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PRACTICE TIPS - EQUINE FLUID THERAPY	Page 2
FELINE HYPERESTHESIA SYNDROME	Page 2
THOUGHT FOR THE MONTH	Page 3
EQUINE GASTRIC ULCER SYNDROME	Page 4
CONTINUING EDUCATION OPPORTUNITIES	Page 6
THE USE OF ASPIRIN IN CANINE DIROFILARIASIS	Page 6
SUCCESS IS NO ACCIDENT	Page 8

Kent C. Roberts, DVM
Extension Veterinarian

PRACTICE TIPS - EQUINE FLUID THERAPY

The placing of an IV catheter should be conducted as a sterile, surgical procedure. If properly placed a catheter can remain in a horse for two weeks.

A good catheter flush is 3 ml heparin in 500 ml of physiological saline solution.

Super glue often works better than sutures to hold an IV catheter in place.

A good rule of thumb for giving IV fluid therapy is to run in 2/3 of the total volume rapidly and the final 1/3 slowly. It is possible to produce pulmonary edema in horses and very possible in foals.

Warm cold fluids in a microwave for approximately two minutes prior to administration.

IV fluids in neonates should be given at the rate of 500-1000 cc/hour using a 5-1/2 inch 16 gauge catheter. Saline and dextrose is better for neonates than lactated ringers solution. They may suffer from severe electrolyte imbalance.

The best vessels, other than the jugular vein, for IV administration are the lateral thoracic and then the cephalic vein.

Best results are most often attained when the patient has good renal function, receives isotonic fluids in adequate amounts and is well monitored.--**Dr. Doug Byars, Lexington, KY, as presented at IV Nutrition & Fluid Therapy for the Horse, Equine Medical Center, Lessburg, VA, November 1985.**

FELINE HYPERESTHESIA SYNDROME

The feline hyperesthesia syndrome (FHS) is a specific entity in cats that seems to be increasing in incidence. It has been referred to as neurodermatitis, rippling, rolling, or twitchy skin syndrome, neuritis and even hyperkinesis. It is a complex problem and is probably an external manifestation of a number of problems. It is doubtful if there is just one cause.

The signs can develop at any age, but usually begin to be a problem between 1-4 years of age. Most of the signs are exhibited in moderation by many cats; but in FHS cats the signs are carried to extremes so that normal activities are curtailed. The cats may show all or any combination of the following: rippling the skin of their back; biting or licking at their tails, flanks, or pelvis; apparently hallucinating; going glassy-eyed with widely dilated pupils; meowing frantically; swishing their tails; stopping to lick violently when running or walking or eating; running crazily around the house; and attacking objects including the owner without provocation. The cats may even have generalized seizures and associated social problems such as urinating elsewhere than the litterbox. Signs may last a few seconds to a few minutes, may occur at specific times of the day, may vary in incidence from month to month, and may occur once every few days or almost constantly all day. Between episodes the cats seem peaceful, but they may wake up suddenly at night and attack their owners. Both sexes, neutered or intact, are affected.

Many cats, especially kittens, do some of these things so the FHS diagnosis is probably overused. Diagnosis is difficult and is based largely on the history and ruling out other differential diagnosis. Many of these cats exhibit an excessive response (glassy eyed, dilated pupils, biting themselves or the clinician, licking their flanks, or twitching their back) when scratched by one finger over the L₇S₃ area in the midline. Normal cats react similarly but to a lesser extent to this presumably sexual stimulation.

Occasionally these cats may run a fever (less than 106°) but exhibit no white blood cell changes. In a few severe cases, marked EEG changes are apparent which disappear if treatment is successful. In cats we have necropsied, no significant lesions have been found.

