



Newsletter

Volume 4, Issue 3

September 1999

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

From the Director

Dr. James Collins has been named as the new head of the Department of Veterinary Science and Microbiology. Dr. Collins is already interacting with the members of the department on some important issues and he plans to be on board full time approximately November 1, 1999. We welcome his input to improve the efficiency and effectiveness of, not only the department, but also the veterinary diagnostic laboratory. Dr. Collins is a PhD microbiologist and has been the director of the virology and at some times the entire microbiology section of the diagnostic laboratory at Colorado State University. He has a number of years of experience and has developed a reputation for promoting interaction between the laboratory and its clients. He has stated a strong interest in developing interactive relationships with our various user groups. Dr. Collins has been instrumental in developing a number of new tests at the CSU Diagnostic Laboratory and we look forward to his participation in helping us upgrade some of our capabilities. He has been particularly effective in developing PCR testing which we are soon to bring on line here.

We hope newsletter readers will take every opportunity to get acquainted with Dr. Collins. We also want to thank our many constituents and practicing veterinarians who participated in the search for a new department head, especially Drs. Gerry Ault, Joe Yearous, Richard Panzero, Bill Wallace, Jack Quick, Chuck Boreson, Howard Moore, and Ray Reed. This was a time consuming commitment and it is truly appreciated.

Other good news includes a commitment from Dean Sander to begin the search for another diagnostic pathologist for the AzVDL. We look forward to this as an opportunity to become more creative and to participate more effectively in outreach activities.

The Dilemma of Sudden Death

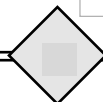
To reach a diagnosis of the cause of death of an animal a joint effort by the owner, attending veterinarian, and diagnostician is usually required. A careful observation by the owner of any changes in an animal's environment and behavior, as well as a good medical history and a detailed clinical evaluation by the veterinarian, often generate invaluable diagnostic information, without which a necropsy and laboratory testing may not be sufficient to fully characterize the cause of death. It is not uncommon for us to receive submissions of animals where most information is missing. The primary history (or most often the **only** history) is "sudden death". In a number of these cases the "sudden" death is the result of a progressive clinical disease, which was simply not observed by the owner, but significant lesions were observed on necropsy. Cats and birds seem to be less likely to let their owners know how distressed they are. A good example is a case where there was a severe chylothorax but the cat was "found dead". In some cases, a true acute or peracute condition such as trauma, poisonings with substances physically identifiable in the ingesta, or a gastric torsion can be easily diagnosed on necropsy. But it is not uncommon

to face cases where we can only eliminate known identifiable causes of death, and offer, at best, our educated but uncertain diagnosis, which is a situation particularly frustrating for owners. Some specific comments regarding various species follow.

Canine: Dogs of various ages are often presented with the history of sudden death. In the warmer months in Arizona, these are often related to some type of heat stress or water deprivation. There are no specific lesions of these conditions and we usually eliminate other likely causes and then depend on what history is available to try to conclude the possibility of heat exhaustion. Unfortunately, these

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animals are frequently found in the sun during hot weather and the specimens may be quite autolyzed making our search more difficult. There is another type of sudden death in dogs that are in apparent good health and are turned out in the backyard for exercise. Then a few minutes to a few hours later they are found dead. Sudden death can also occur as a very distressing sequela to anesthesia or even post anesthetic recovery. In these rare but distressing deaths we sometimes suspect heart failure, but the diagnosis is only solidly positive when we can find lesions such as actual cardiomyopathies. The dramatic myocardial infarctions typical of human "heart attacks" are extremely rare in animals. It is not possible at this time to do a very good job of evaluating anesthetic technique or dosages in the postmortem situation. Often the most important thing we can do is to try to help the people involved to understand that this is a rare event and appears to be basically unavoidable. The anesthetic death is a good example of the need to warn clients that there is always some risk associated with a procedure.

