tissues, as well as post-partum discharges may contain the organism. Domestic pets such as barn cats may inadvertently become infected from these substances, and thus pose a secondary risk to their owners. For reasons still poorly understood, dogs pose much less of a problem for transmission of the organism to man (1).

Resistance of the Q fever agent (C burnetii) to disinfection: C burnetii is very resistant to physical and chemical influences, and evidently can survive in the environment and soil for months (2). It is also very resistant to chemical disinfectants, although it is sensitive to sodium hypochlorite and 1:100 lysol solution (2). After an abortion, and after specimens required for diagnostic purposes have been salvaged, it's best to eliminate all suspected infected membranes, fluids, or fetuses as efficiently as possible – preferably by burning or incineration. While carrying out obstetrical procedures on small ruminants, wear disposable gloves. When obstetrical work is completed, clean your hands thoroughly with an effective disinfectant soap.

Isolation of aborted animals: aborted ewes or does should be isolated from the rest of the group. Remaining tissues or fluids from aborted animals should be promptly

Continued Page 12

Coxiella burnetii (Q fever) and small ruminant abortion

(Continued from page 11):

incinerated. Along with *Coxiella sp*., toxoplasmosis is also of concern in small ruminant abortions, together with chlamydiosis. Both of these diseases are also potentially zoonotic. These 3 agents – *Coxiella burnetii*, *Toxoplasma gondii*, and *Chlamydia sp*, should always be considered as prime suspects in cases of caprine or ovine abortion.

Wildlife and domestic animal cycles: one life cycle of *C burnetii* is found in domestic animals (mainly goats, sheep, and cattle), while the other occurs in wildlife, where the agent circulates between wild animals and their ectoparasites, especially ticks. Interaction between the wild and domestic cycles may occur. In various studies in N America and elsewhere, many species of wild animals, including rodents and hares, have been found to be infected with the organism. In Nova Scotia, Marrie has observed a significant number of seropositive reactors in farm cats (1), indicating their exposure to the organism that originated in either infected domestic animals (primarily goats, or sheep) and/or wild creatures. Cats are presumed to become infected by ingesting infected mice and infected parturient cats, in particular, may be a source of the organism for humans.

Blood testing for Q fever antibody is not readily available: Q fever or *Coxiella burnetii* infection in ruminants, and especially in dairy goatherds, is an ongoing concern for operators in British Columbia. Unfortunately, there is no laboratory in Canada that offers an antibody blood test for this organism. Consideration is now underway to develop a Q fever antibody test here at Abbotsford's AHML, although no firm time lines have yet been drawn; however, it is noteworthy that in the Newfoundland investigation of Q fever in humans and goats, there was no relationship between seropositivity in goats and frequency of abortion (1). The value of an antibody test would therefore be limited in any attempt to screen a herd for exposed animals or carriers.

References:

1. Marrie T J. Q fever – a review. *Can Vet Jour* 1990; 31: 555 – 563

2. Radostits, Gay, Blood, Hinchcliff. *Veterinary Medicine*, 9th edition, year 2000: Saunders Pub Co.

Antibody test for Toxoplasma gondii abortion in small ruminants:

The Animal Health Monitoring Laboratory has a serum ELISA test available for the detection of toxoplasmal antibody in aborting animals. Recently, this test was used to advantage in a group of aborting ewes. Not only were the post mortem lesions observed typical of toxoplasmal abortion - both grossly and microscopically - but toxoplasmal antibody was actually detected at a level of 1:512 in (aborted) fetal heart blood. The case was further confirmed via the PCR technique on placental tissue.

