Virginia Cooperative Extension Service

VIRGINIA VETERINARY NOTES

VIRGINIA-MARYLAND REGIONAL COLLEGE OF VETERINARY MEDICINE

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WHAT'S INSIDE!

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Extension Veterinarian

VVMA Summer Sojourn

The Virginia Veterinary Medical Association summer meeting will be held at Wintergreen in the Blue Ridge Mountains, Nelson County, June 14-16, 1990.

THOUGHT FOR THE MONTH

Failure is the opportunity to begin
again, more intelligently.

--Henry Ford

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. James F. Johnson, Acting Director, Virginia Cooperative Extension Service, and Acting Vice Provost for Extension, Virginia Polytechnic Institute and State University, Blacksburg, Virginia; Clinton V. Turner, Administrator, 1890 Extension Program, Virginia State University, Petersburg, Virginia.

CANINE HERPESVIRUS

Canine herpesvirus (CHV) is similar to other herpesviruses in that it is species specific. Most infections are subclinical during which latency is established. No vaccine is available so control of the disease is mandatory to reduce the clinical problems associated with CHV. A good review of companion animal herpesvirus infections has been provided by J.F. Evermann, Proceedings, Society for Theriogenology Annual Meeting, 1989.

The CHV is spread primarily by aerosol and direct contact of oral nasal secretions. Veneral spread of CHV is possible. Dogs are most susceptible to infection during pregnancy, the first three weeks of life, and other times of immunosuppression. If clinical signs develop in the adult, upper respiratory signs are the most common. They may even be seen in conjunction with the kennel cough complex of parainfluenza virus, adenovirus type 2, and Bordetella bronchiseptica. In neonatal puppies, a severe fatal infection is seen. The bitch and her puppies are unusually susceptible during the last three weeks of pregnancy and the first three weeks after whelping.

Diagnosis of CHV can be accomplished using serology. Serology indicates prior exposure to CHV by the animal but does not correlate well with viral shedding or contagiousness of the virus. Histopathology provides a presumptive diagnosis with intranuclear inclusion bodies seen in the liver and kidney. Necropsy of dead or dying puppies is necessary for a diagnosis. If available, viral isolation can be attempted on oral nasal and/or urogenital secretions.

Control of CHV is the only protection against the fatal forms of CHV. Up to 85% of the dog population has been exposed or infected with CHV. High risk animals are those dogs which come into contact with many other dogs, such as show dogs or dogs housed in multiple dog facilities. Quarantine of the bitch in the last weeks of gestation and quarantine of the bitch and puppies in the first three weeks after whelping will help eliminate neonatal puppy losses. Eradication of the virus is not feasible due to the wide-spread prevalence of the virus. As with other herpesvirus infections, current thinking is that the dog remains infected for life. Herpesviruses are very labile outside the body. Common disinfectants (bleach, water) are effective in controlling the environmental spread of CHV. **--B. J. Purswell, DVM, PhD, Diplomate, ACT, VA-MD Regional College of Veterinary Medicine, Blacksburg, VA**

B-MODE REAL TIME ULTRASOUND OF THE CANINE FETUS

Between 14 and 16 days of gestation, fetal sacs may be detected with close inspection. By day 20 or 21, the fetal pole with a fetal heart beat can be detected. Fetal membranes become evident and change in position between days 21 and 30. Fetal structures also are beginning to be recognized during this time. By day 31 to 33, fetal motion and early fetal mineralization is observed. This mineralization is first detected in the fetal mandible. Mineralization of the fetal skull and spine follow rapidly. Visceral organs such as the heart, liver and bladder become recognizable at around days 33 to 36. There is not a good distinction between the thoracic cavity and the abdominal cavity until approximately day 45. At that time the lungs become more hyperechoic (i.e., seen as white on ultrasound screen). Fetal vessels are identified approximately by day 35. By day 39, the fluid-filled stomach, the ventricles of the brain and the fetal blood flow in the umbilical vein, artery and ductus venosus are clearly defined. The kidneys are identified by day 43. Mineralization of the ribs is sufficient to create shadows over the thorax. By day 53, the visceral organs, including the duodenum, and stomach arteries are distinctly seen. The thymus and cranial mediastinum and faint motility are seen by days 58 to 63.