Horses: The occasional sudden death in a horse may actually be a severe colic that became fulminating without observation. Sometimes the history or the animal's surroundings can help with that diagnosis. Electrocutation may also be a concern and there have been situations where malicious administration of drugs have been involved (some of which are difficult to trace). We recently had a case of a horse that was heavily exercised and became stiff progressing to death within a few minutes. The only finding was somewhat elevated potassium levels in the anterior chamber of the eye. However, clinicians tell us that it is not definitive evidence of hyperkalemia. The final decision was that this was probably representative of a poorly defined syndrome known as "exhausted horse syndrome". This may be a combination of disturbances in electrolyte balances, acid base balances, and other physiologic parameters. Horses do not commonly die of "heart attacks".

Bovine: We must consider some of the common causes such as bloat, acidosis, and lightning strike. Lightning strike is a particularly interesting situation because there frequently are no burn marks and the presumptive diagnosis is often a matter of eliminating other possible causes of death. We recently had a situation where some adult cows were hauled for a period of about two hours and seven out of 60 died within the next several hours. Here again the practitioner involved was unable to find any specific lesions and we did not find anything in the laboratory. The assumption is that there was probably some metabolic problem, perhaps related to relatively warm temperatures.

The diagnosis of most of these sudden deaths will most likely remain elusive because of the nature of the insult, economic or technical limitations on how much testing is possible, or simple autolysis of the cadaver. But, as in the

observation of "frogs in the yard" in the case of bufotenin intoxication described below, the submission of a medical history when available and a careful description of the animal's environment can sometimes greatly facilitate the diagnosis.

by Bob Glock and Carlos Reggiardo

Arizona Arbovirus Update

Every year from May through October, public health officials in Arizona conduct surveillance for mosquito-borne encephalitis viruses, including western equine encephalitis (WEE) and St. Louis encephalitis (SLE). Mosquito samples are collected by participating agencies (county, state, Indian Health Service, Tribal Governments and pest abatement districts) and submitted to the Arizona Department of Health Services – Vector-Borne & Zoonotic Diseases Program (ADHS-VBZD) where they are identified to species. Mosquito samples with *Culex* species are submitted to the Arizona State Health Laboratory (ASHL) for tissue culture and mouse inoculation. As of August 13, five samples have tested positive for arboviruses at the ASHL, including three WEE positive samples from Tempe, one WEE positive sample from southwest Phoenix (Maricopa County) and one SLE positive sample from the Gila River Indian Community (Pinal County). All of the positive samples were collected in mid-July. Mosquito control efforts have been stepped-up in the affected areas. To date, no human or equine cases of mosquito-borne encephalitis have been reported. For more information on mosquito-borne diseases, contact the ADHS-VBZD Program at (602) 230-5918.

by Craig Levy, ADHS-VBZD

(Note: If you need diagnostic assistance with a horse exhibiting neurological signs we suggest you contact the AzVDL at 520 621-2356.)

Diagnostic Update

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



Bovine

Pasteurella multocida and *Mycoplasma* sp. were the cause of pneumonia and severe otitis media in three-week-old dairy calves with a history of head tilt and droopy ears.



Equine

Colitis X was the diagnosis in a ten-year-old Arabian gelding. This animal had a history of being off feed for 36 hours and lethargic for twelve hours with terminal respiratory distress and convulsions. Severe diffuse colitis with mucosal congestion, edema and limited hemorrhage were observed at necropsy. The diagnosis is

based entirely on history, gross lesions, and microscopic lesions. The cause of this disease is not specifically known although various bacteria including *Clostridium* spp. and *Salmonella* spp. have been suggested as possible participants. In this case clostridial populations were very low. Additional research is needed on this rather frustrating disease syndrome.



Small Ruminants

An acute **fibrinopurulent bronchopneumonia** was the cause of death of a five-month-old Nubian doe. A pure culture of *Pasteurella hemolytica* was isolated from the lungs and thoracic lymph nodes as well as from areas of muscle hemorrhage in the neck and shoulders.

Caprine arthritis encephalitis was diagnosed in a four-year-old LaMancha doe, which died following a month-long history of weight loss and respiratory disease. Necropsy lesions were confined to a chronic interstitial pneumonia, with characteristic filling of the alveoli by lipoproteins and diffuse lining of the septa by type II alveolar cells. There were no lesions in the CNS or the joints.