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A preliminary microbiological and serological investigation into *Brucella* spp in neonatal and juvenile Harbour seals (*Phoca vitulina richardsi*) presented to the Vancouver Aquarium Marine Mammal Centre and Saltspring Island Wildlife Natural Care Centre:

Stephen Raverty (AHC – BCMAFF), Craig Stephen (Center for Coastal Health), W Fiessel and K Carlsen (AHC – BCMAFF) ,

Pacific harbour seals (*P vitulina richardsi*) are distributed along the western seaboard of North America from California to Alaska, and to the Aleutian and Pribilof Islands. Since implementation of the United States *Marine Mammal Act* in 1972, there has been a substantial increase in the numbers of this species, with an annual growth rate of approximately 7.2% recorded since 1996 (G Ellis, personal comm.). A recent census estimates the entire population between 312,000-317,000, with approximately 260,000 animals in Alaska, and 45,000-52,000 throughout the south coast of British Columbia.

The recent detection of *Brucella* spp antibody titers and bacterial isolation from wild harbour seals (*Phoca vitulina richardsi*) in Washington state in 1999, coupled with subsequent demonstration of the abortifacient and zoonotic potential of this group of bacteria in cattle and humans, respectively, resulted in a preliminary serologic and microbiologic investigation to assess the prevalence of this agent in wild and rehabilitated harbour seals from the south coast of British Columbia.

During the 2000 pupping season, 39 neonatal and juvenile harbor seals were submitted to the Vancouver Public Aquarium Rehabilitation Center (VPA), while 47 were submitted to the Saltspring Island Wildlife Natural Care Center (SIWNCC). On initial presentation to either facility each animal was clinically evaluated, individually identified, and separately maintained in fiberglass enclosures. Blood samples were collected and submitted for routine clinical chemistry and hematology, or preserved in storage for possible future use.

In addition, blood samples obtained from 6 animals at the SWINNC and 28 seals at the VPA were retained until the conclusion of the pupping season, then forwarded en masse to the Canadian Food Inspection Agency Laboratory in Nepean, Ontario for serology. Titers for *Brucella* spp were ascertained by buffered plate agglutination test (BPAT). Those animals which succumbed during rehabilitation were necropsied at the Animal Health Center (AHC). Tissues from another 17 harbour seals submitted for post mortem evaluation were screened by culture. Post mortem autolysis, incomplete sampling, or freezing precluded microbiological appraisal of the remaining 10 animals.

Of the samples submitted for the BPAT in 2000, 40% were positive, 47% were negative, and 13% were unsuitable for evaluation. Despite comprehensive tissue sampling which included lung, mediastinal lymph node, spleen, brain, kidney, liver, and representative segments of bowel, no *Brucella* spp were isolated on specialized agar, nor observed in histologic lesions. The detectable antibody titers may represent nonspecific, false positive results, passive transfer of maternal antibodies in colostrum, or other yet-undefined factor(s). Failure to propagate the bacteria *in vitro* may be due to inappropriate media, insufficient time for incubation, antemortem antibiotic administration, or absence of bacteria within the sampled specimens.

Continued Page 14

Microbiological and serological investigation into *Brucella* spp

(Continued from page 13)

Although the harbour seal *Brucella* spp isolate is considered distinct to that isolated from Cetaceae, recent detection of elevated titers to this bacteria in a stranded grey whale together with demonstrated zoonotic potential of a European isolate, raises concerns about possible human infection arising from contact with wildlife or direct interaction of humans with marine mammals during rehabilitation. The potential for human exposure to *Brucella spp* may be of particular concern to First Nation communities that harvest seals for food. It is hoped that further investigations will be undertaken to resolve the epizootiology* and pathology of this organism in marine mammals along the BC coast.

*Epizootiology is the scientific study of factors in the frequency and distribution of infectious diseases among animals. Ed.

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Detection of putative Koi Herpesvirus infection in recently imported juvenile goldfish (*Carassius auratus*):

S Raverty, AHC

Tropical fish, especially ornamental goldfish (*Carassius auratus*) are among the most popular pet animals in North America. A large proportion of imported stocks originates from Southeast Asia. In the past, koi fish imports were predominantly from Taiwan and China; increasingly however, fish are being acquired directly from Japan. In 1995, an outbreak of putative koi herpesvirus with subsequent massive mortality was reported in Japan. Similar outbreaks more recently recognized in private culture facilities in the north Atlantic and western United States and Israel. This case report describes a possible herpesvirus infection in recently imported juvenile goldfish originating from Japan.

