



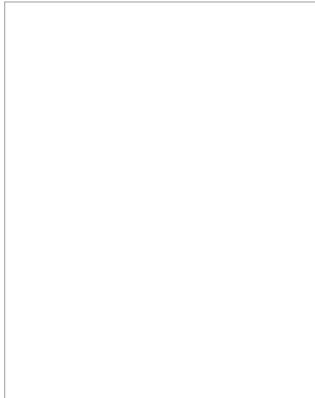
Newsletter

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Quarterly Publication of the **Arizona Veterinary Diagnostic Laboratory** and Cooperative Extension

From the Director:



Robert D. Glock
Director of the
Arizona Veterinary Diagnostic
Laboratory

My first three months as laboratory director have been a real opportunity to learn. Fortunately, I've had lots of help from people in the Diagnostic Laboratory as well as on the campus. I've had a number of inquiries about what happened to Dr. Reggiardo. He is happily transitioning out of administration and into diagnostics, research and continuing supervision of all microbiology. My objectives as director are to assist in enabling our faculty and staff to continue to provide excellent service and to seek ways to improve our client interactions through good practical diagnostics supported by good communications. I encourage input on how we can accomplish these goals. If you have ideas, please call me at 520-621-2356 or stop by.

In the "what's new?" category, we have had an opportunity to upgrade outdated and worn-out equipment in the office, histopathology and microbiology. We are also acquiring equipment to allow us to provide expanded service with ELISA testing and toxicology. Look for more specifics in the next newsletter.

We recently assisted in hosting a meeting of the Southern Arizona Cattlemen's Protective Association. It was an excellent opportunity to get better acquainted with some of our laboratory users. We hope to do more of this type of interaction in the future. We see an opportunity for expanding our outreach in continuing education efforts throughout the state. Our available personnel are limited but we can expand our capabilities by working with other campus services as well as Cooperative Extension.

Submission tip

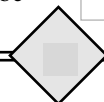
The onset of hot weather presents additional problems for veterinarians submitting unfixed tissues and carcasses for necropsy to the AzVDL. Inadequately cooled specimens rapidly decompose in transit and when received they are frequently less than satisfactory for diagnostic purposes. (1) More thorough cooling can be accomplished by bagging specimens and small cadavers in plastic and then packing them in ice immediately for thorough cool-down. Direct contact of the bagged specimen with ice is a more rapid means of cooling a specimen. (2) When the specimens are thoroughly cooled and ready for shipment, they can be transferred to the shipping container and packed with plenty of leak-proof frozen commercial "ice paks".

For those outside the Tucson area, a number of courier services are available for submissions to AzVDL. The University of Arizona provides a free courier service from Phoenix with daily pick-ups from the Maricopa County Extension Office, 4131 E. Broadway. Specimens must be

delivered to the Maricopa office before 10:30 AM. All specimens must be properly sealed and refrigerated in leak-proof insulated containers or the courier will not be able to transport them. Call us to inquire about the delivery of heavier specimens. We usually receive these submissions by 3:00 PM of the same day. Commercial couriers can be used state-wide. You may use Federal Express at our contract rates by charging the shipping to our account (check the "Bill Recipient's Account" box and write in the AzVDL account number: 1381-3559-4). All packages should be received at this facility Monday through Friday.

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Diagnostic Update

The following are selected samples of cases submitted to the AzVDL during the spring months of March, April, and May.



Bovine

Nitrate poisoning was diagnosed in two cases. In the first several dead cattle had been fed sudan hay a sample of which contained 1.8% d. w. nitrate (forage values of 1% or more are considered toxic to ruminants). Both serum and ocular fluid from one animal contained significant amounts of nitrate (>100 ppm). In the second case several range cows which were given access to a "new" water source delivered in a salvaged storage tank whose previous use was unknown. Aqueous humor from the dead cows contained significant amounts of nitrate (>100 ppm) and water from the storage tanks contained approximately 5000 ppm (0.5%) nitrate. It is reported that nitrate concentrations in water in excess of 1500-3000 ppm (0.15-0.3%) are capable of causing nitrate toxicity in cattle.



