



# Newsletter

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## From the Director:

The newly constructed I-10 frontage Road is open to the public, and we now have direct access to and from I-10 (Exit # 255). Our entrance gate is on the east-bound frontage road, one hundred yards (or so) south of the Miracle Mile overpass.

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

*Carlos Reggiardo, Director*

## Ionophore Poisoning in Horses

An episode of monensin poisoning at an Arizona equine training facility (see "Diagnostic Update") warrants a review of this uncommon form of intoxication in horses.

The ionophores are feed additives used as growth-promoting agents in ruminants and as coccidiostats in poultry and other birds. There are several drugs in this group: monensin, lasolocid, laidlomycin, narasin and salinomycin. Monensin is marketed under the trade name Rumensin and is commercially produced in large quantities for addition to premix and pelleted or bulk feeds fed to ruminants. Horses are extremely susceptible to ionophore poisoning. The LD50 for monensin in this species is in the range of 2 to 3 mg/kg whereas cattle have a larger safety zone (LD50 = 20-34 mg/kg). Figures for other species are dogs, 5-8 mg/kg; sheep and goats, 10-12 mg/kg; and poultry, 90-200 mg/kg. Poisoning by the ionophores is usually the result of mixing errors at the mill resulting in higher than acceptable levels in feed for ruminants or inadvertent addition to food intended for monogastric animals. Horses used to work feedlots have been poisoned by consuming correctly mixed cattle feed containing monensin.

The pharmacologic action of the drugs is the inhibition of sodium and potassium ion transport across cell membranes. This leads to mitochondrial failure, decreased ATP production, failure of calcium ion retrieval from the cytosol and, ultimately, myofibrillar hypercontraction and necrosis. The highly energetic tissues of the body such as myocardium and skeletal muscle are primarily affected.

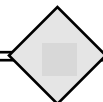
The clinical signs begin 12-24 hours after consumption of an acutely toxic dose but may be delayed for days or weeks in the case of chronic low level intoxication. The signs of acute intoxication in horses may include some or all of the following depending on dose and the individual: colic, intermittent sweating, ataxia, muscle weakness, tachycardia, myoglobinuria, polyuria followed by oliguria, respiratory distress, and recumbency. Again, depending on dose and individual susceptibility, death can occur in less than 24 hours. Animals surviving the acute intoxication and those with chronic intoxication may exhibit signs of progressive congestive heart failure, poor growth, and poor weight gain due to the toxic effects on the myocardium. Sudden deaths in the weeks or even months following intoxication have been reported.

Clinical pathologic changes reflect injury to the myocardium, skeletal muscle and kidneys. Specifically, there are increases in CPK, LDH, SGOT and ALK PHOS (bone isoenzyme). BUN and creatinine may be mildly elevated. Decreases in serum calcium and potassium may be present. In the early stages, the muscle enzyme elevations may be slight and difficult to differentiate from those seen in any recumbent horse. There are no ante-mortem diagnostic tests for detection of ionophores.

Gross necropsy lesions may be absent in animals dying within 24-36 hours. Similarly, histologic muscle lesions are often not present in animals dying with the acute form of the disease. With time, gross lesions of pallor and pale streaking of skeletal and cardiac muscle will be present along with changes associated with heart failure; ascites, hydrothorax, hydropericardium and pulmonary congestion. Microscopic lesions of myofiber degeneration and/or signs of muscle fiber repair will be present at this stage. Ionophores can be detected in stomach contents using thin-layer chromatography. Suspect feeds can be tested by the same method.

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Treatment of ionophore poisoning is primarily supportive.<sup>1</sup> Activated charcoal per os to block absorption is a priority and saline cathartics to encourage elimination may be helpful. Aggressive fluid therapy to correct hypovolemia and support cardiac and renal function are suggested. If clinical laboratory support is readily available, supplemental potassium to correct hypokalemia is indicated. Calcium supplementation is not recommended.

