



Newsletter

Volume 6, Issue 3

September 2001

A Quarterly Publication of the **Arizona Veterinary Diagnostic Laboratory**

From the Director:

Our Mission

Our mission is generally defined as “providing veterinary diagnostic services to the veterinarians and animals owners of the State of Arizona.” However, our collective training and experience allows us to interact in unique ways with our colleagues on the campus and with various groups off campus. A good example is the current involvement of Drs. Bradley, Dial, and Noon who have primary roles in teaching courses for our veterinary science curriculum. This enhances our interaction with our colleagues on the campus and provides pre-veterinary students with an invaluable exposure to veterinary professionals with a wide range of professional experiences that can make the learning experience more meaningful. We also interact by cooperating with researchers on campus in developing and utilizing new diagnostic procedures. Very often we are dependent on those people to assist us with new and complex diagnostic tests. An example is the support we receive from Dr. Songer’s lab with anaerobic diseases such as Clostridial disease. There are also significant efforts with other enteric diseases by individuals such as Dr. Joens who works with enteric pathogens, and Dr. Sterling, who works with enteric protozoa. Dr. Collins provides valuable collaboration and support with molecular diagnostics and Dr. Lightner helps us solve problems with aquatic animals. We also participate in numerous departmental and university committees in the hope of helping the productive activities of the department and university. Occasionally we see ourselves somewhat as outcasts because a lot of University operations are designed for support and regulation of research laboratories rather than service laboratories. Our commitment to outreach activities has also escalated with the addition of Dr. Cuneo as our extension veterinarian. We hope we are utilizing our efforts productively in these various endeavors in addition to regular diagnostic work. I am always interested in comments from those out-

side the diagnostic laboratory that may have ideas with regard to how we can better utilize our resources. Feel free to contact me with your comments. (520-621-2356)

Equine CNS Disease

We had a comment in our last newsletter about notifying the diagnostic laboratory or the Arizona Department of Health Services if West Nile Virus was suspected in equines. That statement was somewhat inappropriate and we offer the following comments by Dr. Willer, the Arizona State Veterinarian, as a more appropriate method of reporting CNS disease in equines; “The correct point of reporting any suspected or diagnosed equine viral encephalomyelitis is the State Veterinarian’s Office. The State Veterinarian passed a rule several years ago mandating the reporting of certain livestock diseases to the State Veterinarian, either within 4 hours, or on a monthly basis, depending on the disease. Equine encephalomyelitis must be reported within 4 hours of being suspected or diagnosed. For a complete listing of reportable livestock diseases, contact the State Veterinarian’s Office at 602-542-4293.”

West Nile Virus Update

The spread of West Nile Virus to the Southwest is probably just a matter of time. This summer we have seen the virus spread to Canada and ten more states as of September 7, 2001. Alabama, Florida, Georgia, Illinois, Indiana, Kentucky, Louisiana, Michigan, Ohio, and Wisconsin have all reported cases of West Nile Virus.

PCR Testing

Our introductory pricing for PCR testing is coming to an end. We will be publishing a new price list for all of our services soon and PCR testing will increase. Our objective is to be more in line with our costs and the fees charged for PCR in other diagnostic laboratories.

Dr. Robert D. Glock, Director

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Important Telephone Numbers:

<i>After hours assistance</i>	520-349-5534
<i>AzVDL cell phone</i>	520-349-6361

Submission Tip:

Courier Service

The University of Arizona Courier Service has asked that we remind our customers in the Phoenix area of the policies and procedures for using their services. They transport specimens to us for necropsy as a free service. However, they do have some requirements. Please keep these requirements handy for referral when using this service.

1. Because the driver is alone, a weight limit of 75 pounds must be implemented. Keep in mind that this will include the weight of the animal, the shipping container, and the ice.
2. All specimens, including blood samples, must be delivered to the pick-up location in a *sealed, leak-proof, insulated shipping container*.
3. Leaking, or otherwise inappropriately packaged specimens will be refused for transport to the AzVDL.
4. Live animals will not be accepted under any circumstances.
5. All shipping containers must be properly addressed, including a return address and phone number. Properly packaged specimens should be delivered the same day, at a time prior to the arrival time of the courier as stated in the schedule below.
6. And finally, under NO circumstances will specimens be packaged or stored at any of the locations.

