Laboratory Services Division
Animal Health Laboratory
University of Guelph
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AHL Newsletter

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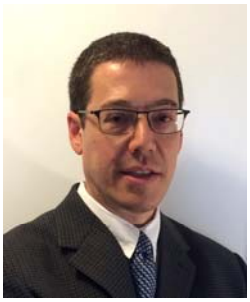
Dr. Felipe Reggeti, new AHL Clinical Pathologist - Clinical Toxicologist

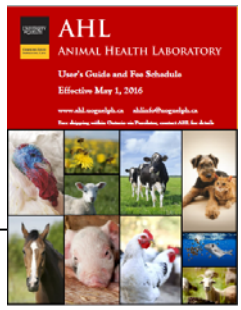


We will be welcoming Dr. Felipe Reggeti to the AHL as our Clinical Pathologist - Clinical Toxicologist, effective mid-July, 2016. Felipe graduated in veterinary medicine in Venezuela in 1992, taught biochemistry while earning an MSc in 1997, and earned his PhD in OVC Pathobiology in 2005. He taught clinical pathology at OVC for 3 years and passed the Clinical Pathology certifying examination of the American College of Veterinary Pathologists in 2008. For the past 7 years, Felipe has worked as a veterinary clinical pathologist in Toronto.

Dr. Brent Hoff will be mentoring Dr. Reggeti in Toxicology before retiring (again) this fall. We're delighted to have Dr. Reggeti join our team at the AHL.

May 1, 2016, AHL User's Guide and Fee Schedule

Includes more test information, new tests (bentpnl—see next page), and much more!
Also available on-line at http://www.guelphlabservices.com/AHL/#



DSP Client Outreach Technician

The AHL is pleased to announce that **Josie Given** has been named as Client Outreach Technician, a new 1-year position funded through the **Disease Surveillance Plan (DSP)** to provide a range of outreach activities for the lab. **Her position will focus on a voluntary pilot AHL milk bacteriology quality program.** Her clinic visits will include preanalytical aspects of laboratory submissions - sample handling and shipping, electronic and customized submission forms, premises ID, and submission supplies. Josie will be able to assist in all areas of in-clinic laboratory quality assurance, including clinical pathology and parasitology. This support should help clinics provide the best possible results to their clients.

Josie brings a wealth of experience from her current position as Client Services Technician at the AHL. Please help us in welcoming Josie to this position.

PID summer student

We are pleased to welcome **Kassie Dusome**, who will be working out of our AHL-Kemptville laboratory. Kassie is a first-year veterinary student at OVC with experience in the dairy industry. She will be helping to increase the use of Premises Identification numbers (PIDs) on food animal submissions this summer, along with other OAHN projects.

As an Ottawa native, Kassie is excited to be working back in eastern ON.

New comprehensive anticoagulant rodenticide (AR) screen

Nick Schrier

With the support of the Disease Surveillance Plan (DSP) - funded by the OMAFRA-University of Guelph Strategic Partnership under a joint federal-provincial Growing Forward 2 project - the toxicology section has implemented a new comprehensive anticoagulant rodenticide (AR) screen. The new screen is based on liquid chromatograph tandem mass spectroscopy (**LC-MS/MS**) instrumentation with limits of detection in the low parts-per-billion (ppb) range.

Anticoagulants included in the screen are: **difethialone, flocoumafen, bromadiolone, brodifacoum, difenacoum, chlorophacinone, coumachlor, diphacinone, dicoumarol, warfarin, coumafuryl (fumarin), coumatetralyl, pindone,** and **valone**.

The diagnosis of AR intoxication requires both the presence of one or more AR in appropriate samples (e.g., liver or serum) and antemortem or postmortem evidence of a coagulopathy unrelated to another identifiable causes of hemorrhage (e.g., trauma).

Submit 10 g liver, or 2 mL serum or plasma (not whole blood), or 20 g suspect material, with liver and serum being the best samples. Sample should be frozen. Price is $90 per sample and turnaround time is 5-10 business days. *AHL*

New bovine enteric panel *Jim Fairles*

This panel combines the 3 most common tests used in **neonatal bovine diarrhea workups**:

◊ Sucrose wet mount for *Cryptosporidium*.
◊ Bacterial culture for *E. coli, Salmonella, Clostridium perfringens.*
◊ Bovine Rotavirus/ Coronavirus PCR.

The sample required is feces in a sterile leakproof container.
The fee of $78 is discounted from the individual test fees ($84.50) and the short code is **bentpnl**. *AHL*

AHL Newsletter
June, 2016 - Volume 20, Number 2

Editor: **Grant Maxie,** DVM, PhD, Diplomate ACVP
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The *AHL Newsletter* is published quarterly (March, June, September, December) by the Animal Health Laboratory, Laboratory Services Division, University of Guelph.

Its mission is to inform AHL clients and partners about AHL current activities, and laboratory-based animal disease events and disease trends.

