



Newsletter

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From the Director

We are very pleased that **Dr. Sharon Dial** has joined the Arizona Veterinary Diagnostic Laboratory as a pathologist. She began June 1st and brought with her a strong background of knowledge with regard to morphologic pathology and clinical pathology. Dr. Dial received her DVM PhD at Colorado State University and is a diplomate in the American College of Veterinary Pathologists with a specialty in clinical pathology. She will be doing general pathology and diagnostic work as well as filling a void with regard to our understanding of the subtleties of interpretation of clinical pathology. Dr. Dial is well known to many in this area as a dedicated and communicative person who is enthusiastic about helping practitioners seek solutions to difficult problems. Dr. Dial will also be spending some of her time developing a research program and sharing her broad knowledge with some of our students as a teacher. She has teaching experience at Colorado State University, The University of Wisconsin, and Louisiana State University. She most recently has been active in diagnostic work in association with commercial laboratories in Arizona. We know she will help us increase our ability to better serve our clients.

Animal Cruelty

The AzVDL has had a recent upturn in requests for necropsies associated with suspected animal cruelty. This stems from recent meetings in this area that have brought many people's attention to the fact that people who abuse animals frequently also abuse humans. In addition, certain types of animal cruelty are now punishable as felonies and law enforcement agencies are taking a much more serious look at animal cruelty. An **Animal Cruelty Task Force** has been formed. Leadership is being provided by Mr. Marsh Myers at the Humane Society of Southern Arizona. That organization can be contacted at 321-3704. Local practitioners who have been active in the task force include Dr. Michael Lent and Dr. David Edsall. They are helping to coordinate the task force objectives with clarification of a veterinarian's role in identifying and reporting abuse cases in animals and humans. Questions involving abuse can be directed to Pima Animal Control Center, where personnel are very active in investigating suspected animal cruelty.

Polymerase Chain Reaction Testing (PCR)

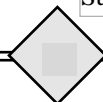
The current hot topic in diagnostics is **polymerase chain reaction (PCR) testing**. This highly sensitive and highly specific method of determining the presence of specific genetic material is finding widespread use as a rapid and efficient diagnostic technique. We are very fortunate to have a new department head, Dr. Jim Collins, who has expertise in this area. He was active in developing numerous PCR tests while at Colorado State University. A great deal of that technology has been transferred to The University of Arizona and the diagnostic laboratory is now accepting submissions for canine distemper, parvovirus, herpesvirus, and hepatitis virus. Feline agents that we now test for include herpesvirus, chlamydia, mycoplasma, and panleukopenia. Tests for equine herpesvirus types 1 and 4, chlamydia, malignant catarrhal fever, and ovine progressive pneumonia virus are also available. A recent mailing contained details of submissions, tests available, and expectations. If you have an interest in PCR testing and did not receive one of these mailings, please

contact us. Additional tests are in development and we are anxious to receive input from anyone who has ideas as to what tests may be valuable and useful. We are especially interested in input regarding the needs of the livestock industry.

Robert D. Glock, Director

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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



Hemophilus somnus was isolated from acute fibrinopurulent bronchopneumonia in an eight-week-old Limousin calf. The interesting feature of this case was the sudden onset and rapid death of the affected calf, which was the second to die in the herd in the past few days. The calf seemed normal and was seen nursing the day before it died. *Hemophilus somnus* is a common pathogen in cattle and frequently is associated with acute bouts of pneumonia, CNS disease, and arthritis.

Two adult Holstein cows died quite suddenly with no recorded evidence of clinical observation of disease or treatment. Inflammatory changes were observed in liver, lung, and intestine. **Salmonellosis** was the diagnosis and a group C-2 *Salmonella* was identified in cultures.

Polioencephalomalacia was diagnosed by microscopic examination of brain tissue from crossbred cattle that had a history of wasting, blindness, and recumbency while grazing on harvested broccoli pasture. There was no information available on the sulfur content of the forage or water but excess dietary sulfur intake would be an important consideration when “polio” cannot be associated with ingestion of thiaminase containing plants.