The damage to cardiac muscle is irreparable. Animals surviving intoxication have a guarded prognosis for long term survival and return to fitness.

1. Raisbeck MF. 1992. Feed-associated poisoning. *In*: Current Therapy in Equine Medicine. 3<sup>rd</sup> ed. NE Robinson ed. W.B. Saunders, Philadelphia Pa. pp. 366-377.

*By Gregory A. Bradley DVM, Diplomate ACVP*

## Diagnostic Update

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



### Bovine

**Intestinal perforation** with resultant **peritonitis** was diagnosed in a mature Brown Swiss dairy cow that was submitted for necropsy. The cow had been treated for a bout of severe diarrhea. At necropsy, the abdominal cavity contained a large amount of brownish, malodorous fluid. Fibrinous adhesions were present on the serosae of small intestine, rumen, and greater omentum. Ingesta-like plant material was present within the peritoneal exudate. A group C2 salmonella was isolated in cultures of the gut mucosa adjacent to the perforation. Dissection revealed a perforation of the distal jejunum and microscopic examination of sections from the area of the perforation revealed an enteritis and a peritonitis. Enteritis due to salmonella infection is likely to have caused the diarrhea, and perhaps an ulcer formed which subsequently perforated.

**Salmonellosis** is a major cause of death losses in young Holstein calves raised in confinement for replacement or for beef production, particularly in cases where there is no administration of colostrum. The source of an ongoing salmonella infection was investigated in a "calf ranch" which had sustained heavy losses in calves purchased locally from dairies in central Arizona. Most of the calves had no colostrum immunity. Fecal cultures demonstrated that none of the calves were carriers of the infection on arrival, but were exposed to the infection soon after arrival, with 30% shedding salmonella in the feces at six days post-arrival. After culturing a large number of

environmental samples, which included calf hutches, flies, milk replacers and milk replacer mixing equipment, it was concluded that environmental contamination was the main source of infection. Flies (trapped in the calf hutches and in the area of milk replacer mixing) and fecal-soiled calf hutches appeared to be the most important sources. Successful fly control and strict environmental hygiene are, most likely, key factors in the prevention of the disease.

**BVD** virus infection can also be a major source of morbidity and mortality in "calf ranches". An explosive outbreak of the hemorrhagic form of BVD virus affecting young calves just over four weeks of age was recently diagnosed in central Arizona. Over 400 death losses have been recorded so far, and possible sources of the infection are being investigated.



### Equine

Eight horses at a training facility died within a 36 hour period following acute onset of ataxia, paralysis and tachycardia. One horse exhibited signs of colic and sweating. Supportive therapy including fluids and antibiotics was administered but all horses died; usually within 12-24 hours of onset of the signs. Antemortem blood chemistries on two animals had mild to moderate elevations in AST, LDH, CPK and total bilirubin. The horses were being fed only alfalfa hay. Water was from a well that was also used for the household. Gross necropsy examination of four horses revealed blood tinged foam accumulated around the nostrils with smaller amounts in the trachea of some. No specific microscopic lesions were present. The myocardium and skeletal muscles were all within normal limits. Gas chromatography of stomach contents identified **monensin** in three of the four horses. The source of the monensin had not been determined at the time of this writing.

**Envenomation due to snakebite** was diagnosed in a 5-month-old quarterhorse filly that died after being bitten by a rattlesnake the previous day. The referring veterinarian thought the offending snake was a Mojave rattlesnake. The filly developed "muscle spasms" and seemed to be "photosensitive." She improved after receiving treatment which included two vials of antivenin but then died unexpectedly. At necropsy, there was prominent swelling of the upper lip and nose that extended about one third the way up the muzzle. Fang marks were located on the inside of the upper lip on the buccal mucosa. The pharyngeal, submandibular, and peritracheal lymph nodes were enlarged and hemorrhagic. Microscopic examination of sections of the muzzle lesion revealed severe edema, hemorrhage, and necrosis along with erythrocytolysis. Necrosis of blood vessel walls

