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VIRGINIA-MARYLAND VETERINARY NOTES

Veterinary Teaching Hospital, Virginia-Maryland Regional College of Veterinary Medicine

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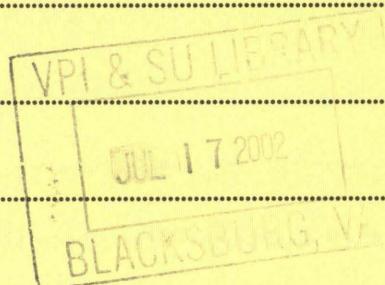
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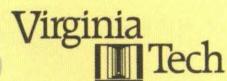


Kent. C. Roberts, D.V.M.
Extension Veterinarian

Would You Believe?

Tuberculosis continues its spread around the world. The number of cases of TB in 1999 was estimated to be 8.4 million with projected increases in 2005 to 10.2 million and to 11.6 million by 2010.

K.C. Roberts, D.V.M., Blacksburg, VA, Dec. 2001



VIRGINIA POLYTECHNIC INSTITUTE
AND STATE UNIVERSITY

This newsletter is published quarterly in support of the outreach program of the Veterinary Teaching Hospital VMRCVM, Blacksburg, VA and is prepared for and distributed to veterinarians in the Mid Atlantic Region



CANINE IDIOPATHIC DILATED CARDIOMYOPATHY

Dilated cardiomyopathy (DCM), characterized by chamber dilatation and myocardial systolic and diastolic dysfunction, is one of the most common heart diseases in dogs. Gross pathology examination of dogs with DCM generally shows dilatation of either all four cardiac chambers or predominant dilatation of the left chambers. Myocardial eccentric hypertrophy is evident by an increased heart weight/body weight ratio, together with a decreased ratio of left ventricular wall thickness to chamber diameter. Echocardiographic indices of left ventricular volume, but not fractional shortening (FS) or E-point septal separation (EPSS), proved to be prognostic markers in 31 dogs with DCM. A study of survival and prognostic indicators in 189 dogs with DCM reported survival rates of 17.5% at one year and 7.5% at two years. The most significant negative predictive variables were young age at onset of clinical signs, followed by presence of dyspnea and clinically detectable ascites.

DCM is often referred to as being breed specific for Boxers, Doberman Pinschers, English Cocker Spaniels and other breeds. Review of reports on histopathologic findings in canine DCM reveals two histologically distinct forms of DCM: (1) cardiomyopathy of Boxers and Doberman pinschers, corresponding to the "fatty infiltration-degenerative" type, and (2) the form seen in many giant, large- and medium-sized breeds, including some Boxers and Doberman Pinschers, which can be classified as the "attenuated wavy fiber" type of DCM. The classification of canine idiopathic DCM according to histologic findings seems superior to classification suggestion breed-specific syndromes, as some breeds (i.e. Boxers and Doberman Pinschers) may be affected by both diseases. However, antemortem etiological diagnosis of DCM is difficult. DCM carries a poor prognosis in dogs, and few prognostic indicators have been identified.

The etiology of idiopathic dilated cardiomyopathy is, by definition, uncertain or not known. There is, however, a diverse spectrum of suspected and known causes of myocardial hypokinesis. DCM is considered to be hereditary in 20 to 35% of human patients. Nutritional abnormalities causing myocardial hypokinesis, i.e. carnitine and/or taurine deficiencies, have been described in humans, dogs, and cats. Metabolic disorders associated with DCM include diabetes mellitus, pheochromocytoma, and hypothyroidism. Immunological processes may be involved in the pathogenesis of DCM, as auto-antibodies have been detected against the cardiac *B*-receptor, the mitochondria, the mitochondrial ADP/ATP translocator, and o`- and 6-myosin heavy chains, both in humans and experimental animals. It is well accepted that acute myocarditis can progress to a state of myocardial hypokinesis. Drug- and toxin induced cardiomyopathies include cardiotoxicity caused by doxorubicin and other anti-neoplastic agents, ethanol, cobalt, lead, catecholamines, histamine, methylxanthines, and vitamin D. Several studies on experimentally induced tachycardia (>200 beats/min) report development of CHF and chamber dilatation, and reversible hypokinesis in dogs.

