

Winter 2000

FROM THE ASSISTANT DIRECTOR

W.G. Van Alstine, DVM, PhD

Remember Y2K? Seems like a long time ago, doesn't it? Since our last newsletter, we were able to continue to utilize enough of our existing computer system that ADDL activities were not hindered. We will rely on the current computer system until July 2000, when we anticipate that the new system will be online. You will see changes in reports and billings and we trust the changes will be positive. If you have any suggestions for improving our new report format, please give us a call.

Springtime means 4-H Fair time is near. As caseload in the serology lab typically triples from mid-May to mid-July, we encourage you to submit samples as early as possible. If you have questions regarding serology turnaround time during fair season, please feel free to call our serology lab. Guidelines for submitting fair serum samples appear; in this issue of Diagnostic Forum.

Dr. Kanitz, ADDL virologist, will host Dr. M.M.A. Samie in his laboratory this summer. Dr. Samie comes from the Suez Canal University in Ismalia, Egypt. He will be here to learn various diagnostic virology and molecular biology techniques in Dr. Kanitz's laboratory.

This winter we have had six veterinarians take advantage of our practitioner sabbatical. They found their time at ADDL both enjoyable and informative. Visiting veterinarians always enrich the teaching environment of the ADDL. If you are interested in a practitioner sabbatical at ADDL, please contact me or Dr. Thacker. You are always welcome.

As always, we welcome suggestions from you. Our wish is that you find this newsletter informative and useful.

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Hepatic Abscesses in Feedlot Cattle

Liver abscesses negatively affect feedlot cattle performance both in the feedyard and on the rail, causing financial loss to cattle feeders and packers through decreased feed intake, average daily gain, feed efficiency, dressing percentage and liver condemnation.

Causative agents of this condition include *Fusobacterium necrophorum*, a gram negative obligate anaerobe which is the most commonly isolated bacterium (up to 100%), and *Arcanobacter (Actinomyces) pyogenes*, a gram positive facultative anaerobe which is the second most commonly isolated (35%) pathogen. Other bacteria which may be cultured include *Staphylococcus spp.*, *Streptococcus spp.*, and *Bacteroides spp.*

The two subspecies of *F. necrophorum* are subsp. *necrophorum* (biotype A) and subsp. *funduliforme* (biotype B). Of these, the subspecies *necrophorum* is the most pathogenic. The two virulence factors of major importance are leukotoxin and endotoxic lipopolysaccharide which help to prevent phagocytosis of *F. necrophorum*.

F. necrophorum and *A. pyogenes* are part of the normal bacterial flora of the rumen. Damage to the wall of the rumen secondary to rumen acidosis leads to bacterial colonization. Subsequently, the bacteria enter the bloodstream via the portal circulation, which seeds bacteria throughout the liver. The bacteria readily proliferate forming abscesses. Liver abscesses begin as areas of coagulative necrosis that develop into encapsulated abscesses over time. Eventually, these areas may heal by forming a fibrous scar.

Cattle with liver abscesses usually do not show clinical signs; however, some cattle may be febrile, anorexic, depressed and experience weight loss. Cattle with ruptured liver abscesses may be severely depressed, anorexic, and febrile due to peritonitis. Sudden death may occur due to anaphylactic shock if an abscess ruptures and releases a large amount of purulent material into the

bloodstream. In these cases, the lungs will appear markedly congested and edematous at necropsy. In some instances, abscesses may involve the posterior vena cava, causing phlebitis and, eventually, thrombosis. Clinical signs of caudal vena caval thrombosis include chronic diarrhea, emaciation, mild ascites, and distended subcutaneous abdominal veins. Clinical signs of pulmonary thromboemboli include epistaxis and/or hemoptysis, coughing, dyspnea, tachypnea, anemia, and melena.

Diagnostic techniques available to diagnose liver abscesses include laboratory changes and ultrasonography. Common laboratory changes include increased gamma glutamyltransferase (GGT), sorbitol dehydrogenase (SDH), globulin, bilirubin, fibrinogen, and leukocyte counts, as well as hypoalbuminemia and significantly decreased sulfobromophthalein clearance. Ultrasonography is a useful diagnostic tool, although it may be unable to detect abscesses in some areas of the liver. Ultrasonography detects areas where the hepatic parenchyma has coagulative necrosis and therefore has a necrotic fluid center. These changes may be visible as soon as three days after experimental infection.

