VIRGINIA COOPERATIVE EXTENSION SERVICE

EXTENSION DIVISION - VIRGINIA POLYTECHNIC INSTITUTE AND STATE UNIVERSITY - BLACKSBURG, VIRGINIA 24061

VIRGINIA-MARYLAND
REGIONAL COLLEGE
OF
VETERINARY MEDICINE

VIRGINIA VETERINARY NOTES

September-October, 1986 No. 23

WHAT'S INSIDE!

Kent C. Roberts

Kent C. Roberts, DVM
Extension Veterinarian

Issued in furtherance of Cooperative Extension work, Acts of May 8 and June 30, 1914, and September 30, 1977, in cooperation with the U. S. Department of Agriculture. W. R. Van Dresser, Dean, Extension Division, Cooperative Extension Service, Virginia Polytechnic Institute and State University, Blacksburg, Virginia 24061; M. C. Harding, Sr., Administrator, 1890 Extension Program, Virginia State University, Petersburg, Virginia 23803.

PHOTOSENSITIZATION "PHOTO"

Cattle, sheep, goats, horses, swine, and white-tailed deer grazing certain improved pasture and range forage primarily in the spring and summer may be affected by one of two different kinds of photosenitization. In the case of primary photo, the photodynamic agent in the blood of the skin is a chemical compound not normally ingested by the animal and is absorbed directly from the digestive tract and not completely detoxified or excreted by the liver. In the case of photo due to liver damage, a product of chlorophyll digestion called phylloerythrin, which is normally absorbed from the digestive tract and excreted in the bile, reaches the blood in the skin due to dysfunction or damage to the liver. Common causes for liver damage are grazing toxic plants, eating moldy hay or forage, and liver flukes. With either type of photo, the photodynamic agent in the skin predisposes the non-pigmented areas of the skin to sunburn.

Although primary photosensitized animals seldom die, weight loss, damaged udders, refusal of nursing young, and secondary infections cause considerable economic losses. General treatment for photosensitization is to remove animals from the suspect pasture, place in the shade, give dry feed, and paint affected areas with 2% aqueous methylene blue solution to protect from sunlight. **--from TAEX fact Sheet "Photosensitization of Livestock and Deer in Texas" & University of Georgia Veterinary Newsletter, adapted for VMNL. Veterinary Newsletter, July 1986, University of Florida.**

MAST CELL TUMORS - DEADLIER THAN THEY LOOK

Mast cell tumors are potentially malignant, often insidious, and relatively common tumors in dogs. They are, in fact, so common (comprising 20% of all canine skin tumors) and so innocent-looking (often appearing as solitary, well-delineated masses) that they may be treated too casually. With surgery alone, 1/3 of all mast cell tumors can be expected to recur along the incision line 2 weeks to 6 months after wide surgical excision. In fact, 38% of all dogs with mast cell tumors can be expected to die within 30 weeks of surgery.

No reliable treatment is known. Post-surgical treatments proposed previously, such as radiation and/or corticosteroids, give dogs no better chance of survival than surgery alone. Cats have no better prognosis than dogs. For example, a small, solitary firm mass on a cat's head usually metastasizes in 3 to 6 months.

Figures such as these emphasize the value of pre-surgical diagnosis by fine needle aspiration of the suspected tumor. The aspirate is smeared on a slide, air-dried, and stained with Harleco' Diff Quik or Wright's Stain. Numerous mast cells with their characteristic blue to purple granules are easily seen microscopically under high dry and oil immersion. This analysis of fine needle aspirates is so easy, rapid, relatively painless, and inexpensive that it should become a routine procedure in every private practitioner's laboratory. **— Dr. R.L. Grier, Professor, Iowa State University News, Feb. 1986. Veterinary Newsletter, July 1986, University of Florida.**

SUDDEN DEATH IN CATTLE

Often, the only history available to the prosector is "found dead". This doesn't indicate whether the animal actually died acutely without clinical signs, of if clinical signs were not observed. Some of the more common causes of sudden death in cattle are described below.

One such cause is a ruptured abscess at the liver hilus. Two syndromes may result from this rupture. In all cases the abscess has ruptured into the vena cava and may be partially healed. Usually there are small (2-10 mm) abscesses scattered throughout the lung (embolic shower). Initially, these are more easily palpated than visualized. The abscesses may be encapsulated, indicating the liver abscess ruptured days to weeks ago. If there is marked interlobular edema of the lung, the abscess ruptured a second time, trigerring an "anaphylactoid reaction". The resulting pulmonary edema is sufficient to essentially drown the animal. In other cases the pulmonary abscesses erode through the vessel wall and into the lung parenchyma. The cow may have several episodes of epistaxis prior to death from severe blood loss.

