



Newsletter

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

From the Director

The Arizona Veterinary Diagnostic Laboratory is part of the Veterinary Science and Microbiology Department at the University of Arizona. Therefore, the leadership of that department is very important to us. We feel that is also of vital interest to the people of the state of Arizona and particularly the veterinary community. Dr. Charles Sterling has announced that he will be resigning as department head on July 1, 1999. Dean Sander has formed a committee to search for a suitable replacement. Dr. Gerry Ault of Tucson has agreed to serve on that committee at the request of Dean Sander. We thank him for his willingness to take the time to participate. We also are pleased that he will be helping to represent the interests of veterinarians and animal owners in the state of Arizona. He will be in an excellent position to help transmit thoughts and ideas from any of you who may wish to communicate with him. Input from those outside the University is helpful in our goal of service to the people of the state of Arizona.

The AzVDL will be closed for the holidays on Thursday, December 24th and Friday, the 25th and on Friday, New Year's Day, January 1.

Robert D. Glock, director

Field investigation update

There are occasional situations where it seems beneficial to have diagnostic laboratory personnel visit premises where there are significant problems. This service is most appropriate when there are problems that may have fairly broad implications in the animal community. Our objectives are to assist the practicing veterinarian in identifying the source and potentially the solution of significant problems. We often coordinate these efforts from the diagnostic laboratory but it is sometimes beneficial to have a diagnostician actually on the scene. We have rather limited numbers of personnel available so we cannot always respond immediately but we are most certainly willing to try to provide this type of support. Dr. Bicknell as extension veterinarian has filled this role frequently in the past. There are others in the diagnostic laboratory that have interests and specialties that lend themselves to field investigation. The cost to the user is generally about the same as if animals are submitted to the laboratory plus the cost of transportation. These types of investigations frequently result in a learning experience for all involved and may help us to better serve others with similar problems. If you are interested in this type of service, please give us a call at the diagnostic laboratory.

Instructions to fill the Federal Express Airbill are as follows:

- In **payment** box (area #3), check #2
- In "Bill Recipient's account" write in the AzVDL account number: 1381-3559-4

If our account number is not used you will be charged the regular Fed X fees (approximately triple our rate).

Diagnostic Update

The following are selected samples of cases submitted to the AzVDL during the fall months of September, October and November.



Bovine

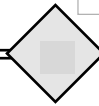
Silverleaf nightshade ingestion was the apparent cause of death in several adult cows. Several others were sick or were ill with neurologic signs, tremors, and general debilitation. No specific lesions were found in these animals but

Federal Express Submissions

For your convenience you may ship specimens to us via Fed X. We will then add the shipping charges to our service fees and bill them to your account. You can experience significant savings on Federal Express shipping by charging our account number.

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Silverleaf nightshade was identified in the surroundings as well as in rumen contents. Quantities as small as 0.1 % nightshade in rations can cause symptoms similar to those observed in this case.

Dallis grass intoxication was diagnosed in a herd of yearling cross-bred cattle that were turned into an irrigated mixed-grass pasture in southern Arizona that contained substantial amounts of mature Dallis grass (*Paspalum dilatatum*). The syndrome occurs when the ovary of maturing grass is parasitized by the fungus *Claviceps paspali* which forms a sclerotia or ergot in place of a seed. The sclerotia contains a tremorgen which causes the intoxication. Clinical signs include variable but often severe muscle tremors, recumbency, and repetitive seizures. Aspiration of rumen contents into the lungs occurs in severe cases as was the case in two of the animals that were necropsied. There are no diagnostic lesions or tests. Diagnosis is based on the grazing history and the finding of ergotized plant material in the rumen. Treatment includes keeping lesser-affected animals quiet in the shade and manually feeding and watering them until they recover, which can require as much as three weeks. While probably uncommon in irrigated pastures in Arizona, Dallis grass is an otherwise palatable, useful grass that must be managed carefully to avoid intoxication of grazing animals. We are told that the syndrome is common in cattle in wet years in east Texas. The toxicology section at the TVMDL recommends ripping the seed heads off with a mechanized shredder, after which it can be utilized. Note: the following Arizona range grasses have been reported to become ergotized: wheatgrasses (*Agropyron*), redtop (*Agrostis*), bromes (*Bromus*), bluegrass (*Poa*), tobosa (*Hilaria mutica*), and galleta (*Hilaria jamesii*) ref. Livestock-Poisoning Plants of Arizona.