Histologically, liver abscesses have a necrotic center containing leukocytes, hepatocytes and cellular debris. The area surrounding the necrotic center contains macrophages and multinucleated giant cells. The next layer contains plasma cells, degenerating hepatocytes, immature fibroblasts, neutrophils, macrophages, and immature collagen strands. The capsule consists of fibrous connective tissue. At necropsy, the liver may contain a single abscess or numerous abscesses that range in size from pinpoint to over 15 cm in diameter. Peritonitis may be present if an abscess has ruptured into the abdominal cavity. The liver may be adhered to the diaphragm with fibrous connective tissue.

In a study conducted by Lechtenberg et. al, *Fusobacterium necrophorum* was found to be generally susceptible to penicillins, tetracyclines (chlortetracycline and oxytetracycline), lincosamides (clindamycin and lincomycin), and macrolides (tylosin

and erythromycin), and was resistant to aminoglycosides (kanamycin, neomycin, gentamicin, and streptomycin), ionophores (except narasin), and peptides (avoparcin, polymyxin, and thiopeptin). However, long term antibiotic therapy using approved antimicrobials is of questionable benefit.

Fortunately, feeding diets supplemented with antimicrobials can reduce the incidence of naturally occurring liver abscess. Oxytetracycline, chlortetracycline, bacitracin, methylene disalicylate, virginiamycin, and tylosin are antimicrobial feed additives labeled for the prevention of liver abscesses in feedlot cattle. Another preventative measure may include gradually increasing the amount of concentrate in the ration to reduce acidosis. Research by Saginala et al demonstrated that a leukotoxoid vaccine reduced the incidence of liver abscesses; however, this vaccine and other similar vaccines are not available commercially.

-by Jennifer Fairchild, Class of 2000

-edited by Randy White, DVM, PhD

Canine Ehrlichiosis

Ehrlichiosis, also known as “Tropical Canine Pancytopenia” or “Canine Rickettsiosis”, is a tick-borne disease caused by obligate intracellular bacteria of the genus *Ehrlichia* of the family *rickettsiaceae*. Dogs can become naturally infected with several species of *Ehrlichia* including *E. canis*, *E. equi*, *E. risticii*, *E. platys*, and *E. ewingii*. *E. canis* is the most common and causes the most severe clinical disease. Dogs seropositive for *E. canis* have been identified throughout most of the U.S., but most cases occur in areas with an increased concentration of *Rhipicephalus sanguineus*, the brown dog tick, such as the Southwest and the Gulf coast. Canine ehrlichiosis is principally of importance in Africa, Asia, and India.

Ehrlichia canis was discovered in Algeria in 1935. The first case in the United States was reported in 1963. It was not until about 1968-1970, during the Vietnam war, when the full pathologic potential of *E. canis* was first recognized. A severe epizootic episode

of Ehrlichiosis occurred among U.S. military dogs resulting in hundreds of cases of morbidity and mortality.

Transmission: The arthropod vector of *E. canis* is *Rhipicephalus sanguineus* and transmission is transstadial. Ticks acquire *E. canis* by feeding, as either larvae or nymphs, on infected dogs and transmit the infection as nymphs or adults. The organism can also be transmitted by blood transfusions.

Pathogenesis: The life cycle of *Ehrlichia canis* is not completely understood. There are three intracellular forms. Initial bodies are small spherical structures (1-2 microns) which are believed to develop into larger multiple units known as morulae. The morula is thought to dissociate into small granules called elementary bodies.