was occasionally seen. The lesions were compatible with crotalid envenomation

A 13-year-old, half Arabian mare was euthanized following a three day history of colic. Gastric reflux was present on initial exam but not subsequently. Forty-eight hours after presentation there was pyrexia (104.4°), a pulse of 54 bpm and a respiratory rate of 36/min. An abdominal tap contained 8000 WBC's/mm<sup>3</sup>, and protein levels of 3.2 g/dl. Treatment with ceftiofur resulted in a normal temperature within 24 hours. The mare continued to be more painful and was euthanized. **Anterior enteritis** was diagnosed at necropsy.



## Small Ruminants

**Bluetongue** was diagnosed in two sheep flocks, both from Pinal county. Several animals in each herd were affected. Clinical signs included pyrexia, hyperpnea, serous nasal discharge and intermandibular edema. Necropsy lesions were those of petechial to ecchymotic hemorrhages in the mucosa of the rumen, urinary bladder and root of the pulmonary artery. Edema of the intermandibular subcutaneous tissues and of the mediastinum were also found. The virus was isolated from the spleen of animals from both flocks. Both isolates were serotype 11.

**Hepatic lipidosis** was diagnosed in tissues collected by a veterinarian during a field necropsy of two commercial sheep which were both ewes. A urine sample from one of the animals was positive for ketones. The findings are compatible with **pregnancy toxemia (ketosis)** which occurs during the last 2 months of gestation in ewes on a declining plane of nutrition. Other stressors such as weather extremes and parasitism may be contributory.

**Coccidiosis** was diagnosed in a group of fourteen 40-day-old Suffolk-cross lambs. Eight of the lambs exhibited a severe diarrhea of sudden onset, two dying before they could be treated. Necropsy of one of the lambs demonstrated a severe enteritis with a heavy coccidial infestation. *Rotavirus* were also observed in the intestinal contents. The lamb was both selenium (liver 0.087 ppm) and vitamin E (serum 0.48 µg/ml) deficient, factors believed to have been significant predisposing factors for the explosive onset of the coccidial infection.



## Avian

**Coccidiosis** was diagnosed in two six-week-old Bantam chickens submitted to the AzVDL by a veterinarian in Vermont. Illness had become apparent about four days prior to death and a number of birds died. At necropsy, the paired ceca were hemorrhagic and contained hemorrhagic

cores of necrotic, sloughed, tissue debris. Numerous coccidial intermediate stages were present in section of the ceca and oocysts were numerous in luminal areas.

**Salmonellosis** was diagnosed in one of two budgies submitted for necropsy from a pet store. At necropsy, there was atrophy of the pectoral musculature and foci of yellowish discoloration were present in both lungs. Microscopic examination revealed the discolored lung areas to be abscesses. Adjacent lung tissue was pneumonic, and there was an air sacculitis. Heavy growths of a group B salmonella were isolated in cultures of lung and small intestine.

Septicemic **colibacillosis** was diagnosed in a flock of pigeons experiencing heavy early squab mortality. Affected birds were very young (under a week of age) and often had omphalitis. *E. coli* was isolated from the dead squabs and was also heavily shed in the feces of three of four clinically healthy adults. Good sanitation and avoidance of environmental stress are the only practical preventative measures to reduce the incidence of colibacillosis in pigeons.

**Impaction of the proventriculus and ventriculus** with rocks, alfalfa stems and plastic carpet fibers was observed in four emaciated four to six-week-old ostriches from a group with heavy mortality. The birds were offered a complete, pelleted feed, but were more attracted by chopped alfalfa, fibers from an indoor-outdoor carpet in the pens, and by rocks. This is a common observation in young ostriches, particularly when the feeding of a pelleted feed is delayed for a few days after birth or when distracting elements such as excessive hay, rocks, a carpet (as in this case) are present in the pens where hatchlings are raised.

