



# Newsletter

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## From the Director:

I extend a very warm welcome to Dr. Robert Glock, distinguished colleague and friend of many years, who will join the AzVDL faculty in March as the new director of the unit. A veterinary pathologist with many years of diagnostic laboratory experience in Iowa, Arizona and Colorado, a former Director of the diagnostic laboratory at CSU, and an internationally recognized food animal health consultant, Dr. Glock will be the new administrator of this unit and will collaborate in the pathology services.

*Carlos Reggiardo, Director*

## Diagnostic Update

The following are selected samples of cases submitted to the AzVDL during the winter months of December, January and February.



### Bovine

**Ionophore poisoning** was diagnosed in a group of 60 day old Holstein calves. Eleven calves died over a two day period. They were being fed grain containing lasalocid and had access to molasses/mineral blocks containing monensin. Post-mortem examination of three of the dead calves revealed pleural and peritoneal effusion and mesenteric edema. One calf also had pericardial effusion. Histologically there was myocardial necrosis and centrilobular hepatocellular necrosis. Analysis of the grain ration revealed 1219 grams lasalocid per ton of grain. This is approximately 20 times the recommended level for this ionophore.

Beginning two weeks after arrival from out-of-state 25 of 45 pregnant Limousin cows at a sale barn developed signs of weakness, weight loss and hind limb pain leading to recumbency and death. The cows had been shipped during a storm and arrived in cold, rain/snow mix weather. Six cows eventually died. Two were necropsied. Lesions included multifocal bronchopneumonia with pulmonary abscesses, vegetative endocarditis, necrotizing myositis and pseudomembranous rhinitis all with bacterial colonies evident; a **group C *Streptococcus*** was isolated. In the lung, but not elsewhere, the *Streptococcus* sp. was commingled with *Hemophilus somnus*. Other findings included chronic cholangitis consistent with prior liver fluke infection and deficient liver copper and selenium levels. This case was a primer on the contribution of environmental, transport and nutritional stresses in the production of severe clinical disease.

Multiple eight-month-old, spayed heifers in a feedlot exhibited clinical signs of lateral recumbency and eyes rolled back in the head. There was no rigidity or hyperesthesia. All received treatment with thiamine, dextrose, tylosin and spectinomycin. The feed included silage, ground hay, cottonseed and gin trash. Postmortem examination of two heifers revealed red/yellow foci of infarction in the thalamus. Microscopically, there was multifocal hemorrhagic necrosis of the thalamus with vascular thrombosis, vascular necrosis and massive infiltration of neutrophils. Aerobic and anaerobic cultures of the thalamic lesions were negative. The gross and microscopic lesions are compatible with **thromboembolic meningoencephalitis** due to *Haemophilus somnus* infection. The organism is very sensitive to antibiotics and often cannot be recovered from animals receiving treatment prior to death.

Five, six-month-old, Beefmaster heifers from a herd of sixteen died suddenly over a seven day period. A field necropsy examination performed by the referring veterinarian revealed ecchymotic and petechial hemorrhages on multiple organs and free blood in the abdomen and thorax. Histologic examination revealed acute necrotizing myositis of the myocardium, random hepatocellular necrosis in the liver, multifocal splenic hemorrhage and acute interstitial pneumonia. Small blood vessels in the lung and liver sinusoids contained many gram-positive bacilli. Small numbers of bacilli were present in the interstitial tissues of the myocardium. Frozen sections of lung and liver were fluorescent antibody positive for *Clostridium chauvoei*, the causative agent of **blackleg**.

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**Oleander poisoning** was diagnosed in a cow that was found dead unexpectedly. The cattle were being fed a grass-alfalfa hay but had access to a dump where oleander, cedar, and pine yard trimmings had been placed. **Oleandrin** was detected in a sample of rumen ingesta from the cow by thin-layer chromatography. Ingesta from the alimentary tract is the preferred specimen for testing. Urine (50 ml minimum) is sometimes positive as well if the animal survives long enough to excrete oleandrin. All parts of the oleander plant are exceedingly toxic. One reference states that mature cattle and horses can be poisoned by eating an ounce or less of the green leaves. The effects of oleandrin are similar to those of the glycosides of the digitalis group and clinical signs include a rapid heart rate, weakness, and a sometimes bloody diarrhea. In the horse, colic and sweating are also reported. Treatment is similar to that of digitalis poisoning and is often said to be unrewarding.