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Our continued thanks to all of the non-author AHL clerical, technical, and professional staff who contribute to the generation of results reported in the AHL Newsletter.

Ontario Animal Health Network

"Your comprehensive source for animal health information."

OAHN Update June 2016

What's new and exciting at OAHN?

◊ Network surveillance projects are underway - the 10 projects were announced in the March 2016 AHL Newsletter.

◊ New podcasts:

- Veterinary mental health podcast
- Recognizing signs of abuse in clients
- Rabies update
- Getting the most from your lab samples

◊ Upcoming podcast topics: *Cysticercus ovis* review, producer mental health, OMAFRA food and animal protection teams.

◊ Top animal health links of the week. Weekly rabies update.

◊ May 7th Small Flock Poultry Veterinarian Training day.

To keep up with what we are doing, please follow us on Facebook and Twitter, and sign up for the OAHN Updates Newsletter.

BEES

The OAHN Bee network held its most recent quarterly conference call on March 9. A report should be out soon.

SMALL RUMINANTS

The Q1 2016 survey was distributed and completed by 27 veterinarians, with a successful distribution across Ontario. The network had its Q1 conference call on April 25. Top items of discussion were: Cache Valley virus and diarrhea in goat kids. Check the OAHN website for veterinary and producer reports.

BOVINE

The OAHN Bovine network had its Q4 quarterly call in March and released its veterinary and producer reports. The report outlined the new *Salmonella* Dublin project. The Q1 2015 bovine survey will be available June 1.

FISH

The OAHN Fish network had its 6th quarterly conference call in April. A technical/producer report will be released shortly– stay tuned!

SWINE

The OAHN Swine network completed its Q1 2016 veterinary survey and had its quarterly conference call in April. Stay tuned for the report, which will be released soon!

ALTERNATIVE SPECIES

The Alternative Species network had its latest quarterly conference call on May 13. Open to any practitioner interested in mink. Interested? Email us!

POULTRY

The OAHN Poultry network held its Q2 2016 conference call at the end of May. The Q1 2016 (Nov 2015—Jan 2016) reports are available on OAHN.ca. As well, the OAHN Poultry network ran a successful Small Flock Poultry Veterinarian Training session on May 7, with >30 veterinarians attending in person and online. The course notes can be accessed here: **http://tinyurl.com/** (You must be logged in to OAHN to see the content).

Want to connect with other small flock poultry vets? Email oahn@uoguelph.ca

EQUINE

The OAHN Equine network held its Q1 2016 quarterly conference call at the end of April, and the veterinary and owner reports are available now on OAHN.ca. The main discussion points in these reports were the OAHN Lyme research study, recent cases of hydrops in mares, EHV-1, and respiratory agent testing.

WILDLIFE

CWHC continues to share quarterly reports with OAHN networks. Additionally, an OAHN Wildlife group is currently being explored.

COMPANION ANIMALS

The OAHN companion animal network had its Q1 2016 conference call at the end of April, and released its veterinary report in the first week of May. Disease issues discussed included: giardiasis, tick and tick-borne disease update, salmonellosis in BC, and a rabies update.

Check out the new Tick risk infographic here (please note that you must be signed in with a veterinary account): **http://tinyurl.com/j9bbf3v**

Want to receive veterinary reports? Email oahn@uoguelph.ca

AHL Lab Reports

RUMINANTS

AHL camelid autopsy diagnoses, 2006 to 2016

Janet L Shapiro

Between 2006 and 2016, 13 llamas and 126 alpacas, were submitted to the AHL-Guelph and AHL-Kemptville locations for postmortem examination.

Abortion/Stillbirths		**1-14 d-old**		**15 day to 1-y-old**		**>1-year-old**		**Total**
alpaca	llama	alpaca	llama	alpaca	llama	alpaca	llama	
17	0	23	0	23	3	63	10	
17		23		26		73		**139**

Abortions: In 16 of the 17 abortion/stillbirth submissions, no infectious etiology was found. Three of the abortions were associated with umbilical cord torsion. A protozoal etiology was suspected in a mid-gestation abortion submission with nonsuppurative encephalitis, multifocal hepatic necrosis, mild placentitis, placental necrosis, and mineralization and from which no bacteria or viruses were isolated. Congenital cataracts have been reported in camelids, and we received 1 stillbirth with bilateral congenital cataracts from which no bacteria, viruses, *Mycoplasma* spp. or *Chlamydia* spp. were detected.

