



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

From the Director:

West Nile Virus

We have received some questions regarding West Nile Virus and its possible impact on animal health in Arizona. The incidence of infections with West Nile Virus in birds, horses, and humans has increased over the last two years with significant illness and death loss in all three species in the northeastern part of the United States. The Arizona Department of Health Services officials tell us that it is likely that the virus will find its way to Arizona through migratory bird routes. We are participating with ADHS in monitoring disease in birds. Our primary input will be to perform necropsies on selected dead birds that will be acquired through ADHS in cooperation with a number of other agencies. We will examine the birds for WNV lesions and selected tissues will be sent to other labs to determine if WNV is present.

We are also interested in participating in any potential diagnostic work regarding CNS disease in horses. It is likely that the virus will be detected through the efforts of ADHS in wild birds, sentinel chickens, or mosquitos before it appears in equine populations. However, it would be helpful if any unusual CNS disease in horses would be reported to the AzVDL or the ADHS. There are plans for ADHS to do some monitoring of equine sera but details have not been worked out. Meanwhile, there appears to be little risk of West Nile Virus infections in horses at this time. However, this could change. Continued vigilance is suggested. Currently, there are no vaccines available for West Nile Virus. This may change as efforts are made to control problems in the northeast. As always, any protection of animals from mosquitos and other arthropods is a good practice along with the use of other vaccines to prevent arthropod-borne diseases.

Dr. Robert Glock, Director

Submission Tip:

For those of you who may be interested, we can report cases via E-mail instead of faxing. If you are interested in receiving your reports in this manner, please supply us with an E-mail address and we will begin reporting to you in that manner immediately. We will follow up with a hard copy (signed by the diagnostician) of the report for your records. A note of clarification — If we E-mail your reports, we will discontinue faxing.

Rabies Alert:

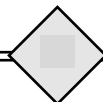
Rabies is a fatal neurologic disease that affects mammals. It is caused by a Rhabdovirus. Within regions of the United States, strains have some degree of species preference. These are host species and the type of rabies isolated refers to the host species in that area. In the southwest, the primary host species are bats and skunks. Currently Arizona is experiencing an outbreak of skunk rabies in the Flagstaff area, Santa Cruz County and southern Pima County.

Veterinarians should inform animal owners of the increased prevalence of this disease and encourage appropriate prophylactic vaccination. In particular, horse owners should be informed of the risk of rabies. Clinical signs of rabies in the equine are often nonspecific and may be attributed to colic. The owner may be exposed before diagnosis is made. Rabies should be included as a possible differential diagnosis in cases of progressive, fatal neurologic disease. The definitive test for rabies is fluorescent antibody-stain of the CNS (hippocampus and cerebellum). The test is done by the Arizona Department of Health Services, at laboratories in Phoenix and Tucson. The Arizona Veterinary Diagnostic Laboratory does help in cases of rabies suspects by providing removal of the brain for testing.

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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.

Equine



Colitis X was the diagnosis in a fourteen-year-old Quarter horse gelding. The animal was euthanized after a few days of colic. The large intestine had severe congestion with edema and significant thickening of the wall. The mucosa appeared to be intact in most areas but there was an area in the distal colon where there were some small ulcers. Aerobic cultures were negative for pathogens. Anaerobic cultures produced large populations of *Clostridium difficile* and both A and B toxins were identified. The etiology of colitis X has not been completely elucidated. However, *Clostridium difficile* is considered a likely cause and certainly appears to be involved in this particular case.

Enterolithiasis resulting in rupture of the large colon was diagnosed in an eight-year-old Paint mare. Necropsy revealed a five-inch diameter struvite enterolith lodged in and obstructing the lumen of the transverse colon. The colonic rupture occurred approximately eight inches proximal to the enterolith and was approximately eight inches long in a transverse direction.

Botulism was diagnosed in an eleven-year-old Arabian gelding from central Arizona. It had received a routine vaccination two weeks before death and developed an abscess at the injection site. It had been off feed for the last five days and unable to rise for the last three days. Peracute aspiration pneumonia was the ultimate cause of death, presumably because of dysphagia. The diagnosis was confirmed by the detection of *Clostridium botulinum* type C toxin in serum by the mouse bioassay test. The detection of botulism toxin in the serum of affected horses is very rare. We have detected it on only one other occasion, from the serum of a horse that died after a clinical course of less than 24 hours. Although no culture or toxin isolation was attempted from the vaccination site, wound botulism is suspected in this case due to the history and the relative ease of detection of the toxin in the serum of a horse clinically affected for several days. Although very seldom diagnosed in animals, wound botulism is increasingly observed in humans, particularly in infected injection sites in drug addicts. The laboratory confirmation of botulism usually requires the submission of serum (at least 5 cc), feed, stomach, and intestinal content. Wound tissue (castration sites, penetrating wounds, etc.) should also be submitted in cases where wound botulism is suspected.