Once the organism has been transmitted, there are three clinical phases of Ehrlichiosis: acute, subclinical, and chronic. The acute phase begins after an incubation period of 8-20 days and lasts 2-4 weeks, during which time the organisms multiply in reticuloendothelial cells, lymphocytes, and monocytes. Infected mononuclear cells marginate in the small vessels or migrate into endothelial tissues and vasculitis ensues. Immunologic and inflammatory mechanisms are involved with increased platelet consumption. Platelet-associated IgG and antibodies that recognize platelet proteins in dogs with *E. canis* infection may play a role in the thrombocytopenia. In addition, platelet migration-inhibition factor (PMIF) has been found to exist in dogs with Ehrlichiosis and its level is related inversely to the platelet count. The acute phase usually resolves spontaneously. The subclinical phase can persist for years. Immunocompetent dogs may be able to eliminate *E. canis*; however, the organism persists intracellularly in most dogs, leading to the chronic phase. This phase may be mild to severe. In the mild form, there is vague illness and weight loss. Bone marrow hypoplasia leading to pancytopenia occurs in the severe chronic form. The severity of the disease depends on the dog's age (i.e., young dogs are more susceptible), strain of the organism, the presence of concurrent

disease, and breed (e.g., German shepherds) are more likely to be infected.

Clinical signs: Clinical findings in dogs with Ehrlichiosis vary with the phase of the infection. During the acute phase, nonspecific signs such as fever, oculonasal discharge, anorexia, weight loss, dyspnea, and lymphadenopathy may occur. Clinical signs commonly seen during the chronic phase include depression, weight loss, pale mucous membranes, abdominal pain, hemorrhage, lymphadenopathy, splenomegaly, dyspnea, increased lung sounds, hepatomegaly, arrhythmias, pulse deficits, polyuria, polydipsia, and stiff, swollen, painful joints. Ocular abnormalities such as perivascular retinitis, hyphemia, retinal detachment, anterior or posterior uveitis, and corneal edema may occur. Abnormalities of the CNS, including meningeal pain, paresis, cranial nerve deficits, and seizures have been reported.

Diagnosis: Ehrlichiosis is an important differential diagnosis for pancytopenia. Hematological changes for infections caused by *E. canis* generally include nonregenerative anemia, thrombocytopenia, and leukopenia. Serum chemistry abnormalities include hyperproteinemia with hyperglobulinemia, and elevated alanine aminotransferase and alkaline phosphatase. Other clinicopathologic findings include proteinuria, hematuria, and prolonged bleeding time. CSF analysis in dogs with CNS signs shows an increased protein level and predominant lymphocytic pleocytosis.

A definitive diagnosis of Ehrlichiosis can be made by demonstration of morulae in leukocytes from blood smears or tissue aspirates from spleen, lung, or lymph node.; however, finding morulae on smears is often difficult and time-consuming. A diagnosis of Ehrlichiosis is usually based on positive results of the indirect FA test on serum. This test detects serum antibodies as early as 7 days post-infection. Serum antibody levels in untreated dogs peak at 80 days after infection. Most laboratories measure an IgG titer. A titer of 20 or greater is generally considered to be evidence of infection and/or exposure. An ELISA test has also been developed to detect antibodies and

circulating antigen in dogs with *E. canis*. Cross-reactivity occurs between several of the *Ehrlichia* species. For academic interest, Western immunoblotting and PCR may be used to characterize different organisms.

Pathologic findings: Ehrlichiosis is not characterized by specific pathologic findings, but gross lesions may include petechial and ecchymotic hemorrhages on the serosal surfaces of the gastrointestinal and urogenital tracts and kidneys, edematous or hemorrhagic enlargement of most lymph nodes, and edema of the limbs. Dogs are generally emaciated at death and may have signs of epistaxis. Splenomegaly and/or hepatomegaly may be observed.

Histopathologic findings include widespread perivascular accumulations of lymphoreticular and plasma cells, particularly in the meninges, kidneys, liver and lymphopoietic tissues. Multiple Kupffer cell hyperplasia and degeneration and acute centrilobular necrosis of the liver may be seen. Lesions of the CNS include hemorrhage and plasma cell accumulations in the meninges and occasionally lymphocytic and plasma cell infiltrations are present in the brain parenchyma. Other microscopic findings may include crescent-shaped perifollicular hemorrhages in the spleen, bone marrow hypoplasia, interstitial pneumonia, and glomerulonephritis.

Ehrlichia organisms are difficult to detect histologically. Ultrastructurally, morulae in blood monocytes are intracytoplasmic inclusions made up of numerous organisms. The organisms are round, ovoid, or elongated and are surrounded by a double membrane.