Pituitary abscesses are another cause of sudden death. There are usually no clinical signs associated with this problem, although occasionally bradycardia may be detected. The pituitary abscess may be the only lesion observed. In some cases there may be coning of the cerebellum from increased intracranial pressure as a result of the space occupying lesion. The pituitary abscess may be intact or may have ruptured and be associated with suppurative meningitis. This syndrome occurs in other ruminants but has not been documented in other types of animals.

Acute septic mastitis, another cause of sudden death, is frequently overlooked at necropsy. Subcutaneous edema and emphysema may be present at the base of the udder. The cut surface usually appears dry and dull. The gland may be dark green brown or it may initially be red-tan. If the cut surface of the gland is left exposed to the air while completing the necropsy, it will turn green because of the bacteria present. There is usually a distinct odor of fermentation present.**--Lois Roth, DVM, PhD, Diplomate ACVP, VA-MD Regional College of Veterinary Medicine.**

EFFICACY OF IVERMECTIN AGAINST <u>DIROFILARIA IMMITIS</u> LARVAE

Forty-two beagles, 14 to 15 weeks of age, were injected subcutaneously with 50 infective larvae of <u>Dirofilaria immitis</u> and were allotted by weight, within sex, to 6 treatment groups. Group 1 served as nonmedicated vehicle-treated controls; groups 2 through 5 were given an oral tablet form of ivermectin at dosages of 0.3 µg/kg, 1.0 µg/kg, 2.0 µg/kg, and 3.3 µg/kg at 30 days after inoculations; group 6 was given the 2.0 µg/kg dosage at 45 days after inoculation. Dogs were euthanatized and necropsied 154 days after treatment (day 139 for dogs in group 6) and examined for heartworms. On the numerical bases of helminths recovered in the groups, the efficacies for preventing heartworm maturation were 0% (group 2), 53/2% (group 3), 97.2% (group 4), 98.1% (group 5), 63.8% (group 6). Drug-related adverse reactions were not detected.**--by A.J. Paul, D.V.M., M.S.; J.W. McCall, Ph.D.;and K.S. Todd Jr., Ph.D. as presented at the American Heartworm Symposium '86, March 21-24, 1986 in New Orleans, Louisiana. Small Animal Professional Topics, 1986, Vol II, No 1.**

Invermectin was given per os to 48 beagle puppies 45 days after they received 50 infective larvae of Dirofilaria immitis. Within each of 8 replicates formed by sex and weight, animals were randomly allocated to receive placebo or ivermectin at 3.3, 6.0, 12.0, 25.0 or 50.0 mcg/kg once, respectively. Blood samples were examined for microfilariae prior to infection and 3 and 7 months after treatment. Necropsies were done at 7 months after treatment (215 days) for a total count of heartworms.

Each of the 8 control dogs had microfilaria and heartworms (geometric mean 36.26) found at 8.5 months after infection. One and two male heartworms were found, respectively, in one dog in each of the ivermectin 12.0 mcg/kg and 3.3 mcg/kg groups. All other ivermectin treated dogs were negative for heartworms. There were no adverse reactions to treatment.

In a second study, 42 beagle puppies were infected and treated following a protocol similar to the above trial. In this study ivermectin was 100% effective in preventing heartworm infection at all dose levels evaluated. None of the treated dogs had worms at necropsy, while all control dogs had worms. No adverse reactions to ivermectin were noticed.--**by A.J. Paul, D.V.M., M.S.; J.W. McCall, Ph.D.;and K.S. Todd Jr., Ph.D. as presented at the American Heartworm Symposium '86, March 21-24, 1986 in New Orleans, Louisiana. Small Animal Professional Topics, 1986, Vol II, No 1.**

AN ETIOLOGIC AGENT OF CANINE DERMATOSES

Staphylococcus intermedius has been identified in recent studies as the major coagulase-positive species associated with canine dermatoses. The bacterium is normal flora of the skin and has been shown to be biochemically and epidemiologically distinct from S. aureus. Approximately 90 percent of the staphylococci isolated from canine dermatoses are now identified as S. intermedius and the remaining 10 percent are identified as S. aureus. The coagulase-negative staphylococci are seldom incriminated as etiologic agents of canine dermatoses, but are part of the normal flora of the skin.