As a reminder, the personnel at the extension centers are not affiliated with the courier service. They only “loan” their office space for holding our packages. Please do not ask them to assist you in any way with the handling or packaging of specimens.

1. *Pinal Extension Center specimen should arrive before 9:00 am (M, W, F)*
2. *Maricopa County Extension Center before 10:00 am*
3. *Maricopa Agricultural Center before 9:30 am*

Feature Article:

Nitrate Poisoning

Poor quality hay containing pigweed (*Amaranthus palmeri*) sold as low-cost “cow hay” was the likely cause of nitrate poisoning in several cattle during this past reporting period. The term “cow hay” implied that the hay was safe for cattle when in fact it was not. This type of hay, in our experience,

has often been the cause of nitrate poisoning.

Oswailer, et. al. state that “acute poisoning may be expected when forage nitrates exceed 1% (dry weight) or 1,500 ppm in water.” These values serve as a general guide but it is well to remember that the effects are dose-related. Oswailer et. al. state that, although variable, the approximate lethal dose of nitrate in ruminants is approximately 0.5 gm/kg body weight (note 1% = 10,000 ppm = 10,000 mg/kg).¹

Ruminants are most susceptible to nitrate poisoning because of the greater efficiency of rumen microorganisms in converting nitrate to the more toxic nitrite form. The horse is far less susceptible than the ruminant, probably because microorganisms in the hindgut are less efficient in converting nitrate, making poisoning less likely, but still possible. When absorbed, nitrite converts hemoglobin to methemoglobin, which renders it incapable of transporting oxygen and results in signs of hypoxia. When large amounts of nitrate-containing forage are ingested, death occurs fairly rapidly in ruminants. However, clinical signs vary according to the ingested dose of nitrates and the resulting degree of methemoglobin formation. Nitrate-poisoned animals, if examined while moribund or recently dead, may have chocolate-colored blood that does not clot. It is reported that in animals dead longer than five hours the typical discoloration of the blood fades. It is our experience that discoloration of the blood is not commonly observed at necropsy due to the substantial post-mortem interval between death and necropsy that is typical of our bovine case material. There are no other diagnostic necropsy lesions. Nitrate poisoning in the bovine must be differentiated from urea poisoning, cyanide poisoning, acute bovine pulmonary emphysema, sodium chlorate poisoning, and other causes of unexpected death. Animals suspected of dying following consumption of weedy forage can be evaluated for nitrate poisoning by submitting (in order of preference): aqueous humor from the eye, cerebrospinal fluid, serum or heparinized whole blood, or rumen contents. Rumen contents should be collected within two hours of death and be refrigerated or frozen immediately to inhibit microbial activity. Suspicious forage should be submitted for testing as well. Dried forage retains its toxic potential.

Note: An excellent new book about poisonous plants containing color pictures is now available. It is entitled *A Guide to Plant Poisoning of Animals of North America* by A. P. Knight and R. G. Walter. It is available from booksellers or Teton New Media, Post Office Box 4833, Jackson, Wyoming 83001 (1-888-770-3165).

By T. H. Noon

¹Clinical and Veterinary Toxicology 3rd Edition (1976) by G. D. Oswailer, T. L. Carson, W. B. Buck, and G. A. Van Gelder. Kendall Hunt Publishing Co., Dubuque, Iowa.

Diagnostic Update

The following are selected samples of cases submitted to the AzVDL during the summer months of June, July, and August.

Bovine



Neospora canis was diagnosed as the cause of abortion in two bovine fetuses submitted from an Arizona dairy. PCR testing was positive for chlamydia in both fetuses as well. Lesions in both fetuses were very suggestive of *Neospora* sp. infection but immunoperoxidase staining was positive in only one of the fetuses. Lesions consistent with *Neospora canis* were also found in the heart and skeletal muscle of two Holstein fetuses from another Arizona dairy. Canids have been determined to be the definitive host for this parasite. Dogs should be prevented from defecating on feed intended for pregnant cattle.

Small Ruminants



A four-year-old doe had a history of milk depression, weight loss, fever, and upper respiratory distress. The dark, firm, heavy lungs did not collapse. Histologic lesions of diffuse interstitial pneumonia with extensive lymphoid infiltrates are typical of infections with **caprine arthritis and encephalitis virus (CAE)**. This virus occasionally produces lesions similar to those associated with ovine progressive pneumonia in sheep. Both syndromes are caused by a lentivirus. The viruses from sheep and goats have minor differences.