Porcine



We received tissues from two-day and five-day-old pigs with a history of marked edema in the mesocolon associated with diarrhea and some death loss. Small intestines were histologically normal but there was extensive edema and inflammation in the mesentery of the large intestine. This was accompanied by areas of erosion and exudation in the mucosa of the large intestine. Numerous large gram-positive rods were present. *Clostridium difficile* was isolated and tested positive for toxins A and B. The diagnosis was enteritis caused by *Clostridium difficile*.

Small Ruminants



A one-month-old Ramboulett mix lamb had a brief history of right hind limb lameness followed by listlessness. Its body temperature was 106.8 degrees F. It was treated with Banamine® and penicillin. It was found dead the following morning. Gross lesions included thick mats of yellow fibrinous material in multiple joints including the hocks, stifles, and the atlantooccipital joint. Ecchymotic hemorrhages were present on the serosa of the small intestine. *Mycoplasma mycoides sub sp. mycoides (LC)* was recovered from the joints and multiple visceral organs.

A four-year-old Suffolk ewe had dystocia with two dead lambs. Approximately six weeks later, she was presented with weight loss, debilitation, and some indication of CNS signs. Necropsy revealed severe diffuse purulent **peritonitis**. There were extensive adhesions between the abdominal viscera including the uterus.

Avian



Avian **Marek's Disease** was diagnosed in a young adult chicken. This pet chicken had a history of increasing debilitation and emaciation. The diagnosis was based on histopathology with infiltration of lymphocytes in the sciatic nerves, the spleen, and the lung.

Chlamydiosis was diagnosed in an adult female African Grey parrot that was submitted for necropsy. There were no prior signs of illness and the bird was found dying on the bottom of its cage. Necropsy revealed some atrophy of the pectoral musculature and an enlarged spleen. Multifocal whitish discolorations were scattered throughout the parenchyma of liver. Gimenez stain imprints of liver tissue were positive for chlamydia.

Trichomoniasis was diagnosed in a white tipped dove from an aviary in an Arizona zoo. The aviary had a history of trichomoniasis and treatment had been instituted. A typical trichomonad-induced canker was found in the dorsal pharyngeal area and trichomonads were isolated from the lesion on culture.

Zinc poisoning was the diagnosis in a one-year-old goose. The owner reported losing one other goose with similar clinical signs that included ataxia and stiffness. Two more geese were also sick. At necropsy, two zinc pennies (all pennies minted after 1983 contain zinc) were found in the ventriculus. The pancreas contained 1007-ppm zinc.

Feline



A **pulmonary carcinoma** at the base of the middle and caudal lobes of the left lung resulted in infarction and subsequent necrosis of the lobes. The affected portions of the lung were collapsed and firm with a tan color and extensive fibrosis. This is an uncommon finding and appears to be the result of a primary pulmonary carcinoma with a secondary obstruction of vasculature.

Retroperitoneal and perirenal **hematoma** formation was diagnosed in a one-year-old neutered male feline. The owner suspected the cat had been hit by a car five days previously. Hypovolemia was likely to have been the cause of death.

An adult female feline was euthanized after several days of unstable ambulation, erratic behavior, and dilated pupils. On necropsy, the lesion responsible for the clinical signs was a single, 5-mm pyogranuloma of the brain stem that protruded into the fourth ventricle and the overlying cerebellum. Numerous *Coccidioides immitis* spherules were found in the lesion. No other lesions of coccidioidomycosis were found in the cadaver.

Canine



Acute onset of lethargy, anoxemia, and pale gums in a seven-week-old Great Dane was accompanied by a mild sternal systolic murmur. **Right heart dilatation** was observed at necropsy and secondary lesions included a swollen liver, congested lungs, and fluid in the body cavities. No additional lesions were identified grossly or microscopically and the cause of the right heart dilatation was not specifically determined.

Infectious canine hepatitis is still diagnosed in Arizona

with relative frequency in non-vaccinated puppies. In our latest case, ICH was the cause of death of seven mixed breed puppies out of a litter of nine four-week-old puppies found abandoned in the Phoenix area. Affected puppies exhibited lethargy, occasional vomiting, and diarrhea (often hemorrhagic). The disease was confirmed by the microscopic lesions with viral inclusions, and by PCR test on liver tissue.

