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From The Director

R.J. Lewis

I have recently returned from several meetings in Ottawa regarding current animal health issues. The meetings were organized by the Canadian Food Inspection Agency (CFIA) and attended by representatives from the provincial governments and the livestock industries. A wide range of discussions of importance to each of these groups took place over the four days of meetings.

One of the interesting presentations was on the recent *simulation* exercise in which an outbreak of foot and mouth disease (FMD) was diagnosed. This was the largest disease exercise ever attempted; three countries (Canada, USA, and Mexico) were involved with a total of 300 participants, of whom 150 were from Canada. The scenario had FMD occurring in garbage-fed swine in southern Texas soon spreading in the air to nearby cattle. Some of these cattle were shipped to Mexico and several cowboys from Alberta attending a rodeo in Texas brought the virus back to Alberta. At the same time, a cattle liner that had not been properly disinfected crossed the border into Ontario. Within a very short period of time, the disease was diagnosed in all these areas.

The disease from this simulated exercise spread very quickly and soon hundreds of farms were involved. Projecting several weeks ahead, the exercise indicated thousands of cattle would have been destroyed. Numerous questions arose from this exercise including:

- how to dispose of several hundred thousand dead cattle
- how to communicate effectively to stakeholders and the general public
- how to effectively compensate producers
- best methods of disinfection
- where to access vehicles and the tons of supplies that would be necessary

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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)*

- how valuable is vaccination in the face of an outbreak and how to quickly access the very large numbers of required vaccines

Of course, during this time and likely for several months afterwards, Canada would be unable to export livestock products with a consequent severe economic impact. Other questions arise such as how to effectively zone the affected areas to allow the rest of Canada to continue to export animal products.

Over the last few years, we have seen classical swine fever (hog cholera) cause major disruptions in Holland, FMD has devastated the livestock industry in Korea, Taiwan and, recently, South Africa. With the rapidity of air travel and increasing numbers of global travellers, we have been very fortunate that we have not already had a foreign animal disease in Canada. The frightening part of this exercise was the realization that it could occur at any time with devastating results.

Early in February, Dr. John Coates will take part in a ten-day foreign animal disease training exercise at CFIA's new National Centre for Foreign Animal Disease in Winnipeg. Diagnostic laboratories and veterinarians must always be alert to the very real possibility of a foreign animal disease incursion in Canada. Livestock producers must also be aware of major global disease problems and prevent people from affected countries visiting their farms. We all have a role to play to prevent foreign animal disease.

As part of increasing awareness and with the support of CFIA, we hope to develop a seminar on foreign animal diseases this year in Abbotsford.

On behalf of all the Animal Health Branch staff, our very best wishes to you and your families for a very happy and successful 2001!

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A summary of equine diagnoses between 1993-1999 at the Animal Health Centre:

Contact – S Raverty, W Fiessel, and K Carlsen, AHC

Over the last decade, there has been a substantial increase in the number of horses and horse owners in British Columbia. To provide practitioners pertinent information regarding equine diagnoses derived at the Animal Health Center (AHC), case summaries from 1993-1999 were reviewed, and a profile of morphologic diagnoses and etiological (causative) agents was generated. The number of horse cases submitted per year varied from 70 in 1993 to a high of 110 in 1996 (table 1), with these accessions accounting for approximately 0.01-0.02% of the annual AHC caseload. The predominant breed submitted was the thoroughbred with fewer quarter horses, standardbreds and smaller numbers of Arabs, draft horses, miniature and Shetland ponies, mules and donkeys (table 1).

During this time interval, there were a total of 600 horse submissions comprised of either whole animals, or aborted fetuses and tissues harvested from field necropsies (table 2). Blood serum submitted for trace mineral or vitamin analysis, as well as serology results were excluded from this review, as these data are not encoded as diagnoses within the AHC vet lab database.

Table 1. Summary of equine submissions according to breed

Year	Thoroughbred	Quarter Horse	Standardbred	Other breeds*
1993	20	14	6	30
1994	24	5	11	42
1995	26	10	7	43
1996	35	18	3	54
1997	32	15	6	26
1998	20	12	6	33
1999	18	16	2	47
Total	175	90	41	275

***other breeds include Arabs, draft horses, miniature ponies, mules, donkeys, and Shetland ponies**

Table 2. Summary of equine submissions to the AHC, 1993-1999

Year	Total number of submissions (per cent AHC total)	Number of Tissue submissions	Number of fetal submissions	Number of whole animal submissions
1993	70 (0.01)	23	7	40
1994	82 (0.02)	32	22	28
1995	96 (0.02)	38	13	45
1996	110 (0.02)	42	18	50
1997	89 (0.02)	28	23	38
1998	71 (0.01)	16	12	43
1999	82 (0.02)	24	17	41
Total	**600 (0.01)**	**203**	**112**	**285**

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Table 3. Per cent diagnoses of horse submissions to the AHC according to organ system

System	Per cent Diagnosis	Number of Animals
Gastrointestinal	26	245
Urogenital	22	208
Cardiovascular	13	123
Musculoskeletal	10	91
Respiratory	8	85
Integumentary	4	41
Hemolymphatic	4	41
Endocrine	4	41
Nervous	3	32
Open diagnosis	2	24

Pathological processes referable to the gastrointestinal, urogenital, cardiovascular, and musculoskeletal systems were the most commonly identified anatomic systems, with fewer cases involving the respiratory, integumentary, hemolymphatic and other predilection sites (table 3).

The gastrointestinal tract was the system most commonly involved with disease processes. Lesions varied from non-specific inflammation of the tongue (6 horses), stomach ulceration (18) and stomach perforation (7), intestinal torsion and/or incarceration (47), undefined inflammation of the intestinal tract/enteritis (30), inflammation of the colon/colitis (21), and colitis X (3). There were 19 cases of enteritis; 17 of these had no significant bacterial isolates, 3 were linked to parasitism, 2 were related to toxin ingestion and 1 was diagnosed as ulcerative enteritis. There was a single case of intestinal mycobacteriosis (*Mycobacterium avium*) diagnosed in 1995. Hepatitis (liver inflammation) was noted in 13 animals including two isolates each of *Streptococcus equi* subsp *zooepidemicus* and *A suis,* as well as non-specific bacterial isolates. Tyzzer's disease (*Clostridium piliformis*) was diagnosed in 7 cases. Eight cases of emaciation were diagnosed, and 4 animals presented with severe starvation.