Taken from: Tidholm, A., et al Vet J 162 92-107, 2001, as reported in VetMed, Vol 8, Issue 2, January 2002, Iowa State University, Ames, IA

CODLING MOTHS

Codling moths are the most destructive and widely distributed pest of apples, pears & walnuts in the world. The moths were accidentally introduced into this country from Europe in the 1700's. Uncontrolled, the moth larvae can destroy up to 95% of an apple crop and up to 60% of a pear crop.

Department of Agriculture ARS, June 2001

EQUINE PROTOZOAL MYELOENCEPHALITIS

Equine protozoal myeloencephalitis (EPM) is a serious and often fatal neurologic disease of equids. Animals affected by EPM can demonstrate a variety of clinical abnormalities, and signs can vary tremendously in severity. Classically, horses with EPM develop a variety of asymmetric neurologic deficits including gait abnormalities, ataxia, weakness, and focal muscle wasting. However, symmetric neurologic abnormalities are also seen frequently. The disease may be focal or multifocal in nature and may be manifested less frequently as a head tilt, facial paralysis, seizures, or even apparent behavioral changes. Horses of all ages can be affected, but horses are usually at least 6 months old when first diagnosed with EPM.

Sarcocystis neurona is the parasite thought to be the principal causative agent of EPM. Recent work suggests that *Neospora caninum* and/or *Neospora hughesi* infections can also cause neurologic disease that is clinically indistinguishable from that caused by *S. neurona*. However, *S. neurona* infections are apparently a much more frequent cause of EPM. The life cycle of *S. neurona* is complex and is not fully understood. Horses are considered an aberrant host for these parasites. The definitive hosts of *S. neurona* are believed to be members of the opossum family (*Didelphis virginiana* in North America and other members of the Didelphidae family in Central and South America). The natural range of opossum species corresponds well with areas where EPM is commonly identified. EPM has been recognized in horses on other continents, but to date only in horses that originated from North America, Central America, and South America. A recent experimental study showed that the domestic cat can serve as an intermediate host, but the natural intermediate hosts for *S. neurona* remain unknown.

EPM is a growing problem for the horse industry in the U.S. This disease has been diagnosed with increasing frequency by veterinarians throughout the U.S. during the past decade since commercial tests to detect *S. neurona* infection became available. Treatment of EPM is quite expensive, and both mildly or severely affected horses often require treatment for extended periods. Despite aggressive treatment, horses with EPM can be affected so severely that they must be euthanized, or they can develop permanent problems which affect their ability to be used for athletic activities.

Making a definitive diagnosis of EPM in the live horse is challenging. Currently, there are no universally accepted methods for making a definitive diagnosis. It is important to understand that EPM, the disease, is different from documented exposure to *S. neurona*, the organism that causes the disease. A presumptive diagnosis of EPM can be made in some horses if they show neurologic signs that would not likely be caused by other diseases, such as cervical vertebral malformation or herpesvirus myelitis. In many other horses, however, these clinical signs could be caused by one or more other diseases. Many veterinarians make a presumptive diagnosis of EPM if *S. neurona* or antibodies to the parasite can be detected in cerebrospinal fluid (CSF) collected from horses with neurologic abnormalities, assuming that the CSF was not contaminated by blood during collection or because the blood-brain barrier has been injured. Recent studies suggest that some normal horses can have antibodies to *S. neurona* in CSF. Horses might also be assumed to have EPM if they respond favorably to treatments that are believed to kill the parasites causing this disease. A definitive diagnosis of EPM can be established after horses die by examining tissues of the brain and spinal cord using special techniques. But even necropsy tests can miss an infected animal due to the small number of organisms required to cause disease.

Some of the methods used to identify exposure definitively to *S. neurona* include detection of specific antibodies (using the immunoblot or direct agglutination assays), detection of DNA from the organisms (using polymerase chain reaction assay), or identification of organisms in tissues (using culture or immunodiagnostic techniques). The most common method used to identify horses specifically that have been infected is the immunoblot assay. This diagnostic test became available commercially in 1992 and since then has been used widely in the U.S. to detect antibodies to *S. neurona* in serum and in CSF. Identification of *S. neurona*-specific antibodies in serum indicates that the animal has been exposed previously to *S. neurona*. Results of seroprevalence studies indicate that in some regions (California, Colorado, Florida, Kentucky, Missouri, Michigan, Ohio, Oregon, and Pennsylvania), approximately 30-60 % of horses tested were exposed previously to *S. neurona*. Not all exposed horses develop the disease EPM, but hospital based studies suggest that exposure can be documented more frequently in groups of EPM horses compared to clinically normal horses.