The fish stock had been imported from Japan, and was delayed up to 14 hours en route to Vancouver. On arrival, the fish were crowded and the water was turbid with a pungent ammoniacal odor. The stock was immediately introduced into a quarantine facility with high water flows, injected oxygen, and optimal stocking rates. Mortality rates within the first 1-2 days were within acceptable standards. The stock remained stable for a three-week period, then experienced a sudden and sharp increase in mortality rate. Fish either exhibited no clinical signs prior to death, or demonstrated vague symptoms including listlessness, lethargy, crowding at the water intake, and inappetance. Due to ongoing, persistent mortalities that were unresponsive to broad-spectrum antibiotics, a small subset of fresh dead and moribund goldfish was presented to the AHC for analysis.

The fish were generally in good body condition. In multiple fish, the abdominal walls were mildly to moderately distended and the coelomic cavity contained 1-5 mL of red, turbid fluid (serosanguinous ascites). Spleens were often enlarged, friable, and featured ragged margins. There was also variable enlargement of the liver and kidneys. Patchy hemorrhage was noted within abdominal serosal surfaces. There was multifocally extensive subcutaneous edema along varying levels of the body wall, occasionally with raised scales. These regions were frequently demarcated by a thin,

hyperemic margin; in more severely affected fish, there was central ulceration of these areas.

The most significant microscopic lesions were localized to the oral cavity, gills and skin. In multiple fish, there were individual and small clusters of cells with enlarged nuclei scattered throughout the oral mucosa, respiratory epithelia and epidermis. These cells featured marginated nuclear chromatin and a central amphophilic to eosinophilic inclusion. In more severely affected fish, there was moderate to marked transmural vasculitis (blood vessel inflammation) throughout the submucosa of the oral cavity, involving numerous small to intermediate size blood vessels. The inflammatory reaction occasionally obliterated the lumen of vessels. Throughout the spleen, liver and kidney there were variably extensive regions of acute hemorrhage, fibrin deposition, and neutrophilic infiltrates. Occasional histiocytes containing small numbers of coccobacilli were also observed. Numerous fish exhibited mild to moderate respiratory epithelial hyperplasia of the gills, with blunting and fusion of secondary lamellae.

Aerobic culture of the liver, spleen and kidney yielded variable growth of *Aeromonas hydrophila*. Direct electron microscopy using negative-stained tissues revealed a number of herpesvirus-like particles. Based on microscopic and ultrastructural findings, a presumptive diagnosis of herpesvirus infection was made.

Experimental work undertaken at the University of California, Davis, has revealed that after an outbreak, subclinical carriers of this virus can persist and transmit the virus to susceptible tank mates. Typically, after introduction of new stock, there is an initial primary mortality precipitated by the stress of transport, followed by a secondary wave of affected fish due to horizontal transmission and infection of naïve stock. Efforts are currently underway to develop serological tests to screen for this pathogen, for health-certification. In the case described here, additional samples have been forwarded to UC Davis, for additional molecular studies.

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Collateral Expertise:

R Puls

In diagnosing livestock problems, the final answer or cure is not always found on the necropsy table, nor in the findings of the associated laboratory sections. Often, further expertise is required to pinpoint the cause of a problem, and a farm visit by a specialist may be necessary. In this regard, the Ministry of Agriculture, Food and Fisheries has an array of expertise upon which to draw. Many of these skilled persons are present within the Abbotsford Agriculture Centre.

Recently, a case of Alsike clover (*Trifolium hybridum)* poisoning was diagnosed in a horse in the Fraser Valley, yet no Alsike clover had knowingly been fed to the horse. Alternative diagnoses were explored. In the meantime, additional blood tests from several valuable and clinically healthy horses on the same property indicated abnormal serum enzyme profiles compatible with a diagnosis of Alsike clover poisoning, despite their healthy outward appearance. The veterinarian called the Animal Health Centre for assistance, and was directed to our Weed Specialist, Roy Cranston. Mr Cranston visited the property and quickly determined that Alsike clover was indeed growing in the pasture on which the horses were grazing. From this point

Continued Page 16

on, remedial action was then taken by the owner to remove either the clover or the horses from the pasture, whichever was deemed most practical.