Equine



An *Escherichia coli* septicemia was the cause of death of a neonatal (one-day-old) foal. No gross lesions were observed on necropsy. Histologic lesions included a severe embolic suppurative nephritis with emboli in glomerular capillaries, a septic hepatitis, and lymphoid necrosis in the spleen. Microabscesses were observed in the interstitium of the kidney and liver. *Escherichia coli* was isolated from all the affected tissues.

An **abscess** measuring approximately 15 centimeters in the thoracic inlet resulted in acute respiratory distress and death of a one-year-old Quarter horse. There was a history of the animal having strangles about two months previously. *Streptococcus equi* subsp. *equi* was isolated from the lesion.

Equine influenza was the cause of death of a yearling horse. The horse was one of thirteen horses in a herd of thirty to develop an acute respiratory illness. In the yearling, the illness was refractory and ended in death after an eight-day duration of "heaves." The presence of the virus was con-

firmed by the Directogen® test on a swab of tracheal exudate.

Monensin (Rumensin®) was found in a pelleted equine feed supplement that had been fed to two horses, both of which became ill and one of which died. A necropsy was not conducted and so a definitive cause of death could not be established. Retrospectively, an estimated dose was calculated and was found to be slightly below the reported toxic ranges of this compound for horses. Although a definitive cause of death was not established, it has been well established that monensin is an unusually hazardous compound for horses. The toxic dose for horses is far lower than other livestock. As most are aware, monensin ionophore is not approved for use in horse feed but is a registered feed additive for growth promotion purposes for use at specified levels in cattle feeds.

Avian



Turkey pox was identified in one-month-old turkeys that were very thin as a result of 0.5 to 1.0 centimeter nodules located primarily around the eyes and mouth region.

A **severe gastric spiruroid nematode infestation** was diagnosed in a group of pigeons (Homers and Indian fantails) with a history of weight loss and weakness leading to death of six of the hens in a period of two to four weeks. Large numbers of spiruroids tentatively identified as *Dispharynx nasuta* were found in the proventriculus and ventriculus of two hens euthanized for necropsy. The worms burrow into the mucosa of the stomach causing necrosis and inflammation. Their life cycle requires an insect intermediate host.

Visceral gout was the cause of death of a six-month-old Amazon parrot. The bird had a chronic pyogranulomatous pyelonephritis (ascending infection) from which a pure culture of *E. coli* was isolated.

Liver cirrhosis has been found several times in necropsies of psittacines. Although infections or toxic causes such as mycotoxicosis are often mentioned in the literature as a possible cause, etiologic agents are seldom identified in necropsied birds. Lesions of chronic, end-stage liver disease are non-specific; toxicologic analysis of feeds is of little use to detect exposure to a toxin that often takes place months before; and infectious agents are rarely identified. Three unrelated such cases were observed during this reporting period. One involved two parrotlets undergoing treatment for mycobacteriosis. Although either the infection and/or the treatment could have precipitated the condition, no cause and effect relationship could be established at necropsy. The other cases involved a parrotlet (of a different breed) and a lory. No likely cause for the condition was identified at the

necropsy of either case.

Necrotizing hepatitis, encephalitis and myocarditis were the findings of the necropsy of a five-week-old African Grey parrot. Three members of the clutch had died in the days following an accidental overheating. Numerous vibrio-like organisms were observed in silver-stained sections of the affected tissues. Although the causative agent could not be isolated because microaerophilic cultures were not attempted at necropsy, the lesions and the morphology of the organisms were highly suggestive of a *Campylobacter jejuni* infection.

Adenovirus infection was the cause of death in two, three-month-old racing pigeons presented for necropsy. The loft of one hundred sixty birds lost seven birds in two weeks. Hemorrhagic intestines were noted grossly in one of the birds. The other had no gross lesions. Hepatic necrosis with intranuclear viral inclusion bodies characteristic of adenovirus was found in both birds.

