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Extension Veterinarian

THOUGHT FOR THE MONTH

With longing or regret we are tempted to look back.
With fear or hope we peer into the future.
But the time for accomplishment must forever be now.

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.

THE ROLE OF INSECT AND TICK CONTROL IN THE CONTROL OF POTOMAC HORSE FEVER

Can the knowledge we have acquired since last summer help us control Potomac horse fever (PHF) this year?

We noW know that the disease is caused by a rickettsial organism Ehrlichia sp., and can regularly reproduce the disease using this organism. We already knew that the disease was seasonal, occurring in the summer months. Many other rickettsias, and particularly other members of the tribe Ehrlichieae, are transmitted by ticks. Examples are Ehrlichia canis, transmitted by the brown dog tick, Ehrlichia phagocytophila, transmitted by the sheep tick Ixodes ricinus, and Cowdria ruminantium, transmitted by ticks of the genus Amblyomma.

Studies so far have not been able to prove that PHF is tick transmitted, but it still seems the most likely route. We do know that under laboratory conditions, virtually all horses appear susceptible to the disease, and undergo some form of clinical illness. When this is coupled with the field picture of a sporadic disease, it seems reasonable to speculate that if a tick or insect is involved, there is a very low infection rate of these invertebrates with the Ehrlichia.

Recently, we have shown that the disease can be transmitted by the intradermal route, and result in classic PHF with an incubation period of 9-14 days, the same as the intravenous route. This not only adds more weight of evidence that the disease is arthropod borne, but also suggests that mechanical transmission by biting flies or lice may possibly occur as well as the more likely biological transmission.

Last year's study of what arthropods are biting horses offers some general guidance as to when and where to apply arthropod control.

Adult ticks on horses were found principally from May to late July, with a definite peak in numbers during June. Low numbers of ticks were found on some farms much later. The tick found on horses in the region is the American dog tick, Dermacentor variabilis. This tick has favorite sites for attaching, which are the tail and the mane. This means that they often go unnoticed, and these are places which are sometimes missed by the spray or the sponge.

Other flies, such as blackflies, fed in large numbers on horses at pasture. Guidelines for the control of ectoparasites in horses produced by the Extension Service at Virginia Tech offers some useful general advice to follow, and are available from local extension agents in Virginia, or from Dr. J.E. Roberts, 215 Price Hall, Virginia Tech, Blacksburg, VA 24061.

When the mechanism of transmission has been elucidated, more specific control advice will be possible.--**Brian D. Perry, BVMS, MSc, DTVM, MRCVS and Jorgen Hansen, DVM, PhD, College of Veterinary Medicine, Virginia Tech, Blacksburg, VA; E. Craig Turner, Department of Entomology, Virginia Tech, Blacksburg, VA.**

CHOCOLATE POISONING IN DOGS

Chocolate intoxication is not rare in dogs as judged from the calls received by the National Animal Poison Control Center (NAPCC). In the last year (1983-84), close to 100 calls have been received by the NAPCC regarding chocolate ingestion and toxicosis in dogs.

The toxic principle in chocolate is theobromine, a methylxanthine compound related to caffeine and theophylline (Aminophylline®). Milk chocolate contains about 44 mg of theobromine per ounce of chocolate, while unsweetened baking chocolate contains about 450 mg per ounce. For this reason, a dog ingesting unsweetened chocolate is at a far greater risk of experiencing toxic effects.

The approximate median lethal dose (LD_{50}) for 50% of dogs exposed to theobromine is reported to be between 250 and 500 mg/kg. However, there is one case report of death in a dog after ingestion of as little as 114 mg/kg of theobromine. Therefore, to reach a hypothetical minimum lethal dose of 100 mg/kg, a 10 kg dog would have to ingest approximately 1000 mg of theobromine, which would be about 2-1/4 oz. of unsweetened chocolate or about 22 1/2 oz. of milk chocolate (about 1-1/3 lb.).

At one time, theobromine was used as a diuretic in dogs at 20 mg/kg. Therefore, according to the data available, ingestion of this amount would be expected to produce diuresis, but no other clinical signs.