Equine

Burroweed (*Isocoma tenuisecta*) poisoning was diagnosed in an eight-year-old Arabian mare. The mare had a five day history of trembling, ataxia and lingual paralysis which began after being placed in a pasture containing burroweed. The owner reported that the horse consumed larger amounts of the plant, primarily the flowers, than any of her other horses. Serum chemistries revealed elevated AST (12,280 U/L) and CK (40,740 U/L) levels. The mare deteriorated and eventually died. Necropsy examination revealed excess, clear pericardial fluid and marked, multifocal streaking of the myocardium by pale foci of necrosis. The lungs were edematous and foamy white fluid filled the trachea and nares. Microscopic lesions included severe necrosis of myocardial muscle and less severe necrosis of skeletal muscle of the tongue and axial and appendicular musculature. Two possible causes of muscle necrosis are oleander and ionophores. These were ruled out. The lesions of fatal

burroweed poisoning of horses have not been reported. However white snakeroot poisoning, which occurs in the eastern half of the US, has been described in horses (JAVMA 185: 1001-1003) and the two plants share in common the same toxic principle, tremetol. The lesions of white snakeroot poisoning are identical to those seen in this horse.

Chronic hepatotoxicity was the cause of death of several horses in two stables in Pima county. All horses died after exhibiting signs of hepatic failure for a period of approximately one week. The animals were febrile, icteric, dehydrated and some had photosensitization. Elevated liver enzymes and thrombocytopenia were the most significant hematological findings. Necropsy lesions included icterus, visceral and muscular hemorrhages, small, yellow-green livers, and enlarged kidneys. The basic microscopic lesions were hepatic necrosis with fibrosis and diffuse nuclear (karyomegaly) and cytoplasmic gigantism (megalocytosis). These changes are characteristic of intoxication by **Pyrrrolizidine alkaloids**. Most pyrrrolizidine alkaloid containing plants occur in three families: Boraginaceae, Compositae and Leguminosidae. Threadleaf groundsel (*Senecio longilobus*) and Fiddleneck (*Amsinckia intermedia*) are common examples in southern Arizona. No suspect plants were found on the premises where the horses were kept, but parts of an unspicated *Senecio* sp. were found in hay believed to have been fed to the affected horses.



Small Ruminants

Twenty feeder lambs died in a flock of 600. The animals had been sick for approximately one week after they had been trucked from New Mexico and turned into an alfalfa pasture. Clinical signs included separation from the flock, "lotsa time at water trough", "lowered heads", nasal discharge, and mucoid diarrhea. Necropsy by the submitting veterinarian revealed multiple abomasal erosions. **Mycotic rumenitis** was diagnosed when microscopic examination revealed the ruminal lesions to be transmural ulcers that were heavily colonized with fungal organisms along with mixed bacterial organisms. Invasion of nearby blood vessels by fungal organisms was also seen. A previous episode of ruminal acidosis occurring prior to arrival in Arizona was suspected as a cause. Over-consumption of high concentrate-type (grain) rations in unaccustomed animals is a common cause of ruminal acidosis.

Rumen acidosis was diagnosed as the cause of death in a four-year-old Pigmy goat. This was one of two dead goats from a herd of 30 animals. Another goat was sick. Clinical signs included acute onset of pasty, yellow diarrhea, recumbency, panting and teeth grinding followed by death within 24 hours. Histologic lesions included multifocal rumenitis with necrosis of rumen epithelium.



Avian

Avian **tuberculosis** was diagnosed in a quail which at necropsy had numerous, variably-sized granulomatous foci scattered throughout the liver. Acid-fast staining of tissue sections revealed the causative mycobacteria which were intracytoplasmic in swollen macrophages within the lesions. Avian mycobacteria can be zoonotic.

Hemochromatosis resulting in hepatocellular necrosis and iron pigment accumulation in hepatocytes and myocardium was diagnosed in a male, Red-billed toucan. The liver contained 7700 ppm iron.