Several treatments have been used in horses diagnosed with EPM, and others are being investigated currently. Responses of horses with EPM treated at a few referral veterinary hospitals have been reported, but results of objective efficacy studies have not been published to date. A major emphasis of treatment is the elimination of the parasites from neurologic tissues. The treatment that has been reported to be used most often is a

combination of antibiotic/ antiparasitic drugs (a sulphonamide and pyrimethamine). Another antimicrobial drug that has been used infrequently is oxytetracycline. New drugs that are being investigated to determine their efficacy for eliminating the causative agent include diclazuril, toltrazuril, and nitazoxanide. Antiinflammatory drugs (e.g., phenylbutazone and flunixin meglumine) and nutritional feed additives (e.g., vitamins such as folic acid and vitamin E) have been used as adjunct supportive treatments in horses with EPM. Acupuncture has also been used in an effort to diagnose and treat horses with EPM.

Because the sporocysts of *S. neurona* are passed in the feces of the opossum, it is likely that infective oocysts are introduced into the feed and water supply of horses and intermediate hosts. Preventing access of opossums to hay, grain, pasture, and water sources may decrease the risk of exposure and disease. However, it is not clear that eliminating direct exposure of horses to wild opossums will eliminate completely the risk of disease because horses may be exposed to the parasite through other sources such as contaminated feed.

Taken from: EPM in the US, NAHMS, 2001 USDA:APHIS:VS, CEAH, Fort Collins, CO as reported in VetMed, Vol 8, Issue 2, January 2002, Iowa State University, Ames, IA

HYPERTHYROID CATS TREATED WITH IODINE 131

This study provides estimates of duration of survival for cats successfully treated for hyperthyroidism with radioactive iodine, which can be useful in assisting with client treatment decisions. Two hundred thirty-one cats treated with radioactive iodine at the Texas Veterinary Medical Teaching Hospital were followed for a median of 25 months. All cats referred for high thyroid hormone concentrations underwent radionuclide imaging of the thyroid. An abnormal image was defined as increased uptake (as compared to the salivary glands) in 1 or both thyroid glands. This included increases in the number or size of the normal thyroid gland(s). Iodine 131 as sodium iodine was injected into a peripheral vein to achieve thyroid ablation. The mean dose of I131 was 4.9 miC (range, 2.8-8.9 miC). An attempt was made to avoid exceeding 1 miC/0.45 kg bodyweight. Mean age at diagnosis of hyperthyroidism was 13 ± 2.5 years with a range from 4 to 21 years. Mean weight at diagnosis was 3.6 ± 1 kg with a range from 2 to 7 kg. The cats included 105 males and 126 females.

At the time of diagnosis 17 (7%) cats had no or minor clinical problems, 99 (43%) had only cardiac-related clinical problems, 87 (38%) had cardiac & other major problems, 12 (5%) had both cardiac & renal problems, and 16 (7%) reported other major problems not included in the above areas. Therefore, a total of 198 (86%) cats were diagnosed as having a cardiac-related problem.

Based on data available at the time of diagnosis of hyperthyroidism in this population of cats, the predictors of survival of older cats included only age at diagnosis and sex of the cat. Cardiac problems were likely not predictors of survival because almost all cats had these problems and most resolved with treatment of the hyperthyroidism. The relative risk for each increase in 1 year of age was 1.2. Males had poorer survival across time than females. The relative risk for females compared to males was 0.68 with females having a lower risk of death than males. A male cat diagnosed at 12 years of age had only a 59% chance of surviving 2 years compared to a 70% chance for a female diagnosed at the same age. Because treatment with I131 is expensive, time consuming, and stressful for the patient, this information should prove useful for clients making treatment decisions:

The median life span of these cats was 15 years and the median survival time in our study was 25 months (range 3 days to 8 years). Renal-related problems and neoplasia of any type were by far the most common health problems at death, as well as being the only significant predictors of survival among all health problems. Other problems, including central nervous system and gastrointestinal disease, contributed to morbidity but were not predictors of survival. Each unit of increase in the number of problems increased the risk of dying almost 2-fold.