Mid-term abortions due to *Neospora caninum* infection of the fetus were diagnosed on three different dairies. The history from one of the farms included the presence of many dogs on the farm. Dogs have recently been identified as the definitive host of the parasite and fecal contamination of foodstuffs as the source of infection for dairy cattle.

Lesions compatible with **ruminal tympany (bloat)** were found at necropsy in the carcass of a Hereford cross steer that was presented to the AzVDL for necropsy in March. The animal had been fed a total mixed ration that included long hay and was simply found dead in the morning. Necropsy findings included bloating in the carcass, a considerable amount of ingesta in the rumen, a pale liver, congestion of the lungs, and diffuse congestion of the tracheal mucosa. There was extensive hemorrhage in the submucosa of the trachea. A distinct bloat line was present in the esophageal mucosa at the level of the thoracic inlet. The anterior mucosa of the esophagus was discolored dark red, and the interior thoracic portion of the esophageal mucosa was pale. The nasal turbinates were intensely congested and oozed blood when incised. No other gross lesions were found.

A severe, necrotizing **metritis**, with an associated retained placenta and mixed bacterial infection that included *Clostridium chauvoei* was diagnosed at necropsy in a mature cow which had been pastured on permanent fescue pasture that included some Bermuda on a ranch southwest of Tucson. The cows had been supplemented to some extent with “good alfalfa hay.” Two other cows in the herd had also died. Clinical signs reported by the owner included: “weak, can’t get up, go down, eyes roll back, staggered, go down.” At necropsy, it was noted that the cow was lactating but there was no evidence of mastitis. Internally, the uterus was only partially involuted and contained most of the placenta, which was loosely attached to the uterine caruncles. A small amount of dark red, turbid, slightly malodorous fluid was present in the uterine lumen. Additional lesions included numerous locally extensive hemorrhagic foci on the parietal pleura and locally extensive submucosal hemorrhage in the trachea. The lungs were discolored dark red and appeared to contain numerous foci of hemorrhage. Several foci of epicardial and endocardial hemorrhage were also evident. Gram stained sections of uterus revealed superficial necrosis and extensive colonization of the necrotic areas by mixed bacterial flora, which included spore-forming organisms. Fluorescent antibody staining of sections of the uterine caruncle was positive for *Clostridium chauvoei*. Nitrate and cyanide poisoning and magnesium deficiency were ruled out on the basis of toxicologic testing. Rabies testing was also reported as negative. Of interest in this case, is the fact that the animal was severely copper deficient (liver copper 1.2 ppm, the reported “adequate” range for bovine liver copper is 25 - 100 ppm) and had a marginal selenium status (liver selenium 0.13 ppm, the reported “adequate” range for liver selenium in the bovine is 0.25 - 0.50 ppm). It is reported that retained placenta and reduced humoral and phagocytic immune responses may be associated with copper and selenium deficiencies in cattle (per *Mineral Levels in Animal Health*, by R. Puls).

Equine



Clostridium perfringens type A was the cause of an acute necrotizing enterocolitis in a four-day-old Quarterhorse. The foal had a two-day history of diarrhea and colic. Gross lesions included dark red small and large intestines with tan pseudomembranes on the mucosal surface. The contents were red, watery fluid.

We received an aborted equine fetus with a crown rump measurement of forty-eight centimeters. The diagnosis was abortion due to **equine herpesvirus**. There were typical necrotic lesions in the liver with basophilic intranuclear inclusions. There was also inflammatory change in the heart, lung, lymph nodes, and kidney. The Appaloosa mare had a history of having been vaccinated against equine herpesvirus but the specific times of vaccination were not known.

A pure culture of *Rhodococcus equi* was isolated from a tracheal wash of a six-week-old Arab foal with a two-week history of bronchopneumonia. *Rhodococcus equi* infections seem to be less common in southern Arizona than in other parts of the country, which could be related to climate and soil composition. The present case is from a farm where horses have been raised for many years in relatively large concentration.