Previously, the genus Staphylococcus was classified into three species: S. aureus (coagulase-positive), S. epidermidis (coagulase-negative), and S. saprophyticus (coagulase-negative). The identification criteria for these three species were based on coagulase production and biochemical tests. Rabbit plasma has traditionally been used to test staphylococcal isolates for coagulase production. Most veterinary bacteriology laboratories reported the coagulase-negative staphylocci as S. epidermidis and the coagulase-positive staphylococci as S. aureus. More definitive taxonomic studies have resulted in the genus Staphylococcus being reclassified and includes: two coagulase-positive species, S. aureus and S. intermedius; one coagulase-variable species, S. hycius, and ten coagulase-negative species, S. capitus, S. cohnii, S. epidermidis, S. haemolyticus, S. hominus, S. saprophyticus, S. sciuri, S. simulans, S. warneri and S. xylosus. The commerical API STAPH-IDENT system has proven suitable for the identification of these Staphylococcus species, and is based on fication of these Staphylococcus species, and is based on biochemical tests. Because many S. intermedius strains are coagulase-negative for rabbit plasma but are coagulase-positive for dog plasma, the staphylococcal isolates should be tested using both rabbit plasma and dog plasma.

Canine S. intermedius strains have demonstrated antimicrobial resistance for ampicillin, carbenicillin, chloramphenicol, clindamycin, erthromycin, gentamicin, kanamycin, penicillin G, stroptomycin and tetracycline. Resistance to two or more antimicrobial agents has been frequently observed and has been hypothesized to be plasmid mediated. Antimicrobial susceptibility tests should be routinely performed on the clinical isolates by determining the minimum inhibitory concentrations of selected antimicrobials or with the standardized Bauer-Kirby disk diffusion procedure. **-- Charles M. Scanlan, D.V.M., Ph.D., Texas A&M University, College Station, Texas. Texas Extension Service, Veterinary Quarterly Review, Summer 1986.**

POTOMAC HORSE FEVER TESTING

At a recent meeting it was decided, that until further notice, all blood (serum) samples submitted for testing with the fluorescent antibody test for Ehrlichia risticii from Virginia or Maryland, should be sent to the following address:

Maryland Department of Agriculture
Animal Health Laboratory
4901 Calvert Road
College Park, MD 20740
Tel. (301)454-3631

Two samples should be taken from suspected cases, one during the acute phase of the disease, and the second during the convalescent period, at least two weeks after the original sample was drawn. The cost will be $20.00 ($10.00 per sample) and this will be charged to the referring veterinarian.

This will be the only laboratory which will carry out this test for practicing veterinarians and your cooperation in this matter will greatly enhance the speed with which you will receive your results.

SUCCESS IS NO ACCIDENT

Marketing veterinary services is a term we encounter quite often these days. What is marketing? Do you have a marketing strategy in your practice?

In what is probably an over simplification, marketing can be defined as determining people's (client's) needs, as opposed to selling, which doesn't necessarily take the needs of the customer into consideration. A good marketing strategy would be to first find out what your good client's needs are and then to organize your practice around meeting those needs.

Just the act of talking/communicating with clients can be a valuable and enlightening exercise for any practitioner and his staff. Determining client needs is one way to demonstrate that you care about them and their animals.

Try it -- it could be a very worthwhile effort.**--Kent C. Roberts, DVM, VA-MD Regional College of Veterinary Medicine.**

VETERINARY COLLEGE LABORATORY SERVICES

The Laboratory Services Section of the Veterinary Medical Teaching Hospital offers clinical support in the following areas:

Surgical Pathology

Routine histopathologic evaluation of surgical biopsies or necropsy tissues is available. Please submit samples in wide-mouth, screwtop, leak-proof, nonbreakable jars containing 10% neutral buffered formalin. Be sure to include an adequate history and description of the lesion.

Clinical Cytology

Routine evaluation of cytologic preparations is available. Please submit air-dried, stained or unstained glass slide smears in suitable mailing folders. Mark "hand cancel" on packages to avoid breakage. Be sure to include an adequate history and description of the lesion.

Bacteriology

Routine aerobic, anaerobic and fungal culturing is available. Please submit samples in appropriate sterile culturettes or containers under refrigeration. Be sure to include an adequate history, particularly listing previous drug therapy.