-By Jeanine Peters, Class of 2000
University of Georgia

-edited by Evan Janovitz, DVM, PhD



Abortions – Why Have Them Necropsied?

The success rate in identifying causes of abortion by conducting a post mortem on the fetus and placenta is low compared to other diseases. This is particularly true for cattle, sheep and pigs, the species most frequently submitted to laboratories for fetal pathological analysis. A unique diagnostic feature of abortions lies in the intimate relationship existing between dam and fetus, simultaneously involved and affected by the event, often unequally. While the fetus is ultimately affected and prematurely expelled, the triggering cause of abortion in many cases is disease involving the dam and her environment. The diagnostic conundrum presented by abortion requires critical examination of these relationships.

Why examine fetus and placenta?

For owners of affected animals, negative results are of equal importance to positive ones. Clearly it becomes important to know if an aborted calf is affected with IBR or Neospora, or that lambs were aborted because of toxoplasmosis or coxiellosis. An accurate diagnosis allows the practitioner to respond using solid medical information in determining treatment and preventative measures for specific diseases. On the other hand, negative results – despite full pathological and microbiological examination-offer reassurance that several infectious diseases, including important zoonoses, have been partially or completely ruled out.

Examination of the aborted fetus can provide insight into noninfectious diseases affecting the unborn like congenital anomalies incompatible with fetal survival, placental hemorrhage arising from trauma, fetal heart failure caused by dietary selenium deficiency and hemorrhage as a result of sweet clover poisoning. A full term, underdeveloped fetus may reveal maternal problems like inadequate nutrition through the later half of pregnancy, or placental disease. The detailed necropsy supplemented by histology and other tests involving virus isolation or immunohistochemistry help differentiate noninfectious disease from things like congenital exposure to BVD.

Genetic defects in livestock are occasionally expressed as fetal deformities that result in death of the fetus and abortion. A few specific genetic defects may be identified through examination of fetal tissue. Osteopetrosis, arthrogryposis of cattle and “spider-lamb” chondrodysplasia in sheep are in this category. Non-heritable chromosomal defects with malformation of multiple body systems may be diagnosed by karyotyping viable cells collected from fetal tissue.

Autolysis is a major enemy of the pathologist. Fetal submissions are received in various states of autolysis more frequently than any other type of specimen, due in large part to *in utero* decomposition of the dead fetus that may not be expelled for a period of time while maintained at the dam’s body temperature. Despite this reality, pathologists are repeatedly surprised to find characteristic lesions of IBR (liver), Neospora (brain) and other infections when autolyzed tissue is examined histologically.

Even the autolyzed fetus can be a valuable diagnostic tool.

-By Dr. James P. Orr, Pathologist
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the Animal Health Expositor,
Prairie Diagnostic Services,
Saskatoon, Saskatchewan



Equine Polysaccharide Storage Myopathy

Equine polysaccharide storage myopathy (EPSM) is a form of rhabdomyolysis classified as a metabolic disease that results in the accumulation of high muscle glycogen and abnormal polysaccharide in skeletal muscles.

The occurrence has presently been documented in Quarter horses, American Paint Horses, Quarterhorse crosses, warmbloods, draft horses and draft crosses. Presently, EPSM is believed to be

transmitted as an autosomal recessive disorder with mares being more frequently diagnosed than geldings. Geldings may be more commonly affected with non-diagnosed clinical cases.

Horses that are affected generally are referred to as having a calm demeanor and being heavily muscled. Signs often occur 10-20 minutes after light work in 2-4 year olds starting training, but signs can also occur at any stage of life. The episodes may occur once or twice a year to every time the horse is exercised. A common complaint is that horses are exercise intolerant, especially at high speeds. In mild cases, horses show a tucked up abdomen, muscle fasciculations in the flank, and a camped-out stance. If exercise is continued, profuse sweating, front and hindlimb gait asymmetry, and reluctance to move are seen. In severe cases, horses may refuse to move forward, buck and lie down to avoid exercise. When returned to the stall they may show signs of colic, such as rolling or pawing. Rarely, myoglobinuria can be seen. The gluteal, biceps femoris, semitendinosus, and epaxial muscles may be tense and painful when palpated.