Porcine



Tissues were received from three five-month-old pigs with a history of bloody diarrhea and weight loss. Gross and microscopic lesions were typical of porcine **proliferative enteritis**. *Lawsonia intracellularis* was identified by PCR. Proliferative enteropathies are common in swine and hamsters. The causative agent appears to be identical or very similar. Other animals such as lambs, foals, and calves have been identified with this disease but it is seen only sporadically in these species.

Avian



A five-year-old, male Rose-breasted cockatoo had a four-month history of progressive neurologic deficits and poor body condition. Necropsy lesions included marked muscle wasting and atrophy of fat stores. The brain had bilateral atrophy of the cortex of the cerebrum and cerebellum. Microscopically there was malacia with glial and neuronal necrosis and gliosis in the cerebral cortex, midbrain, cerebellum, and medulla. Karyomegaly with pale intranuclear inclusions typical of **polyomavirus** was seen in glial and ependymal cells. The infection was confirmed by PCR testing of liver and lung tissue. Clinical polyomavirus infection in adult birds is rare. There are a few descriptions of similar central nervous signs and lesions in adult birds in the literature and we have seen two such cases at the AzVDL in the last ten years.

Hemorrhage into the clavicular and thoracic air sacs was the cause of death of a juvenile Barhead goose with a traumatic injury to the humeral region of the left wing. Bleeding into the connecting pneumatic cavity of the humerus was the source of the blood found within the air sacs.

An aviary had mortalities in several canaries. Clinical signs included respiratory distress and “undigested sands” in the feces. A few birds that became ill eventually recovered. Necropsy examination of a three-month-old canary revealed non-suppurative myocarditis with intranuclear inclusion bodies and the presence of similar intranuclear inclusion bodies in neurons and adrenal gland cells. The lesions were considered consistent with **avian paramyxovirus type 3** but

attempts at virus isolation were unsuccessful. PMV3 infection in canaries and finches typically causes clinical signs of conjunctivitis, anorexia, yellowish diarrhea, and dyspnea. Some birds die within a few days. Clinical signs in psittacines usually involve the central nervous system and include torticollis and circling.

A two-and-one-half-year-old chicken was euthanized due to declining condition and ascites. At necropsy, the coelomic cavity was filled with clear amber fluid. Firm white nodules measuring two to five millimeters in diameter were scattered on the surface of the stomach, intestine, and oviduct. The ovary was replaced by a two to three centimeter diameter mass of similar tissue. The lesions were histologically compatible with an **ovarian carcinoma**. Ovarian carcinoma is the second most common tumor of the ovary in chickens, the first being lymphoma associated with the herpesvirus of Marek’s disease.

Gastric perforation secondary to a **gastric carcinoma** of the proventriculus was the cause of death in an eleven-year-old Gray Cheek parrot with a history of acute weakness and anorexia.

Synovial cell sarcoma of the wing was diagnosed in a twenty-five-year-old Moluccan cockatoo that had a large mass encompassing the entire humerus.

Hepatic lipodosis was the cause of death of an obese male African Gray parrot. The fatty changes were severe and diffuse affecting virtually every hepatocyte. This condition is usually dietetic.

Aspiration pneumonia was diagnosed in an eleven-year-old male Macaw, which showed signs of respiratory embarrassment shortly after feeding the female. Males feeding females is part of the pair bonding process in some psittacines but it can result in aspiration of food.

Neuropathic gastric dilatation was the cause of death of a seven-year-old African Gray parrot. Lymphocytic infiltration was observed in ganglion cells of the proventriculus, ventriculus, duodenum, and adrenals. Gliosis and lymphocytic cuffing of blood vessels was observed in the brain.

Feline



A twelve-week-old kitten had been adopted two weeks previously from a rescue group. It died “suddenly.” There were no gross lesions but there was significant focal hepatocellular degeneration and inflammation in the liver. The diagnosis was **salmonellosis** and a group B *Salmonella* sp. was isolated.