Psittacosis was diagnosed in a Amazon parrot. At necropsy, there was diffuse, fibrinous airsacculitis, a swollen liver with scattered pale foci of necrosis, splenomegaly and watery green intestinal contents. *Chlamydia psittaci* was isolated in tissue culture from the liver, lung and spleen.

Airway obstruction due to *Aspergillus fumigatus* infection of the syrinx was the cause of acute dyspnea and death in a ten-month-old, Green wing macaw.

Severe, bilateral **hydrocephalus** of the lateral ventricles with cerebrocortical atrophy was found at necropsy of a six-month-old, female, Rainbow lory. The bird had a four month history of debilitation and incoordination with poor color and poor feather growth.

A 24-year-old, Blue and Gold macaw died following acute onset of dyspnea. The owner reported that the room housing the bird had been power washed just a few days prior to onset. The bird had remained in the room during the procedure. The owner noted that the fine mist from the power washer was so thick that she could not see into the room. At necropsy, there was pulmonary edema and the lungs contained myriads of tiny, firm white foci. These translated microscopically into granulomas containing phagocytized, birefringent foreign material compatible with dust or silica (paint?). The cause of death was granulomatous bronchopneumonia and pulmonary edema due to **inhalation of aerosolized foreign material**.



Canine

A two-year-old, Brussels griffon was presented with a history of seizures which became uncontrollable. Laboratory work included a normal CBC and chemistry panel and negative *E. canis* and *C. immitis* serology. The owners elected euthanasia. At necropsy there was evidence of brain edema characterized by coning of the cerebellum. The meninges were congested. Microscopically there was marked, **multifocal necrotizing, nonsuppurative encephalitis** affecting both the grey

and white matter. Bacterial and viral cultures were negative. Morphologically, the lesions closely resembled those of "Pug encephalitis." A disease with these characteristics has been described in the Pug and Maltese but not in the Brussels griffon. We note with interest that Pugs were used in developing the Brussels griffon breed.

Chronic, suppurative tubulointerstitial nephritis was the cause of death of a Jack Russell terrier following a short episode of vomiting and anuria. **Leptospirosis** diagnosis with the aid of silver stains was based on the observation of numerous leptospirae in sections of the affected kidney. Although no fresh tissues were available for culture, high MA titers against *L. grippityphosa* were detected in a serum sample collected from this dog prior to death, and in the serum of another dog in the premises which recovered following treatment. *L. grippityphosa* is the leptospira serotype most commonly associated with wildlife infections in Arizona (rodents, javelina). The affected dogs were from the Tucson area but were frequently taken to a ranch in southeastern Arizona.

Wound infection due to *Nocardia* sp. was diagnosed in a five-month-old stray female Chihuahua-mix canine. The dog was euthanized and submitted for necropsy with a history of poor response to treatment for multiple puncture wounds which were presumed to be bite wounds of the right shoulder and chest areas. Despite local wound treatment and administration of Clavamox® and Baytril® the animal continued to deteriorate and began seizing repeatedly. Necropsy revealed drainage of purulent exudate from the largest of the wounds and multiple granulomatous foci scattered throughout liver parenchyma. The lungs were discolored a plum color. Microscopic examination revealed a locally extensive wound-associated **cellulitis** in which gram-staining revealed numerous gram-positive, beaded, filamentous bacterial organisms. Similar organisms were present in the granulomatous foci in liver and in exudate around the choroid plexus of the lateral ventricle of the brain. *Nocardia* sp. was isolated in cultures of the wounds and from a liver lesion.

Bufotenine intoxication (toad poisoning) was diagnosed in a two-year-old Yorkshire terrier poodle mix weighing 25 pounds. The dog started barking and whimpering during the night. Upon investigating the owner found the dog "spread-eagled" and suspected toad poisoning and immediately washed the dog's mouth out with a garden hose. Despite the treatment, the dog died and necropsy revealed considerable froth in the tracheal lumen, mucus, and a small amount of ingesta in the stomach. Both GC/MS and TLC screening of stomach contents were positive for 5-methoxy-bufotenine, which is part of the toxin complex secreted by the Colorado River toad. Some of the toxins can produce ventricular fibrillation, especially in smaller dogs.