The clinical signs occurring in dogs with theobromine (chocolate) toxicosis often include: 1) vomiting, 2) depression and lethargy, 3) diuresis, 4) muscular tremors, 5) diarrhea, and 6) death. Death can often be related to respiratory or cardiac arrest as theobromine can have profound cardiac effects, causing atrial tachycardia and premature ventricular contractions (PVC's). Also, it is reported that theophylline (a related xanthine) toxicosis in humans can cause cardiac arrhythmias before showing any outward clinical signs. On at least one occasion, this may have occurred in a canine chocolate toxicosis reported to the NAPCC, resulting in the acute death of the dog a day after other clinical signs had disappeared.

Treatment of theobromine poisoning in dogs is primarily based upon delaying or preventing absorption, hastening elimination, and symptomatic and supportive treatment. The treatment regimen is as follows:

1. Make certain the animal is breathing adequately, and support the cardiovascular system with fluids if needed.

2. Induce vomiting unless the animal is comatose or has lost the gag reflex, in which case, institute gastric lavage after placing an endotracheal tube and inflating the cuff. It may be difficult to recover all of the ingested chocolate because it can melt and form a ball in the stomach, not allowing it to be regurgitated or evacuated by lavage.

3. Give multiple doses of activated charcoal at 0.5 to 1.0 g/kg every 3 hours for 72 hours (perhaps shorter if the animal is clinically much improved). It has been shown in humans that multiple dosing of activated charcoal in theophylline poisonings results in a marked shortening of

the plasma half-life. It is reasonable to assume that plasma theobromine concentrations in dogs can be similarly affected. The activated charcoal is continued for 48 hours because of the very long half-life of theobromine in dogs (17 hours compared to 7 hours in humans).

4. Give a saline cathartic. Give one dose initially and a second dose at 3 hours after the beginning of the treatment. Sodium or magnesium sulfate are suggested at 0.5 g/kg diluted in at least 5 times as much water and administered orally or by stomach tube.

5. Monitor the EKG for cardiac abnormalities and treat tachycardias and/or PVC's symptomatically.

6. Secure in place a urinary catheter and allow a free flow of urine. Recent work in dogs has shown that a clinically significant amount of a related methylxanthine (theophylline) can be reabsorbed from the urinary bladder. Since 50% of a theobromine dose is excreted unchanged in the urine and another 30% is excreted as an active metabolite, this may be of some value.

7. Monitor fluid status and give replacement fluids if needed to prevent dehydration since theobromine is a diuretic.

Theobromine can be detected in plasma or serum. For submission, 2 to 3 ml of serum or plasma should be collected, frozen (tilt tube during freezing) and shipped on ice to the University of Illinois diagnostic lab where the test can be performed.**--Steve Hooser, DVM, Small Animal Prof., Topics 1984, Vol 9, #4, University of Illinois.**

WILDLIFE MAKE UNSATISFACTORY PETS

Human beings risk severe illness from a common intestinal roundworm of raccoons warns the American Veterinary Medical Association's (AVMA) Council on Public Health and Regulatory Veterinary Medicine.

According to the Council, the parasite Baylisascaris procyonis has recently been the cause of fatal infections in other wildlife species and two children. Milder infections in adults have resulted in eye damage and other symptoms. People, especially small children who habitually insert dirty hands or objects into their mouths, may become infected by ingesting parasite eggs found in contaminated dirt or food. The eggs eventually hatch into larvae, migrate through the body organs, especially the brain, eyes, and/or spinal cord and cause death.

Since raccoons are frequently found in residential and recreational areas, raccoon fecal contamination in these areas is likely and exposure to Baylisascaris procyonis is a possibility. The parasite eggs may remain infective for long periods of time.

The AVMA recommends that pets and children should not be allowed access to areas where raccoons may have been nested or caged. In addition, people should wash their hands immediately after contact with dirt or waste material.**--University of Georgia, Veterinary Newsletter, December 1984.**

FELINE HYPERTHYROIDISM

Hyperthyroidism has been recently reported to occur quite frequently in older cats. This disease is very important because thyrotoxicosis can mimic hypertrophic cardiomyopathy in the cat.

The disease is generally caused by the presence of one or two functional adenomas of one or both lobes of the thyroid. These adenomas result in elevated levels of T_3 and T_4 (>300 ug/dl and >5,9 ug/dl, respectively).

