## ₹ VIRGINIA VETERINARY NOTES

VIRGINIA-MARYLAND REGIONAL COLLEGE OF VETERINARY MEDICINE

NOV 13 1990

November-December 1990																No. 4	8
WHAT'S INSIDE!	The state of the s				The state of		Management										
CANINE LYME DISEASE: HOW REAL THE T	HREA'	т.							•			•				Page	2
A DROP OF VERMOUTH IN A 16,000 GALLO	N MA	RTI	NI					•		•	•				•	Page	3
THOUGHT FOR THE MONTH							٠									Page	3
VON WILLEBRAND FACTOR			•													Page	4
ROUNDWORMS IN KIDS AND PETS				•												Page	5
ANTIBIOTICS OFFER PROMISE FOR UTERIN	E IN	FEC	rio	NS						•						Page	6
RABIES VACCINE TRIAL										•	•					Page	6
CONTINUING EDUCATION OPPORTUNITIES.														•		Page	7
VIRGINIA ASSOCIATION WINTER MEETING									•							Page	7
EASTERN STATES CONFERENCE																Page	7
FOURTH ANNUAL LARGE ANIMAL MEDICINE	SHUB,	г с	TIR	SF	TN	C	OTJ	IME	RTA		SC	,				Paga	7

Kent C. Roberts, DVM Extension Veterinarian

Virginia Tech and Virginia State

Virginia's Land-grant Universities

#### CANINE LYME DISEASE: HOW REAL THE THREAT

Although a critically important human disease, Lyme borreliosis may not be a disease of dogs or cats at all, but only an immunologic response to an organism that remains of questionable pathogenicity of these species. Although this suggestion may represent heresy to veterinarians who routinely diagnose Lyme disease, I would like to offer several form of indirect evidence that provide the basis for this concern. Borrelia burgdorferi has been recovered from several species of wild and domestic animals, but despite extensive research efforts, no one has yet successfully induced the disease in dogs by experimental administration of the organism. In fact, with the exception of skin lesions resembling erythema chronicum migrans in guinea pigs and rabbits, polyarthritis in infant rats, and multisystemic disease in the severe combined immunodeficiency mouse, experimental inoculation B. burgdorferi has not resulted in apparent illness in an immunocompetent animal host.

In endemic regions, the organism is nearly ubiquitous in ticks, canine exposure is extensive, and many seropositive dogs remain asymptomatic, despite demonstration of spirochetemia. In contrast to human borreliosis, immunoglobulin M titers to B. <u>burgdorferi</u> remain positive for several months in asymptomatic dogs, and antibody titers fail to decrease following "appropriate" antibiotic treatment in symptomatic dogs. Immunoblot analysis of the IgG response to B. <u>burgdorferi</u> indicates that naturally exposed dogs from different geographic regions experience exposure to similar organisms, but there is no difference in the immunoblot pattern between symptomatic and asymptomatic dogs with high B. <u>burgdorferi</u> antibody titers. Serologic support for a pathogenic role for the organism in dogs is lacking.

Although the initial case report of canine Lyme disease was convincing, two of Koch's postulates were not satisfied, i.e., induction of disease in a susceptible dog with the cultured organism and reisolation of the organism from the experimentally infected animal. Much of the literature that has followed this initial report is based upon a serologic diagnosis of Lyme disease in dogs from endemic regions where seroprevalence may reach 80%. Since seropositivity, rather than seroconversion was used as the criterion for diagnosis, the relevance of these cases to the clinical spectrum of canine or feline Lyme disease is highly suspect. Even more recent reports, in which the organism was isolated or demonstrated by monoclonal antibody technique in tissues, fail to demonstrate an absolute cause and effect relationship. This concern would not be quite so bothersome were it not for the fact the Dr. Burgess (in the reference cited above) was able to isolate the organism from blood in 8/111 healthy dogs from USDA licensed vendors in Wisconsin. Considering the historical difficulty in growing the organism in modified Kelly's medium, it is possible that even more dogs may have been spirochetemic. This means that the demonstration of antibodies or antigen in dogs from endemic regions during the tick season may merely reflect exposure to the organism rather than being diagnostically significant. Carefully structured, long-term, controlled studies are needed to establish whether antibody or antigen-positive dogs subsequently develop disease.