Polyomavirus infection was the cause of death in a fledgling Blue and Gold macaw and two unrelated Indian Ringneck parakeets. This disease most often affects birds less than sixteen weeks of age. There is usually a sudden onset of weakness and rapid decline in affected birds. Ecchymotic hemorrhages can often be seen in the skin of thorax, abdomen and upper legs. Post mortem lesions are distinctive and include multifocal hemorrhages in the epicardium, liver and spleen with splenic and hepatic necrosis and intranuclear viral inclusion bodies.

Aspergillosis caused emaciation with lesions in the lungs and air sacs of a three-year-old lory. Iron levels were excessive (1417 ppm) in the liver. This is sometimes referred to as hemosiderosis. We are uncertain whether this is secondary to the aspergillosis.

Salmonellosis was diagnosed in a cockatiel. The bird was one of seven that became “weak” and died within a three-week period. No other outward signs were noted. Group B Salmonella organisms were isolated in cultures of the intestines and lung. Multifocal coagulation necrosis of hepatocytes with extensive bacterial colonization of affected areas was also evident microscopically. Bacterial colonies were also evident within capillary lumens of lung sections. Lymphoid depletion and multifocal necrosis were present in the spleen.

Botulism was diagnosed in a young adult Ruddy Headed shell duck from a private zoo. The history provided with the case indicated that several other ducks were listless, ataxic, and were unable to walk. At necropsy there were no gross lesions and serum taken prior to death by the submitter was positive for *Clostridium botulinum* type C toxin (mouse bioassay).

Malignant melanoma was diagnosed in an adult African Grey

parrot. The owner noted that the “bird stayed in the nest box most of the time”. The bird was ultimately found dead. At necropsy, the liver was enlarged and had a roughened irregular surface, with dark, almost black tumor-like foci embedded throughout the parenchyma. Microscopic examination of sections revealed a typical malignant melanoma.

A **necrotizing salpingitis, internal laying,** and resultant **serositis** were diagnosed in a duck that was submitted after being confiscated in a suspected animal cruelty case. The salpingitis was due to mixed bacterial and yeast infection and organisms were evident throughout sections of the affected tissue. Yolk droplets and extensive inflammation were evident on serosal membranes covering abdominal viscera.

Aspiration pneumonia was diagnosed in a juvenile Green Cheeked conure that was reported to have “neurologic signs for approximately eight hours”. Plant fragments, amorphous food-like material, colonies of mixed bacterial organisms, and numerous macrophages were present on the serosal surfaces of the proventriculus. Plant fragments and mixed bacterial organisms were also present in the airways of the lung.

Feline



Small cell lymphoma is a fairly frequent finding in older cats. We recently have had a fifteen-year-old and a sixteen-year-old cat submitted with histories including diarrhea, cachexia, jaundice, and abdominal swelling. This form of lymphoma can be very difficult to identify clinically and the lesions frequently are not obvious grossly. The diagnosis is based on histologic identification of heavy populations of small lymphocytes in the intestine.

Approximately 40 cats were affected by an outbreak of **respiratory disease** of high morbidity and mortality in an animal control pound in eastern Arizona. Calicivirus, *Mycoplasma* sp. and a group G *Streptococcus* sp. were isolated from the pneumonic lungs of one of the affected cats, a twelve-week-old Siamese kitten submitted to AzVDL for necropsy.

Cardiomyopathy was diagnosed in a six-to-seven-year-old Domestic Shorthair feline. The owner found the cat dead in the yard. Vaccination history was up to date and no prior clinical signs had been noted. At necropsy, the heart was of normal overall size but the left ventricular walls were hypertrophied leaving only a slit-like left ventricular chamber. Microscopically there was multifocal endocardial and myocardial fibrosis with disorganization and atrophy of myofibers.

Panleukopenia (feline parvovirus, feline enteritis, feline distemper) was diagnosed in three kittens that were submitted for necropsy. Vomiting had been noted and megaesophagus was one of the clinician’s differential diagnoses. At

necropsy, there was fecal soiling of the perineum in two of the kittens. Internally, congestion of the serosal vessels, mucoid luminal contents, and dilation of the intestinal loops in two of the kittens was evident. In the third kitten there was locally extensive hemorrhage of the cerebral cortex and intra-abdominal hemorrhage. Microscopically, lesions in all three kittens were those of a necrotizing enteropathy that was compatible with panleukopenia. It was reported that PCR testing of gut tissue was positive for nucleic acid sequences specific for panleukopenia virus in all three kittens.