The urogenital system was the second body system most commonly involved. A large proportion of these consisted of abortion submissions (154); 91 of these were idiopathic (due to unknown causes). Bacterial infection (18) was the most commonly recognized cause of abortion. Organisms identified included *Strep equi* subsp *zooepidemicus* (11), *Strep equi* subsp *equi* (2), *Strep dysgalactiae* subsp *equisimilis* (1), *Rhodococcus* spp (1) and *Pseudomonas aeruginosa* (1). There were 6 cases of abortion due to equine herpesvirus infection and a single case attributed to aspergillosis (a form of fungal infection). Umbilical torsion was diagnosed in 13 pregnancies, and congenital anomalies were recognized in 9 neonates and fetuses. Stillbirths were noted in 9 cases, nonspecific placentitis (inflammation of the placenta) in 6, and placental degeneration in five. In 11 neonates, there was deficient colostral ingestion of colostrum characterized by hypogammaglobulinemia. Kidney lesions were identified in 21 animals, and were attributed to 10 cases of kidney inflammation (nephritis), 2 end-stage kidneys (nephrosis), 4 horses with nonspecific interstitial nephritis, and 1 case of renal oxalosis. Renal oxalosis is a condition characterized by large numbers of oxalate crystals observed within kidney tissue, and in animals may have a variety of causes including, for example, ingestion of oxalate-containing plants and grasses, or man-made toxins such as ethylene glycol (antifreeze).

The cardiovascular system ranked third in diagnoses. Hemorrhage attributed to trauma, sepsis (the presence in the blood or other tissues of pathogenic microorganisms or their toxins), and other disease processes were apparent in 26

cases. Disseminated intravascular coagulation (DIC) was diagnosed in 8 horses. Anemia was apparent in 5 animals, including 2 confirmed cases of viral equine infectious anemia.

Bacterial septicemia (blood poisoning) involving primarily young horses was identified in 27 cases. The most common bacterial isolates included *Actinobacillus equuli* (6), *A suis* (5), nonhemolytic *Escherichia coli* (3), *Strep equi* subsp *zooepidemicus* (3), *Actinobacillus* spp (2), *Klebsiella* spp (2), *Strep equi* subsp *equi* (1), *Pseudomonas fluorescens* (1) and a single case of mixed *infection including A suis* and *Strep equi* subsp *zooepidemicus*. There were 29 cases of peritonitis that were usually associated with gastrointestinal compromise or accident, with significant bacteria isolated in 7 of these animals including *Strep equi* subsp *zooepidemicus* (3), *A suis* (1), *A equuli*(1), and *Bacteroides* spp (1).

Diagnoses related to the respiratory system included 21 cases of bronchopneumonia. Eleven of these yielded bacterial agents including *Strep equi* subsp *zooepidemicus* (5), nonhemolytic *E coli* (2), *Rhodococcus equi* (2), *Klebsiella* spp (1), and one case yielding a mixed flora including *Pasteurella* spp, *Strep zooepidemicus*, and *Actinobacillus suis* (1). Aspiration pneumonia was noted in 11 animals with isolation of *Strep zooepidemicus* (3), nonhemolytic *E coli* (2), and *Actinobacillus* spp (2) isolated from 7 of these submissions. Pulmonary emboli were noted in 3 animals. *Strep zooepidemicus* was isolated from 2 of these, and 1 specimen yielded no significant growth. There were 6 cases of pleuritis associated with *Rhodococcus equi* and *Strep equi* subsp *zooepidemicus*; a single case was associated with *Salmonella hadar*. Only 3 cases of guttural pouch empyema were diagnosed over the 7 years, including identified isolates of *Strep equi* subsp *zooepidemicus, Bordetella bronchiseptica,* and an unidentified fungus. Only 2 cases of chronic obstructive pulmonary disease were diagnosed during the study period.

Musculoskeletal lesions were limited to 8 arthropathy cases (disease processes involving the joints) and 7 laminitis (founder) cases. There were also 9 cases of muscle inflammation associated with *Staphylococcus aureus* (3), and *Clostridium* spp (2); 4 cases yielded no significant growth. Trauma was noted in 15 animals, with 10 of these related to limb injuries, and 2 cases involving vertebral fractures. Fractures linked to pre-existing pathological change were not apparent.

Despite a recognized lack of selenium throughout the province, nutritional cardiomyopathies (diseases of the heart muscle) and skeletal muscle degeneration/ necrosis were noted in only 17 animals. This observation likely reflects diligent supplementation and optimal nutritional management.

Within the central nervous system, 6 cases of bacterial meningitis were reported; 3 had significant bacterial isolates including nonhemolytic *E coli*, *Actinobacillus* spp, and *Strep equi* subsp *zooepidemicus*. Eight cases of encephalitis were also recorded during this 7-year period, including 2 of bacterial origin, 1 of viral etiology, and 4 of undetermined cause. The AHC sentinel case of equine protozoal myelitis was diagnosed on February 16, 1996. The case involved a 10 year-old imported quarterhorse, which had resided in the province since 1991. Although not included in this survey, 2 additional cases of equine protozoal myelitis were confirmed earlier this year (2000).

Over the 7-year span, there were 98 tumors diagnosed. Lipomas (14), pituitary adenomas (14), and lymphoma (13) were the most commonly identified tumors, with fewer sarcoids (10). Other tumors identified included melanomas (6), thyroid adenoma (4), thyroid carcinoma (2), pulmonary carcinoma (3), pheochromocytomas (3), and squamous cell carcinomas involving the skin (4), conjunctiva (1) and urinary system (1).