Equine

Severe, diffuse hepatocellular necrosis of unknown cause but suggestive of Theiler's disease (equine serum hepatitis) was found in liver tissues submitted from a gelding. The horse had been "not doing well", then became icteric, ataxic, and progressed to "stupor". A bladder paralysis was also noted. The animal subsequently became recumbent and unresponsive. Theiler's disease is reported to occur in horses injected with materials of horse tissue origin such as tetanus antitoxin. In this case, the horse had not received any antitoxin during the preceding six months but had received standard equine vaccines.



Porcine

Porcine sunburn is most often observed as a problem in 4-H pigs that may be owned by people without a lot of swine management experience. We see or, most often, hear about this when young pigs are moved from a shaded environment to one with access to direct sunlight or, unfortunately, where no shade is provided. The result may be sunburn which produces totally unexpected clinical signs. Pigs tend to walk normally and then suddenly dip their back until their abdomen nearly touches the ground. They may go down on their knees or sides and they sometimes squeal. They usually soon appear to recover until the next attack. This symptomatology suggests central nervous system (CNS) dysfunction or disease, so it can be quite misleading. These pigs do continue to eat and they recover in a day or two if no other complications occur. Sunburn also occurs on older animals when moved out of shaded environments and can produce quite a marked dermatitis. Prevention is simple. Simply use caution when moving white pigs from a shaded environment.



Small Ruminants

Two cross-bred lambs died from **copper poisoning**. Clinical signs included unsteadiness, labored respirations and dark red urine. Gross necropsy revealed icterus of the subcutaneous fat. The livers were pale yellow/orange and the kidneys were dark red. The urine was clear and dark red. Liver and kidney copper levels in both lambs were in the toxic range. The problem was traced to the consumption of a concentrate ration marketed for growing lambs but which contained an excessive amount of copper.

Ovine progressive pneumonia (Maedi) was diagnosed in a two-to-three-year-old range ewe that had been sick for several months. The animal had labored breathing and had been straggling behind the flock. At necropsy, the lungs did not collapse. When the thorax was opened the lungs had a rubbery consistency and were diffusely discolored a faint bluish-grey. Histology was typical of OPP. OPP (Maedi) is a chronic, progressive pneumonia of sheep caused by non-oncogenic retroviruses (Lentivirus). Sheep and goats are both susceptible.

Scrapie was diagnosed in a seven-year-old Suffolk ewe from the Tucson area. It had a six month history of weight loss and pruritus with loss of wool. The diagnosis was based on the typical microscopic lesions of neuronal vacuolation in the brain stem. It was confirmed by immunohistochemical staining at National Veterinary Services Laboratories.

A pure culture of *Streptococcus pneumoniae* was isolated from brain, lungs and liver of a seventeen-day-old goat which died with lesions of acute interstitial pneumonia and multifocal hepatic necrosis after a short febrile episode. *Str. Pneumoniae* infections are an infrequent but well documented cause of death in kids.



Avian

Avian tuberculosis was the cause of weight loss in a ten-year-old, male White-cap pionus. Gross necropsy findings included emaciation, dilated and flaccid intestines with watery green contents. Pinpoint white foci were scattered throughout the intestinal mucosa and liver.

Verminous gastritis due to spirurid nematodes was the cause of four, velvety mucosal masses in the proventriculus of a nineteen year old Sulphur crested cockatoo. Bleeding from the masses had resulted in anemia which was the original presenting complaint. Nematode species in the super-family Spiruroidea associated with this lesion include *Spiroptera incerta* and *Dispharynx nasuta*. The proliferative masses are the result of mucosal hyperplasia as a reaction to mucosally embedded parasites. Clinical signs reportedly include chronic vomiting and weight loss.

The biology of the parasite is not well understood but an insect intermediate host is suspected.

Salmonellosis, characterized by necrotizing hepatitis and bacteremia was the cause of death in a Molocan cockatoo. The presenting complaint prior to death was anorexia and diarrhea. Gross necropsy lesions included a swollen, green liver with scattered tan foci, a pale spleen, a slight excess of pericardial fluid and dark green/black, pasty intestinal contents. A group B *Salmonella* was isolated from the liver, small intestine and spleen.