A severe **pulmonary hemorrhage** caused the death of a four-year-old Moluccan cockatoo, after an illness of a few hours duration. The cause of the hemorrhage was a *Pseudomonas aeruginosa* septicemia which produced a generalized, severe necrotizing vasculitis with heavy bacterial colonization of the wall of the affected vessels. Such an aggressive *Pseudomonas* infection is usually precipitated by stress or immunosuppression. In this case, there was a concurrent chlamydial infection, most likely immunosuppressive.

**Egg peritonitis** was diagnosed in a 44-week-old hybrid research chicken. This diagnosis is reported more frequently in production type hens as they age.



## Canine

**Hemangiosarcoma** of the right atrium of heart with multiple implants to epicardium and metastases to the

lungs was diagnosed in a seven-year-old, neutered male Golden Retriever that died after being anorectic and slightly depressed for two days. When presented to the referring veterinarian, the dog was noted to be "tachypneic" and had a tachycardia and pale mucous membranes. The dog was weak and appeared to have some dropped beats in his "pulse rate". A leukocytosis (31,000) was also noted. At necropsy, the abdominal cavity was full of serous ascitic fluid, and the pericardial sac was filled with unclotted blood.

Severe **intra-abdominal hemorrhage** from a **subcapsular hematoma of the liver** was diagnosed in a 13-year-old neutered Shepherd mix canine that died after being "presented in shock" to the referring veterinarian. Blood from an abdominal tap had a PCV of 48%. Peripheral blood PCV was 16%. At necropsy, the abdomen was full of unclotted blood, and the liver was very friable and light bronze in color. A circular separation of the capsule from underlying parenchyma was evident on the concave surface of the right medial lobe. Hemorrhage was present under the affected area of capsule, which was thin and had ruptured. Microscopic examination revealed a cholangiohepatitis and diffuse disruption of hepatic cords with individualization of many hepatic cells. There was no evidence of trauma. It was assumed that the hematoma occurred as a spontaneous event as a result of capsular separation from the underlying, abnormal parenchyma.

A **splenic hemangiosarcoma** with multiple implants to adjacent mesentery, omentum, and diaphragm was diagnosed in a nine-year-old neutered female Labrador, which was presented to the referring veterinarian with a history of lethargy of two days duration. The PCV was noted to be "low." At necropsy the abdominal cavity was full of unclotted blood.

Canine **parvovirus** infection was diagnosed in two Greyhound puppies. The pups were among 27 that were sick in a group of 48. The lesions were typical and parvovirus particles were seen in preparations of gut contents examined by electron microscopy.

**Group G Streptococci** (*Streptococcus canis*) were isolated in March of this year from a case of neonatal septicemia in a Great Dane breeding colony. This was the third litter lost to abortion or neonatal deaths in as many weeks (AzVDL Newsletter, June 1997). Sometime later, pharyngeal and vaginal swabs were cultured from three of the bitches in the colony, one of which had recently lost her litter. Group G streptococcus was isolated from one of the vaginal swabs. It had been collected from the bitch that had aborted. Additional neonatal infections were observed during the summer. The attending veterinarian collected a semen sample from the sire that had bred all the bitches in the colony. Large numbers of group C

streptococci were isolated from the semen. Group G streptococcus causes the majority of streptococcal infections in puppies and kittens. The vagina of the bitch or queen is usually the source of infection for the neonate, but little is known about how the infection spreads in a population. The present case emphasizes the potential significance of venereal transmission in the spread of the disease.



## Feline

A 12-week-old, male, domestic shorthair kitten was presented with acute onset of hypothermia and hypovolemia. Laboratory results included neutropenia, hypoglycemia and hypoalbuminemia. Gross necropsy revealed severe, hemorrhagic enteritis. Microscopic examination revealed necrotizing enteritis characteristic of **feline panleukopenia** virus infection.