Listerial (L. *ivanovii*) encephalitis in a llama:

Contact – J Coates and G Thiele, AHC

Listerial meningoencephalitis due to the bacterial organism *Listeria ivanovii* was diagnosed in a 10 year-old llama. According to the owner, the animal had a brief clinical history of illness for about 2 days characterized by circling, recumbency, and finally by terminal convulsions. Three other mature llamas in the same group were not affected.

The most significant microscopic findings on the animal were observed within the brain tissue, where there was severe inflammation (meningoencephalitis). This was most apparent within the medulla and brain stem area. The inflammatory reaction was both lymphocytic and suppurative, characterized by frequent cuffing of vessels, numerous microabscesses, and swelling of brain tissue. (Practitioners take note: if you are requesting an examination of brain tissue for *Listeria* in any animal species, always submit the brain stem, cerebellum, and medullary tissue for both culture and histopathology; random slices of cortex are inadequate and often yield negative or inconclusive findings.)

As reported in past issues of this newsletter, listeriosis has been regularly diagnosed in animals submitted to the AHC for pathological examination. The organism is widely distributed throughout the environment, and has been isolated from soil, plants, as well as decaying vegetation and silage (ph over 5.5) in which it mulitiplies. Asymptomatic fecal carriers occur in man and many animal species. Silage is commonly implicated in outbreaks of listeriosis in cattle and sheep. (PJ Quinn, ME Carter et al; *Clinical Veterinary Microbiology*, 1999: Harcourt Publishers Ltd, Toronto, New York).

This animal had been fed grain and a hay ration prior to the onset of illness in mid-November. Although it appears that listerial cases develop in domestic animals when there are stress-related seasonal changes occurring, this laboratory diagnoses listerial encephalitis in submissions at any time of the year.

Aside from the fairly brief clinical course and brain lesions anticipated with a listerial bacterial encephalitis, the organism isolated form this camelid case was *L ivanovii*, whereas the vast majority of isolates are usually *L monocytogenes*. *L ivanovii* is differentiated from L monoctyogenes on the basis of biochemical tests performed on cultures of the isolate (*Clinical Vet Microbiology*, 1999, Quinn et al). *L ivanovii* has also been linked to abortion in sheep.

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Spirochaetal colitis in grower pigs:

Contact– J Coates, J Waddington and C Byra (Greenbelt Veterinary Services)

Loss of body condition, reduced growth rate, and limited death losses were observed in a group of 100 grower pigs aged 12 – 14 weeks or older. The animals had a variably soft to loose stool; no blood was observed grossly within feces. Although most of the pigs were bright, alert, and continued to eat, affected individuals gradually became slab-sided and developed a prominent backbone; a few developed secondary rectal prolapses.

Gross necropsy on 3 submissions revealed mild bronchopneumonia. All had dilated spiral colons, with yellowish-green contents that adhered to the underlying mucosa; there was no evidence of blood. The small intestinal tract (including the ileum) of all

animals appeared essentially normal on gross necropsy. Extensive sampling of tissues followed, for additional laboratory studies.

Escherichia coli (*E coli*) predominated within the intestinal microflora, although *Clostridium perfringens* was also present in light to moderate numbers. All cultures were *Salmonella sp* negative. All tests for porcine TGE coronavirus or other viruses were negative. Fecal flotation for coccidial species indicated a moderate level of coccidial oocysts in one of the 3 pigs. Fecal samples were all negative for the cecal worm *Trichuris suis*.

Microscopic examination of spiral colon (utilizing both standard H&E and Warthin-Starry stain) revealed a patchy, dense matting of the colonic mucosa with numerous spirochaetal organisms. In some areas the organisms assumed a picket fence appearance, arranged perpendicularly to the colonic enterocytes; others were arranged loosely in interweaving or parallel masses to the mucosa, or within the glandular crypts.

Colon contents were positive for the spirochaetal organism *Serpulina pilosicoli*, via the PCR test procedure. No spirochaetes were observed within the small intestine. Moderately numerous *Balantidium coli* protozoal organisms were also observed microscopically within colon contents, together with a predominantly Gram-negative bacterial flora. A few coccidial organisms (*Eimeria spp*) were observed within the small intestinal mucosal cells of one specimen. A mild diffuse enteritis was also present in these animals, with mild to moderate villus atrophy. Although no lesions were observed either grossly or microscopically that were consistent with proliferative ileitis (intestinal adenomatosis) in any of these animals, the identification of L*awsonia intracellularis* via PCR does indicate the high sensitivity of this test.

It is noteworthy that the clinical progression of the disease observed in these animals, and the colitis observed microscopically, was distinct from swine dysentery, which is attributed to the spirochaete *S. hyodysenteriae*. In addition, the numbers of bacteria seen within the lumen of both the small and especially the large intestine, combined with an obvious proliferation of the protozoan *Balantidium coli*, indicated a significant disturbance of the normal gut microflora.

Microscopically, the characteristic feature of spirochaetal colitis is a densely formed mat of spirochaetal bacteria which adhere to the colonic epithelium. The lesions of spirochaetal colitis resemble a mild form of swine dysentery (Radostits, Gay, Blood & Hinchcliff: *Veterinary Medicine*; 9th ed, 2000; W B Saunders Co). From a veterinary practitioner's point of view, spirochaetal enteritis/colitis is a significant differential diagnosis in grower or feeder pigs with persistent clinical signs of looseness of stool and loss of body condition.

In summary, it is likely that the enterocolitis observed in these animals, including the colitis associated with *S. pilosicoli*, was linked to poor sanitation within the pens. In discussions with the owner and on-site visits by the consulting veterinarian, it was learned that there have been ongoing mechanical problems in the barn with keeping flush gutters clean, resulting in incidents of pit overflow and excessive exposure of the animals to manure. Some animals had been noticed by the owner to be ingesting accumulated feces.