Many similar scenarios occur each year. The following is an overview of some of the expertise the Ministry can supply:

1. ***There are three veterinarians in the Health Management and Regulatory unit of the Animal Health Branch.*** **604 556-3014.** Although they have other duties, these individuals are available for special investigations of cases, if requested by the practicing veterinarian attending the case. Incidentally, each of these veterinarians has just completed a three-week tour of duty to Britain, where they were involved in assisting in the eradication of foot and mouth disease in that country.
2. ***Plant Pathology Laboratory:*** **604 556-3126.** This laboratory is used by the AHC to culture moulds from feed samples suspected of containing mycotoxins. Determination of the genus of mould present (if any) helps the Toxicology or Monitoring Labs decide which mycotoxins could be present, and which of the toxins (if any) should be tested for.
3. ***Pesticide specialist:*** **604 556-3027.** Madeline Waring has all the information on pesticide use, including rodent and pest control. In addition, she has access to all the Material Safety Data Sheets (MSDS's) covering toxicity to livestock.
4. ***Weed specialist:*** **604 556-3066.** Roy Cranston helps identify poisonous plants or weed seeds in livestock feeds that could cause toxicities.
5. ***Entomologist:*** **604 556-3031.** Bob Costello is often asked to identify and recommend control measures for insects in feed, on livestock, or in and around barns or other farm buildings
6. ***Livestock specialists:*** these individuals, allocated to various livestock commodities, are available for nutritional and management advice. Improper nutrition is often at the root of many livestock diseases or deaths. Livestock specialists at the Abbotsford Agriculture Centre are:
    i. Ron Barker 604 556-3087: Dairy Cattle.
    ii. Stewart Paulson 604 556-3083: Poultry.
    ii. Basil Bactawar 604 556-3081: Sheep, goats, horses, pigs.
7. ***Agriculture Engineers:*** **604 556- 3096.** These persons can assist with building plans for disease control, waste disposal, mineral feeders, and anything else associated with the design of livestock production facilities.
8. ***District Agriculturist*****: 604 556-3086.** Mark Robbins is the district agriculturalist for the Lower Mainland.

This is by no means a comprehensive list. These are just some of the skilled personnel located in the Abbotsford Agriculture Centre, in addition to those in the Animal Health Centre, whom you may call for advice or assistance.

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Animal Health Monitoring Laboratory (AHML) News:

L Curley

CA*E test (caprine arthritis encephalitis virus):* the AHML offers a CAE competitive-ELISA test, with sensitivity greatly increased over the previous AGID test, combined with excellent specificity (false positives have been a problem with some other CAE ELISA tests). The AHML provides services to veterinarians. We strongly encourage goat owners to work with a veterinarian in collecting serum samples and developing a CAE control program.

Brucellosis testing: there has been some confusion lately about the BPAT test used at the AHML. The AHML is a Canadian Food Inspection Agency-accredited Brucella testing lab and the rules regarding Brucella testing are set by CFIA. This test can be used to certify all species for *Brucella abortus*, *B. melitensis*, or *B. suis,* for export purposes. It cannot be used, however, to certify sheep and goats that require testing for *Brucella ovis*. These samples must be sent to the CFIA's Brucella Centre of Expertise at Nepean, Ontario, for complement fixation (CF) antibody testing.

Artificial insemination is the only allowed purpose for Brucella testing of non-export animals. If a veterinarian suspects brucellosis, it is a reportable disease and the CFIA District Office assumes responsibility for the case. Testing "at owner's request" samples is not allowed at the AHML, and CFIA will not test them, primarily because it is a drain on financial resources. CFIA has ongoing surveillance testing at northern Alberta and B.C. auction markets, and performs a triennial survey.