## Equine

A twelve-year-old Quarterhorse mare was euthanized following progressive clinical signs of ataxia. The mare had an elevated WBC of 24,200/ml with an absolute lymphocyte count of 14,278/ml. The lymphocytosis was composed of small and intermediate-sized lymphocytes. The total plasma protein was 7.9 g/dl with an A/G ratio of 0.82. Serum electrophoresis demonstrated elevation of the beta fraction; 2.8 g/dl (N= 0.7-2.5 g/dl). Gross lesions included a 4 cm diameter, pale, tan, fleshy mass in the mesocolon. Scattered 1-2 mm diameter, red/gray plaques were present on the dura mater of the spinal cord. Microscopically, the diagnosis was **malignant lymphoma** involving the meninges and substance of the brain and spinal cord, spinal nerve roots, stomach, small intestine, colon and a colonic lymph node.



## Avian

**Tuberculosis** was diagnosed in four-year-old female Pacific Parrotlet that was found dead. Moderate bilateral atrophy of the pectoral musculature was evident at necropsy and there were multifocal pale areas in the liver. Microscopic examination revealed multifocal accumulations of macrophages laden with acid-fast bacilli in lung, liver, ovary, and skin. In the intestine, there was mucosal effacement and lamina propria of mucosa contained severe diffuse infiltrates of macrophages laden with acid-fast bacilli. Although human infection is reported to be uncommon, avian tuberculosis is considered to be a zoonotic disease.

**Trichomoniasis** along with a pneumonia was diagnosed in a four-day-old Homing pigeon squab. *Pasteurella* sp. was

isolated from lung tissue. The loft owner reported that squabs were dying 3-6 days after hatching. The parents were vaccinated with paramyxovirus and paratyphoid vaccines 7-10 days prior to hatching. There was multifocal necrotizing inflammation of the crop mucosa and cultures of the crop contents were positive for trichomonads.

**Lymphoid leukosis** was diagnosed in a male show chicken of unspecified breed that had been purchased at a show. The bird was originally from Florida. Anorexia, diarrhea, and emaciation were noted prior to death. Necropsy revealed an enlarged, tumorous liver. Microscopic examination revealed that infiltrates of neoplastic lymphoid cells had obliterated most of the normal architecture of liver, splenic, kidney and bursa of Fabricius.

**Urolithiasis with severe renal atrophy, visceral gout, and pyelonephritis** were diagnosed in several laying hens used for research purposes. The causes of urolithiasis leading to renal failure and various associated secondary changes in chickens are reported to include calcium excess in the diet, dehydration, and early infection by infectious bronchitis 'virus. In this case, feed analysis determined that both dietary calcium and phosphorus were low and that there was a relative calcium excess (10:1 versus desired 4:1). Dietary calcium should be at least 3.25-3.50 % and phosphorus about 0.75 % for laying hens.

**Histomoniasis** was diagnosed in a seven-year-old male Plover from a zoo. Gross necropsy findings included miliary, 1-3 mm diameter, caseous tan foci in the liver. *Histomonas meleagridis* was cultured from the liver.

*Salmonella typhimurium* was isolated from the tissues of a lovebird received for necropsy. This was one of ten birds to die in the past three months. Clinical signs were that of birds sitting on the ground followed by death in about twelve hours. Necropsy lesions included multifocal hepatocellular necrosis and multifocal necrosis in the spleen with bacteria in the cytoplasm of macrophages.

A large, pedunculated **nephroblastoma** of 3 cm in diameter was found free in the abdomen of a seven-week-old African grey. Rupture of the thin vascular stalk which attached the tumor to the kidneys produced an acute intraabdominal hemorrhage, causing the death of the bird.



## Canine

A diffuse, **cardiac lymphoma** extending to much of the left ventricle was found during the necropsy of a four-year-old female Rottweiler which died of cardiac arrest. Cardiac lymphomas are considered metastatic in most species, but no primary foci were found in this case.