An **esophageal perforation** by a bone fragment with a resulting acute mediastinal hemorrhage was found in the necropsy of a seven-year-old female Toy poodle. The animal had been “shaking, not acting right, vomiting” and with a “tender anterior abdomen” for three days. A v-shaped flat bone fragment was found lodged in the thoracic esophagus above the heart. Both extremes were sharp and had perforated the esophageal wall, one of them producing a venous puncture in the base of the heart. The bone appeared to be a fragment of a chicken’s pectoral girdle, either a clavicle or a scapula/coracoid bone.

Cardiomyopathy was diagnosed in a six-and-a-half-year-old Brittany spaniel. The owner found the dog dead in the yard in the morning. The lungs were discolored dark red and the tracheal lumen was filled with froth. Microscopic examination of the heart revealed multifocal areas of myofiber atrophy and degeneration typical of cardiomyopathy.

Autoimmune hemolytic anemia was confirmed in a ten-year-old female Shepard that had been diagnosed clinically with the disease. Treatment had included splenectomy and steroids, antibiotics, and a blood transfusion. Necropsy revealed thrombosis of the portal vasculature with associated venous infarction of a forty-inch length of mid jejunum. Intravascular thrombi, hemorrhage, and congestion were also evident in gastric mucosa. There was also focal necrosis of one adrenal gland. Intravascular thrombosis is reported to be sometimes associated with cases of autoimmune hemolytic anemia in dogs.

Severe focal **necrotizing granulomatous encephalitis** was diagnosed in a one-and-a-half-year-old male Shepard mix. The animal had been submitted from the Humane Society and the history was sketchy. Necropsy revealed a yellowish, partially liquified lesion in the midbrain. Microscopic examination of the lesion revealed necrosis and granulomatous infiltration along with fungal elements. Fungal cultures of a swab of the lesion yielded growths of the fungus *Xylohypha bantiana* (formerly *Cladosporium bantianum*) from the lesion.

Brodifacoum intoxication was diagnosed in a four-year-old neutered female Husky that was presented to the submitting veterinarian “depressed, vomiting, anorectic”. The serum alkaline phosphatase and ALT were elevated and there

was a neutrophilic leukocytosis. At necropsy, both thoracic and abdominal cavities were full of unclotted blood. Analysis of liver tissue was positive for brodifacoum (0.013 ppm). Brodifacoum is an anticoagulant compound used in some rodenticide products.

A five-year-old, female Golden retriever died unexpectedly. Gross lesions included bloody fluid on the hairs of the perineum. The entire small intestine and colon were red/black with an edematous mucosa and watery red/black fluid contents. Anaerobic cultures of the small intestines yielded a heavy growth of *Clostridium perfringens* type A (enterotoxigenic). Histologically, there was diffuse hemorrhage in the wall of the small intestine with loss of epithelium from villi. Rod-shaped bacteria colonized the surface of villi. The lesions were consistent with **hemorrhagic gastroenteritis**. This is a poorly understood disease with a rapid onset of clinical signs that may be fatal if not treated early and aggressively. Various causes have been postulated including hypersensitivity reactions but none have been conclusively proven. *Clostridium perfringens* has been implicated in some instances by culture and histologic identification of the organism covering necrotic intestinal villi. This organism is part of the normal intestinal flora of dogs implying that a precipitating factor(s) must be involved in disease causation but the exact nature of those factors is not known.

Canine herpesvirus infection was the cause of death in a six-day-old, male Labrador retriever puppy. The puppy was presented for crying, pain, dyspnea, and hypothermia. It died three hours later. Gross lesions included petechiae and ecchymoses on the surfaces of the kidneys and serosa of the small intestine. The spleen and liver contained scattered pale foci. The lungs were red, wet, and heavy. Microscopic lesions of necrosis and intranuclear inclusion bodies were present in the liver, spleen, kidney, lung, heart, and brain.

Exotics



Coccidioidomycosis was diagnosed in a four-year-old male Lemur. The animal had posterior ataxia and a lumbar vertebral lesion. Serology for *Coccidioides immitis* was positive at 1:32 (IgG). IgM serology was negative. It was also noted in the history that the animal was diabetic and was being given insulin. Necropsy revealed multifocal, coalescing granulomatous foci in both lungs. There was a lumbar vertebral lesion draining purulent material into the sublumbar retroperitoneal area. Spherules of *Coccidioides immitis* were found in all lesions examined.

Staphylococcal septicemia was diagnosed in a guinea pig. The animal had been under treatment for a swollen, infected

front paw lesion that developed a draining tract in the ventral footpad area. *Staphylococcus aureus* was obtained in cultures of lung tissue and a swab of the core of the foot lesion.

A female guinea pig died following a brief history of diarrhea. At necropsy, there was red watery fluid in the intestine. Microscopically, the mucosa of the large intestine contained foci of severe epithelial necrosis and colonization by coccidia consistent with *Eimeria caviae*.