1-14-day-old camelids: In 7/23 neonatal crias, the most common problem was congenital defects, of which the most common was choanal atresia. Presenting signs were dyspnea and mouth-breathing since birth. Other developmental anomalies in this group included unilateral renal agenesis, renal cysts, multiple congenital heart defects, including persistent truncus arteriosus and ventricular septal defect. One cria, paretic since birth, had kyphosis, scoliosis, and subluxation of 2 cervical vertebrae. Systemic bacterial infection was caused by *E.coli* in 5/6 cases, and was associated with omphalitis in 1 case. Intestinal cryptosporidiosis, associated with a history of severe diarrhea, was diagnosed in a 10-day-old cria. The cria also had severe acute renal tubular necrosis; gentamicin renal toxicosis was suspected. Lymphosarcoma involving liver, kidney, spleen, bone marrow, lung and lymph nodes was diagnosed in 2 14-day-old crias, for which the predominant presenting sign was diarrhea in one and fever in the other. Lymphosarcoma is one of the most commonly diagnosed neoplasms of llamas and alpacas. *West Nile virus* (WNV) was detected by PCR and immunohistochemistry in a 14-day-old cria with meningitis, but its clinical significance was unknown as the cria also had lesions consistent with a systemic bacterial infection in liver and lung.

15-day to 1-year-old camelids: The most common clinical signs in this group were neurologic, and diagnoses included idiopathic encephalitis, meningoencephalitis, meningomyeloencephalitis and meningitis, as well as vertebral osteomyelitis, paravertebral abscesses, and cervical spinal cord degeneration resulting from previous neck trauma. An arachnoid cyst, dysgenesis of the occipital condyles, and hydrocephalus with secondary cerebral and hippocampal atrophy was diagnosed in an 8-mo-old alpaca that had a 1 mo clinical course of blindness and staggering.

Septicemia caused by *Streptococcus equi* subsp *zooepidemicus* was diagnosed in 2 cases. This is the etiological agent of "**alpaca fever**" and is one of the most important diseases of camelids in some regions in South America. Transmission is via ingestion from contaminated objects or direct contact with infected animals, and systemic disease is often associated with physiologic stressors. Additional diagnoses in this group were mesenteric torsion, emaciation secondary to impaction of stomach compartment 3, urolithiasis, severe coccidiosis, systemic bacterial infection, and dilated cardiomyopathy in an 8-mo-old alpaca that also had partial collapse of the trachea interpreted as a congenital defect.

>1-year-old camelids: Neurologic disease was the most common diagnosis in camelids >1 y-old. Lesions consistent with parasitic meningoencephalitis caused by "meningeal worm", *Parelaphostrongylus tenuis,* were found in 7 alpaca cases. Some affected herds had multiple cases of parasitic meningoencephalitis in consecutive years. Also diagnosed were polioencephalomalacia (2 cases), WNV infection (3 cases), cerebrocortical or intervertebral disc abscesses (3 cases), intervertebral disc protrusion with compression of the spinal cord, and degenerative myelopathy associated with

Continued on p. 17

Alpaca autopsies—continued from page 16

previous severe neck trauma. A nursing alpaca with a 5-day course of anorexia, incoordination, and abnormal mentation had disseminated parasitic myositis and mild nonsuppurative encephalitis with large intramuscular organisms resembling *Sarcocystis* sp., possibly *S. aucheniae*. The significance of the intramuscular parasite is not known; infection with many species of *Sarcocystis*, including *S. aucheniae*, is usually an incidental finding.

Digestive tract disease was the second most common diagnosis in this age group. Dental diseases, including excessive tooth wear, malocclusion, tooth root abscesses, complicated by maxillary or mandibular osteomyelitis and/or septicemia, were causes of anorexia, lethargy, or emaciation. Two alpacas were euthanized because of choke. Gastric ulceration involving stomach compartment 3 was a frequent diagnosis, occasionally as resulting in death from bacterial and/or mycotic invasion, or perforation with secondary septic peritonitis, as well as an incidental finding in animals dying from other causes or associated with the use of non-steroidal anti-inflammatory drugs. Multiple alpacas in one herd had gastritis attributed to grain overload. Vague non-localizing clinical signs were caused by gastric squamous cell carcinoma in a 13-y-old alpaca. Lethargy or colic were the clinical presentations of mesenteric torsion in 2 cases, 1 complicated by jejunal rupture, and a case of diaphragmatic herniation of compartment 1. Mixed gastrointestinal parasitic infections were a cause of emaciation or weight loss resulting in euthanasia, and were also incidental findings in animals dying of other diseases. Intestinal parasites identified including *Nematodirus* sp., *Trichuris* sp., *Capillaria* sp., and *Hemonchus* sp., as well as *Monezia* sp., *Eimeria* sp., *Eimeria punoensis,* and *E. macusaniensis.*

Emaciation was the third most common disease diagnosis. In >50% of cases, there was no predisposing disease found, and a primary nutritional etiology was suspected. Emaciated animals were found dead in 6/15 cases, but others had a short clinical course of lethargy or weight loss for a few hours to a few days before dying or being euthanized.