Septicemia due to mixed bacterial infection (group G *Streptococci* and hemolytic *E. coli*) was diagnosed in an eleven-day-old Yorkshire terrier pup that died suddenly. Septicemias are a common finding in “sudden deaths” of puppies necropsied at the AzVDL.



Feline

Feline Infectious Peritonitis was diagnosed in six independent necropsies. Three of the cases were the classical, effusive peritoneal form. A fourth case was a Persian cat with a history of lethargy, anorexia and dyspnea. Radiographs revealed a pleural effusion. At necropsy, the chest contained 20 ml of clear fluid. The sternal and hepatic lymph node were enlarged and contained multifocal, firm tan areas. Histologically, the nodes exhibited necrotizing pyogranulomatous inflammation compatible with FIP. The fifth case was the dry form of FIP in a seven-month-old male cat. Gross lesions included white foci on the liver and kidneys. Ataxia and paresis were interesting parts of the clinical history. Pyogranulomatous infiltrates were found in various organs including the brain. The sixth case had only pyogranulomatous brain lesions.

A five-year-old, twelve pound domestic short-hair feline that was being boarded when it had an “acute onset” of facial, tongue and lip swelling. The cat progressively worsened and “respiratory arrest” occurred. The animal was resuscitated and stabilized temporarily but then had a “fit”, became agitated, acted “demented”, then died. During resuscitative procedures the attending veterinarian noted that the mucosa of the tongue was discolored and sloughing. Necropsy revealed moderate swelling of the lips, swelling and tan discoloration of the tongue, edema of the retropharyngeal tissues, and edema and necrosis of the esophagus throughout its length. **Poisoning due to ingestion of a quaternary ammonium disinfectant compound** was diagnosed when GC/MS analysis of stomach contents revealed the presence of both N-alkyl dimethyl and N-alkyl dimethyl benzyl ammonium compounds. This matched a sample of quaternary ammonium disinfectant used by the kennel. From the distribution of the lesions it was assumed that accidental ingestion of the disinfectant probably occurred. Precisely how this occurred is uncertain. Quaternary ammonium compounds in appropriate concentrations can be severe irritants and may produce substantial tissue damage. This case suggests that it is important to train kennel personnel in the proper use of disinfectant compounds, including proper dilution of disinfectants and flushing of surfaces that have been treated. A similar, although less severe, case involving **accidental ingestion of Chlorox®** (sodium hypochlorite) by a cat has been seen. The cat was accustomed to drinking water from puddles in its owner’s shower and ingested Chlorox® solution which had been used in cleaning the shower but had not been flushed

away. The cat lived but required treatment for severe damage to the oral mucosa.

Obstruction of the common bile duct of unknown etiology was diagnosed in an 8½ week-old American Curl kitten that was presented for necropsy. The kitten had been fine until 7½ weeks of age when there was a sudden onset of diarrhea, anorexia, and dehydration lasting five days which was followed by the animal’s death. Necropsy revealed generalized icterus. The tissues and the liver were swollen and pale. The gall bladder contained a small amount of thick, viscous bile. The common bile duct was not patent to the duodenum and fecal remnants in colon were pale indicating a lack of bile excretion into the intestine.

Septicemia due to group G streptococcal infection was diagnosed in a six-month-old female domestic long-hair feline from a cattery with about 30 other cats. The animal had an “acute onset of fever and limping of the right rear leg”. Abscess formation was also noted and the cat did not respond well to antibiotic treatment. Necropsy revealed extensive edema in the subcutis of both hock joints and purulent exudate in the deeper periarticular tissues of the right hock. There was a fracture of the left distal femoral epiphysis which was also surrounded by purulent exudate. Microscopic examination revealed numerous chain-forming, gram-positive bacteria within both hock lesions and bacterial invasion of the marrow cavity from the fracture site. Coccoid bacteria were also present in lung in which there was a necrotizing pneumonia. Bacterial thrombi were present in glomerular capillaries and in a few capillary lumens in the renal cortical interstitium. Cultures yielded group G *Streptococci* from both hock lesions and from lung. The septicemia likely occurred as a result of embolic spread from the epiphyseal fracture which may have occurred as a pathologic event resulting from the spread of virulent *Streptococci* from a cellulitis of the subcutis of the hock joints. This amply illustrates the pathogenic potential of this organism. The cattery has had a persistent problem with streptococcal infections.