Two, one-week-old, goat kids born to the same doe developed swollen joints and fever (106°F). Multiple joints were affected. An eight-day treatment regimen including penicillin and Rimadyl® was not effective. Both were euthanized and one was submitted for necropsy. The atlanto-occipital, left carpal, right hock and left stifle joints contained copious amounts of cloudy fluid with clumps of fibrin. **Mycoplasma mycoides**, large colony type, was isolated from the joints.

An extensive **purulent meningoencephalitis** affecting most of the frontal region of the left cerebrum was the cause of death in a month-old goat. The infection had originated in the site of dehorning. This is a common complication in the dehorning of baby goats, a procedure, which requires experienced personnel due to the cranial anatomy in this species.



Avian

Chlamydiosis was diagnosed in several birds submitted for necropsy, including Amazons, Eclectus, cockatiels, lovebirds and starlings. Most were relatively acute infections in young birds.

There has been an apparent increase in the number of cases of **polyomavirus** infections this summer, with three necropsy diagnoses (in two Eclectus and one Blue and Gold macaw) in one week alone. In one of the cases (a three-month-old Eclectus) there was an acute chlamydiosis coexisting with the polyoma infection.

Botulism due to *Cl. botulinum* type C was diagnosed in a swan received from the Phoenix area and in a duck from the Tucson area.

Aspiration pneumonia was the cause of death in a seven-month-old psittacine of unspecified breed. The bird was being hand fed by the owner and had been sick for several days. Food-type debris and bacteria were found in the air sacs and lung airways. The bird also had a focal mycotic pneumonia along with mild hemosiderosis or iron accumulation in the liver (929 ppm iron, 60-300 ppm reported to be “adequate” in poultry), which probably accounted for its ill health prior to its death. In our experience, unexpected deaths in birds that are ill and are being hand-fed are often due to aspiration pneumonia. Excessive dietary intake of iron may account for the hemosiderosis, as at least some psittacine birds reportedly absorb iron very efficiently. A retrospective diet analysis to determine dietary iron levels would be indicated in any case where hemosiderosis has been diagnosed.

Hemosiderosis of the liver (1531 ppm iron, 60-300 ppm reported to be “adequate”) was diagnosed in a psittacine fledgling that died suddenly. The bird was fed a commercial diet by the breeder who also reported having problems with crop stasis in conures and macaws, and sporadic death losses in conures fed the same diet. Again, retrospective diet analysis to determine iron levels might have been helpful in this case.

Marek’s disease was the diagnosis in a layer hen that died unexpectedly. She had a history of never producing eggs. Lesions included neoplastic lymphocytic infiltrates in the brain, kidney, spleen, liver, ovary, oviduct, lung, small intestine, thymus, proventriculus and colon. Grossly recognizable lesions included an enlarged and pale liver and spleen.

Psittacine proventricular dilatation syndrome was diagnosed in a macaw with a history of chronic crop stasis. The only significant gross finding was an ulcer in the crop. Microscopy indicated the presence of inflammatory infiltrates associated with nerve plexuses in the wall of the proventriculus, ventriculus and intestine. This is a fairly common diagnosis in psittacine birds.



Canine

The first case of **bufotenin intoxication** (Colorado river toad poisoning) was recorded during the first week of summer rains. Bufotenin was detected in the stomach content of a small Yorkie mix found dead in the morning after spending the night outside. The owner had noticed several “frogs” in the yard.

Severe locally extensive **demodicosis** of the skin of the

head, neck, and dorsal trunk area was diagnosed in an approximately three-year-old neutered female Rottweiler that had been treated extensively for Malassezia infection. Skin scrapings were reportedly negative on four occasions for mites. The dog had persistently elevated serum AST values, became anorectic, and began pacing and wandering aimlessly. Sections of skin revealed a chronic active dermatitis and numerous mite and mite egg cross-sections consistent with demodex. Also present was a chronic active hepatitis and glomerulonephritis.

An acute **metritis** due to mixed bacterial infection by *Clostridium perfringens* type A and β hemolytic *E. coli* was diagnosed in a six-year-old Doberman that developed dystocia during labor. Non-surgical attempts to relieve the dystocia were unsuccessful. A caesarian was elected. The animal died while under anesthesia. Necropsy also revealed non-metastatic transitional cell carcinoma of the urinary bladder.