Canine



Diphacinone **rodenticide poisoning** resulted in the death of a three-month-old Beagle. This anticoagulant resulted in severe hemorrhages in the thoracic and abdominal cavities. This type of rodenticide is being used more frequently. The development of numerous types of rodenticide poisons, including anticoagulants, over the past few years has made analysis for these toxins more complex.

A large **chemodectoma** or so-called heart-based tumor interfered with cardiac function in a fifteen-year-old Coon hound. This resulted in severe ascites and some liver pathology.

A **pheochromocytoma** originating in the adrenal gland was associated with extensive aortic aneurism and restricted blood flow in the posterior vena cava of an eight-year-old miniature Schnauzer. There was a history of abdominal and scleral hemorrhages associated with thrombocytopenia and a depressed packed cell volume.

Pheochromocytoma of the adrenal glands was also diagnosed in an adult female Chow that was submitted by a municipal animal control agency in a case involving suspected animal cruelty. At necropsy there were bilateral, nodular, tumorous enlargements of both adrenal glands and multiple tumor foci were present in the parenchyma of the liver. Microscopic sectioning of the lesions was typical of pheochromocytoma. It was suspected that the cause of death in this case may have been tachyarrhythmias resulting from chronic, excessive, catecholamine production by the adrenal pheochromocytomas.

Purulent prostatitis, peritonitis and septicemia produced by *Staphylococcus aureus* were the cause of death of a

nine-year-old Rottweiler. History indicated that the animal had collapsed shortly after being picked up from a boarding facility. The prostate was enlarged, with a thin-walled abscess, and there was a ruptured, necrotic periprostatic cyst where the infection had extended.

Three poodle puppies from a litter of six died after “fading away”. All three were smaller than their litter mates. Necropsy findings in one of the dead puppies included black tarry feces in the colon and pasted on the hair of the perineum. Small amounts of digested blood were present in the stomach and small intestine. Internal fat stores were absent. Microscopically, bacteria were colonizing the epithelium of intestinal villi and the surface epithelium of the colon. Erosion, exfoliation, and epithelial cell degeneration were seen in the colon. *Escherichia coli* was isolated.

Papilloma of the choroid plexus was diagnosed in an eight-year-old Chow mix. It was presented to a veterinarian circling from right to left. The dog had been blind for ten days. At necropsy, there was yellowish, gelatinous fluid in the lateral ventricles of the brain. A one-to-two centimeter sized reddish, nodular, tumorous mass with an irregular surface was present and attached to the anterior-lateral aspect of the left lateral ventricle of the brain. Microscopic examination was typical of a papilloma of the choroid plexus.

Severe, diffuse, necrotizing, hemorrhagic and abscessing pneumonia was diagnosed in a five-year-old neutered canine that had been boarded for ten days. At necropsy, there was thin, bloody fluid present in the thoracic cavity. The lung lobes were discolored dark red to black and were consolidated. Culture of the lung tissue yielded coagulase-negative beta hemolytic *Staphylococcus* sp. and beta hemolytic *E. coli*.

Canine parvovirus infection was diagnosed in four unrelated cases during this reporting period.

Wildlife



Hemosiderosis was diagnosed in a five-year-old Peregrine falcon that was found on the north side of Phoenix. The bird died two days after being brought to a rehabilitator. Diffuse, cytoplasmic accumulations of brown, granular, iron-positive pigment were present in the cytoplasm of hepatocytes and Kupffer cells. Formalinized liver tissue contained an excessive amount of iron (1,135 ppm; reference level approximately 300 ppm).

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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Exotics



Acute hemorrhage from an active right ovary was the cause of rapid decline and death in a three-year-old, female Bearded dragon.

Cardiovascular collapse was evidently the cause of death in a polar bear that was immobilized with Telazol® and Metomidine® at recommended dosages for radiography of a forelimb lameness problem. Respiratory arrest occurred about thirty minutes after immobilization. Resuscitation was unsuccessful. At necropsy, there was congestion of the spleen, intertubular vessels of the kidney, and the sinusoids of the liver suggesting that cardiovascular collapse occurred. No other lesions were found.

Compiled by Greg Bradley, Sharon Dial, Peder Cuneo, Bob Glock, T. H. Noon, Carlos Reggiardo, and Barbara Pickard

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

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