Blood sample quality: for quality control, various samples are repeat-tested to ensure that the results fall within allowable variances. If they don't, we often discover that the original blood sample had been submitted in very poor condition. For bloods not received in the AHML within a few hours of sampling, the following guidelines will ensure that you receive the most accurate and precise serology results possible.

If serology is to assist in diagnosis of an acute infection, remember that 10-14 days are required for the development of demonstrable antibodies. If a blood sample is drawn during the acute phase of a disease, a second sample should be drawn 10-14 days later. Freeze the acute serum and submit the paired sera (acute and convalescent) together. Of course, this cannot apply to such testing as herd surveys for *Neospora sp* antibody.

It's best to send serum only, not whole blood or clots. Allow at least 0.5 ml serum per requested test. Collect blood in clean sterile tubes, ¾ full, and allow to stand, thus permitting a solid clot to form and retract. Spin the tubes and pour the serum into a clean tube, cap it tightly, and refrigerate until shipment. It is not necessary to remove the serum from SST tubes prior to shipping.

Errors in animal identification are avoided if the tubes are numbered consecutively, and the tube number and corresponding animal I.D. are clearly written on the submission form. Pleaase ship your samples overnight with gel icepacks during the warmer months, with sufficient enclosed packing material to protect the contents. To avoid damage due to leakage of the gel-pack or serum vials, place the submission form in a plastic zip-lock bag.

Fecal samples for Johne's testing: should be collected aseptically and placed in sterile plastic bags (Whirlpak) or wide-mouthed, screw-top containers. Seal tightly. Please do not send samples in plastic gloves or sleeves. Chill the samples from time of

Continued Page 18

Animal Health Monitoring Laboratory *(Continued from page 17):*

collection until they arrive at the laboratory. High contamination rates have been a problem with large dairy herds, so we are attempting to place the fecals into HPC (decontaminant solution) as soon as possible, right at the farm. If you wish, call 604 556-3135 and we will supply screw top tubes containing HPC. Sinply add 2 grams fecal matter (thumbnail size), shake well, and ship immediately. However, these particular samples should not be refrigerated or put on icepacks, as HPC precipitates at low temperatures.

Please call us in advance, if you anticipate collecting large numbers of samples, either for serology or Johne's fecal culture.

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Focus on Staff:

Julie Bidulka, molecular diagnostic scientist

Ms. Julie Bidulka joined the Animal Health Centre in September 2000, and is currently employed full-time in the molecular diagnostics section of the Virology department. A graduate of Simon Fraser University, Ms. Bidulka has a BSc degree in biochemistry as well as a co-operative education degree from SFU. Julie has also acquired specialized training in fin-fish diagnostics

Julie originally came to work at the Animal Health Centre as part of SFU's co-operative education program. After receiving her degrees, she was employed at St. Paul's Hospital, where she investigated the causative agent of human cardiovascular disease development in type II diabetic patients. She subsequently returned to the AHC and accepted her current position last year. Julie has worked on ELISA techniques for identification of BVD virus in sera of bovine herds.

Ms. Bidulka is responsible for animal pathogen identification, using nucleic acid extraction and polymerase chain reaction (PCR) techniques. In addition to routine diagnostic testing on a wide range of animals, Julie is studying ways to optimize existing PCR assays, and to develop new diagnostic PCR techniques, such as additional porcine PRRS virus strain typing/identification.

Julie's other interests include dragon boat racing, fishing, cooking, gardening and travel. She enjoys her work at the AHC very much, and looks forward to new challenges each day. We welcome Ms Bidulka to the AHC, and look forward to utilizing her expertise in the increasingly diverse area of PCR molecular diagnostics.

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Did you know?

— that myoglobinuria does not occur commonly in enzootic muscular dystrophy (vitamin E-Se related disease as in calves, lambs, kids), possibly because there is insufficient myoglobin in the muscles of young animals. Radostits et al, *Veterinary Medicine*, 9th ed.