Ascites and right ventricular failure syndrome was the cause of death in a five-week-old Cornish-cross chicken. This was one of seven birds that died out of a flock of 35. Clinical signs began as lethargy and anorexia followed by labored respiration and death. Grossly the bird had marked right ventricular hypertrophy and dilation. The abdomen was filled with gelatinous, clear yellow fluid. This syndrome is becoming increasingly important in broiler flocks. The cause is poorly understood but is hypothesized to involve pulmonary hypertension leading to right ventricular hypertrophy as a compensatory reaction. Eventually, right heart failure occurs. Rapid growth is suspected as the precipitating factor in broilers by increasing oxygen requirements which leads to pulmonary hypertension. Reducing growth rates can decrease the incidence of the disease in the flock. Other contributory factors appear to include cold temperatures, excess dietary sodium and pellet type diets.

Large basophilic intracytoplasmic inclusions characteristic of **Circovirus** were found within macrophages of the spleen, lungs and kidney of a pigeon. Circovirus is an immunosuppressive agent and pigeons dying of the disease usually exhibit coexistent infections. In this case, a herpesvirus esophagitis and candidiasis were also observed.

Extensive feather loss, anemia, weight loss and death was produced by **skin mites** in a finch.

Acute pancreatic necrosis is an uncommon, idiopathic disease of psittacines. Nonetheless, we had two unrelated diagnoses of this condition in this period, both involving two-year-old Quaker parakeets. In the first case a female was presented dead with a one day history of decreased activity and regurgitation of red/brown fluid. Necropsy examination revealed large volumes of turbid red/brown fluid in the coelomic cavity, air sacs, and lungs, and a pale pancreas. The second case was a male presented to the clinic after one day of being listless, sitting in the cage. It died a few hours later in spite of supportive treatment (tube feeding, antibiotics). Necropsy revealed a clearly demarcated area of yellow-green discoloration extending to most of the organ. Microscopically there was diffuse pancreatic necrosis with necrosis of adjacent fat in both birds.

Lead poisoning was diagnosed in a Prairie falcon owned by a falconer for the past two years. The bird had been fed feral pigeons collected from the downtown Tucson area. Clinical signs began with abnormal behavior and “hiding”, anorexia, diarrhea and weight loss. Three weeks later seizures developed. The lead level in whole blood was very high (11.4 ppm). As of this writing, the source of the lead is not known conclusively.



Canine

A six-year-old, male Chow was being treated for autoimmune hemolytic anemia but exhibited poor response to treatment. The dog developed gastric distention, severe neck pain and epistaxis. Necropsy lesions included a severe suppurative myocarditis, nephritis and orchitis with vascular thrombosis and fungal hyphae **phaeohyphomycosis**. *Bipolaris* sp. fungi were cultured from the kidney and heart. These are opportunistic infections which usually occur only in hosts with compromised immune function.

Nodular esophagitis due to *Spirocera lupi* infection was found as an incidental lesion during postmortem examination of a three-year-old, female Australian cattle dog. Two, 2 cm diameter nodules were present in the wall of the esophagus over the base of the heart. A nematode parasite protruded from an opening in the mucosal surface of one nodule. This parasite is found in the southeastern United States and has a dung beetle as an intermediate host. Canids acquire the infection by consuming a vertebrate transport host. Mesenchymal neoplasms develop in the wall of the esophageal granuloma in some dogs. Tumor types reported include fibrosarcoma and osteosarcoma. Hypertrophic pulmonary osteopathy is an occasional concomitant lesion. The travel history, if any, of this dog was not available.

A probable case of **congenital bullous emphysema** was diagnosed in a twelve-week-old, female Bassett Hound puppy that died after experiencing persistent respiratory disease that began at five weeks of age. Clinical signs included severe dyspnea, gagging and forced exhalation. The submitting veterinarian reported radiographic findings of increased “linear patterns” in lung fields and a short, incomplete sternum that lacked sternbrae. In a previous litter, several other puppies had similar clinical signs. At necropsy protruding emphysematous blebs were evident on the pleural surfaces of both lungs. Alveolar emphysema, edema and diffuse hypercellularity of interalveolar septa were evident microscopically.