A severe chronic **gastritis**, which became hemorrhagic, was diagnosed in a 9½-year-old neutered male Great Dane, which had bloody vomiting and diarrhea for about six days. *Clostridium perfringens* type A was cultured from the stomach contents but few organisms were seen when sections of the stomach were examined microscopically, making the finding somewhat questionable. However, the dog had been treated with antibiotics, which perhaps reduced the numbers of the organisms. Incidental findings included an aspiration pneumonia, which probably resulted from the severe persistent vomiting, a chronic interstitial nephritis, adenomatous hyperplasia of the adrenal (non-functional) and thyroid glands, and a cardiomyopathy.

Malignant lymphoma was confirmed in an eleven-year-old male Chow that had been ill for about five months. The dog was previously diagnosed with the neoplasm. The dog presented with pleural effusion and enlargement of the abdominal lymph nodes. It was treated with chemotherapy (adriamycin, vincristine, and Cytoxan®) that resulted in a partial remission. It later presented with respiratory distress, generalized lymph node enlargement, and neurologic “deficits” of the hind legs. Necropsy lesions were typical with tumorous involvement of the prescapular, mandibular and anterior mediastinal lymph nodes. There was also tumorous invasion of the proximal end of the pancreas. Microscopic examination also revealed neoplastic involvement of the wall of the stomach, liver, spleen and perivascular spaces of the cerebrum. The brain lesions probably accounted for the neurologic deficits noted clinically in the hind legs.

A ten-week-old, male Poodle died following a five-day history of diarrhea and anorexia. Severe colonization with **coccidia** resulted in epithelial necrosis in the small intestine.

Lesions compatible with *Ehrlichia canis* infection were found in a twelve-year-old, castrated male Shih Tzu that was euthanized following a brief history of listlessness, anuria, vomiting and seizures. The lesions included chronic glomerulonephritis, lymphoplasmacytic meningitis and plasmacytic infiltrates in the spleen, kidney, liver and lung.

A three-year-old, Peruvian hairless dog was presented with a one-week history of vomiting. The BUN and creatinine were 149 and 5.4 mg/dl respectively. The dog did poorly at home and was hospitalized and placed on intravenous fluids. The dog developed seizures and died two days later. At necropsy, severe, bilateral adrenocortical atrophy consistent with **Addison’s disease** was found.

Heat stroke was the cause of sudden death in three dogs from different premises. All three deaths were unobserved and occurred between June 24 and June 25. Necropsy findings were non-specific and included advanced post mortem decomposition, pulmonary congestion and (in one dog) ecchymotic serosal hemorrhage. There are no pathognomonic lesions of heat stroke and the post-mortem diagnosis often requires a “leap of faith”, especially when the death is unobserved. Elimination of other causes of sudden death and a history of exposure to high environmental temperatures, poor ventilation or unaccustomed exercise in warm weather are important clues. The risk of heat stroke is also greatest during the first few hot weeks of the season as some adaptation to the heat takes place after this time. Increased incidence also occurs in the fall following an abrupt rise in temperature after a cool spell.

Streptococcus equi subsp. **zooepidemicus** and **canine distemper virus infection** was the cause of fatal pneumonia in a litter of four-month-old Bull Dog puppies shipped in from out of state. The puppies began coughing three to five days after arrival. At post-mortem, the lungs were firm, red and meaty and contained irregular tan foci of necrosis. The intestinal contents were watery green fluid.

A mixed infection by **canine parvovirus** and **group C Salmonella** was the cause of fatal enteritis in a six-week-old Shar Pei puppy with a two-day history of illness. The gross and microscopic lesions were indistinguishable from those caused by parvovirus alone and included dull red intestinal serosa, watery red intestinal contents, necrosis of crypt epithelium and lymphocyte necrosis in Peyer’s patches and spleen.