The **endomyocardial form of feline cardiomyopathy** was diagnosed in a five-year-old, female, domestic shorthair with a history of lethargy and rapidly developing pulmonary edema and pleural effusion. Radiographically, there was a “valentine-shaped” heart. Necropsy revealed “bruising” of the myocardium. Examination of formalin-fixed tissue collected by the referring veterinarian revealed characteristic endomyocardial inflammatory infiltrates with acute myocardial necrosis and hemorrhage and thickening of the endocardium by proliferation of fibroblasts. This form of feline cardiomyopathy occurs most often in young cats (mean age of 3.4 years) and there is often a history of stress in the recent past.

Respiratory disease by **feline herpesvirus** was the cause of respiratory distress and death in two unrelated cases in adopted stray kittens. The infection was confirmed in both cases by virus isolation and by our newly developed PCR test, which is specific and highly sensitive.

Canine



Heat stroke was the cause of death in a two-and-one-half-year-old, female Labrador mix. The dog was presented with acute onset of vomiting, diarrhea, and collapse that followed a short jog to the park followed by five minutes of retrieving a ball. On presentation, the dog was in shock and had gaseous distention of intestinal loops and increased borborygmi. Fluid therapy was begun but the dog developed bloody diarrhea, vomited blood and had cold extremities. There was progressive depression and the dog became agonal within 24 hours of presentation. It was euthanized. Necropsy lesions included multifocal hemorrhage including free blood in the abdominal cavity and multifocal necrosis in visceral organs, especially myocardial necrosis in the heart, diffuse centrolobular to mid zonal necrosis of the liver and crypt necrosis in the small intestine. Necrosis in multiple organs (variably including liver, brain, heart, skeletal muscle and kidney) and hemorrhage are fairly characteristic lesions of heat stroke in dogs that live long enough (at least 6-12 hours) for the morphologic lesions to develop. The necropsy diagnosis is much more difficult in dogs that die rapidly from overheating (such as being locked in a car on a hot day) since there is insufficient time for cellular damage to become recognizable as morphologic lesions. In addition, post-mortem decomposition occurs at an accelerated rate. Adequate history and exclusion of other causes may allow a presumptive diagnosis in those cases.

A practitioner diagnosed interstitial pneumonia in a thirteen-year-old Poodle based on dyspnea and radiographs. The dog then died suddenly and the lungs were removed for histopathology. Histopathology supported the diagnosis of diffuse interstitial pneumonia. There were numerous macrophages that contained refractile particles. The diagnosis was **silicosis**.

Chronic extensive **glomerulo-interstitial nephritis and nephrosis** was diagnosed in a seven-year-old spayed female Yorkshire terrier. The kidneys were grossly small, pale, and very firm. A specific cause was not determined but this type of lesion is often the result of immune mediated disease.

Canine distemper was diagnosed in a five-month-old female mixed breed canine weighing thirty-seven pounds, which was described by the submitting veterinarian as “semicomatose” and having pneumonia and “chewing gum seizures.” Necropsy revealed that the apical, cardiac, and diaphragmatic lobes of the left lung were consolidated and sank in fixative. No other gross lesions were noted. Microscopic changes in the affected areas of lung included a severe, acute bronchopneumonia. Additional findings included foci of gliosis and neuronal necrosis in brain. Pale, eosinophilic inclusions compatible with those of canine distemper were encountered in some of the glial cells, inflamed areas of the brain, and in transitional epithelium of the urinary bladder. The cause of death in this case was pneumonia secondary to canine distemper virus infection.

Drowning was suspected as a cause of death in a six-year old female Wheaton terrier that had no prior history of illness. The dog was found dead in the owner’s swimming pool. At post mortem, there was a moderate amount of free blood in the thoracic cavity, which prompted testing of liver tissue for anticoagulant rodenticides. This was reported as negative and there were no other significant lesions referable to active disease. The hemorrhage noted in the chest cavity might have occurred agonally. A diagnosis of drowning is difficult and often must be made by exclusion of other causes of death.