Hemopericardium from a ruptured atrial **hemangiosarcoma** was the cause of death of an adult female Golden Retriever which died suddenly after a history of shortness of breath for a couple of months. Numerous metastases were present throughout the lungs; additional metastases

were also observed in brain, spleen, intestine and pancreas.

Stomach contents from a three-year-old Rottweiler/Shepherd mix canine were received for strychnine testing. It was noted in the history that the "owner was awakened at 3:30 am by the sound of the animal hitting the glass door. The animal was bucking and slamming himself into a brick wall repeatedly until death occurred at 5:30 am". The stomach contents were **positive for strychnine (by TLC)**.

Tissue from a seven-year-old spayed female Shepherd mix canine were received for histopathologic examination. The animal had been euthanized following an illness of two weeks duration during which there was anorexia and a fifteen pound weight loss. Clinical signs included progressive deterioration with vomiting, bloody diarrhea, and muscle tremors. Renal failure was diagnosed. Microscopic examination revealed **severe diffuse renal amyloidosis**. It is reported that amyloidosis is most common in older dogs and is usually idiopathic, although some cases appear to develop in association with chronic suppurative and granulomatous lesions in other tissues. Renal failure with proteinuria develops in affected dogs.



Feline

Group G *Streptococcus* sp. was isolated from three aborted, six weeks gestation feline fetuses.

A four-year-old, domestic mediumhair cat was euthanized following a seven day clinical history of deteriorating neurologic signs. The onset was characterized by proprioceptive deficits in the right foreleg. The condition responded to corticosteroids within 24 hours. Two weeks later, the cat was again presented and had tetraparesis. It was euthanized. At necropsy, a large portion of the medulla oblongata was replaced by a 0.25 cm diameter, gray tan mass which extended from the obex caudally to the right dorsal portion of the brainstem at the foramen magnum. Similar masses of varying sizes were present in the liver and spleen. ***Coccidioides immitis*** was cultured from the lesions.

Pasteurella multocida infection was the cause of acute interstitial pneumonia and suppurative myocarditis in a one-year-old, male Persian cat. The cat was presented three days after elective surgery. It had respiratory signs and died later that night. The surgical sites were grossly and histologically unremarkable. The organisms were cultured from brain, kidney, liver, lung, spleen, heart, lymph nodes and pericardial sac. A hematogenous route of dissemination is apparent but the site of entry was not determined.

Chronic suppurative cholangiohepatitis was the cause

of death in a six-year-old, female Persian feline that died after a brief illness that followed delivery of kittens. At necropsy, the liver was pale, multinodular and firm. ***E. coli*** was cultured from the liver. Bacterial colonies and marked suppurative infiltrates were present in bile ducts and liver along with bile duct proliferation and portal fibrosis. In retrospect, the owner noted that the cat had not had a good appetite for a long time.

Systemic amyloidosis and feline infectious peritonitis were both factors in the death of a young Abyssinian cat. Gross necropsy lesions included emaciation, icterus, pinpoint pyogranulomas on all serosal surfaces in the abdomen and thorax and lymphadenopathy. Deposits of amyloid were visible microscopically in the spaces of Disse' in the liver, the walls of splenic arteries, the adrenal cortex and glomerular tufts.



Wildlife

Trichomoniasis was the cause of death in a two-and-one-half year-old, female Harris Hawk. The bird was presented to the referring veterinarian for weakness and inability to fly. It was emaciated, pale and had cardiac dysrhythmia. Gross lesions included serous atrophy of fat and a 3 cm diameter pale yellow/tan focus in the mucosa and wall of the distal esophagus. ***Trichomonas* sp.** were cultured from the lesion.

Two comatose coyotes were found by a rancher. Previously one horse had died on the ranch and another horse had been euthanized with unspecified neurologic disorder. The rancher suspected that the coyotes were transmitting a disease to the horses. Blood was drawn from the coyote before euthanasia and necropsy. Necropsy revealed the stomach was full of flesh and horsehide. Gas chromatography demonstrated pentobarbital in the blood of the coyote. There were no lesions in the coyote. The diagnosis was **pentobarbital poisoning** caused by ingested of tissue from the euthanized horse.