Toxicology

Routine toxicological evaluation of gut contents, blood, feces, urine, organs or feed for heavy metals, mycotoxins, insecticides, rodenticides and common plant toxins is available. Please contact the VMTH Laboratory Services Central Receiving Desk (703-961-4320) for specific sample requirements prior to sample submission. **-- William Chickering, D.V.M., Ph.D. - VMTH Laboratory Services VA-MD Regional College of Veterinary Medicine, Blacksburg, VA.**

VETERINARY COLLEGE RESIDENT AND INTERN PROGRAM

The Virginia-Maryland Regional College of Veterinary Medicine inaugurated a clinical residency program on July 1, 1986. Three residents are now at work in their specialty areas in the Teaching Hospital. They are:

William Hay - Small Animal Medicine - Auburn '85
John Payne - Small Animal Surgery - Ohio '82
Christina Boyd - Anesthesiology - Saskatchewan '85

The College has its third class of clinical instructors (interns) at work in the Teaching Hospital. They are:

Julie Hass - Small Animals - Michigan '86
Christiane Jennes - Small Animals - Hanover '86
Ann Rashmir - Large Animals - California '86
Wilbur Frank - Large Animals - Georgia '86
Patricia Malick - Small Animals - VPI '85

VIRGINIA-MARYLAND REGIONAL COLLEGE OF VETERINARY MEDICINE
TEACHING HOSPITAL CLINICAL FACULTY

Small Animal Medicine

Deborah Davenport, DVM, MS, Diplomate ACVIM	Internal Medicine/Oncology
Michael S. Leib, DVM, MS, Diplomate ACVIM	Gastroenterology
W. Edward Monroe, DVM, MS, Diplomate ACVIM	Internal Medicine
Kay L. Schwink, DVM	Ophthalmology
Linda G. Shell, DVM, Diplomate ACVIM	Neurology
Jeff R. Wilcke, DVM, MS	Clinical Pharmacology
Jerry A. Woodfield, DVM	Cardiology

Small Animal Surgery

Mark J. Dallman, DVM, PhD	Peter K. Shires, BVSc, MS, Diplomate ACVS
Robert a. Martin, DVM, Diplomate ABVP	Claire J.A. Spackman, DVM, MS
Dale L. Rigg, DVM, MS	

Large Animal Medicine

Karen H. Baum, DVM	W. Kent Scarratt, DVM, Diplomate ACVIM

Large Animal Surgery

Robert Holland, DVM	Frank "Bimbo" Welker, DVM, MS
Paula D. Modransky, DVM, MS	

Anesthesiology

R. Eddie Clutton, BVSc, MRCVS	D. Leighton Richards, BVSc, MRCVS, DVA
Charles J. McGrath, DVM, Diplomate ACVA	

Radiology

Don L. Barber, DVM, MS, Diplomate ACVR	Martha L. Moon, DVM
Colin B. Carrig, BVSc, PhD, Diplomate ACVR	

Clinical Pathology

William R. Chickering, DVM, PhD	Cytology, Hematology
Jorgen W. Hansen, DVM, PhD	Parasitology
Ann Zajac, DVM	Parasitology

Theriogenology

John M. Bowen, BVM, FRCVS, Diplomate ACT
William B. Ley, DVM, MS, Diplomate ACT
Beverly J. Purswell, DVM, MS, PhD, Diplomate ACT
David J. Sprecher, DVM, MS, Diplomate ACT

Epidemiology

Pierre R. Lessard, DVM, MPVM	Brian D. Perry, BVMS, MSc, DTVM, MRCVS

Ambulatory Service/Production Management Medicine

Gareth A. Moore, DVM	Craig D. Thatcher, DVM, MS, PhD
Clifford F. Shipley, DVM	W. Dee Whittier, DVM, MS

CONTINUING EDUCATION OPPORTUNITIES

September 24-25-28	Small Animal Medicine Update Norfolk (24) Charlottesville (25) Charleston, WV (28)
October 2-3, 1986	Bovine Practitioners Seminar Holiday Inn - Frederick, MD
October 17-18, 1986	Small Animal Fracture Repair Lecture/Wet Lab - Blacksburg, VA
October 22, 1986	Local Associations Meeting Feline Medicine - Roanoke, VA
October 24, 1986	Equine Practitioners Seminar Anesthetic Techniques - Charlottesville, VA
November 21-22, 1986	Practical Eye/Ear Surgery Lecture/Wet Lab - Blacksburg, VA

For more information on the programs, contact:

Kent Roberts, DVM
VA-MD Regional College of Veterinary Medicine
Blacksburg, VA 24061
(703)961-7666

Virginia-Maryland Regional College of Veterinary Medicine Extension Staff:

Dr. J.M. Bowen - Extension Specialist - Equine
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
Barbara B. Jones, Managing Editor of VIRGINIA VETERINARY NOTES

COOPERATIVE EXTENSION SERVICE
U. S. DEPARTMENT OF AGRICULTURE
VIRGINIA POLYTECHNIC INSTITUTE
AND STATE UNIVERSITY
BLACKSBURG, VIRGINIA 24061