Feline

Acute **bacterial meningitis** was diagnosed in a five-year-old female DLH cat euthanized after a day of rapidly progressing neurologic disease of acute onset. *Acinetobacter sp.* was isolated in culture. Members of this genus are widely distributed in nature, but they usually

produce disease in weak or immunosuppressed animals, rarely in healthy ones. Since this cat had received at least one injection of penicillin, it is possible that the treatment may have inhibited the growth of more likely agents of meningitis in cats.

A chronic **purulent rhinitis** was the necropsy finding in the postoperative death of a young cat, which had undergone dental cleaning, and removal of a nasopharyngeal polyp. There was an obstruction of the nasal passages by a thick, sticky mucopurulent exudation, which most likely interfered with the breathing of the animal still under the effect of the anesthesia. A pure culture of *Pasteurella multocida* was isolated from the exudate.



Wildlife

A **renal carcinoma** was the cause of death of an adult male Mallard duck found dead in a residential backyard in central Arizona.

Electrocution was the cause of death in a Harris hawk found dead underneath a power pole in the Cave Creek area. Burn marks on the feet were the only post-mortem findings.



Exotics

A **cholesterol granuloma**, identical with those found in the choroid plexuses of aged horses, was found in the midbrain of a nine-year-old kingsnake. The mass was approximately 2 mm in diameter and resulted in significant displacement of brain tissue. It had produced neuromuscular signs resembling those observed in inclusion body disease.

Disseminated **lymphoma**, affecting heart, spleen, lymph nodes, lungs, the urinary tract and adrenals, was the cause of death of an adult pet ferret.

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. Here is an example. A bronchopneumonia due to infection by *Pasteurella* sp. was diagnosed in a two-month-old lamb identified as a Barb. At necropsy, there was bilateral pneumonic involvement of the anteroventral portions of the lungs and specimens of the affected tissue sank in fixative. Microscopic lesions were typical. Of interest in this case is that death occurred on May 18 and the owner presented the carcass for necropsy on May 26 but had kept the carcass packed in ice until delivering it at the AzVDL. Preservation of the tissues was generally very good and the owner's diligence in preserving the carcass resulted in useful diagnostic information. 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. As illustrated by this case, carcasses can usually be held for several days if the ice is periodically replenished. 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.

by T. H. Noon

New AzVDL Web Site

There will be a wealth of information on our new web site <http://www.microvet.edu>, click on AzVDL. The web site will be online very soon and initially will contain information about the AzVDL, a user guide, price list, an email address (azvdl@ag.arizona.edu), an animal health alert, past newsletters, and a down-loadable submission form. As construction of the website progresses, an informational "special advisories" button as well as other features will be added. We hope you will find it useful.

By T. H. Noon and Natalie Furrey

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.

The University of Arizona

College of Agriculture

Department of Veterinary Science/Microbiology

2831 N. Freeway

Tucson, Arizona 85705-5021

Fax: (520) 626-8696 Phone: (520) 621-2356

email azvdl@ag.arizona.edu

We welcome any subscriptions, comments or suggestions.

Editor:

Natalie Furrey

Contributing editors:

Greg Bradley

Robert Glock

Craig Levy

Ted H. Noon

Carlos Reggiardo

Sarah Swanson

Faculty and Staff of the AzVDL

Director: Robert D. Glock DVM, PhD, Diplomate ACVP

Diagnostics:

Greg Bradley DVM, Diplomate ACVP

Ted H. Noon DVM

Carlos Reggiardo DVM, PhD, Diplomate ACVM

Administrative Staff: Barbara Hiers

Sarah Swanson

Necropsy: Melvin Perry

Owen Halferty

Chemistry: Dana Perry Betzer MS

Barbara Rickert

Histology: Esther Kerr MT(ASCP)

Microbiology: Natalie Furrey MT(ASCP)

Virology: Mark Shupe

Thea Meeker

Adjunct Faculty:

Sharon Dial DVM, PhD

Howard Frederick PhD, PAS

Emeritus Faculty: Raymond E. Reed DVM, Diplomate ACVP

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The University of Arizona
Dept of Veterinary Science and Microbiology
Arizona Veterinary Diagnostic Laboratory
2831 N. Freeway
Tucson, AZ 85705-5021