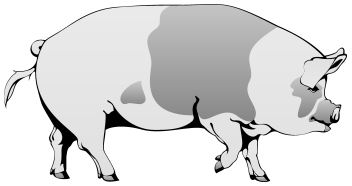
This case demonstrates the complementary role of both the consulting veterinarian and the diagnostic pathologist/laboratory, working together to solve or ameliorate a livestock owner's veterinary health concerns. It also demonstrates the dependent relationship that exists between raising healthy animals of any species and the cleanliness of their environment. Where this relationship is allowed to deteriorate, health problems can be anticipated.

Serpulina Spp PCR testing at the AHC:

Dr. John Robinson, Head of the AHC's Molecular Diagnostics Section, reports that a polymerase chain reaction (PCR) test is now available for *Serpulina pilosicoli*, the bacterial agent linked to spirochaetal colitis in pigs. The test is highly accurate, and targets a unique 16S ribosomal target. The PCR test identifying *S pilosicoli* will not cross-react with *S. innocens*. The test has recently been used successfully at the AHC on suspected necropsy samples, and is used in conjunction with a positive control that was kindly provided by a colleague of Dr. Robinson's from the State of Kentucky. Dr. Robinson notes that a PCR test for *S hyodysenteriae*, the causative spirochaetal agent of swine dysentery, is also available at the AHC.

Farm biosecurity and multiple outbreaks of Porcine Transmissible Gastroenteritis (TGE) in the Fraser Valley:

Contact – J Waddington, Health Management Veterinarian, BC Ministry of Agriculture, Food and Fisheries, AHC.



There have been two outbreaks of the viral disease TGE (Transmissible Gastroenteritis) on 3 separate pig farms in the Fraser Valley since November 15th, 2000. Because this agent is spread via fecal contamination (a manure-based virus) it would be worthwhile for veterinary practitioners to review the farm biosecurity measures of their clients so that the likelihood of animals contracting this disease is reduced.

In general terms, farm biosecurity is the exclusion of disease-causing organisms or their potential carriers from a farm site or facility. In a practical sense, biosecurity on a farm site implies limited and restricted access to animals (e.g., pigs) that have been secured within a controlled environment. Restricted access applies to farm workers, visiting persons and their vehicles, other animals both wild and domestic, as well as birds, rodents, and insects.

TGE virus may be introduced to a pig farm by contaminated manure. This is most commonly carried onto the farm site via boots, shoes, clothing, truck beds and tires, or anything else to which manure adheres. Rodents, cats, dogs, birds (starlings), and possibly flies can also spread the virus between locations. Feed products may also be a source of the live virus, if in any way contaminated by infected manure. The smallest amount of manure, under the right conditions and at the right time, can initiate the infection. Carrier pigs are also a potential source of the virus.

TGE can enter a facility at any point, affecting a pig of any age. In the two most recent outbreaks within the Fraser Valley, signs of mild to moderate diarrhea were noted in the finisher and grower barns. Once established in the pig population, spread of the virus is usually quite rapid, although in the recent Fraser Valley outbreaks movement through the various age groups within barns was slower than had been seen in previous cases. The reasons for this are unknown at this time. Clinical signs are most severe in neonatal piglets, where it causes extremely high pre-weaning mortality, usually within the first two weeks of life. Most outbreaks of TGE result in at least three weeks of total production loss. Chronic TGE causes additional ongoing losses, and generates a larger proportion of poor-doers.

Because the virus is quite stable at low temperatures, this disease is most common during the winter months. One of the first observed clinical signs is a watery, yellowish diarrhea, commonly associated with vomiting in younger animals. Often these signs are seen in one or more sows, followed immediately by similar signs in their newborn piglets. Unfortunately, this is usually followed within 24 - 48 hours by piglet death.

Spread through a barn is usually rapid. Containment to a particular area of the pig farm is almost impossible. Most animals over 1 month of age will show mild signs, and then recover. If the virus does become established within a facility, there are proven strategies available for minimizing the impact of the disease and for eradicating it. However, a much less costly approach is to prevent the introduction of the organism.

Prevention by biosecurity:

The basis of any biosecurity program is to minimize the introduction of unwanted infectious organisms onto the farm-site and into the barn. For this reason, two separate but interconnected strategies are used: *farm-gate biosecurity* and *barn-door biosecurity*:

Farm-gate biosecurity:

1. All vehicles entering the farmyard should be cleaned and disinfected. This includes your own, as well as any commercial trucks hauling pigs, feed, or manure.
2. Dead-stock haulers **should not** be driving to the barn door; a disposal facility should be placed at the road. Better yet, farm employees should do the hauling, to ensure responsibility for the cleanliness of the vehicle when it is returned.
3. Any vehicle not required for barn/barn door access should not be used for this purpose.
4. Restricted access signs to the barn should be posted. If possible, a gate should be erected to prevent unwanted traffic approaching it.
5. Separate clothing and footwear that must be used by employees while marketing pigs. Marketing boots and cover-alls should not be worn in the cab of the truck to prevent vehicle contamination.
6. Any transfer of animals for purchase or sale should be via an off-site truck-to-truck transfer, using the same clothing and footwear precautions as described above. Separate coveralls and disposable boots should be used for this purpose.
7. Any equipment that may have seen previous use at a hog facility, including any used or rented equipment, must be thoroughly cleaned and disinfected before it is brought onto the farm.

Barn-door biosecurity:

1. All areas outside the barn or beyond a defined perimeter fence should be considered “contaminated”, and treated as such. For this reason the easiest, and least costly biosecurity insurance you can buy (if a shower in/shower out policy is not in place) is a separate set of boots and coveralls for all staff (including kids!) for areas inside and outside the barn.
2. If a shower (in/out) is not in use, a well-defined and rigidly-maintained (boot and clothing) change area should be used. This area should be located at the main entrance door, which should be the *only* human entry/exit. No cross-traffic should take place beyond this point. If any cross-traffic occurs, either with boots or stocking feet, the primary purpose of this inside/outside barrier is defeated. If the farm has more than one building without enclosed walkways, this change should occur at each barn.
3. All persons entering the barn must use the biosecurity system that has been put in place - including children!
4. Consistent and aggressive control of rodents, birds, and flies should be ongoing.