Fetal death is identified by the lack of a fetal heart beat. There may be differences between fetuses in size and maturity that may suggest a potential problem with a fetus. A slowed heart beat may be indicative of probable fetal demise. After fetal death, the normal echoic appearance between the thoracic and abdominal cavities (after fetal age of 45 days) become confluent. Visceral organs become indistinct and hypoechoic. The fluid in the fetal sac changes from anechoic (i.e., seen as black on ultrasound screen) to echoic. Gas may be detected in the fetus. **--Kathy Spaulding, DVM, North Carolina State University, Raleigh, North Carolina, as reported in Veterinary Quarterly Review, Texas A & M, Winter, 1989-90.**

USE INSECT REPELLENTS CAREFULLY ON CATS, CORNELL PHARMACOLOGIST WARNS

Cat owners, concerned that their pets may bring ticks carrying the organism responsible for Lyme disease into the house, may poison their cats if they apply excessive amounts of insect repellents with the chemical DEET on the animals, a veterinary pharmacologist at Cornell University warns.

Instead, concerned cat owners should keep their pets indoors. Pharmacology professor Wayne S. Schwark has advised veterinarians to be on the lookout for signs of DEET poisoning in cats. Symptoms include seizures, tremors, or loss of coordination, salivation and vomiting.

Health officials in New York, New Jersey, and Connecticut issued a warning in August about excessive use of DEET repellents on human beings, especially children. Overexposure to DEET causes neurological problems in humans, including seizures, comas, and slurred speech.

DEET is the abbreviation of N, N-diethyl-M-toluamide, an ingredient in some insect repellents now being advertised as protection against the deer tick that carries Lyme disease bacteria in the northeastern United States.

When deer ticks carrying the Lyme disease bacteria were found on white-footed mice in the Northeast, some cat owners became concerned that their mouse-catching pets could spread the infection to the family. "That's no reason to slather the cat with insect repellent," Schwark said.

"Cats are more vulnerable than humans--or even dogs--to most toxic substances," he explained. "They don't have an abundance of the enzymes that break down toxic substances in the liver. That's why cats are easily poisoned by overdoses of drugs such as aspirin that are usually considered innocuous." Kittens and old cats are particularly susceptible to toxic substances.

"Lyme disease is one more reason to keep cats indoors," the pharmacologist said, citing feline leukemia, rabies, and automobiles as other hazards of the outdoor life. If confining a cat indoors is not possible, said Dr. John Saidla, extension specialist in the Cornell Feline Health Center, some of the pinhead-sized ticks may be removed with a fine-tooth flea comb. Many of the antiflea products approved for cats have some repellent action against ticks, he added. **--Cornell University College of Veterinary Medicine Veterinary Viewpoints, Number 4, 1989. As reported in Animal Health Beat, University of Nevada, Reno, Jan. 1990.**

THE MENINGEAL WORM

Parelaphostrongylus tenuis is also called the deer meningeal worm, deer worm or meningeal worm. It can live in the white-tailed deer (Odocoileus virginianus) without causing clinical signs. The white-tailed deer is a definitive (natural) host for P. tenuis. It exists predominately in eastern North America.

The infective larvae (L3) are consumed by the host (deer), migrate to the CNS (central nervous system) where they develop into adults. Eggs are laid and develop into L1 larvae, which migrate within the deer. L1 can be coughed-up, swallowed and passed in the feces of infected deer.

The L1's passed in the feces of the definitive host (white-tail deer) gain entry into terrestrial gastropods (snails or slugs), where two molts occur resulting in infective larvae (L3). The L1's enter the gastropods through ingestion (being eaten with normal forage) or through penetration of the feet or other body surface. The snails and slugs, which are intermediate hosts, generally favor a moist or wet environment of organic material with a diet of leaf litter or vegetation (some species prefer lettuce and carrots!) plus available dietary calcium carbonate. The proper environment (including moisture) and its dietary requirements plus extended daylight are important for optimal fertility and activity of these terrestrial gastropods.

The survival of free-living L1's are reduced by repeated freezing or drying. Experimentally, a similar parasite displayed similar survival of L1's at 45% relative humidity in air as in water at 5°C (41°F) and 14°C (59°F). Under optimal conditions there was a 50% survival for this L1 parasite an average of 200-300 days!

Once the larvae molt in the snail, survival of L3 outside the snail is thought to be short-lived, especially if not in water. The question arises as to how long the larvae can remain in the gastropod (snail) and what triggers their exit.