Chitridiomycosis was diagnosed in native Arizona Leopard frogs (***Rana yavapiensis*** and ***Rana chiricahuensis***). Recently, there has been high mortality in adult frogs of these species. The lesions noted included cutaneous erythema and edema, especially of the ventrum. Microscopically, there was chronic, hyperplastic dermatitis with hyperkeratosis and intracorneal fungal organisms. Consultation with the Armed Forces Institute of Pathology and the Department of Pathology of the National Zoological Park resulted in identification of the organism as a species of chitrid fungi. Until the last few years, these organisms had not been recognized as an agent of disease in vertebrate animals. They normally live on decaying vegetation. The factors (environmental, stress, toxic) that lead to infection with the organism have not been identified but the infection is being recognized with increasing frequency in many parts of the US and Australia.

Strychnine poisoning was diagnosed in an adult female Great-horned owl. The bird was found lying on its back underneath a tree where it usually perched during the day. Necropsy revealed a ball of fur in the lumens of the proventriculus/ventriculus. Examination of the ball of fur revealed that it contained a few milo-like kernels of grain. There were no other lesions. Thin-layer chromatography testing of the fur ball and grain kernels was positive for strychnine. Presumably the owl ingested a rodent which had been poisoned by one of the commercially available strychnine-containing baits.

Strychnine poisoning was diagnosed in a female Red-tailed hawk found near a golf course in the Tucson area. As with the previous case, the lumens of the proventriculus/ventriculus contained a fur ball that contained a few kernels of milo-type grain along with the bones of a small mammal. Thin-layer chromatography testing of the fur ball material was negative for strychnine but further testing using a more sensitive technique (GC/NP) was positive for strychnine. Again, secondary poisoning from ingestion of a rodent poisoned with a commercially-available bait was assumed.

Numerous encysted **spirurid nematodes** were found in the proventriculus of an immature Black Crowned heron found weak and emaciated in Central Arizona. Parasite debris were also observed within distended bile ducts surrounded by a granulomatous reaction.



Exotics

Chronic necrotizing bronchopneumonia and fibrinonecrotic stomatitis and tracheitis

caused by **herpesvirus** infection was the cause of death in an adult, male Desert Tortoise. The tortoise emerged from hibernation with nasal discharge, ptialism and conjunctivitis which did not respond to antibiotics.

Coccidioidomycosis was confirmed in an eleven-year-old Mandrill (primate) The animal had been treated for a periorbital infection involving the right eye. *Coccidioides immitis* had been identified. At necropsy, a granulomatous nodule containing *C. immitis* was found in the right lung. This was assumed to be the original route of entry. There was also extension to the base of the brain where granulomatous inflammation and *C. immitis* were identified. The

infection presumably accessed the brain via the optic nerve.

Necropsy of a twelve-year-old zoo ostrich was conducted after the bird died despite treatment for a vague illness of four weeks duration. Lameness referable to the left leg had also been noted in the history. Lesions included severe diffuse hepatic **lipidosis** and a **myopathy** of the flexor and extensor musculature of the left tibial area. **Liver failure** was the likely cause of death. The cause of the myopathy was not apparent however trauma was considered as an etiologic possibility.

A purulent pneumonia and septicemia was the cause of death of a captive Pallid bat. The causative organism was an oxidase positive, indole negative non-fermenter gram negative coccobacillus identified as a **Moraxella sp.** The same organism has been repeatedly isolated before from localized granulomatous lesions in joints and lungs of other bats in the same colony. We have found no descriptions of this organism in the reference materials available to us.

Dystrophic calcification of the myocardium and kidneys and **visceral gout** were the cause of death of a Panther chameleon. Excess vitamin D in the diet was believed to be the cause of the condition.

An eight-year-old Lazuli Bunting died after a short episode of weakness and inability to fly. Death was precipitated by a severe intraabdominal hemorrhage originated in a large, very anaplastic **Renal Carcinoma**.

Pseudomonas aeruginosa was the cause of a necrotizing dermatitis, cellulitis and septicemia in an adult female rabbit. Selenium and/or copper deficiency could have made this animal more susceptible to infections since hepatic levels of both minerals were very low.

A trichobezoar was the cause of a duodenal obstruction and death of an adult rabbit.

compiled by; Greg Bradley, Robert Glock, T. H. Noon, Carlos Reggiardo

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

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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