Clinical signs include weight loss in the face of increased appetite, hyperexcitability, polydipsia/polyuria, diarrhea, more frequent defecation, and increased size of one or both lobes of the thyroid gland. Radiographic signs may include mild to marked cardiomegaly and occasionally pleural effusion if congestive heart failure is present. Two thirds of the cats present with sinus tachycardia (>260 beats per minute) in the absence of excitement. Most have left ventricular enlargement (R wave >0.9 mv) and a few patients may have atrial or ventricular premature beats.

Cats affected with cardiac thyrotoxocosis are generally older (>8-9 years) while cats with idopathic hypertrophic cardiomyopathy are younger (6.5 years). T_3 and T_4 levels will render the definitive diagnosis.

The increased levels of T_3 and T_4 cause increased numbers of B_1 adrenergic receptors in the heart, increased sensitivity of those receptors to endogenous catecholamines, and increased levels of catecholamines in the myocardium. Thyroid hormones also have direct inotropic and chronotropic effects on the heart.

The treatment of choice for this disease is a combined medical and surgical approach. Propranolol, 2.5-5 mg T.I.D., should be administered for 7-14 days in order to reduce the heart rate to the normal range. Surgical excision of the adenomas should then be accomplished. Some animals may require thyroid replacement therapy following surgery in order to maintain the euthyroid state.--**James C. Keith, Jr., DVM, PhD, Cardiovascular Pathophysiology, VA-MD Regional College of Veterinary Medicine.**

PREVENTION OF FELINE LEUKEMIA VIRUS INFECTION THROUGH TESTING AND VACCINATION

Until recently, the control of feline leukemia virus (FeLV) infection in cats has depended on the accurate identification of persistently-infected (and infectious) cats and their removal from the population at-risk. This has resulted in a substantial reduction in FeLV-associated diseases and deaths in catteries and multiple-cat households where such measures have been implemented.

Presently, two tests are available to detect FeLV infection, both of which detect the group-specific p27 core antigen of the virus. The indirect fluorescent antibody (IFA) test detects p27 antigen as part of whole virus infecting circulating neutrophils and platelets. In almost all cases, a positive IFA test is consistent with persistent viremia. The enzyme-linked immunosorbent assay (ELISA) test detects p27 antigen in whole blood, plasma or serum.

It has become evident that there is discordancy in some cats between ELISA and IFA test results. Occasionally, cats are found to be transiently or persistently ELISA(+) while remaining IFA(-). Although the exact reason for this discordancy is unclear in many cases, transient self-limiting infections or persistent non-viremic "latent" infections are likely explanations. Proper evaluation of the FeLV status of ELISA (+) asymptomatic cats depends on the use of both the ELISA and IFA tests at the time of initial evaluation and repeated after 3 months.

The recent introduction of a soluble tumor cell vaccine for the prevention of FeLV (Leukocell® - Norden Laboratories) should provide the practitioner with a second means for reducing the prevalence of FeLV in the feline population. The vaccine consists of individual viral and cellular proteins which stimulate immunity to viral infection and FeLV-induced tumor formation. Since no whole virus is present in the vaccine, immunosuppression associated with FeLV protein p15e is not expressed.

It is the author's opinion that vaccination for FeLV should be incorporated into the routine care of every healthy cat, and not used selectively. This recommendation obviously depends on the observed safety and efficacy of the vaccine following commercial introduction. The efficacy of FeLV vaccination will be easier to assess if the FeLV status of cats is determined before vaccination is initiated. The author currently recommends a screening ELISA test on all cats before or at the time of the first vaccine. Further vaccination of ELISA(+) cats is then withheld for 3 months until serial ELISA and IFA tests have been evaluated. Vaccination of persistently-infected viremic cats appears to offer no health benefit.--**John R. August, B.Vet.Med., M.S., M.R.C.V.S., Diplomate A.C.V.I.M., VA-MD Regional College of Veterinary Medicine, Blacksburg, VA.**

SULFATHIAZOLE TOXICITY IN MARYLAND CALVES

A dairyman in Frederick County, Maryland had about 25 head of Holstein calves of about 150 to 400 pounds in a paved barnyard. Around December 5, weakness and coughing was noticed in some of the younger calves, and an outbreak of pneumonia was suspected.