Pulmonary congestion and edema were the morphologic diagnosis in a ten-month-old spayed female Blue Heeler canine, which had been euthanized after an illness of two weeks duration. Clinical signs included decreasing weight and anorexia. The dog was noted to have a severe cardiac arrhythmia. An EKG interpretation rendered by a consulting cardiologist reported a significant conduction abnormality, which was interpreted as AV block or AV dissociation. There were no gross or microscopic lesions evident in the heart that would account for the functional disorder. The cardiac arrhythmia was considered to be idiopathic.

Renal dysplasia was diagnosed in a three-month-old female mixed breed canine that was found with its littermates in the desert. The animal was described as a “poor doer” and had a depressed appetite. Renal parameters including BUN, creatinine, and phosphorus were elevated (372, 14.6, and 26.4 respectively). At necropsy the kidneys were smaller than normal, pale, and lobulated. Microscopically, the changes were typical of renal dysplasia. An incidental finding was mild, chronic, focal encephalitis of undetermined etiology. Although not diagnosed in this case, canine distemper may have been a cause for the encephalitis, as there were no

known prior vaccinations.

Severe diffuse dilated **cardiomyopathy** was diagnosed as the cause of death in an eleven-year-old neutered male Dalmatian weighing approximately eighty-five pounds.

Renal failure produced by **amyloidosis** was the cause of death of a female Shar Pei with a history of chronic renal disease and elevated proteinuria. Amyloid deposition was almost exclusively glomerular and affected virtually all glomeruli. This is a component of Shar Pei fever syndrome.

Torsion and rupture of the spleen was the cause of death of a four-year-old male Irish wolfhound, which collapsed after vomiting several times, and then developed a bloody diarrhea. Lesions of a chronic cholangiohepatitis were observed in the liver, suggesting the possibility of a primary digestive upset with vomiting, leading to the splenic torsion.

Gastric dilatation was the cause of death of a fourteen-year-old Labrador mix. An **aortic-iliac thrombosis**, similar to those observed in horses and cats, was found on necropsy. It had produced several chronic renal infarcts, and it was possibly related to the gastric dilatation since blood perfusion was abnormal in much of the abdominal viscera. These thromboses are very rare in dogs. Its cause was not apparent in this case.

Exotics



Tularemia was the cause of death of five tamarins; two Cotton tops, one Red Handed, one Golden Headed and one Golden Lion, from a zoo. The animals died after acute onset of fever and lethargy. Necropsy lesions included scattered pale foci in the liver and caseous yellow foci in the spleen, mesenteric lymph nodes, tonsils, and Peyer's patches of the small intestine. Necrosuppurative hepatitis, splenitis, lymphadenitis, enteritis, and tonsillitis were present in histologic sections of these

organs. *Francisella tularensis* was isolated from the lesions. Wild rodents and rabbits are reservoirs of infection. Transmission may occur by direct contact, ingestion or via the bite of ticks and other insects. The source of the infection in this group of primates is currently under investigation.

An adult Star tortoise had a history of rapid decline and weight loss. Multiple one millimeter white nodules scattered through the liver were the result of secondary infection with *Salmonella stanley*. There was a large amount of clear fluid in the body cavity. This is a common finding in debilitated tortoises.

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

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

Submission Tip

Good carcass preservation in hot weather is possible. We at the AzVDL routinely recommend bagging smaller carcasses in plastic (to prevent water logging) and promptly packing them in ice to cool them down rapidly. Carcasses can usually be held for several days if the ice is periodically replenished. Smaller carcasses can be held in an old picnic cooler, which can also be used for their transport to the AzVDL. The ice should be replaced by ample leak-proof "cool packs" when a specimen is shipped in an insulated container by common carrier. The "icing down" method of preliminary cooling is preferable to refrigeration in a conventional household refrigerator, which is usually neither cold enough nor cools rapidly enough to prevent decomposition. Large carcasses can be similarly cooled by placing them in a large poly-tarp in the bed of a trailer or pick-up and packing an ample supply of bagged convenience store ice around them, followed by covering with the loose flaps of the tarp, and ideally, an old blanket for insulation. *T. H. Noon*

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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