**Histoplasmosis** was diagnosed in a one-year-old Siamese cross neutered male. The cat had a history of swollen limbs and painful walking, and an elevated white cell count. X-rays revealed osteolysis of the left distal radius and of the right distal ulna. Histologic examination of the affected bones revealed extensive granulomatous inflammation of the lytic areas with numerous fungal organisms within macrophages. *Histoplasma capsulatum* was isolated in culture from both bones. Although histoplasmosis is endemic in the Ohio-Mississippi Valley, sporadic cases of the infection can be observed in many other regions of the country including Arizona. This cat was raised on a tree-shaded property in Yuma where pigeons had been raised in the past, most likely creating a microenvironment favorable for the growth of the organism.

**Poisoning by Aldicarb** (Temik ®) was diagnosed in a cat from central Arizona. The owner reported losing three cats and a dog in eight weeks. They were found dead or dying after a short illness. Brain acetylcholinesterase activity was only slightly depressed in this animal (3.98  $\mu\text{mol}/\text{min}/\text{g}$ ), but Aldicarb poisoning was nonetheless suspected because of the finding of a few black granules in the stomach content, similar to Temik ® granules. The presence of Aldicarb was confirmed by chemical analysis of the stomach content. Brain acetylcholinesterase activity can be a poor diagnostic test in poisoning by Aldicarb and other carbamates because the inhibition of the enzyme by these agents is easily reversible. In this case, the enzyme activity increased to 6.85  $\mu\text{mol}/\text{min}/\text{g}$  (well within "normal" limits) after a 12 hour incubation of the brain tissue at 37° C, a typical finding in carbamate poisonings.



## Wildlife

**Amyloidosis** was diagnosed in three Masked Bobwhite quail that had been “sick for about a week.” Amyloid deposits were most prominent in liver but were also present in spleen. Multiorgan chronic inflammatory lesions were present in all birds, but an etiologic agent responsible for these could not be identified, as the preservation of the specimens was poor. Amyloidosis in birds is usually of the secondary type, occurring in association with chronic, inflammation.

**Herpesvirus** infection was the cause of death in a sub-adult Red Tailed hawk that died suddenly at a rehab facility. At necropsy, there were raised, tan, plaques on the oral and pharyngeal mucosa. Microscopically, there was severe fibrinonecrotic pharyngitis, stomatitis, esophagitis, tracheitis and enteritis. Epithelial cells at the margins of the lesions contained intranuclear inclusion bodies.



## Exotics

Enteritis caused by *E. coli*, and encephalitis and nephritis caused by *Encephalitozoon cuniculi* was the diagnosis in an eight-week-old Dwarf rabbit that died after a short illness characterized by “respiratory signs” and diarrhea.

Septicemia due to a **group G streptococcal infection** was diagnosed in a 17-year-old female Giant anteater weighing 66 pounds. Necropsy revealed a generalized lymphadenopathy. Streptococci were isolated along with other, mixed flora in cultures of lung, axillary lymph node, spleen, bone marrow, pericardial sac, heart blood, and gut.

**Clostridial gastritis** was diagnosed in a one-year-old pet sugarglider, which died after a brief period of illness characterized by the attending veterinarian as an acute sepsis. There was an acute hemorrhagic gastritis with extensive hemorrhages of the wall of the stomach, thrombosis, edema and areas of necrosis. Large numbers of clostridial organism were observed in the affected areas. Although no anaerobic cultures were attempted, roughly half of the flora present were identified as *Clostridium septicum* by FA staining of deparaffinized sections of the stomach. Clostridial gastritis (by *Cl. septicum* alone or in combination with other clostridia) are sometimes seen in neonatal ruminants precipitated by diet and/or environmental problems. No probable precipitating factors were identified in this case.

*compiled by T. H. Noon, Greg Bradley, Carlos Reggiardo*

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

## Vesicular Stomatitis Update

The 1997 Vesicular Stomatitis outbreak appears to have come to an end. There have been no new cases reported since the week of November ninth. Following is the final “tally” of positive premises records: 272 in Colorado, 68 in New Mexico, 38 in Utah, and 2 in Arizona.

### 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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