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Farm biosecurity and multiple outbreaks of Porcine Transmissible Gastroenteritis (TGE) in the Fraser Valley: ***(Continued)***

5. Dogs and cats (those with access to the outside) are kept from barn entry.
6. When loaded onto a truck for shipment, weaner or market pigs should be prevented from re-entering the barn.
7. No unnecessary persons should be allowed into the unit. Those persons allowed entry should adhere to the biosecurity system in place.
8. Entries/exits to the pig operation should be carefully restricted. Removal of dead-stock should be through the most convenient exit. The owner/operator should avoid stepping beyond (i.e., outside) the biosecure area of the barn (admittedly, this can sometimes be awkward with a 450-lb dead sow). Regular boot and cover-all change is then made at the usual access door, thus enabling removal of dead stock and its transport to the collection site.

Having and adhering to a biosecurity plan is in the best interest of the veterinary practitioner and the client's farm enterprise. Implementing and diligently following such a plan can prevent the introduction of diseases such as porcine TGE. The situation of each farm operation will vary, and biosecurity should be customized to fit the unique circumstances of each farm. With or without concern over porcine TGE, the above recommendations are sound practice for any hog farm. Many other infectious diseases, such as swine dysentery or salmonellosis, are also readily introduced to a pig farm by many of the same routes.

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Cardiomyopathy in farmed Atlantic salmon parr:

Contact – S Raverty (AHC) and J Brocklebank (Brocklebank Mobile Services, Courtenay, BC)

With the intensification of the aquaculture industry worldwide, cardiomyopathy in salmon is increasingly being recognized as a production disease not only in growing and harvested stock, but also in the early- development stages of production. Cardiomyopathy is a general diagnostic term referring to primary disease of the heart (myocardium). Although cardiomyopathy has been previously recognized in saltwater netpen sites in British Columbia, this unusual case is believed to be one of the first instances recognized in a hatchery facility.

Fish stocks involved were crossbred (Cascade x Movi) Atlantic salmon. The eggs had originally been incubated in 8°C surface water until the yolk was internalized and resorbed (buttoning up), and were then maintained at 9°C until first feeding. Following the introduction of feed, the fish were acclimated to 11-12°C recirculated water. Affected parr were destined to be either S0's (smoults of less than a year of age) or early S1's (yearling smoults).

Close evaluation of the stock failed to reveal any clinical indication of disease within the hatchery. Water quality and stocking rates throughout the early production phases were considered optimal. Because stock raised under similar environmental conditions in the previous year had developed marked cardiomyopathy and mortality during the second seawinter growing period, fish were collected as part of a pre-purchase examination.

On gross necropsy, internal and external examination of the stock failed to reveal any significant lesions. Gill wet-mounts and skin scrapings were negative for ectoparasites. The midventral abdomen of necropsied specimens was incised, enabling the preservation of the fish in 10% neutral buffered formalin.

Histopathology of representative samples disclosed variably extensive heart muscle degeneration (cardiomyopathy) characterized by loss of myofiber sarcoplasmic striations, cytoplasmic vacuolation, compensatory nuclear hypertrophy and scattered endothelial accumulations of macrophages and lymphocytes. There were no other significant findings within the remaining tissues.

Cardiomyopathies have been previously reported in farmed Atlantic salmon fry raised during the egg stage at elevated water temperatures. However, this is believed to be one of the first instances in which eggs that were incubated within the normal, reference range for the species developed cardiomyopathy following acclimation (as recently hatched fry) to higher water temperatures for promotion of accelerated growth. The precise pathogenesis of the degenerative changes within the heart muscle have not been fully resolved. Because these animals are described as ectothermic (the regulation of body temperature is influenced by the external environment rather than by internal metabolism), increased metabolic demands associated with elevated environmental temperatures may exert undue physiologic stress on the heart. Heart muscle decompensation over a protracted period of time may then follow, with subsequent acute mortality.

Without refinements in current management strategies, perennial losses of these fish stocks are anticipated. Additional efforts to sample and evaluate the progression of these fish stocks will be attempted, to further characterize this disease process.

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Pneumonia in neonatal and juvenile harbor seals (*Phoca vitulina*) due to *Streptococcus phocae:*

Contact – S Raverty and W Fiessel, AHC

As part of an ongoing collaboration with the Department of Fisheries and Oceans (DFO), the Vancouver Aquarium Marine Mammal Center, and various coastal wildlife rehabilitation centers, post mortem examinations are carried out periodically at the Animal Health Centre (AHC) on neonatal and juvenile harbor seals. These are specimens that have been found dead along the seashore, or that have succumbed during attempts at rehabilitation. Approximately 202 harbor seals have been necropsied during the last 10 years. This summer, an unusual bacterial pathogen was isolated in 4 animals, and was presumptively diagnosed histopathologically as the causative agent in an additional harbor seal specimen.

One adult and 4 juvenile harbor seals from the Lower Mainland were submitted for necropsy, together with 1 specimen from Tofino, Vancouver Island. The animals generally presented in fair to poor body condition suggestive of hypoglycemia and possibly hypothermia. Three of the animals had massive swelling of the lung tissue, which was consolidated and frequently exuded frank blood (hemorrhage) on sectioning. The lesions were present within the distal half to third of lung parenchyma of either the left or right cranial lobes, or within the right middle lung lobes. Regional lymph nodes were occasionally enlarged and swollen (edematous). In more severely affected seals, there was evidence of bacterial septicemia.

Pneumonia in neonatal and juvenile harbor seals: *(Continued)*

Multifocally extensive expansion of alveolar spaces was observed microscopically immediately below the visceral pleura of the lung, and throughout the lung tissue. These areas were frequently flooded with serofibrinous exudate admixed with mild to moderate accumulations of degenerate and necrotic neutrophils, fewer macrophages, and random accumulations of Gram-positive bacterial coccoid organisms. Similar bacteria admixed with fibrin were occasionally noted within the lumen and along the endothelial lining of small to intermediate-caliber blood vessels.