Taken from: Slater, M: R., S. Geller, and K Rogers J Vet Intern Med /5., 2001 in ISU Vet Med, May 2001 as reported in Veterinary News, July 2001, Penn State University, University Park, PA

THROMBOLYTIC THERAPY IN DOGS AND CATS

Thrombolysis begins when the injured tissues and vascular endothelium respond to thrombus formation with slow release of tissue plasminogen activator (tPA) which converts the inactive proenzyme, plasminogen, into the fully active serine protease plasmin. Plasmin is a proteolytic enzyme that catalyzes fibrin breakdown. Tissue plasminogen activator normally exists in low concentrations in the plasma, and secretion is controlled by physical and hormonal factors such as venous pressure and vasoactive substances including thrombin. Thrombin concurrently stimulates the release of an endogenous inhibitor of t-PA, plasminogen activator inhibitor 1 (PAI-1), from the endothelium and platelets. Tissue plasminogen activator and PAI-1 bind to each other and circulate in plasma until the resultant complex is adsorbed onto the fibrin meshwork. Plasminogen activator inhibitor is then released back into the circulation, and the t-PA transforms the thrombusbound plasminogen to plasmin. The enzymatic activity of t-PA is greatly enhanced by transformation of fibrin to fibrin polymer.

Streptokinase acts by binding to plasminogen in a 1:1 ratio, forming an active enzymatic complex capable of subsequent conversion of other plasminogen molecules to plasmin. Streptokinase is produced by beta-hemolytic streptococci and is potentially antigenic. Streptokinase does not possess any direct fibrin binding properties, activating both thrombus-associated and free-plasminogen. In addition to the thrombolytic activity, streptokinase causes rapid degradation of the circulating fibrinogen pool and subsequent accumulation of fibrinogen degradation products. These degradation products may then interrupt newly forming fibrin networks by substituting for fibrinogen in the process of fibrin polymerization. Coagulation factors including V, VIII, and prothrombin are also degraded, giving streptokinase the potential to cause a massive coagulation defect. Although the half-life of streptokinase is approximately 30 minutes, depletion of fibrinogen may continue for 24 hours.

Recombinant tissue plasminogen activator (tPA) was originally synthesized from c-DNA obtained from a human melanoma cell line. The mechanism of action of t-PA involves formation of a complex between fibrin, t-PA, and plasminogen. Tissue plasminogen activator has a high affinity for fibrin due to a fibrin binding site, preferentially activating thrombus-associated plasminogen. Although the fibrin specificity of t-PA increases the likelihood of effective thrombolysis without production of systemic plasma proteolysis, the benefits of this specificity are limited. Importantly, the fibrin specificity of t-PA is relative, and when the dose of t-PA is increased to achieve effective thrombolysis, the risk of plasma proteolysis and excessive bleeding increases. Tissue plasminogen activator has a half-life of 2 to 3 minutes, but prolonged fibrinolytic activity may occur due to enhanced binding of t-PA to fibrin, providing protection of both fibrin-bound t-PA and plasmin from their inhibitors.

Clinical experience with thrombolytic agents in small animals is limited to streptokinase and t-PA. It is possible, that as in humans, canine and feline patients with PTE and right ventricular dysfunction may benefit from thrombolytic therapy but there are no veterinary studies to support this theory to date. Successful use of streptokinase has been documented in a small number of canine patients with systemic thromboembolism. Thrombolytic therapy is relatively efficacious in cats with aortic thromboemboli but is associated with a high mortality rate. With regard to use of t-PA in veterinary medicine, the small number of animals treated with varying protocols makes it impossible to provide safe and effective dose recommendations at this time.

Taken from: Thompson, M., et al J Vet Emerg Crit Care 11:111-121, 2001, as reported in VetMed, Vol 8, Issue 2, January 2002, Iowa State University, Ames, IA

What are the "Perks" of Practice Ownership?

Nearly every practice in existence today, including those owned by corporate consolidators, was built, bought or nurtured to success by young veterinarians much like you. Today's challenges are different and difficult, but no more so than ownership endeavors of your predecessors. There are many good reasons to own a practice.

You can practice quality medicine. Utilization of the ever-increasing number of referral facilities and specialists will allow every practice owner the opportunity to provide excellent standards of care, even without the latest

and greatest gadget or training.

You will earn more. Your level of responsibility increases only slightly over that of a well compensated "Chief of Staff." associate. As an owner, you have the benefit of equity accumulation and the opportunity to accumulate practice profit over the years for investment and savings.

You will be the beneficiary of a number of money saving "perks". Special tax savings reserved for the business owner are numerous.

Flexibility of an owner as your own supervisor gives you the opportunity to take the afternoon off you want, go to your child's special school event, and even have your children with you at work if necessary (without asking for permission).