Wildlife



Capture myopathy was diagnosed in an antelope that had been darted for game management reasons. Necropsy revealed pale irregular foci in the myocardium and numerous linear, pale areas in the longissimus dorsi muscle. The liver selenium level was subnormal (0.14 ppm; the reference range for antelope is reported to be 0.25 - 0.90 ppm, similar to the "adequate" range reported for the domestic goat). Selenium deficiency has been associated with the occurrence of capture myopathy.

Chlamydia sp. infection was the cause of death in a yearling, male Red-tailed hawk. The bird was found on the ground and treated but died the following day. Necropsy findings included opacity of the pericardial sac and thickened air sacs covered with yellow exudate. Microscopically there were multiple foci of necrosis in the liver, spleen, pancreas, proventriculus, small intestine, and brain associated with intracellular microorganisms consistent with *Chlamydia* sp. The infection was confirmed by PCR on tissues from the bird.

Comments on Diagnostic Update can be directed to
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Feature Article:

Biosecurity; What has Foot and Mouth Disease Taught Us?

The recent outbreak of Food and Mouth Disease in the United Kingdom has attracted the attention of the media, governmental officials, and livestock owners throughout the Southwest. The United Kingdom has spent a tremendous amount of time and money to control this virus. Reports show that more than 3.1 million animals will be destroyed by the U. K. in an attempt to stop FMD. Other nations have placed an embargo on livestock and animal products from the U. K. to prevent the introduction of this virus. Travel restrictions within the U. K. have seriously affected the tourist industry. Consumer confidence throughout Europe in livestock products has been shaken. The net result of this outbreak may be the permanent loss of 30-40% of the livestock producers in the United Kingdom. Concerned observers in the United States question what allowed this disease to spread with such a devastating impact. The primary cause was delay in recognition of the disease and the inability to halt livestock movement within the country effectively. The U. K. had two major risk factors that simplified the spread of FMD:

- 1) The widespread practice of feeding garbage to swine. Uncooked garbage may have allowed contaminated meat to be fed to pigs. These pigs were in close contact with large numbers of sheep and FMD was able to spread from swine to sheep.
- 2) A very large population of sheep are actively traded and transported around the U. K. Sheep do not usually show overt clinical signs of FMD and the recognition of FMD in sheep was delayed.

The response by the United States Department of Agriculture has been to educate the public and livestock producers about FMD and to increase surveillance to prevent the entry of this virus. With the specter of FMD in the U. K. and Asia, livestock producers in the United States have become more aware of biosecurity. The concern for biosecurity has been reflected in popular press. The May 2001 editions of *Dairy Today*, *Drovers Journal* and *Western Dairy Business* all featured articles on biosecurity. There has been a response by Arizona producers to concerns about biosecurity. Feedlots and dairies are restricting access to livestock, closely monitoring truck movement, and preventing visits by foreign tourists. These are appropriate responses to reduce the possible introduction of diseases such as FMD.

What is meant by the term "biosecurity" and how does an effective biosecurity program go beyond these measures? Biosecurity is simply those management practices that are used by an animal owner to prevent the introduction of infectious agents from entering their animals. The principles of biosecurity can and should be applied by any animal owner. The introduction of Equine Viral Arteritis into non-vaccinated pregnant mares can have a devastating impact on a horse breeding program. Bird owners are at risk to introduce *Chlamydia* (psittacosis) in their home aviary by bringing home an infected bird.

The first phase in the development of an effective biosecurity program is to understand the population at risk. Every population of animals will have different types of risk, depending on age, reproductive status and the intended use for that animal. Once the population has been identified, a full assessment of possible disease problems that target that population can be made. With the population clearly identified, risk analysis can be done. Risk analysis determines areas or factors that lead to the spread of a disease. For example, the income for a cow-calf producer is based on net income on each pound of weaned calf. The introduction of purchased bulls that are carrying trichomoniasis would reduce income by reducing the number of calves born. In this example part of a biosecurity plan would be to only purchase virgin bulls to prevent the introduction of this disease.

Biosecurity plans should be based on a thorough understanding of diseases, how they are transmitted, how they are prevented, the impact of vaccination, and what diagnostic tests are available. Any disease process is the result of the interaction of three components; the animal, the pathogen, and the environment. Biosecurity is the analysis of each of these components and how they interact for each disease process. Each disease will present unique problems to biosecurity issues. An effective biosecurity program examines these problems and responds to them. Plans can best be worked out through a cooperative effort involving animal owners and their veterinarians. Information must be current and biosecurity plans must be reexamined frequently to reflect changes in understanding of diseases or improved diagnostics. The FMD outbreak in the United Kingdom is a graphic example of what can happen when biosecurity programs fail.

The next newsletter will focus on diagnostic testing and how it is used in biosecurity programs.

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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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