There are a few things that this disease is not; it is not caused by lesions of the tail, anal glands, flank, back, skin, gut, ovarian or uterine stump ligatures or clips. Topical local therapy is pointless. The most likely causes seem to involve an interaction of the cat's basic personality (affected cats are often nervous, loners, hyperexcitable), frustrations (not being allowed outside, boredom, being ignored) and environmental pressures (overcrowding, sexual competition, another cat has cystitis, or is spraying). The signs seem to be external manifestations of mental problems. Rarely does any systemic disease seem to be the cause. However, some cats have obvious EEG abnormalities (slow waves, dysrhythmias, spike discharges) that disappear when treated. Some cats seem to respond to a simpler diet with fewer preservatives. Hence, it is likely that FHS can be seen from a combination or a variety of single causes. FHS apparently lies mainly in the area of the behaviorist, but may also have or be exacerbated by organic causes (toxins, dietary preservatives, minimal brain damage). It is in an area between sleep, dreaming, behavior, hallucinating, and epilepsy.

Treatment involves test therapy with all of, a combination of, or one of the following: megestrol acetate 1 mg/kg/day, prednisolone 1-2 mg/kg/day, phenobarbital 4-10 mg/kg/day in divided doses, diazepam 2-5 mg/kg/day in divided doses. In addition, behavioral or environmental pressures should be reduced and a food with less preservatives used. In some cats, I have found that more attention when the cat is normal combined with a whack (or waterspray) on the rump whenever signs are seen helps to reduce the problem. Once a successful treatment is found, the dose or combination of the drug(s) should be reduced as far as possible. Megestrol acetate can usually be cut to 1 mg/kg given every 3-4 days and prednisolone to 1-2 mg/kg given every 2-3 days. The cats can often be taken off drugs for prolonged periods but signs usually recur at irregular intervals. One must explain the side effects of these drugs to the client; polyphagia, polyuria, obesity, diabetes.—(by Alan Parker, DVM, PhD, DACVIM, University of Illinois College of Veterinary Medicine. Small Animal Professional Topics, V9 #4, 1984) Kansas State University, Notes From Extension Veterinarians, July 1985.

THOUGHT FOR THE MONTH

What lies behind us and what lies before us are small matters compared to what lies within us.

Ralph Waldo Emerson

EQUINE GASTRIC ULCER SYNDROME

The recognition of gastric ulcers in horses ranging in age from day-old neonates to yearlings and older has increased in the last few years. This recognition is probably due to an enhanced awareness of the problem rather than an increase in the incidence. In fact, the actual incidence has not been determined and varies considerably from one farm to another. This inability to characterize the actual incidence has occurred in part because most affected animals probably do not even demonstrate any clinical signs of having ulcers. For instance, a recent report from the University of Florida evaluated necropsy findings on 322 foals. Of these, 23% had gastric and/or duodenal ulcers. Of those with ulcers, only 40% had demonstrated any clinical signs associated with ulcers. Most foals affected were less than 2 months old, but weanlings were also affected.

The causes of gastric/duodenal ulcers in the equine species are, as in man, multifactorial. And, as in man, the direct cause of the production of the ulcer is not known. However, the occurrence of ulcers in horses is frequently associated with some stressful condition or circumstances, such as concurrent illness, surgery, weaning, or being at the low end of the herd or group social hierarchy.

Recognized clinical syndromes include the following:

1. **"Silent" ulcers** have no observable clinical signs.
2. **"Active" ulcer** clinical signs are observed and include inappetance, odontoprisis, foal lying on its back or its side with its front limbs tucked underneath, excessive salivation, and gastroesophageal reflux of feed or milk from the nostrils. Nursing foals with ulcers will frequently begin to nurse, but then abruptly stop. This behavior may be repeated on several occasions.
3. **Perforating ulcer** can occur peracutely with no previous clinical signs. Some foals will die acutely, while in others the omentum may temporarily seal the perforation. Clinical signs can include **fever**, depression, inappetance, intermittent colic, septicemia, peritonitis, and abdominal distension. Peritoneal fluid will have an increased cell count and protein level, and may also contain intra- and extracellular bacteria. The severity of the accompanying peritonitis and subsequent adhesion formation gives this condition a poor prognosis.
4. **Gastric or duodenal obstruction** occurs secondary to fibrosis which accompanies healing of pyloric or duodenal ulcers and can lead to gastric reflux, pancreatitis, and cholangitis. Surgery is the appropriate therapy, with a variety of techniques employed to by-pass the obstructed bowel.