There are obvious deficiencies in scientific information related to the pathogenesis, diagnosis, treatment, and epidemiology of canine and feline Lyme disease that compromise our ability to respond to questions posed by our clients. Regrettably, it is possible that we as a profession are developing a large body

of clinical information based primarily upon serologic evidence that may or may not represent an accurate description of Lyme borreliosis in these species. This information, if inaccurate, contributes to considerable patient morbidity in both endemic and nonendemic regions through misdiagnosis and improper treatment. It may have also contributed to the development and use of a vaccine for a disease that exists only on the pages of our journals. Since the seroprevalance of canine borreliosis in North Carolina is low (less than 4%) routine vaccination for Lyme disease would not seem appropriate unless dogs posed a significant risk for human Lyme disease. A seroepidemiologic survey conducted in a Lyme disease endemic region found that dog ownership was not a risk factor for human B. burgdorferi infection. Additionally, vaccination will eliminate the dog as a valuable serologic sentinel for documenting exposure to B. burgdorferi.

Future research to define the clinical spectrum of Lyme disease in dogs and cats should incorporate a rigidly defined case definition. Until the pathogenicity of <u>B. burgdorferi</u> for pet species is clearly established, veterinarians should maintain a degree of skepticism when contemplating a diagnosis of Lyme disease in their patients. -- Ed Breitschwerdt, DVM, Professor of Medicine, North Carolina State University, Raleigh, NC.

#### A DROP OF VERMOUTH IN A 16,000 GALLON MARTINI

What does a martini have to do with mastitis and milk quality? Residues in milk can now be detected in the parts per billion range (ppb). How much is 1 ppb? In addition to the comparison of one drop of vermouth in a 16,000 gallon martini, the June 1990 issue of The Dairyman magazine listed several other examples of 1 ppb which help put it in a better perspective:

- -- A single kernel in 2,000 tons of corn.
- -- One teat out of all the cows that a 2,000 cow dairy milking 4X will dip in 114 years.
- -- A shot glass in the Alaska oil spill.
- -- A penny out of \$10 million.
- -- 1 second out of 31.7 years.
- -- The residue level that will appear in milk if one cow is treated with antibiotics and the milk is put into the tank with no withdrawal time as long as the other 69,999 cows are clean! --Udder Topics, Volume XIII, #4, August 1990 as reported in Veterinary Newsleter. University of Georgia, Athens, GA, No 264, Sept 1990.

#### THOUGHT FOR THE MONTH

Ideas are funny things - they won't work unless you do.

-- Ron Whitford

#### VON WILLEBRAND FACTOR

Von Willebrand's disease (vWD) is the most common heritable canine and human bleeding disorder. The disease is caused by a deficiency, qualitative or quantitative, of von Willebrand factor (vWf). Von Willebrand factor is necessary for normal platelet adhesion.

Von Willebrand factor is a glycoprotein glycomeric assemblage of polypeptide subunits. It is referred to as a multimer and is produced by endothelial cells and, perhaps, by megakaryocytes. The vWf is essential in platelet adhesion to subendothelial collagen and may be involved in platelet plug formation. Another function of vWf is to stabilize factor VIII (antihemophilic A factor). The combination of vWf and factor VIII is called the factor VIII complex.

In dogs, parturition, liver disease, endotoxin, and drugs such as thyroxine and the vasopressin analog DDAVP (1-desamino-8-D-arginine vasopressin) stimulate increases in plasma vWf.

Virtually all canine purebred breeds have had affected animals. Mixed breed dogs may be affected. Cats may be affected.

Numerous clinical signs have been attributed to vWD. Included are epistaxis, gingival bleeding, hematuria, vaginal bleeding, and gastrointestinal bleeding. Panosteitis, fading puppy syndrome, hypothyroidism, and clinical difficulties with protracted parvoviral disease are associated with vWD. There is increased susceptibility to viral infections and increased propensity to hemorrhage subsequent to administration of many forms of drugs such as modified live viral vaccines, antibacterials, antibiotics, and most nonsteroidal antiinflammatory products.

Laboratory Evaluation of von Willebrand Factor

Plasma vWf\* - Blood should be drawn directly into a plastic syringe containing 3.8 percent trisodium citrate (1 part citrate to 9 parts blood; the ratio being critical). Blood should be transferred to plastic tubes for centrifugation utilizing plastic pipettes. Plasma should be kept cold unless transferred to a laboratory immediately. Plasma may be frozen. Any hemolysis results in major unpredictable error in vWf quantitation.