A stray, fourteen-week-old Siamese cross kitten in a foster home died suddenly. At necropsy there was bile stained fluid on the hair around the mouth. The stomach contained mucoid watery fluid. The small intestine was flaccid and filled with pink, watery fluid. The colon contents were liquid green fecal material. Microscopically there was necrotizing cryptitis in the small intestine characteristic of **feline parvovirus infection (feline panleukopenia)**



Wildlife

Bluetongue virus serotype seventeen was isolated from the spleen of a captive Desert bighorn ewe with an acute onset of subcutaneous edema of the head and ventrum, depression and anorexia.

Aspergillosis was diagnosed as the cause of death in a Burrowing owl that was found in an urban area of Phoenix. The bird had flown into a window and suffered "head and spinal trauma" and had improved markedly following treatment. After 2½ weeks of treatment the bird was "standing and active but refusing food" and then suddenly went into convulsions and died. Necropsy and histologic examination revealed lesions typical of a mycotic pneumonia and *Aspergillus fumigatus* was isolated from lung tissue in culture.



Exotics

A 17½-year-old Red-back squirrel monkey from a zoo collection died following presentation for inspiratory dyspnea. Chest radiographs revealed an enlarged left atrium. At necropsy, **atherosclerosis** of the aorta and coronary arteries was found and had resulted in multifocal **myocardial infarction and fibrosis**. Also present were multiple scabs and sores on the skin of the hands and feet. The lesions were small ulcers with intranuclear inclusion bodies in adjacent epithelium consistent with simian **herpes-T virus** (Cebid herpesvirus I) infection. Squirrel monkeys are the natural host for this herpesvirus infection. Infections in this species are usually inapparent but may have oral vesicles and ulcers. The infection produces a fatal, generalized disease in owl monkeys, tamarins and marmosets.

Colibacillosis was diagnosed in a two-month-old, dwarf rabbit that died within a few hours of developing mucoid, green diarrhea.

Multicentric malignant lymphoma was the cause of death in a 2½-year-old, male hamster with a history of anorexia, ocular discharge and weight loss. Neoplastic infiltrates were present in visceral lymph nodes, liver and spleen.

Adenovirus hepatitis was diagnosed in three, two-week-old, Bearded dragons. They were from a group of 250, 50 of which died acutely.

Idiopathic cardiomyopathy was the cause of severe ascites and subcutaneous edema in a one-year-old Bearded

dragon which present with a history of anorexia and puffy eyes. The swelling around the eyes was the result of orbital accumulation of edema fluid.

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

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

Notes from the toxicology section

Our new computerized GC/MS (gas chromatography/mass spectrometer) has proved to be a tremendously useful asset for the detection of a wide range of organic toxicants/toxins. It should be noted that despite all the computer-assisted bells and whistles that accompany this machine, a human operator (i.e. an experienced analytical chemist) having considerable skill and knowledge is needed if maximal benefit is to be derived from its use. Also, a good history and in the case of animal death, thorough necropsy procedures to rule out other types of disease are essential. The history should provide information on known or suspected drug usage or chemical exposure. A tabular summary of diagnostically useful findings with this equipment will be presented in the toxicology summary in an upcoming newsletter.

Practitioners might be interested in knowing that client compliance in the administration of controlled drugs to animals can be monitored using the GC/MS. For example, usage of phenobarbital for seizure control in dogs can be verified by submitting a serum sample that can be collected during routine re-evaluations of epilepsy cases.

Screening of birds for both lead and zinc intoxication can be performed on a single 0.5 ml sample of blood submitted in a **Microtainer®** with either EDTA or heparin as an anticoagulant. **Do not** submit samples in standard rubber-stoppered blood collection tubes as stopper-origin zinc contamination is likely to occur. Blood collected with syringes should be transferred **immediately** to the Microtainer®, again, to minimize rubber plunger-origin zinc contamination. *by T. H. Noon and Dana Perry 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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