Liver lesions interpreted as nonspecific toxic hepatopathy, in 1 case accompanied by lesions of hepatic encephalopathy, were found in 3 cases. Hepatic necrosis and lipidosis was associated elevated liver copper levels and clinical signs of staggering and weight loss in a 7-y-old alpaca. Hepatic lipidosis was often found in conjunction with other diseases, such as local of systemic bacterial infections, enteric parasites, and gastric ulceration. Hepatocellular carcinoma was diagnosed in a 4-y-old alpaca.

Sporadic diagnoses in this group included urolithiasis, skeletal myopathy, idiopathic myocardial fibrosis, and bacterial myocarditis complicated by hemopericardium and cardiac tamponade. *Prevotella melaninogenica* bacteria were cultured from an intervertebral disc abscess and from a case of severe cellulitis of the neck. This organism may be found in the oral cavity, and in our cases, may have caused abscesses as a result of systemic spread from dental disease or bite wound of a carnivore. *AHL*

Overnight prepaid courier services offered by AHL is continuing with Purolator Express Return Labels!

AHL has traditionally offered a prepaid overnight courier service for **Ontario clients** through Purolator Courier.
The good news! This is NOT changing. The method that is used to do this however IS changing.
The prepaid collect AHL courier account number is being retired on June 30, 2016, and is being replaced by Purolator Express Return Labels. We will be using these return labels for all prepaid Purolator Courier packages that are destined for the AHL. Please email ahl.supplies@uoguelph.ca to order your **Purolator Express Return Labels**!
See our LabNote 46 for more details http://www.guelphlabservices.com/AHL/LabNotes.aspx

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Do you recognize this plant? *Margaret Stalker*

Yew (*Taxus spp.*):

Who is at risk? Horses, livestock, and humans.
What part is toxic? All parts of the plant are toxic, except for the fleshy red aril covering the seed.
Clinical signs? Sudden onset of trembling, incoordination, difficulty breathing, slow heart rate, sudden death.
Comments:

- ◊ Yews are hardy evergreen shrubs used widely in landscaping.
- ◊ They contain taxine alkaloids that interfere with heart function, and are exceptionally toxic plants.
- ◊ **Yew poisoning is the most commonly diagnosed plant toxicity at the AHL**, typically causing sudden death in cattle and horses.
- ◊ Yews should never be planted around animal enclosures, and prunings should be kept away from all livestock, as they remain toxic even when dry. *AHL*

See also: http://www.omafra.gov.on.ca/english/livestock/horses/facts/07-055.htm

AVIAN/FUR/EXOTIC SPECIES

Atypical mycotic infection in poultry: *Aspergillus oryzae*

Emily Martin, Alex Weisz, Lloyd Weber, Rachel Ouckama

Since November 2015, there have been multiple cases of atypical mycotic infection submitted to the AHL involving layer and broiler breeder flocks. The flocks had clinical signs of increased mortality and severe runting/stunting.

On gross postmortem examination, there were multifocal to coalescing firm cream nodules throughout the lungs, air sacs, and multiple other organs (Figs.1, 2). These nodules were firm and homogeneous throughout on cut section. On histopathology, these nodules were composed of numerous clusters of multinucleated giant cells surrounding fungal organisms (Fig.3) and occasionally coalesced into larger granulomas (Fig.4). This appearance was quite different from the more common mycotic infections caused by *Aspergillus fumigatus* that occur as large well-developed multifocal to coalescing granulomas. The fungal organisms in these cases affected multiple organs including the lungs, liver, kidney, and brain.

These nodules were forwarded to the AHL Bacteriology Laboratory for mycology, and **on wet mounts *Aspergillus* sp. was identified.** These cultures were directed to the AHL Molecular Biology lab section for 18SrRNA sequence analysis. This initial analysis revealed a 99.0% sequence similarity (474/479 bp) to multiple members of family *Trichocomaceae* (*Aspergillus*, *Penicillium*, *Eurotium*, *Neosartorya*, *Edyuillia* spp., etc.) as well as uncultured clones (i.e., *Aspergillus flavus*, *A. fumigatus*, *A. tamarii*, *Penicillium javanicum*, etc.). For further characterization, the nuclear ribosomal internal transcribed spacer (ITS) region was sequenced and found to have 100% similarity to both *A. flavus* and *A. oryzae*. Then the cytochrome c oxidase subunit I (COI) gene was analyzed to attempt to make a final identification of this fungus. **The COI partial gene (640 bp) sequence revealed that this fungus had 100% similarity to *Aspergillus oryzae*** (and 90% sequence similarity to *Aspergillus flavus*).

Aspergillus oryzae, also known as kōji (Japanese), is a filamentous fungus used in Chinese and East Asian cuisines. It is used to ferment soybeans, to make soy sauce, and make fermented bean paste. It is also used to saccharify rice, grains, and potatoes to make alcoholic beverages.