**Disseminated Intravascular coagulation (DIC)** was the cause of death of a seventeen-year-old female Heeler which died after a hemorrhagic episode of short duration. DIC had been precipitated by disseminated neoplasia; a mammary adenocarcinoma with lymphoid, pulmonary, renal and adrenal metastasis was found in necropsy.

A two-year-old, female mixed breed dog died after acute onset of illness at a boarding kennel. The illness was characterized by lateral recumbency and dehydration. Gross necropsy lesions included cerebral edema and a 2.0 cm diameter, soft, brown mass in the right pyriform lobe of the brain. The mass was histologically compatible with an **oligodendroglioma**.

**Severe anemia** secondary to a heavy tick infestation was the cause of death in a seven-month-old male Springer spaniel received for necropsy. This was one of two dogs to die following signs of lethargy and weakness the prior afternoon. Grossly the dog was thin and covered with ticks. The mucous membranes and viscera were extremely pale. The blood was pale red and watery. Centrilobular hepatocellular vacuolation consistent with hypoxia was found in sections of the liver.

**Pentobarbital poisoning** was the cause of collapse and miotic pupils in a dog. This was one of three dogs which exhibited similar clinical signs over the prior month. The owner requested euthanasia and necropsy examination of this dog. The euthanasia was performed with sodium pentobarbital euthanasia solution. Gross necropsy revealed that the stomach was full of hide, hair, decomposed flesh and horse manure. There were no microscopic lesions. Fortunately, the veterinarian had saved a serum sample collected prior to euthanasia. Pentobarbital was detected in the sample by gas chromatography-mass spectroscopy. The source of the poisoning was determined to be a horse carcass that was on or near the dog owner's property. The horse had been euthanized three months prior using pentobarbital euthanasia solution. The owner had witnessed the dogs chewing on the carcass. Apparently, sufficient pentobarbital remains in a euthanized animal to poison scavenging animals months later.

**Myelofibrosis with partial myelophthisis and anemia**, etiology not determined, was diagnosed in a four-year-old Soft-coated Wheaten Terrier that was euthanized for a non-responsive anemia. Treatment had included Epogen® and despite the non-responsive anemia and partial myelophthisis, there were some areas of bone marrow hyperplasia. Myelofibrosis is reported to be associated with non-responsive anemia, thrombocytopenia, and leukopenia from neutropenia. Myelofibrosis may be a terminal event in myeloproliferative diseases secondary to bone marrow damage or malignancies, or it may be idiopathic. It is also reported to occur as a consequence of prolonged erythropoi-

etic response to pyruvate kinase deficiency anemia in the dog.

**Myocarditis** of unknown etiology was diagnosed in a two-month-old Yorkshire Terrier that died unexpectedly. The pup had received its first vaccination about 2 weeks prior to its death. It was noted by the owner to be "less active" than other pups in the litter, all of which seemed healthy. At necropsy, there was locally extensive, bilateral whitish discoloration of the ventricular musculature and the tissue seemed very firm to the touch.

**Canine parvovirus infection** was diagnosed in a one-year-old Queensland canine that was being treated for dogfight wounds and "Kennel cough" at a dog pound. Lesions were typical of parvo-virus infection but electron microscopy was negative for virus particles. Presumably viral titer in gut contents had dropped below the detection limit of the procedure.

**Anesthetic death, idiopathic**, was diagnosed in a six-year-old male Labrador that had been anesthetized for removal of a large subcutaneous lipoma in the sternal area. A few minimal foci of myocardial fibrosis not associated with any active inflammation were found in epicardium. No significant lesions were otherwise found. An adequate morphologic basis for the dog's death could not be found. This case illustrates the fact that despite adequate pre-anesthetic physical, biochemical, and hematologic exams as well as diligent monitoring of oxygen flow and vital signs during anesthesia, general anesthesia is still a risky procedure.