Diarrhea may occur secondarily in all clinical syndromes and is often the presenting complaint.

There are several diagnostic procedures to detect the presence of gastric ulcers. Fecal occult blood will often be positive with ulcers, although a positive result does not diagnose ulcers. Visible or occult blood in gastric contents is also very suggestive of ulcers. Endoscopy of the stomach is the most accurate and useful procedure, but is limited by the diameter and length of the scope in relation to the size of the patient. Patient cooperation can also be a limiting factor.

Endoscopy can reveal discreet, focal, and multi-focal ulcers, as well as areas of mucosal sloughing. The latter appears as thin layers of epithelium peeling off the mucosal surface.

Contrast radiography of the stomach and duodenum is useful in the diagnosis of obstruction secondary to ulcers, and could be performed on smaller foals in many practices.

Therapy of gastric ulcers in horses has been empirical, being based on treatments used in human medicine. Although gastric acid secretion does not directly initiate ulcers, gastric acid can exacerbate the problem, and treatment has centered on inhibition of acid secretion via specific histamine (H-2) receptor antagonists. Two drugs currently in widespread use are cimetidine (Tagamet, SK&F Labs Co.) and ranitidine (Zantac, Glaxo Inc.). Both are available in preparations for oral and IV use. The oral and IV dosage of cimetidine is 1 mg/lb, QID. In foals, this dose causes rapid inhibition of gastric acid secretion that lasts for 2-3 hours. The dosage of ranitidine in foals is not clearly established, but the oral dose of 1 mg/lb, BID, and an IV dose of 0.25-0.50, BID, appear valid. These dosages should inhibit acid secretion for 6-8 hours. Another apparent advantage of ranitidine is that it lacks the potential feminizing effects associated with the long-term use of cimetidine in some men.

The duration of therapy for either drug in the equine species has not been determined. Some patients have responded to a 2 week course of therapy, while others have required several months of continuous treatment.

Another useful medication is sucralfate (Carafate, Marion Laboratories, Inc.), a sulfated polysaccharide. Sucralfate literally binds to exposed gastric mucosa, and thus protects this vulnerable mucosa from damage by acid. Sucralfate requires an acidic medium to be effective, and if administered in conjunction with cimetidine or ranitidine should be given 3 hours after cimetidine and 8 hours after ranitidine. The usual dosage is 1-2 gm, 2-3 times daily.

It is important to recognize that many foals and weanlings with gastric ulcers must remain NPO until severe clinical signs have abated. These patients will require intravenous fluid therapy, and in the case of neonates, parenteral feeding. Signs of septicemia, if present should also be treated concurrently.

Although gastric ulcers are reported to occur most commonly in young foals, we have seen several cases of gastric and/or duodenal ulcers in weanlings, both ante-and-post mortem. Not all of these had clinical signs of ulcers. Additionally, a few yearlings and one 3 year-old have been diagnosed via endoscopy as having gastric ulcers. Clearly, this problem is more prevalent in the horse population than previously appreciated, and should be considered a potential problem in any young horse either exhibiting appropriate clinical signs or subjected to stressful events that are associated with the development of gastric ulcers.--**Michael J. Murray, DVM, MS, Marion duPont Scott Equine Medical Center, Virginia-Maryland Regional College of Veterinary Medicine.**