Draft breeds tend to show more severe episodes with recumbency often leading to death. In all breeds, chronic cases can show generalized poor muscling or subtle to severe muscle wasting commonly involving the rump and proximal thigh musculature.

EPSM is characterized as a glycogenosis or glycogen storage disease. Affected animals have muscle glycogen levels 1.5-4 times higher than normal animals and abnormal polysaccharide present in skeletal musculature. It is proposed that affected horses have an abnormality of glycogen synthesis regulation that leads to storage of unmetabolized carbohydrate in the muscles. However, further research is needed to determine the exact etiology. The weakness and muscle fiber necrosis evident in EPSM affected animals may be due to an energy "crisis" that results from the inability of the muscle to use the abnormal polysaccharide and result in a catabolic state that leads to the inability to maintain normal muscle fiber size.

The enzymes of creatinine kinase (CK), lactate dehydrogenase (LDH), and aspartate transaminase (AST) are elevated and increases parallel the severity of the episode. Serum enzyme levels can range from 2000 to 200,000 U/L. With typical rhabdomyolysis high CK levels decline to normal within days of resolution, but in cases of EPSM, CK level may remain elevated after the horse is rested and levels can increase due to subsequent subclinical episodes.

Exercise testing is an excellent screening test and can be performed by testing blood enzyme levels after 15 minutes of trotting on a longe line. Usually, horses with EPSM show increases of CK of greater than 1000 U/L 4-6 hours after exercise. Surgical or needle muscle biopsies provide a definitive diagnosis. The preferred sites for surgical biopsy are the semimembranosus or semitendinosus muscles. These sites provide easy access, a high prevalence of abnormal polysaccharide, and a poorly visible scar. The needle biopsy can be taken from the gluteus medius muscle but requires a Bergstrom needle; however, this technique is easy and leaves no scar.

The hallmark of EPSM is PAS-positive inclusions scattered in fast twitch muscle fibers found in horses showing recurrent episodes of rhabdomyolysis or exercise intolerance. Unlike normal glycogen, the majority of the PAS-positive inclusions resist digestion with amylase. Other findings that support EPSM are increased staining of normal glycogen, necrotic fibers, rimmed and subsarcolemmal vacuoles, and centrofascicular atrophy and regeneration of muscle fibers. False negatives can occur and are usually the result of a small biopsy size of less than 200 fibers or the absence of abnormal polysaccharides in young animals since they tend to form in the later stages of glycogenoses. False positives typically result from diagnosing EPSM solely on the basis of increased glycogen without the presence of abnormal polysaccharides. Treatment consists of dietary management and implementation of a defined exercise regime. The goals of dietary therapy are to minimize dietary carbohydrates and

maximize fat intake by providing 20-25% of the dietary caloric requirements from fat. All grain, sweet feed, and molasses should be eliminated from the diet and replaced with high quality forages such as alfalfa hay or grass-alfalfa hay mix diets. Additional calories are generally necessary due to the strict exercise regimen needed to control clinical signs; therefore, fat is added to the diet to provide an alternative energy source. Fat supplements of vegetable oil, powdered animal fat, or corn oil can be used. The recommended 1 lb. of fat/1000 pound horse can be accomplished with 2 cups of oil mixed with alfalfa cubes for palatability. These recommendations must be modified depending on the individual caloric needs of the patient.

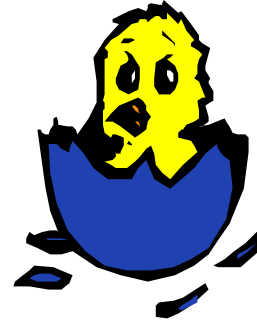
Exercise therapy consists of daily turnout and as little stall rest as possible. The horse should be longed for 15 minutes a day and, if no increases in CK are evident, the workload can be gradually increased. When the horse can be worked for 30 minutes without difficulty, active riding can be initiated.

EPSM horses respond most favorably to both diet and exercise change. Typically, after 3-6 months of therapy, post-exercise serum concentrations of CK are within reference limits. Improvements in muscle function are proposed to be the result of segmental necrosis of fiber segments containing unmetabolized carbohydrates.