**Contrecoup injury** to the brain was diagnosed at necropsy of a five-month-old male Wire-haired Fox Terrier weighing 13 pounds. When presented to the veterinarian the dog had a body temperature of 105.4. It had been noted to be lethargic and depressed with a swollen, painful muzzle about two days before presentation. Two days after presentation the dog was "almost comatose" and subsequently died. At necropsy, palpation revealed excessive lateral movement of the jaw. Dissection revealed bilateral fractures at the angles of the mandibles. Locally extensive hemorrhage was found on the dorsal aspect of the cerebral hemispheres of brain. Diffuse mucosal hemorrhage with ulceration was also present in stomach. It was felt that the cerebral lesions would account for the neurologic signs and it was suggested that they represented contrecoup injury occurring secondary to a blow delivered from underneath the jaw that caused the jaw fractures.

**Severe pancreatitis with associated intra-abdominal fat necrosis** was diagnosed in an eight-year-old female German Shepherd weighing 80 pounds. Prior to its death the dog had elevated values for glucose (447 mg/dl), amylase (3610 IU/L), and lipase (9160 IU/L). Necropsy revealed diffuse edema and hemorrhages in pancreas and dilated lymphatics

in serosa overlying the organ. Foci of fat necrosis were present in adjacent mesentery. Lipidosis was prominent in liver. A partially necrotic, mixed mammary tumor was also noted as an incidental finding.



## Feline

**Hepatocellular lipidosis (fatty liver syndrome)** was diagnosed in a four-year-old neutered domestic long-hair feline that was presented to a veterinarian with the history of “vomiting fluid”. A clinical chemistry profile revealed elevated alkaline phosphatase and SGPT (ALT) enzymes. The total bilirubin was 4.8 mg/dl and there was a leukopenia. The triglyceride level was not reported. The cat was obese and lesions were typical, with an enlarged liver that was discolored bright yellow being evident at necropsy. The cat was FeLV-negative. Fatty liver syndrome is a disease typically occurring in obese, nutritionally stressed animals. There is hypertriglyceridemia and the mortality rate is high.

A **chronic pancreatitis** was found in the necropsy of a five-year-old female DSH with a six month history of exocrine pancreatic insufficiency and insulin-dependent diabetes mellitus. There was an almost complete loss of the exocrine parenchyma which had been replaced by fibrosis and inflammatory infiltration. The few surviving islets contained vacuolated beta cells.

**Cardiomyopathy (endomyocardial form)** was diagnosed in a four-year-old neutered Siamese male, which was presented to the veterinarian with tachypnea, depression and ataxia of acute onset. On necropsy, there was congestive heart failure, with pleural effusion and ascites. There was enlargement of the left atrium, regional areas of left ventricle hypertrophy, and thickening of the endocardium of the left ventricle by fibrosis.



## Wildlife

***Corynebacterium pyogenes*** was isolated from a 4 cm diameter cerebral abscess in a five-year-old mule deer buck. The deer had been hanging around a residence in northwestern Arizona. It was not fearful of people and walked in circles within a very confined area. A mountain lion killed the deer before a wildlife manager arrived. Cerebral abscesses due to this organism are recognized with increasing frequency in the US deer population. The pathogenesis of the infection is not well understood. Trauma incurred during fighting seems a likely contributory factor as the condition is seen only in bucks.

**Herpesvirus** infection was diagnosed in a male Great-horned owl. The owl had recently been transferred from the Bronx Zoo to an Arizona wildlife facility. There were no clinical signs prior to death. Necropsy lesions included multifocal caseous plaques on the mucosa of the oral cavity and esophagus. Numerous 1-2 mm diameter yellow foci were present in the spleen and liver. Histologically, epithelial cells of the esophagus, hepatocytes and reticuloendothelial cells of the spleen contained intranuclear inclusion bodies typical of herpesvirus.

**Botulism** was diagnosed by the mouse protection assay of serum from a Mallard duck submitted for necropsy. It was one of ten plus birds that died by a golf course pond.

A four-year-old, female ferret had a two week history of progressive deterioration characterized by dehydration, hypothermia, cachexia and mucoid ocular discharge.