Aerobic bacterial culture of multiple tissues from each of the examined seals revealed either pure growth of *Streptococcus phocae,* or a mixed culture of alpha hemolytic *Strep* spp, *Pseudomonas* spp, and *Escherichia coli*. In most cases the isolates were susceptible to a range of antibiotics, although resistance to trimethoprim sulfa and occasionally to gentamicin was consistently noted.

Strep. phocae was originally identified in harbor seals in Norway, in 1994. A subsequent epizootic of mortality and abortion was reported from Cape Cross, Namibia, in 1999. The cases reported here are considered to be the first isolates of this organism from harbor seals originating from the western seaboard of Canada. Attempts to locate the geographic distribution of this organism in wild stocks via "trace-backs" are currently underway. Additional seals will be evaluated to further characterize the pathogenesis of this infection.

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Canine herpesvirus infection in 4 week-old pups:

Contact – J Coates

Two 4 week-old purebred Labrador puppies were submitted for necropsy following sudden death. One puppy was very fresh, the other had died 12 hours earlier. There were 4 remaining pups in the litter, all of which appeared normal. The dam had given birth to the pups uneventfully.

Necropsy findings in the 2 submitted pups were identical; both had an excess of fluid within the heart sac, chest cavity, and abdomen. Lungs were diffusely congested, edematous, and were uniformly meaty in consistency. Stomachs contained a small quantity of watery mucus. The intestinal tract was empty in both animals, and the intestinal mucosal epithelium appeared normal. Gall bladders were distended. There were areas of congestion and hemorrhage noted within the cortex of the kidneys.

Microscopically, a diffuse, severe, nonsuppurative, and focally necrotizing pneumonia was observed within lung tissue from the pups. There were also discrete, multifocal areas of necrosis seen within the liver, the adrenals, spleen, kidney, and at times within the intestinal mucosa. Brain tissue revealed multifocal areas of mild inflammation. Distinct intranuclear eosinophilic inclusion bodies (INIB) were readily observed within most affected tissues, notably liver, adrenal, and kidney. These histopathological findings were consistent with canine herpesvirus infection.

Tissues were positive for canine herpesvirus via the polymerase chain reaction test (PCR), as well as by the fluorescent antibody (FA) procedure. Specimens were negative for other canine viruses including canine distemper virus. Electron microscopy studies on multiple intestinal specimens were negative for canine parvovirus/coronavirus.

Canine (DNA) herpesvirus is a fatal viral infection of puppies; only canids are known to be susceptible. Transmission usually occurs by contact between susceptible puppies or groups of puppies, as well as by exposure to infected oral, nasal, or vaginal secretions of their dam or other dogs that have been allowed to commingle with the pups during their first few weeks of life. The case reported here originated within a dog-breeding establishment, where the possibility of exposure to the virus from other animals of different age groups was high. Interestingly, acute parvoviral enteritis developed a week or two later within a second group of older pups.

Canine herpesvirus infection usually occurs in pups from 1 - 3 weeks of age, less commonly in pups aged a month or older. Unfortunately, no vaccine is available. Exposed or infected mature females develop antibodies, and subsequent litters should be protected by maternal colostral antibody. Puppies that receive maternal antibodies may be infected with the virus, but disease evidently does not result. Careful sanitation routines within breeding establishments, coupled with strict separation of age-susceptible pups from other dogs on the property, should reduce the likelihood of exposure to this virus. The prognosis of any puppies that survive neonatal herpesvirus infection is often guarded, since damage to multiple body tissues is often irreparable.

For the practitioner and diagnostic pathologist alike, a differential diagnosis to canine herpesvirus interstitial pneumonia is canine parvovirus type 1. A brief commentary on canine parvovirus type 1 follows immediately in this newsletter. The comments are taken from the Fall 2000 edition of *Lablines* (Fall 2000 edition), a news bulletin published by the veterinary diagnostic laboratory of Colorado State University.

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Interstitial pneumonia in puppies:

"… We offer a polymerase chain reaction (PCVR) test for canine parvovirus (CPV) types 1 and 2. The well-known CPV-2 causes acute enteric disease of dogs. The effects of canine parvovirus type 1, also known as minute virus of canines (MVC), are less understood. Experimental oronasal exposure of specific pathogen-free puppies with the CPV-1 resulted in inapparent to severe disease. Those that developed severe illness had lesions that included bronchitis and interstitial pneumonia. Enteric signs or lesions were absent in the experimentally affected animals. Bronchial, bronchiolar, and alveolar epithelial cells appeared to be the sites of viral growth and correlated to the pattern of histopathologic changes… The seroprevalence of MVC hemagglutination-inhibiting antibodies appears to be high in adult dog sera from widely separated geographic areas of the United States…

In our necropsy laboratory, we commonly observed puppies that die after a brief course of illness. Some of these pups had histologically demonstrable interstitial pneumonia… Since no other pathogens were demonstrated (canine distemper virus, canine herpes virus, heavy cultures of bacteria indicative of septicemia/bacteremia) and enteric lesions were absent, it is assumed that CMV is the pathogen involved in these puppy deaths."

D Gould: *Interstitial pneumonia in puppies*. From the newsletter *Lablines*; Colorado State Univ Diagnostic Laboratories: Vol 5, No 1, Fall 2000.