CONTINUING EDUCATION OPPORTUNITIES

January 9-10, 1986	Dairy Cattle Nutrition Short Course - Blacksburg, VA
February 7, 1986	Emergency Care for the Equine Patient CVM Clinicians - Blacksburg, VA
February 21-22, 1986	Virginia Veterinary Medical Association Annual Meeting - Norfolk, VA
March 28-29, 1986	Small Animal Surgery Short Course Blacksburg, VA
April 6, 1986	Pet Bird Medicine Seminar Richmond, VA

For more information on these meetings, contact:

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VA-MD Regional College of Veterinary Medicine
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THE USE OF ASPIRIN IN CANINE DIROFILARIASIS

For the past three years we have been advocating the use of aspirin (5 mg/lb) as an adjunct to other therapy for dirofilariasis. Aspirin decreases obliterative endarteritis, the lesion caused by adult *D. immitis* in the pulmonary arteries, and significantly reduces post-adulticide thromboembolism following caparsolate therapy. Articles concerning this use of aspirin have appeared in Virginia Veterinary Notes, and continuing education seminars on dirofilariasis have been conducted in Virginia, Maryland and West Virginia.

There is a need to obtain and analyze information on the use of aspirin in dirofilariasis. We hope to improve the use of aspirin in effectively treating heartworm diseases through the collection and dissemination of such information. Your cooperation in completing and returning the following survey will be sincerely appreciated.—**James C. Keith, Jr., DVM, PhD, Virginia-Maryland Regional College of Veterinary Medicine.**

Please return completed survey forms to:

Dr. Kent Roberts
College of Veterinary Medicine
Virginia Tech
Blacksburg, VA 24061

Name _____ City _____ State _____

1. How many cases of canine dirofilariasis do you see per year? _____
a. 10 b. 20 c. 30 d. 50 e. 100
2. How many cases do you treat per year? _____
a. 10 b. 20 c. 30 d. 50 e. 100
3. Do you use the standard 2 day Caparsolate therapy followed by Dizan 6 weeks later?
a. Yes b. No
If no, please include your therapeutic regimen.

4. Do you use aspirin as an adjunct therapy in canine dirofilariasis?
a. Yes b. No
5. What percentage of thromboembolic disease, i.e. fever, coughing, depression 8-12 days following caparsolate therapy, were you experiencing prior to beginning aspirin therapy?
a. 1% b. 3% c. 5% d. 10% e. 20%

After beginning to use aspirin in the post-caparsolate period?

- a. 1% b. 3% c. 5% d. 10% e. 20%
6. How many times per day is aspirin administered?
a. once b. twice c. three times
7. What dose of aspirin do you use in the post-caparsolate period? _____
a. 1 mg/lb b. 3 mg/lb c. 5 mg/lb d. 10 mg/lb e. 15 mg/lb
How many times/day? _____
8. Do you use aspirin therapy prior to caparsolate in advanced cases of dirofilariasis where right-sided congestive failure is present? _____
a. yes b. no
9. Do you use aspirin as an alternative to caparsolate therapy in geriatric patients or in patients with hepatic or renal disease? _____
a. yes b. no
a. once b. twice c. three times daily
a. 1 mg/lb b. 3 mg/lb c. 5 mg/lb d. 10 mg/lb e. 15 mg/lb
10. Please make additional comments concerning the use of aspirin in canine dirofilariasis.

Thank you for taking the time to complete this survey.

Please return by February 15, 1986.

SUCCESS IS NO ACCIDENT

Veterinarians are almost unanimous in voicing their concern for quality in their practices. How many achieve this much sought and highly prized goal of a "quality practice"? How is quality measured in a veterinary practice?

The answers to these questions are not available but we do know what lies behind quality performance: human commitment. People caring about their work and how they perform each day. People who are committed to doing their best as they go about each task whether that task is routine or difficult. People who know that what they do is important in the overall effort.

Are you committed to quality in your veterinary practice? Do you help your co-workers understand their importance to the practice and their role in assuring quality veterinary care?—**Kent C. Roberts, DVM, Virginia-Maryland Regional College of Veterinary Medicine.**

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