When unnatural hosts (eg. llamas, goats, sheep, elk, moose, reindeer) ingest the infective L3, migration through the body occurs. Clinical signs recognized are usually referable to the central nervous system, especially the spinal cord (paresis, ataxia, staggering, inability to rise, weakness, etc.). Migration to the CNS in white-tailed deer takes a minimum of 10 days, while experimental data in llamas indicates it takes 8 weeks to see clinical signs following ingestion of L3.

Treatment depends on the duration and severity of clinical signs. Antiinflammatories are indicated to reduce the reaction to the parasite. Phenylbutazone (bute) has been safely used on llamas, especially if the patient is eating, without the hazards of steroids. Dewormers have questionable penetration into the CNS to kill the larvae, but should be effective against migrating parasites, before or after CNS penetration. Ivermectin at the cattle dose or fenbendazole at 30-50 mg/kg daily for 3-5 days orally, with concomitant use of antiinflammatories, seems to be beneficial. Gastrointestinal parasites (except for coccidia) are also killed by these dewormers. Physical therapy needs to be diligent and persistent. Recovery may take several weeks, to months, as the nervous tissue heals very slowly. Some residual signs may be present and these are usually permanent if still present one year following the onset of signs.

Definitive diagnosis is based on identification of the parasite at necropsy. Presumptive diagnosis is based on clinical signs, history (exposure to white-tailed deer and terrestrial gastropods), elimination of other diseases, eosinophils

in the CSF (cerebral spinal fluid) and findings of characteristic changes in the CNS histologically.

There is no commercial vaccine currently available, so prevention is based on management principles. Elimination of exposure to deer, snails and L3 is ideal but often impractical (without relocating geographically). Reduction of exposure along with frequent deworming to kill the parasite before penetration into the CNS seems to be the most feasible program. Although very little work has been done to prove the efficacy of different dewormers, ivermectin and fenbendazole should be effective. These need to be given every 2 weeks to 2 months, depending on habitat, climate and exposure risks. **--Karen H. Baum, DVM, VA-MD Regional College of Veterinary Medicine, Blacksburg, VA.**

FELINE CHLAMYDIOSIS

Infections with Chlamydia psittaci should be suspected when upper respiratory disease in cats is characterized by recurrent or persistent follicular conjunctivitis. Rhinitis, an unusual finding, is mild when present. The organism often affects kittens 5 to 12 weeks of age, and may cause neonatal conjunctivitis. Once enzootic in a group of cats, clinical signs may persist in individual cats for several weeks and recurrences are common. The organism has been incriminated in a chronic respiratory disease with a mortality rate of 33% in kittens 10 to 14 days of age. Most upper respiratory disease in cats is due to infections with feline herpesvirus I or feline calicivirus.

Natural immunity to C. psittaci appears to be incomplete and recovered animals have only partial immunity to reinfection. Control of enzootic disease in catteries depends on identification and treatment of all infected cats, and vaccination. Using modified live cell line-origin strains of C psittaci, available vaccines provide infection lasting 1 year or more. Upon challenge, vaccinated cats may show mild signs of short duration, but do not suffer from the protracted disease typical of natural infections in unvaccinated animals.

Because there is little interference from maternal antibodies, the age of vaccination is not critical, and one immunization usually affords protection against severe disease. When chlamydial conjunctivitis occurs in young kittens, vaccination may be initiated as early as 3 weeks of age. **--By John R. August, Texas A&M Univ. as reported in Washington State University Animal Health Notes, July, 1989.**

CHLAMYDIOSIS IN BIRDS

This is a common disease in several species of birds, both psittacine and non-psittacine. The infection can range from subclinical to fatal. The "droopy bird syndrome" is often used to describe the infected birds. Conjunctivitis is a common sign along with a fecal pasted vent. There may be feather loss, discharge from the nostrils and reluctance to move. Young birds apparently are most susceptible to Chlamydia psittaci.

The lungs are usually edematous and hyperemic. The air sacs are often thickened and edematous. The liver is usually swollen, mottled and off color. Enlargement of the spleen with subcapsular hemorrhage is frequently seen, especially in psittacine birds. **--Ed K. Daniels, DVM, Deborah J. Briggs, M.S. Diagnostic Laboratory, Kansas State University as reported in Animal Health Beat, University of Nevada, Reno and Animal Health Notes, Washington State University, Jan 1990.**

DOG HOOKWORMS TRANSMITTED IN MILK

Hookworms are bloodsucking parasites which infect man and animals. Several species affect dogs but the most common and most pathogenic is Ancylostoma caninum. Adult hookworms attach to the wall of the intestine and feed on plugs of tissue, causing internal bleeding. When several hundred hookworms are present, intestinal bleeding can lead to life threatening anemia, especially in puppies. Signs of anemia include paleness of the gums, dark tarry stool and weakness.