Overall, these were cases of increased mortality and atypical mycotic lesions. The origin of the fungus is not known. *AHL*

Figure 1. Clinical signs of runting/stunting.

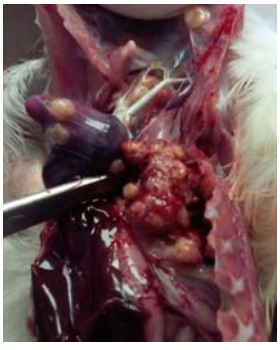


Figure 2. Multifocal to coalescing firm cream nodules affecting multiple organs.

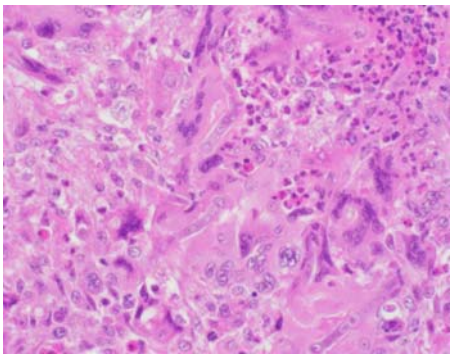


Figure 3. Numerous clusters of multinucleated giant cells surrounding fungal organisms (x600)

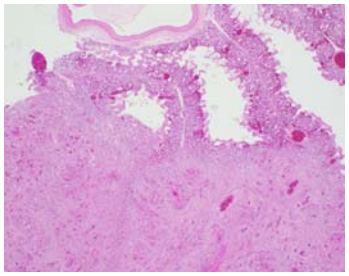


Figure 4. Large fungal masses occupying large areas of lung (x40)

SWINE

Metabolic bone disease and associated testing in finished pigs

Murray Hazlett, Martin Misener, Steve Ensley

Market hogs from a large finishing system that purchases feeder pigs from a 3,700 sow farrow-to-feeder pig farm were having issues with increased losses associated with shipping, including **pigs with fractures at loading and at slaughter**. The 3 pigs in the case came from 3 different barns - 2 were liquid fed and one was a wet/dry feed system. There was no clinical evidence of confounding disease issues.

When these pigs were submitted for autopsy, the ribs would tend to fold and crumple, rather than snap. Several areas of bone were examined histologically, with efforts aimed at the second rib (this is the sample of choice for a metabolic bone profile done in the veterinary diagnostic lab at Iowa State University). Histologically there was thinning of the cortex compared to control pigs (Fig. 1). Areas of fibrosis were present in the medulla and surrounding the cortical bone.

The 3 clinically affected pigs all had the lowest levels of bone ash (Table 1). The value for one of the control pigs was also below reference intervals (RI) but above that of affected pigs. Bone density was higher in affected pigs, perhaps because of replacement of fat with fibrous tissue. Bone ash calcium and phosphorus values were both higher than the control pigs but within RIs, likely a reflection of less bone, but the bone present had a higher percentage of mineral.

It was suspected that the problem in these pigs was due to a **reduced level of micronutrient inclusion for the last 6 weeks of the feeding period**. The formulation was corrected and the problem resolved.

Submissions for bone ash testing can be done through the AHL, and we forward the specimen to the Iowa State Diagnostic Laboratory for analysis. Costs for a typical submission of 4 second ribs from 2 pigs (2 for histology and 2 for bone profiles) would cost ~$363 total (this varies with the Canadian/USD exchange rate). *AHL*

Table 1. Results of bone profile testing (Iowa State Veterinary Diagnostic Laboratory).

Test	Reference interval	Affected pig A	Affected pig B	Affected pig C	Control pig A	Control pig B
Bone ash	58-62%	40	36	37	56	49
Bone density	1.4-1.5 g/mL	1.26	1.23	1.24	1.2	1.2
Bone ash calcium	32-39%	40.8	42.9	31.4	28.5	29.3
Bone ash phosphorus	13-22%	21.2	21.8	16.8	14.2	14.4