Hyperelastosis cutis in horses:

"*Hyperelastosis cutis* of horses is a skin disease characterized by excessive stretchiness of the skin and separation of the skin from the underlying connective tissue. The skin can be pulled away from the body for a distance of 5 cm or more. Seromas form where the skin separates from deeper tissues and the seromas are often the first abnormality noted (*seromas are tumor-like accumulations of serum within tissues. Ed.)* The condition often affects the top-line from the poll to the tail, lateral thorax, and occasionally hind-legs as low as the stifles. The disease is usually recognized in the second year of life. Sometimes, it is noted when the horses are saddled and skin tears under the tack, but some horses tear simply by rolling on the ground. Histologic features are variable and biopsy alone cannot definitively diagnose the condition. Biopsies from several locations of a suspected case are useful, however, particularly in eliminating other causes of skin disease. No treatment is known and most affected horses have been euthanized."

– P Schultheiss: *Hyperelastosis cutis in horses – an emerging problem*. From the newsletter *Lablines;* Colorado State Univ Veterinary Diagnostic Laboratories: vol 5, No 1, Fall 2000.

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A user's guide to pathology services:

"…How many times have you received a pathology report and been dissatisfied with the pathologist because there was an inconclusive result or a questionable diagnosis? Some times we forget that clinicians and pathologists function as a team, and the quality of the submission by the clinician has a direct bearing on the ability of the pathologist to make a definitive diagnosis. Just as there are user's guides to ensure the best use of software on your computer, there are also guidelines on how to make the best use of a pathologist's services. These suggestions are the result of observations made during the past decade in a busy veterinary diagnostic laboratory.

***Impact of euthanasia*:** If you suspect that a cat or dog has pulmonary, pleural, or cardiac lesions, do not inject euthanasia solution into the thorax… the heart can be coated with brown fibrinous material and the lung can become firm and discolored. This artifact can be interpreted as a true lung lesion. Microscopically the lung may have a burned appearance, which can obscure other antemortem pulmonary lesions…

Necropsy procedures: never freeze a carcass or biopsy specimen if you expect a histologic examination to be performed. Freezing tissue creates ice crystals that severely distort the specimen, making it resemble an ice-skating rink that has been scarred by the blades of numerous skaters. This is especially true of tissues with a high water content, such as the liver and brain of neonates. The carcass should be refrigerated until delivered to the laboratory…

Submit the carcass as soon as possible after death, so the tissues are in fresh postmortem condition and not distorted by autolysis… Autolysis can cause even the best pathologist to miss lesions… This is especially true of microscopic lesions in the pancreas and gastrointestinal tract which autolyze quickly after death… Plan an elective euthanasia so the carcass can be submitted in fresh condition… during regular hours of operation…

Include relevant history with the submission, including signs of illness, rather than "ADR (ain't doin' right)" or "sick". Some clinicians feel that supplying as little information as possible makes the pathologist more objective; however, providing critical information to the pathologist *after* gross examination has been completed and the carcass has been discarded does not benefit either party. Pathologists generally are as busy as clinicians and need to use their time efficiently. If a complete history is provided prior to gross examination, it is likely that all questions will be answered by the pathologist's investigation.

If a large animal is euthanatized because of lameness, give specific information about which limb or joint was affected. The pathologist does not have the opportunity to observe the ambulatory abnormalities of the animal once it is dead. It is a daunting task to examine every joint of a horse or cow and, then, to examine the spinal cord if lesions were not observed in the limbs…

On a necropsy submission, always indicate whether the animal was euthanatized and by what method. There can be a lot of time wasted if the history is "found dead" and the pathologist finds signs of gunshot, strangulation, or massive head trauma. Do we contact the police? Or contact the owner to see whether they forgot to tell us something?…

If a field necropsy is performed and tissues are sent to a laboratory for examination by a pathologist, give a complete description of the gross lesions found… Again, do not withhold information from the pathologist with the intent of making the pathologist more intent and more diligent… Pathologists may become more cynical rather than more objective when they learn the truth…

Remember, transport of packages by mail or package handling services may take several days, rendering ice packs woefully inadequate….

Include organs relevant to the clinical signs of illness. That is, if the animal has neurologic signs, submit the brain. A pathologist cannot make a diagnosis of neurologic disease by examination of merely the thoracic or abdominal organs….

In biopsy specimens, submit specimens in neutral-buffered 10% formalin…. labelled as to site of origin…

If the lesion is large, submit several sections from different areas of the lesion. Large tumors may not have a uniform internal structure….. give a gross description of the lesion…. It is an important safeguard and may catch errors in the early stage. A clinician once submitted a lesion from the neck of a dog. Microscopic examination revealed that the tissue was testicle. This prompted an extensive search to find out where the error had occurred….

These suggestions are offered with the intention of improving communication between clinicians and pathologists….. Hopefully, this article will make clinicans realize just how important they are in the practice of pathology…"

– from an article by MB Petrites-Murphy: *User's guide to pathology services*. JAVMA;1998: 212: 362 – 364.

Fatal deer mouse hantavirus infection (pulmonary syndrome) in Peace River farmer:

Dr. Paul Schnee, regional medical officer of the Keeweetinok Lakes Regional Health Authority has issued a new warning for farmers in the Peace region following the death of a 22 year-old High Prairie man as a result of hantaviral infection. Dr. Schnee says the man came in contact with mouse droppings on two occasions; the first in an attic of an old house, the second in a vacant house trailer, about 2 weeks before displaying any symptoms. Two weeks is the usual incubation period of hantavirus infection. Classic hantaviral symptoms of vomiting, diarrhea, and shortness of breath were observed; the affected person died, despite vigorous attempts to save him.

The deer mouse is the primary host of Sin Nombre virus, the hantavirus principally associated with hantavirus pulmonary syndrome. Hantavirus-infected deer mice have been found in nearly every province of Canada. Other hantaviruses have been identified in rodent hosts, such as Prospect Hill virus in meadow voles, but none of these have been associated with human disease. Although cases of hantaviral pulmonary syndrome have been confined to the western provinces, the presence of infected deer mice in eastern Canada suggests a potential for infection across the country.

Deer mouse hantavirus is a zoonotic agent; that is, it is capable of spreading to humans, and causing human disease. Deer mice are distinguishable by their white underbellies and two-tone tails, which are dark on the top and white on the bottom. Prevention of hantaviral pulmonary syndrome can best be achieved through education of the general public on the risks associated with exposure to rodents and their feces, and the adoption of preventive practices.