The dairyman gave the younger and smaller calves a 240 grain bolus of Sulfathiazole (Chem-Vet) each day for five straight days. The dosage was the same each day and no provision for extra water was made.

Calves began dying around December 11 and two calves were submitted for necropsy. Both had greatly enlarged kidneys with grossly visible crystalline material in both the medulla and pelvis. Toxicologic examination of the kidneys was positive for sulfonamides but the level was not quantitated. No evidence of pneumonia was found.

Sulfathiazole has been discontinued for use in the human because frequent toxicosis has occurred. It is still available for use in livestock, however. In this case, the sulfa was used indiscriminately and without veterinary consultation. The primary disease in the calves was thought to be malnutrition.--**Stauffer Miller, DVM, Maryland Department of Agriculture, Animal Health Laboratory, Frederick, MD 21701.**

EARLY FLU VACCINATION FOR FOALS

Drs. Irwin Liu and David Pascoe of the Department of Reproduction, University of California at Davis, have completed a project designed to determine the duration of maternally-derived protection antibodies against influenza in foals. The significance of their study is based upon the fact that once antibodies against influenza in foals have deteriorated due to natural antibody decay, the foal is no longer protected against exposure and infection by the influenza virus. At this stage, vaccination against influenza would be prudent if one is to prevent respiratory infection in foals caused by influenza virus.

Foals, 18 in all, were tested for the presence of protective antibodies against influenza at bi-weekly intervals, from day one to five months of age. Nine of these test foals were born to mares vaccinated against influenza one month prior to foaling date. The nine foals in the second group were born to mares that were not vaccinated against influenza for a period of at least six months prior to foaling.

Laboratory data indicated that at least half the foals in both groups were not protected against influenza by the time they reached two months of age; a few foals from both groups were not protected against influena at two weeks of age. These data suggest that many foals were unprotected against influenza infection at two months of age. This means that the general practice of administering the initial influenza vaccine injection in foals at four months of age may be too late to prevent influenza infection.**--Drs. Irwin Lie and David Pascoe, Department of Reproduction, University of California at Davis, as reported in the Nebraska Veterinary Extension Newsletter, November 1984, University of Florida, Vet Med Newsletter, January 1985.**

REPTILIAN PRACTICE PROBLEMS
Dysecdysis

Reptiles have a scaly skin which is shed periodically. This shedding process is a function of several factors including growth rate, temperature, light, and humidity. Improper shedding of skin is called dysecdysis and can be caused by abnormalities in any of the variables mentioned. Sometimes the problem may be as simple as a lack of a rough surface for the animal to rub against.

The abnormal retention of skin can predispose the reptile to other problems such as bacterial or fungal infections. Occasionally the skin can form a constricting band and cause ischemia in the tail or appendages.

Turtles shed a scale or two at a time and thus very rarely have any shedding problems. Lizards tend to shed in patches and only occasionally have any difficulties. Snakes shed their entire skin at once and it is in this species that dysecdysis is most commonly seen. Clinically, a snake with dysecdysis will usually have a dull, dry skin and cloudy eyes. The cloudy eyes are due to retained spectacles which are a snake's equivalent to eyelids and are normally shed with the rest of the skin. Not uncommonly, a snake will be able to shed all of its skin except the spectacles.

Therapy is based on moistening and softening the skin with water or saline and then gently peeling the skin away with tweezers. Often one can place a reptile in a box with water-soaked burlap (or similar rough cloth) and the animal will remove the problem without further assistance.**--Donald K. Nichols, DVM, National Zoological Park, Washington, D.C.**

CONTINUING EDUCATION OPPORTUNITIES

April 18-19, 1985	VVMA Management Seminars Charlottesville Hilton - Charlottesville, VA
April 19, 1985	Feline Medicine & Surgery Seminar Holiday Inn - Columbia, MD
April 25, 1985	Local Associations Meeting Donaldson Brown Center - Blacksburg, VA
May 3, 1985	Food Animal Medicine Seminar Holiday Inn - Harrisonburg, VA
May 5, 1985	Canine Nutrition Seminar Dr. Francis Kalfelz - Richmond, VA
June 13-15, 1985	VVMA Summer Mini-Convention Bernards Landing - Smith Mountain Lake
June 16-19, 1985	Avian Medicine Conference Columbia Inn - Columbia, MD

For more information on these meetings, contact:

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