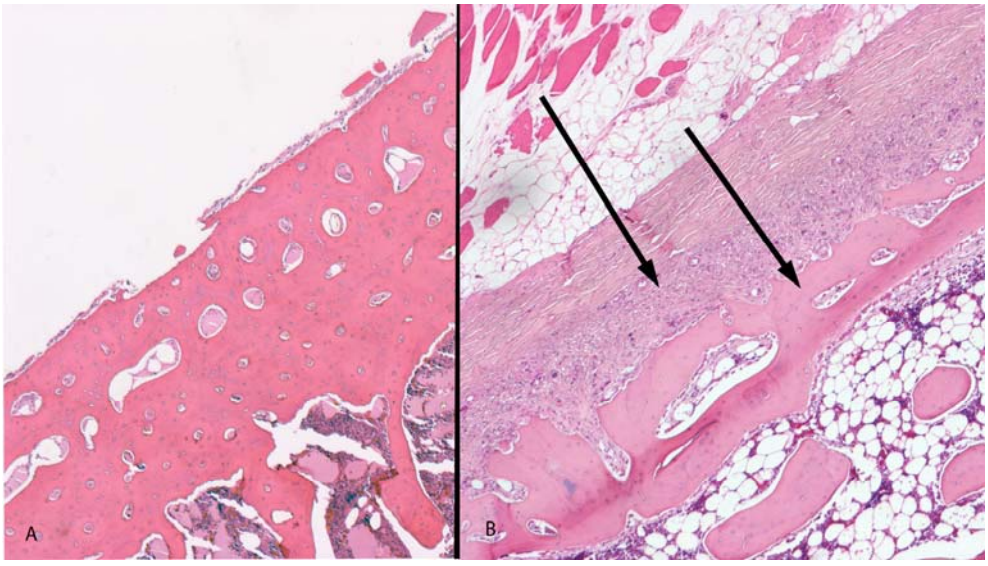


Figure 1. Control rib (left) showing normal cortical thickness and affected rib (right) showing fibrous tissue proliferation (left arrow) surrounding a thin cortex (right arrow).

HORSES

Idiopathic CNS inflammatory disease in horses in Ontario

Murray Hazlett

Equine central nervous system cases are seen on a fairly regular basis at the AHL. From mid-2007 until December of 2015, pathologists from the AHL and Department of Pathobiology have logged 195 cases. Many of these are degenerative diseases such as equine degenerative myelopathy (EDM) or spinal cord malacia associated with vertebral stenosis (wobbler syndrome) (Table 1). There were 54 cases with idiopathic degenerative or inflammatory CNS lesions documented, 24 of these being inflammatory. Many of the inflammatory changes were very mild lymphocytic cuffs in a few sections that the pathologist felt was not significant (*n*=13). **From the reports, 10 cases had significant inflammation with no etiology identified and that the report indicated may be viral.** Depending on the time of year and duration of clinical signs, testing was done for EEEV, EHV, WNV, and *Rabies virus* without an etiologic diagnosis being established.

The age of these horses with idiopathic encephalitis ranged from 6 d to 18 y, with clinical signs ranging from 24 h or less to "months". Three were identified as Thoroughbreds, 2 Standardbreds, and 2 Quarter Horses. No geographic predisposition was seen.

The clinical presentation and lesions seen in these horses varies. The mild "incidental" perivascular lymphocytic inflammation seen in some horses may be a residual lesion from a previous bacterial embolus or a resolving viral lesion. It is a recognized incidental lesion in horses.[1] In reviewing AHL records, perivascular cuffs were not considered incidental if they were present in association with clinical neurologic disease or in relative abundance.

Routine testing on neurologic cases at the AHL is done for *Rabies virus* (CFIA), *Eastern equine encephalitis virus*, *Equid herpesvirus-1*, and *West Nile virus*. Other causes of viral encephalitis are possible and are usually not tested for, including other arboviruses (*Powassan virus, St. Louis encephalitis virus, Snowshoe hare virus,* WEEV) as well as viruses not identified in Canada such as VEEV (Fig. 1). *AHL*

Table 1. Causes of central nervous system disease in equine submissions to the AHL, 2007-2015.

Cause	n
EDM - equine degenerative myelopathy	26
EPM - equine protozoal myeloencephalopathy	23
Wobbler syndrome	21
Fracture/trauma (skull or vertebral	18
Eastern equine encephalitis virus	10
Bacterial	8
Hepatic encephalopathy	7
Neoplasia (including cholesteatoma)	7
CNS disease, no lesions	6
Equid herpesvirus	5
West Nile virus	5
EMN - Equine motor neuron disease	2
Rabies virus	2
Parasitic (*Halicephalobus*)	1
Undiagnosed degenerative or inflammatory	54
Total	**195**

Reference

1. Jahns H, et al. Age-related and non-age-related changes in 100 surveyed horse brains. Vet Pathol 2006;43:740–750.

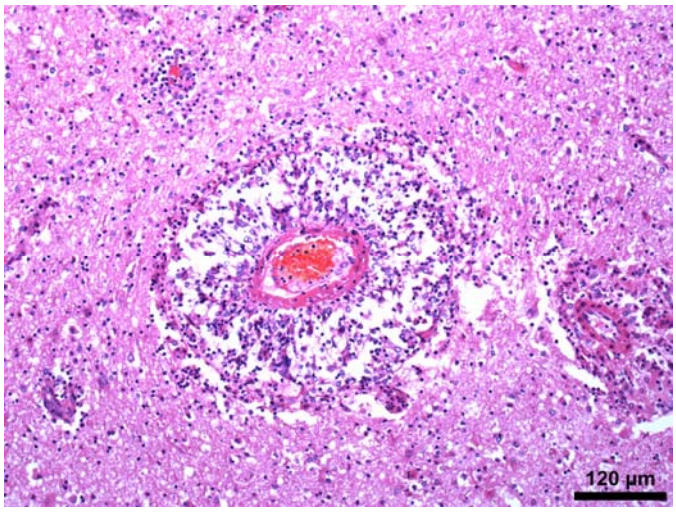


Figure 1. Severe meningoencephalitis in a 4-year-old Thoroughbred.