**Canine distemper virus infection** was diagnosed at necropsy.

Acute **lead poisoning** was diagnosed in a Bald Eagle that was estimated to be five or more years old. When the bird was found near Saguaro lake its breathing was “rattling”. At necropsy, bile staining of the feathers around the vent was prominent. The bird was in good flesh and there were no other lesions. Pesticide intoxication was ruled out. The liver lead level was reported to be 94 ppm (liver lead levels > 6 ppm are reported to be toxic in waterfowl). The source of the lead was not determined. Raptors are commonly victims of secondary lead poisoning; however, it is a result of feeding on lead-poisoned waterfowl. Lead shot has been banned for waterfowl hunting but poisoning among waterfowl undoubtedly still occurs as a result of ingestion of spent lead shot from the bottoms of lakes and ponds where hunting with lead shot has taken place in the past. To the best of our knowledge the current incidence of lead poisoning in waterfowl (and secondarily, raptors) since the lead shot ban has not been determined.

***Hypoderma lineatum***, the common cattle grub, was identified in tissue submitted by a hunter. The larva were found in the subcutis of a deer killed during last fall’s deer season. The common cattle grub is a parasite that is commonly present in range cattle in southern Arizona. Its preferred hosts are domestic cattle. Infestations are rarely reported in other species. The grub is the larval phase of a parasitic fly that is active during the spring months. Infection is acquired when the female fly lays eggs of the haircoat of the host animal. When the eggs hatch larvae emerge, they penetrate the skin and slowly migrate through the host’s tissues, undergoing development as they move. Ultimately they encyst in the subcutis of the back. In the early spring of the following year they eventually pass through a skin pore in the cyst and fall to the ground to pupate, forming a new fly, thus completing the life-cycle.



## Exotics

Tissues from a Phoenix area boa constrictor provided evidence for diagnosis of **inclusion body disease** of boas and pythons. The cause was a virus with characteristics of *Retroviridae*. The snake in this case had eosinophilic cytoplasmic inclusions in liver, kidney, stomach and brain. The snake had a history of recurring respiratory disease over more than a year. Necropsy disclosed *Trichomonas* infection and a caseous lung lesion.

**Pyometra** and a **diffuse fibrinopurulent peritonitis** were diagnosed in a one-year-old English Lop rabbit that was found dead unexpectedly. Purulent exudate filled the uterine lumen. The abdominal cavity contained malodorous, brown, turbid fluid and fibrinopurulent exudate covered the serosal surfaces of liver and uterus.

Acute death in llamas within 24 hours from symptoms was suspected of being caused by **oleandrin toxin**. Leaf remnants found in the stomach contents were identified by the owner and veterinarian as oleander. Of the several

llamas involved only two were necropsied; some recovered. Stomach contents from the two llamas proved to contain **oleander toxin** as suspected. Thin layer chromatography analysis was done by experts at the UC Davis School of Veterinary Medicine. Animals in the herd involved had a history of opportunistic consumption of Aleppo and Ponderosa pine needles as well as rose bushes and citrus leaves. The owner was sure oleander leaves were eaten but wasn't sure how many llamas ate them. Llamas that survived exhibited depression, lethargy, and cardiac arrhythmia for up to a day and one-half. This case is a reminder that long standing, long recognized problems stay with us.

*compiled by; Greg Bradley, T. H. Noon, Ray Reed, and Carlos Reggiardo*

Comments on Diagnostic Update can be directed to Dr. Greg Bradley via e-mail at: [gabrad@ag.arizona.edu](mailto:gabrad@ag.arizona.edu)

## Submission Tip:

The AzVDL routinely determines lead and zinc levels in blood samples submitted from caged psittacine birds suspected of having either intoxication. About 0.2 ml plasma (for zinc) and 0.2 ml whole blood (for lead) are needed for each determination. Submission of a blood sample in a **Microtainer®** is preferred instead of stoppered blood tubes to eliminate the possibility of zinc contamination of the blood sample. One **full Microtainer® (0.5 ml)** of blood in either heparin or EDTA will suffice for both determinations.

*T. H. Noon and Dana P. Betzer*

### Arizona Veterinary Diagnostic Laboratory

Access to the AzVDL: Take Tucson Interstate 10 to the Miracle Mile exit #255. Travel approximately 1/4 mile on the south bound frontage road between Miracle Mile and Grant Rd. Turn west onto the farm at the signed entrance.

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