Adult hookworms in the dog's intestine lay eggs which are shed in the feces. Eggs develop in the feces and hookworm larvae hatch out and continue to develop to the infective stage. Infection of dogs occur when hookworm larvae penetrate the skin or are ingested with contaminated food or water. Thus, dog food should never be placed directly on the ground. Daily removal of feces before the eggs hatch and frequent cleaning of bowls will markedly reduce exposure to infective larvae.

Puppies become infected with Ancylostoma caninum primarily through their mother's milk. Adult dogs, besides harboring adult hookworms in their intestines, accumulate the immature larval stage in muscles and mammary glands. During nursing, these hookworm larvae pass in the milk to puppies. Most of the larvae will pass in the milk during the first week, but some larvae are transmitted throughout lactation. These larvae will grow to egg-laying adult hookworms about 2 weeks after infecting puppies. Therefore, a pup as young as 2 weeks old can be infected and contaminating the environment with hookworm eggs.

If large numbers of hookworms are present, puppies will be anemic at about 2 weeks of age and may die before 3 weeks. Most hookworm infections do not result in death but low to moderate numbers of worms reduce growth and well-being.

Veterinarians deworm puppies at an early age (2, 4, 6, and 8 weeks) to eliminate hookworms as well as ascarid larvae which puppies acquire through the milk of their mother. Adult dogs should be routinely dewormed twice yearly to eliminate adult worms from the digestive tract, but such treatments are not effective against larvae in the tissues. Treatment to destroy these quiescent larvae is difficult. One drug, fenbendazole, reduced ascarid and hookworm burdens by 90 percent in puppies from treated dams, while puppies from unmedicated dams acquired large numbers of hookworms. Treatment for the pregnant female dog should begin about the 40th day of pregnancy and continue daily until 2 weeks after whelping, that is, about 37 consecutive days. Except in special cases, this extensive regimen is not routinely used since it is more practical to treat puppies as described above. **--Dr. Edward Roberson, University of Georgia, College of Veterinary Medicine, Athens, GA. AAVP News Brief, Vol 1, Winter 1990**

RABIES VACCINE LICENSED FOR USE IN FERRETS

On February 17, 1990, the Federal Department of Health and Human Services, Food and Drug Administration and the Department of Agriculture licensed the first Rabies vaccine for use in ferrets of three (3) months old or older. Known by the trade name Imrab and Imrab-1, the vaccines are manufactured by Rhone Marieux, Inc., of Athens, Georgia and distributed by Pittman-Moore, Inc. of Mundelein, Illinois. **--Washoe County District Health Department letter, February 27, 1990 as reported in Animal Health Beat, University of Nevada, Reno, March 1990.**

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

Date	Program	Location	Contact Hours
May 12	Food Animal Practice Seminar	Staunton	6
Sept 27	Small Animal Medicine Update	Charlottesville	4
October 11	Cardiodiagnostics	Charlottesville	6
*Nov 9-10	Gastrointestinal Endoscopy	Blacksburg	8
*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

*Limited enrollment course

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

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COLLEGE NEWS

New faculty members who have recently joined the College of Veterinary Medicine in Blacksburg are:

Bernard Feldman, DVM, Ph.D. is a graduate of the University of Illinois, College of Veterinary Medicine who has earned an international reputation as a clinical pathologist. He completed a residency at Kansas State University and received a Ph.D. from the University of California--Davis, where he was a professor of veterinary clinical pathology before joining the faculty in Blacksburg on January 2, 1990.

David Moll, DVM, MS is a Kansas State University graduate who completed an MS degree at Auburn University following a residency in Ocala, Florida and an internship at Auburn. He is an assistant professor of large animal surgery and is a Diplomate of the American College of Veterinary Surgeons. He joined the Veterinary Teaching Hospital staff on March 1, 1990.

Alumni Gathering

The veterinary college Alumni Association is planning a weekend of continuing education and social events in Blacksburg on September 14-16, 1990. This will coincide with the College's celebration of its Tenth Anniversary.

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
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