COMPANION ANIMALS

Companion animal autopsies at the AHL: What do we do to assist practitioners?

Margaret Stalker, Beverly McEwen, Michael Deane

As part of the AHL data contribution to the new Companion Animal Expert Network of the Ontario Animal Health Network (OAHN), canine and feline submissions to the AHL-Guelph and AHL-Kemptville for the last quarter of 2015 and the first quarter of 2016 were reviewed to determine: 1) reason for submission for autopsy examination, and 2) final diagnosis.

Cases were sorted into 8 general categories based on reason for submission for autopsy examination:

- Investigation of anesthetic-associated deaths (including death during premedication, induction, maintenance, or the immediate recovery period following anesthesia, for a variety of procedures).
- Investigation of deaths resulting from post-surgical complications.
- Investigation of deaths possibly associated with an adverse drug or vaccination reaction.
- Determination of cause of sudden unexpected death, or animal found dead.
- Determination of underlying disease process in ill animal, died or euthanized.
- Determination of underlying disease process in ill animal exhibiting neurologic signs, died or euthanized.
- Determination of cause of abortion/stillbirth.
- Medicolegal/forensic investigations.

Although investigations into cause of sudden unexpected death and cause of illness are the most common case types, medicolegal/forensic cases and investigations into anesthetic-related death also form a significant part of our companion animal autopsy caseload (Fig. 1).

An underlying cause of death for anesthetic-related deaths was determined in 4 of 10 cases in dogs (various diagnoses, including cardiomyopathy and congestive heart failure), and in 2 of 3 cases in cats (cardiomyopathy).

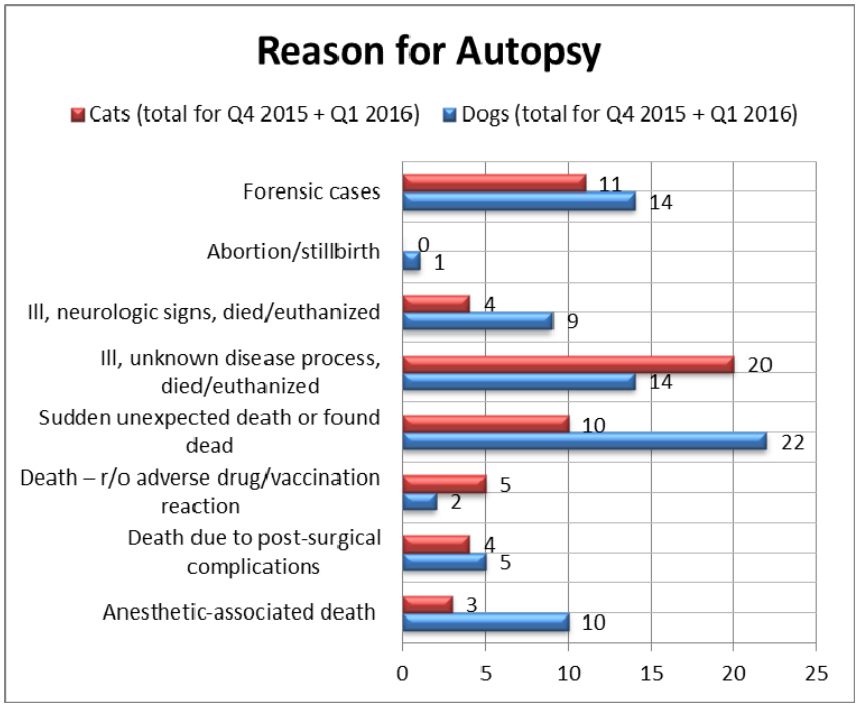
The leading cause of sudden death in dogs is hypovolemic shock or cardiac tamponade due to hemorrhage from a ruptured neoplasm (10 of 22 cases), usually hemangiosarcoma (7 cases).

The leading cause of sudden death in cats was cardiomyopathy (6 of 10 cases).

A wide variety of diagnoses were made in the other categories of investigation.

We continue to track quarterly data in order to identify any emerging trends. For more information on the OAHN Companion Animal Network, see http://oahn.ca/ or email us at oahn@uoguelph.ca . *AHL*

Figure 1. Reasons for autopsy of dogs and cats at the AHL, Oct, 2015 - March, 2016.