Antigenic assays - These assays measure vWf which, when measured by this method, is referred to as vWf:Ag. Concentrations of vWf:Ag are considered to be indicative of the amount of vWf in plasma regardless of biological activity.

Platelet aggregation and agglutination assays - These assays are named after the agent used to induce platelet aggregation or agglutination. Among these are ristocetin cofactor, polybrene cofactor, and botrocetin cofactor. These assays are considered to reflect the function of vWf in platelet adhesion. (contact Dr. Feldman).

Buccal mucosal bleeding time - Although not a specific test, the buccal mucosal bleeding time utilizing a double-bladed bleeding time device gives the best indication of patient primary hemostatic status, that is vWf function, in a vWf deficient patient. --Bernard Feldman, DVM, PhD, Professor of Clinical Pathology, VA-MD Regional College of Veterinary Medicine, Blacksburg, VA.

#### ROUNDWORMS IN KIDS AND PETS

The AAVP teamed up with the Centers for Disease Control (CDC) and Norden Laboratories to provide education materials to pet owners, veterinarians and physicians in the U.S. on the human health problems associated with roundworms (Toxocara canis) in dogs.

Roundworm infections are common in dogs up to about six months of age. Female roundworms in the small intestine produce large numbers of eggs which develop on the ground and become infective in two weeks. In puppies less than six months old, intestinal roundworm infections results when these eggs are ingested; however, when older dogs ingest roundworm eggs, the resulting larvae are distributed to muscle and other organs where they remain without undergoing further development.

When female dogs become pregnant, larvae hidden in their tissues migrate to the fetus; after birth, the worms grow to the adult stage in the intestine of puppies. Also, larvae from the dam's muscles pass through the milk and are ingested by nursing puppies. Thus, most puppies are infected from time of birth.

Since roundworms seldom develop to the egg laying stage in the intestine of dogs older than six months, adult dogs are only a minor source of infection for man. On the other hand, puppies may develope heavy roundworm burdens usually around four weeks of age and constitute a major reservoir of infection for humans, especially children. Children play in sandboxes and parks where roundworm egg contamination may exist due to defecation by dogs.

Infection of children with roundworm larvae causes a condition called visceral larval migrans (VLM). VLM is due to migration of the larvae in the lung, liver and less often the brain; when larvae migrate in the eye, the condition is called ocular larval migrans (OLM). The Centers for Disease Control report about 750 cases of VLM and OLM combined in the U.S. each year.

Visceral larval migrans usually occurs in toddlers with a history of eating dirt or exposure to puppies. Although children usually recover completely, fatal infections may result with larvae invade the central nervous system and heart. Ocular larval migrans occurs when roundworm larvae invade the eye producing impaired vision. Aggressive treatment is aimed at destroying the offending larvae and preventing complications.

Like many diseases, VLM and OLM can be prevented. All puppies should be considered to be infected and should be treated for roundworms as early as two weeks of age and then every two weeks until four months of age. Nursing female dogs also should be treated, however, most drugs that are effective against adult roundworms in the intestine have little effect on the larvae in the tissues of adult dogs. Proper sanitation and adequate supervision of children should be practiced in children exposed to puppies. Disposal of feces of all dogs should be done promptly since eggs do not become infective for two weeks after the feces are passed. Children should wash their hands after handling puppies or playing in sandboxes. --Dr. Ed Couvillion, College of Veterinary Medicine, Mississippi State, MS, AAVP News Brief, Vol. 1, Winter 1990.

#### ANTIBIOTICS OFFER PROMISE FOR UTERINE INFECTIONS

Researchers at the University of California-Davis, School of Veterinary Medicine are exploring a new treatment for uterine infections in mares through the use of natural antibiotics.

Despite the growing number of effective antibiotics, infectious endometritis continues to be the leading cause of infertility in broodmares, costing the horse industry more than \$10 million year.

The new possible form of treatment utilizing natural antibiotics was presented at the annual meeting of the American Association of Equine Practitioners (AAEP) by Marcelo A. Couto, DVM.

According to the university, standard antibiotic treatments occasionally fall short due to failure of the antibiotic to destroy bacteria, insufficient dose for the particular infection, reinfection of the uterus, and/or the appearance of antibiotic-resistant organisms.