The prognosis varies among horses, but dietary and exercise changes almost completely control signs in most EPSM affected animals. The exception occurs in horses of the draft breeds that have become acutely recumbent; 50% do not survive despite aggressive treatment. Currently, affected horses are retired to breeding. Since ESPM may be a heritable condition, this practice should be discouraged.

-By Timothy Galusha, Class 2000

-Edited by Christine Hanika, DVM, PhD



Poult Quality Monitoring

Monitoring day old poult quality is an established practice of companies or individual flock owners who purchase day old poults from outside sources. All seek sources that participate in the National Poultry Improvement Plan, a voluntary monitoring program which originally concentrated on *Salmonella pullorum* and *Salmonella gallinarum*, but also covers *Mycoplasma synoviae*, *Mycoplasma gallisepticum*, and *Mycoplasma meleagridis*. These organisms can be responsible for transovarian disease. Because of the program's success among buyers and sellers, genetic potential of various participating breeds has been enhanced. The Indiana State Poultry Association, in conjunction with ADDL and the Office of the State Veterinarian, put on several "Blood Testing Schools" throughout the state.

In addition to the NPIP standards, individual companies set goals for weekly livability and compare various hatcheries. First week mortality can be the result of breeder flock, hatchery, transportation, grower and management stresses placed upon the individual bird. For example, egg size, time in hatchery and transportation time can determine the state of hydration as the poult arrives on the farm. Consequently, the service man and grower have been asked to pull a number of the worst appearing poults for laboratory diagnostic work. Tests include visual observations of beaks, weights, wing and body feathering, navels, cloaca, legs, toes, and necks for signs of trauma. Internal monitoring include air sac cultures for fungi, yolk and bile cultures for bacteria, as well as serological tests for MG,

MS, and MM. There are many times that routine observations from the laboratory do not correlate to the first week mortality; however, when early infections are apparent on the day one observations, corrective action is more easily accomplished. In some respects, the poult day one monitoring anticipates possible problems in the future.

-By Tom Bryan, DVM, MS, Heeke ADDL

Severe Combined Immunodeficiency (SCID) in Arabian Foals

Severe combined immunodeficiency (SCID) is an important genetic condition that results in the death of a significant number of Arabian foals. The condition is inherited as an autosomal recessive trait and was first identified in 1973. In 1997 it was estimated that 2.5% of Arabian foals died from this condition.

The pathogenesis of SCID is based on a mutation in the allele encoding for a DNA-dependent protein kinase (DNA-PK) that is needed for lymphocyte V(D)J recombination. When both alleles are mutated, the condition develops. V(D)J recombination is essential for expression of antigen receptors on B and T lymphocytes. Without these receptors, B and T lymphocytes do not differentiate and lymphoid tissue fails to develop so severe immunodeficiency results.

SCID foals appear normal at birth and, if they receive colostrum (with maternal antibodies), signs related to their immunodeficiency develop later, when serum antibody levels wane. Clinical signs are related to the development of opportunistic infections, usually of the respiratory tract. These include adenovirus, *Pneumocystis carinii*, and various bacteria such as *Streptococcus*. SCID foals inevitably die before five months of age.

Immunologic characteristics of SCID foals include an absence of B and T lymphocytes, and a lack of serum immunoglobulins. Natural killer cells function normally. On gross examination, severe hypoplasia of the thymus and lymphoid tissue is the distinguishing feature.

The diagnosis of SCID is presumptive when the following antemortem criteria are present: (1) lymphopenia with less than 1000 lymphocytes per μl , (2) lack of serum IgM, and (3) hypoplasia of lymphoid tissue. Postmortem findings of lymphoid tissue hypoplasia and unusual opportunistic infections support this presumptive diagnosis. Definitive antemortem diagnosis

is based on a DNA molecular technique, polymerase chain reaction (PCR) (Vet-Gen

Laboratories, Ann Arbor, Michigan) that can identify the mutant allele of the DNA-PK gene. This test utilizes DNA extracted from leukocytes (from whole blood) or other cells (e.g., obtained from swabs of buccal mucosa). It is considered to be 99.9% accurate and also detects heterozygous carriers of the SCID trait. Identification of carriers is important so that their mating can be prevented.