Update on the diagnosis of canine mast cell tumors

Andrew Vince

Since our previous newsletter article on the diagnosis of canine mast cell tumors (Cytology and surgical biopsy of canine mast cell tumors, 2010), there have been several changes in pathologists' approach to this disease. A commonly used grading scheme for dermal mast cell tumors (MCT), developed by Patnaik in 1984, suffered from a limited concordance (64%) for the diagnosis of grade 1 and 2 tumors, and 75% for the diagnosis of grade 3 tumors, with the majority of mast cell tumors categorized as grade 2. This grading scheme was also often used in the prognostication of subcutaneous mast cell tumors, though it was not originally developed for this purpose and it has long been recognized that MCT originating in subcutis often have a better prognosis (relative to dermal MCT). Since then, **2 studies have evaluated canine cutaneous and subcutaneous MCT as independent entities** with an eye to developing more objective methods of subclassifying these lesions into those more likely to behave in a benign or malignant fashion.

Kiupel et al (2010) subclassified cutaneous MCT into 2 categories, **low-grade and high-grade**, with a weighted agreement between pathologists of 99.3%. **High-grade cutaneous MCT** were characterized by 1 of the following: the presence of 7 or more mitotic figures in ten 400X fields, 3 multinucleated (3+ nuclei) cells in ten 400X fields, 3 or more bizarre nuclei in ten 400X fields, or karyomegaly. Dogs with high-grade cutaneous MCT had a median survival time <4 months, whereas dogs with **low-grade dermal MCT** (Fig. 1) had a median survival time >2 years. Overall, **this new 2-tier grading scheme is a better predictor of survival than the traditional Patnaik grading scheme**, and as such it is a valuable standard to apply to these tumors.

In addition, Thompson et al., 2011, evaluated subcutaneous MCT, and dogs with subcutaneous MCT with <4 mitotic figures in ten 400X fields had a mean survival time >891 days. Dogs with subcutaneous MCT with >4 mitotic figures in ten 400X fields had a median survival time of 212 days, reduced to 140 days with the presence of infiltration and multinucleation. **Because of the prognostic difference between cutaneous and subcutaneous MCT, it is important to collect a sample that will represent this location by including skin and subcutis.**

In a proportion of cases, diagnosis may be aided by histochemical ('special') stains (toluidine blue stain) or by immunohistochemistry (c-KIT/CD117, Ki-67). Additional external assessment is available, including c-KIT PCR and AgNOR evaluation, which may provide additional therapeutic and prognostic information for individual cases. Your surgical pathologist may order special stains on your submission to confirm the diagnosis, and may advise you on further testing recommendations.

Because the cytologic features of canine MCT are usually straightforward, **cytology provides the best option for initial diagnosis prior to surgical planning** (Fig. 2). A very recent study by Camus et al., 2016, has also provided a novel method for grading cutaneous MCT based on cytologic features, principally mitotic indices, presence/absence of binucleate or multinucleate cells, nuclear pleomorphism, or dramatic anisokaryosis. Cytologic grade was found to correlate well with histologic grade, and mean survival times were similar between cytologic and histologic grading categories. Further investigation and broader clinical validation is required before this can be applied as a routine procedure with cytologic examination of MCT. *AHL*

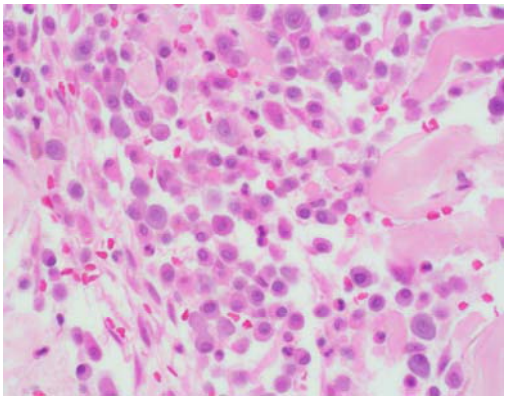


Figure 1. Histopathology from a low-grade canine cutaneous mast cell tumor, 600X magnification, Wright's stain. Numerous well-granulated mast cells separated by edema, moderate numbers of eosinophils and small quantities of hemorrhage.

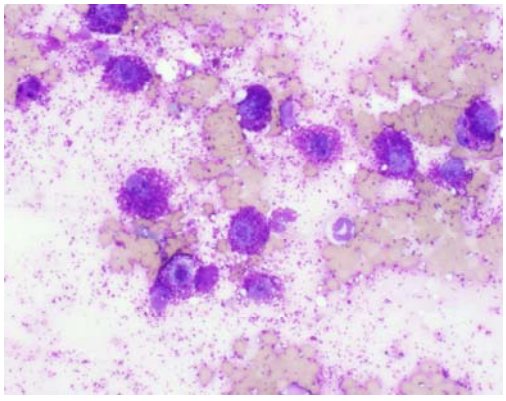


Figure 2. Aspiration cytology from the same low-grade canine cutaneous mast cell tumor, 600X magnification, Wright's stain. Numerous widely spaced and well-granulated mast cells, some hemorrhage, and few eosinophils.