You typically receive status and special respect in the community and instant acceptance into various social circles. There are many, many other similar emotional and personal advantages that you may not otherwise receive.

Possibly, the most significant consideration for practice ownership is the ability to affect change in your practice environment and mold it to suit your personal as well as your professional career objectives. You have the ability to direct your career towards the most fulfilling aspects conceivable. The career alternatives are tremendous within the ownership format.

Practice ownership isn't just for the entrepreneur. It is for anyone who wants the most control of his or her destiny. Practice ownership does not require rocket chemistry. You get to be more the individual you always wanted to be, in your personally sculpted pursuit of excellence. You should think seriously about the benefits of practice ownership and pursue the unique blend of success and independence that can only be found in that fraternity. The sooner the better.

In conclusion, two year grads with good credit and as little often as little as \$25,000 (sometime \$0) available as a down payment are able to purchase very high grossing practices (\$1,000,000 and more) with the ready availability of high-dollar commercial loans. It's easy to do. Veterinarians typically are very good credit risks, and the commercial lenders are aggressive and competitive in making loans to individuals in our profession.

Doyle Watson, DVM and Jim Stephenson, DVM, Simmons & Associates

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WOULD YOU BELIEVE?

Census figures indicate that over two thirds of native born American citizens live in the same state in which they were born. The states with the highest percentage of home grown residents are:

1. New York	82.4%	4. Michigan	78.9%
2. Pennsylvania	81.7%	5. Ohio	76.6%
3. Louisiana	80.3%	6. Iowa	76.5%

K.C. Roberts, Blacksburg, VA

We Need Your Help

I took over as editor of this newsletter about 20 years ago from Dr. Gordon MacInnis who started it in the days of the Veterinary Science Department at Virginia Tech. Having edited over 100 issues of the Virginia-Maryland Veterinary Notes, I hope to continue what I believe is a useful publication for practitioners as I gradually fade into retirement, which officially started in 1995.

The direct costs of printing and mailing the newsletter continue to escalate while our budget at the Veterinary College is taking a serious hit in Richmond because of the economic downturn across America. We dislike having to plead poverty, but there are three things I will suggest which can help us continue publication and control our costs.

1. If you are receiving this newsletter and really would rather not, please tell us so we can remove your name from our mailing list.
2. If the address on your newsletter is incorrect, please provide us with the correct address. I much prefer to mail to practices rather than home addresses with the idea that more than one individual will benefit from the newsletter.
3. We can send your newsletter electronically to your e-mail address at a great savings. If you prefer this delivery method, please send your name and e-mail address to Anne Clapsaddle, aclapsad@vt.edu.

As always, your comments and suggestions are welcome as we try to provide you with a useful and interesting publication. We are pleased to receive articles and/or practice tips from our readers for use in the newsletter. Thank you for your assistance and continuing support for the newsletter and our programs at the Virginia-Maryland Regional College of Veterinary Medicine.

Kent C. Roberts, D.V.M.
VMRCVM
Blacksburg, VA 24061

Opportunities in Continuing Education Spring 2002

<u>Date</u>	<u>Topic</u>	<u>Location</u>	<u>Contact Hours</u>
March 8 & 9	Diagnostic Ultrasonography	Blacksburg	10
March 15 & 16	Applied Ultrasonography	Blacksburg	10
March 22 & 23	Introductory Echocardiography	Blacksburg	10
April 12 & 13	Practical Eye	Blacksburg	10
May 3 & 4	Gastrointestinal Endoscopy I	Blacksburg	10
May 17 & 18	Applied Ultrasonography	Blacksburg	10
May 20 - 24	Intensive Orthopedic Surgical Week	Blacksburg	40
May 31 & June 1	Introductory Echocardiography	Blacksburg	10

Please note:

The courses listed above are limited enrollment and feature a hands-on laboratory experience under the guidance of clinical faculty members. Program brochures provide course details. For registration or more information, please contact **Anne Clapsaddle**, aclapsad@vt.edu (540) 231-5261; or **Conference Registration**, Continuing Education Center, (540) 231-5182.

Virginia-Maryland Regional College of Veterinary Medicine Extension Staff:

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Anne Clapsaddle - Continuing Education/Extension Coordinator

K.C. Roberts, Editor

Anne Clapsaddle, Production Manager of VIRGINIA-MARYLAND VETERINARY NOTES

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COLLEGE OF VETERINARY MEDICINE
VIRGINIA TECH
BLACKSBURG, VIRGINIA 24061

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