— that selenium deficiency decreases neutrophil bactericidal activity and variably affects neutrophil phagocytic activity…. Oral supplemetation of beef cows (initial blood selenium = 45 – 50 ppb) from mid-gestation to calving increased colostral IgG concentrations and postsuckle IgG concentrations in calves. - from *The Compendium*: Oct.1997.

— that eye fluid taken from a recently dead animal can be analyzed for Ca and Mg, as well as for nitrates. In some circumstances, considering the clinical history and other facts of the case, these findings may have diagnostic value. Fetal eye fluid may also be a useful diagnostic tool in abortion cases where subacute or chronic nitrate toxicity is a possibility in pregnant cattle (or mares, sheep, etc) fed a ration (and/or water) containing excessive nitrates.

— that one of the first references to human tuberculosis was by Hippocrates, the "Father of Medicine", in 400 B.C. He used the term "pthisis" to describe human tuberculosis, which he defined as 'a diminution or shrinking of the body, following incurable illness of the lungs accompanied by a small fever'. Countless individuals from all walks of life have succumbed over the ages due to tuberculosis, including the musican/composer Frederic Chopin, the poet John Keats, and the naturalist/ philosopher Henry David Thoreau. - from *Keats*, by Andrew Motion.

— that (in humans) thyroid deficiency during the latter two thirds of gestation and the first months after delivery can result in mental retardation and sometimes neurologic deficits…..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. The reasons for this (iodine) decline include a reduction in salt intake and reductions in the amount of iodine added to bread and animal feeds. New England Jour Med 1999; 341: 601 – 602.

— that the seroprevalence of *Neospora caninum* was compared among cattle from New Brunswick, Nova Scotia, and Prince Edward Island in 1998, 1989, and 1979. In 1998, the seroprevalence was lowest in PEI, where it was the same as in 1979. *N caninum* was present in 1979, but at a lower prevalence. Keefe GP, et al. *Can Vet Jour* 2000; 41: 864-866.

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Thoughts from R W Emerson:

"…Everything in the universe goes by indirection. There are no straight lines."
– from *Society and Solitude: Works and Days*.

"Difficulties exist to be surmounted. The great heart will no more complain of the obstructions that make success hard than of the iron walls of the gun which hinder the shot from scattering. It was walled around with iron tube with that purpose, to give it irresistible force in one direction…"
– from *Letters and Societal Aims: Progress of Culture*.

"…Science has shown the great circles in which nature works, the manner in which marine plants balance the marine animals, as the land plants supply the oxygen that the animals consume, and the animals the carbon which the plants absorb. These activities are incessant. Nature works on a method *of all for each and each for all*. The strain that is made on one point bears on every arch and foundation of the structure. There is a perfect solidarity. You cannot detach an atom from its holdings, or strip off from it the electricity, gravitation, chemic affinity, or the relation to light and heat, and leave the atom bare. No; it brings with it its universal ties."
– from *Society and Solitude: Farming*.

The nature and composition of milk:

"…Milk may be described as the most perfect of foods. It contains every constituent for the natural nourishment of the young, viz, the albuminoids, fat, carbohydrates, and mineral salts, which then taken into the body, produce flesh and bone, heat and energy. It forms an excellent diet for invalids, as it repairs waste and builds up flesh and tissue. In appearance milk is an opaque liquid, creamy in colour, having a sweetish flavour and "cowy" odour. It is heavier and more viscous than water…"

– from *Practical Dairying*, by Dora G Saker; published 1921: Methuen & Co Ltd, London.

A Poet's Vision:

"…Ye who love the haunts of Nature,
Love the sunshine of the meadow,
Love the shadow of the forest,
Love the wind among the branches,
And the rain-shower and the snowstorm,
And the rushing of the rivers
Through their palisades of pine trees,
And the thunder of the mountains,
Whose innumerable echoes
Flap like eagles in their eyries;-
Listen to these wild traditions,
To this song of Hiawatha!…"

– from *The Song of Hiawatha*, by Henry W Longfellow