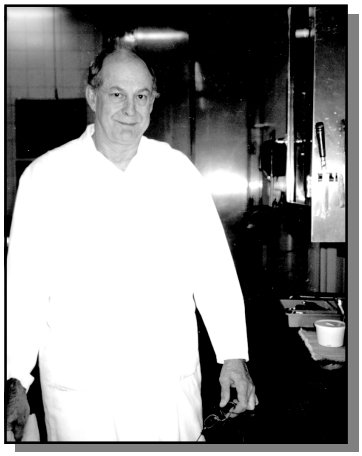
– composite information taken originally from the *Daily Herald Tribune*, an Alberta newspaper, and from *Pro-Med*, an internet website. *Ed*.

Smallpox vaccination trivia:

"Dr. Edward Jenner's 1796 discovery of vaccination, the imuunisation for smallpox, is well known. What is not so well known are the following anecdotes:

> The first child to be vaccinated in Russia was named Vaccinov and educated at the expense of the nation as a tribute to the discovery.
>
> During the 1870-1 Franco-Prussian War, vaccination was compulsory in the Prussian army where only 297 died of smallpox. It was not compulsory in the French army and 23,400 died form smallpox. The French also lost the war."

– taken from *More of Mould's Medical Anecdotes*, 1989, 224 – 225: Adam Hilger publishers, Bristol England.



Robert Hornidge, post mortem (necropsy) technician:

Robert Hornidge is a familiar figure to our clients who bring specimens to the necropsy area for unloading. Bob's duties include the preparation of animal specimens for necropsy by the pathologists. He is also responsible, together with Kim Carlsen, for the efficient processing of the swirl of messages, phone calls, computer data and papers/ reports that accompany each specimen to the necropsy area, and assuring their referral to the assigned pathologist or various laboratory sections. Bob also carries out microscopic examinations on various samples for external/internal parasites, in addition to various other duties such as the preliminary examination of submitted meat tissues from wild game for the zoonotic agent *Trichinella sp*.

Following many years work experience in the Ministry's poultry, dairy, and horticulture sections, Bob transferred to the AHC in 1996. Bob received training in his new work endeavour by Ms. Lisa McLaughlin, our senior necropsy technician at that time, and by AHC pathologists. Mr. Hornidge enjoys working in the necropsy area, for as Bob says, it is a unique combination of "on your feet" work coupled with more exacting demands such as examining parasitology specimens and receiving/processing information.

Mr. Hornidge is married with 7 children. His many outside interests include speed-skating (which he has coached in the past), running, cycling, as well as building and flying radio-controlled model aircraft. Recently, Bob has also been taking additional classes in horticulture. For those of us who work in the necropsy area, Bob Hornidge is a welcome sight each morning; his enthusiasm, natural athleticism and efficiency when preparing larger specimens for post mortem examination are essential ingredients in the efficient operation of the Necropsy room.

Kim Carlsen, necropsy technician:

A native of Burnaby, Kim Carlsen has an extensive background in various areas of clinical veterinary science. Ms. Carlsen has a BSc degree in biochemistry from UBC, and is also a licensed graduate of the University College of the Cariboo's Animal Health Technologist program.

Ms. Carlsen has worked with a wide variety of animal species, "from fish to elephants" as Kim says, for over 20 years - domestic and wild/zoo mammals, bird species, and marine life. Prior to her employment at the AHC, her workday in private veterinary clinics included collecting and interpreting results on biological samples, carrying out anesthesia, as well as aspects of surgery. Ms Carlsen also has practical, hands-on experience in radiology, ultrasound, and emergency medicine. Kim's special interests in her professional work have been Emergency Medicine, large animal anesthesia, and equine reproduction.

Together with Bob, Kim receives specimens from the owner/submitter and channels them appropriately, often carrying out preliminary microscopic studies herself, including parasitology exams, as well as various aspects of clinical pathology such as red/white blood cell analyses. Kim devotes time daily in the trimming of tissues for histopathological examination, as requested by pathologists; she also prepares the various chemical-preservation solutions that are used daily within the necropsy area.

Kim's hobbies are "keeping my children on the honour roll", reading and running, as well as enjoying the company of her dogs, cats, horses, birds, and hamsters. Kim notes that after 20 years of working and taking care of animals, "I still wake up each day wanting to go to work and do my best in a career with animals that I love". Kim is a recent addition to AHC staff, and we welcome her. Her dedication, humour, and talents find ample room for expression within the diverse needs of the AHC's diagnostic laboratory.

It's about Time:

"… We do not know what time is. We have no words ultimately to define it. The great and awesome equations of relativity and the theme of a space/time continuum help us ultimately as little as the classical concept of the Platonic year and its seasons.

…and time itself is under attack. Not a day goes by without my hearing someone say somewhere, 'I am just killing time' – time of which we have so little and which in any case kills all in the end when it is no longer right for them to live…

The rebellion against time shows itself perhaps most in the compulsion to make life faster. There is not so much a love as a lust for speed, for doing things quickly, which totally ignores the fact that time is nothing if not measured, and that every plant, animal, organisation, stone, star, and cosmic system has its own unique measure of time and this measure demands obedience to the rhythm of seasons and renewal. We however, improve on the 'killing time' mentality with the slogan that 'time is money', speeding up all the processes…beginning to manipulate life for our own busy ends so that in systems everywhere 'being' has been taken out of life and a compulsive and frantic 'doing' and busyness put in its place…"

– Laurens van der Post: *About Blady: a Pattern Out of Time*. Published 1991, by Mackays of Chatham, Kent.

Natural calling:

"…Give me a canoe and let me go
Up past Waskesiu to the Wabinow
Give me a canoe and let me go

Take me where the loons are calling
Lake like glass and a starlit sky
Smell of a small campfire burning
Sit and watch the embers die."

– lyrics from *Canoe Song*, by Connie Kaldor