-By Barbara Atkinson, Class of 2000

-Edited by Evan Janovitz, DVM, PhD

ADDL SCHEDULE

Purdue ADDL and Heeke ADDL (formerly SIPAC) will be closed on the following holidays in Spring/Summer 2000.

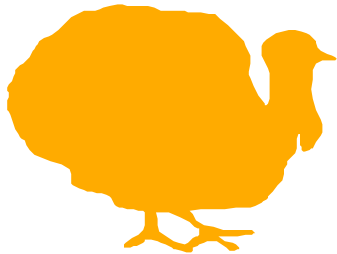
Monday, May 29, Memorial Day
Tuesday, July 4, Independence Day



Please plan accordingly

CORRECTION

The section discussing fluid therapy for gastric dilatation-volvulus in dogs in the Fall, 1999 issue of Diagnostic Forum should have read "... intravenous therapy with 7% NaCl (5ml/kg) in 6% Dextran 70 (HS/D70) initially followed by 0.9% NaCl is superior to 0.9% NaCl alone." Resource is Section 7/Gastrointestinal Disorders, Chapter 4/Disease of the Stomach, pp. 675 of Saunders Manual of Small Animal Practice, edited by Stephen J. Birchard and Robert G. Sherding, W.B. Saunders Company, 1994.



Turkey Coronavirus ELISA

The West Lafayette ADDL is now offering an ELISA test for Turkey Coronavirus (TCV). This test has been validated to have the same accuracy as the IFA test offered at the Heeke ADDL (formerly SIPAC) in Dubois, Indiana. The cost will be \$1.50 per sample. A minimum of six samples is required per flock for testing; however, to better assess the flock coronavirus status, a large number of samples is encouraged. The turnaround time generally should be 24 hours after receipt of the serum samples. Please be sure to identify the owner's name, address, phone and fax numbers, as well as flock identification. Samples should be submitted on an ADDL accession form to the Animal Disease Diagnostic Lab, 1175 ADDL, Purdue University, W. Lafayette IN 47907 by next day air (cold packs are sufficient refrigeration). If you have any questions concerning TCV serology, please contact Dr. T.L. Lin or Ms. Donna Schrader at 765-494-7440.

Other tests for TCV available at ADDL, West Lafayette, include virus isolation, electron microscopy and IFA. The costs and guidelines for the submission of these tests remain the same. Please call ADDL accession desk (765-494-7440) for more information.

- by C.C. Wu, DVM, PhD

Guidelines for Submitting Serology Samples for Fairs and Shows

The season for fairs and shows is fast approaching and plans should be made soon for performing inspections and tests. The following information is a general review for submitting samples to ADDL/Serology.

1. Samples hand-carried to ADDL must be sealed using the veterinarian's label or tape bearing the veterinarian's signature.
2. All regulatory charts must include submitting veterinarian's signature and complete animal identification.
3. ADDL Form 3 (Request for Serological Tests) must be completed and attached to all regulatory test charts. ADDL will run only those tests requested on this form.
4. A health certificate is the only necessary test record for 4-H exhibition. Do not submit duplicate test charts.
5. Use of BD-Vacutainer or Monoject tubes is preferred. Venoject, Jelco or EDTA-treated tubes will not be accepted.
6. Each tube must be identified with a tube number, additional id is desirable.
7. Tube numbers and numbers on chart must match and be in consecutive order and be packaged in consecutive order.
8. Clear serum is preferable to whole blood.
9. Tests for Pseudorabies will be performed daily (M-F). Turnaround time will be 3-6 days (slightly longer at peak testing periods).
10. Brucellosis tests are performed daily (M-F). Turnaround time is 2-4 days.
11. Swine samples for both brucellosis and pseudorabies will be tested for brucellosis first followed by Pseudorabies. Turnaround time will be 4-7 days.
12. ADDL will NOT release results to owners.

13. If you have questions, please contact ADDL/Serology at 765-494-7451 before submitting samples.

-By Charles Kanitz, DVM, PhD

-Karen Crane, Lab Supervisor