The ideal antibiotic would have to destroy a broad spectrum of organisms at relatively low doses and also discourage the emergence of antibiotic-resistant strains, according to UC-Davis.

Small proteins called defensins isolated from neutrophil granules of several species of animals have been shown to fulfill these criteria in the test tube (in vitro), the studies show.

Preliminary test results are encouraging, university officials say. If the extract proves to be effective under controlled conditions in resolving infectious endometritis, this therapy could be applicable in the treatment of susceptible mares prior to breeding and during the postovulatory period before the embryo arrives in the uterus. --DVM, July, 1990 as reported in Communications in Continuing Education, Vol. 6, Number 3, September, 1990.

#### RABIES VACCINE TRIAL

Parramore Island in Accomack County Virginia will be the first US field test site of a genetically engineered vaccine to protect raccoons against rabies. The vaccine uses a piece of genetic material (DNA) from the rabies virus that triggers an immune response from the raccoon. The trial will begin August 20.

Because the vaccine uses a part of the virus that is not infectious, the animal is protected without risk of producing the rabies infection. No adverse effects have been reported from safety tests on over 35 species of animals in captivity. A similar vaccine is being used in Europe to control fox rabies.

Parramore Island was chosen because the test site is uninhabited and the island supports a large raccoon population. Located 4.5 miles from the mainland of Eastern Shore Virginia, the island is owned by The Nature Conservancy, an international nonprofit conservation organization. It has given approval for use of the island. --As reported in Virginia Department of Health Public Health News, August 13, 1990.

# CONTINUING EDUCATION OPPORTUNITIES VIRGINIA-MARYLAND REGIONAL COLLEGE OF VETERINARY MEDICINE FALL 1990

Date	Program	Location	Contact Hours
*Nov 16-17	Orthopedic Surgery/Canine Hindlimb	Blacksburg	10
*Nov 30-Dec 1	Practical Eye/Ear Surgery	Blacksburg	10
*Dec 7-8	Acute Abdomen in the Dog and Cat	Blacksburg	10
*Dec 14-15	The Computer in Veterinary Practice	Blacksburg	10

These courses are limited enrollment and feature hands-on laboratories.

NOTE: Program brochures are mailed six-eight weeks prior to the course date. For course information or assistance, please contact:

Kent Roberts, DVM
VA-MD Regional College of Veterinary Medicine
Blacksburg, VA 24061
(703) 231-7181

#### VIRGINIA ASSOCIATION WINTER MEETING

The Virginia Veterinary Medical Association annual convention will be held February 21-24, 1991 at the Homestead in Hot Springs.

### EASTERN STATES CONFERENCE

The seventh annual Eastern States Veterinary Conference takes place January 12-17, 1991 at the Marriott World Center in Orlando, Florida.

#### FOURTH ANNUAL LARGE ANIMAL MEDICINE SHORT COURSE IN COLUMBIA, SC

The S. C. Association of Veterinarians' Large Animal Academy and Clemson University's Department of Animal and Veterinary Sciences will sponsor a conference, "Focus on Clinical Nutrition", November 14-15 at the Columbia Ramada Hotel, Columbia, SC.

This conference focuses on current nutritional requirements and clinical problems in small ruminants, horses and cattle. Topics are of concern to those practicing throughout the Southeast. Of special interest will be session on mycotoxin in all species and equine colic and laminitis.

For a brochure, contact Dr. John C. Spitzer, Animal and Veterinary Sciences Department, Clemson University, Clemson, SC 29634 (803) 656-5164.

Virginia-Maryland Regional College of Veterinary Medicine Extension Staff:

Dr. J.M. Bowen - Extension Specialist - Equine
Dr. C.T. Larsen - Extension Specialist - Avians

Dr. C.T. Larsen - Extension Specialist - Avians
Dr. K.C. Roberts - Extension Specialist - Companion Animals

Dr. W. Dee Whittier - Extension Specialist - Cattle

K.C. Roberts, Editor Maura M. Wood, Production Manager of VIRGINIA VETERINARY NOTES

VIRGINIA POLYTECHNIC INSTITUTE AND STATE UNIVERSITY VIRGINIA COOPERATIVE EXTENSION BLACKSBURG, VIRGINIA 24061-0512

Nonprofit Org. U. S. Postage PAID Blacksburg, VA 2406 Permit #28