LETTERS TO THE EDITOR

LETTRES À LA RÉDACTION

Cold Exposure of Sheep

Dear Sir:

I am writing this letter to express my concerns regarding the article "Responses of pregnant ewes and young lambs to cold exposure" which appeared in Can Vet J 1987; 28: 181-186. The objectives of this study were "to compare the clinical signs, certain biochemical changes and postmortem lesions in lambs exposed to cold and warmer climates". The experiment also compared clinical signs, certain biochemical changes and the performance of cold exposed, shorn ewes to data from ewes kept at warmer temperatures.

This experiment did not conform to regulations of the Canadian Council on Animal Care nor the U.S. Guide for the Care and Use of Laboratory Animals. Lambs exposed to 0°C and −10°C became weak and anorexic and death was associated with hypothermia and starvation. Cold exposed lambs displayed subcutaneous hemorrhage and edema of the distal limbs. The lambs must have been in considerable distress at the time of death or when euthanized.

The exposure to cold to induce pathology clearly constitutes a painful experiment and this kind of experimentation would not be approved by any Canadian Animal Care Committee. The data, although less controlled, could be obtained by field work and indeed much of this data has been previously reported (see references in article). I believe that the use of animals in experimental procedures is necessary but we all must be careful to be humane. Clearly, the ethics of animal experimentation in the CCAC Guide to the Care and Use of Experimental Animals was not followed. I believe that at least four guiding principles were neglected:

— In test procedures the investigator should be especially cautious with tests which may cause pain and distress. Acceptance should not be based on cheapness and ease of application.
— Investigators have a moral obligation to abide by the humanitarian dictate that experimental animals are not to be subjected to unnecessary pain or distress.
— Experiments involving the withholding of food and water should be short-term and have no detrimental effect on the health of the animal.
— Investigators must be especially prudent in their use of the following procedures:
  a) experiments involving withholding pre and postoperative pain-relieving medication;
  b) paralyzing and immobilizing experiments where there is no reduction in the sensation of pain;
  c) electric shock as negative reinforcement;
  d) extreme environmental conditions such as low or high temperatures, high humidity, modified atmospheres, etc.

  It must be understood that the degree of pain involved should never exceed that determined by the humanitarian importance of the problem to be solved by the experimental study.

I wish to remind all individuals submitting papers to CVJ or CJVR, where animals are used in experimentation that a statement regarding the compliance of the Guidelines of the CCAC is necessary.

Sincerely,

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Author's Reply

Dear Sir:

I appreciate the remarks made by Dr. M.E. Olson regarding the paper I coauthored with Drs. Parker, Lea-Master and Dixon (Can Vet J 1987; 28: 181-186). Cold stress of young lambs is a problem that is common with many commercial operations yet producers often fail to take preventive measures to protect their animals from this environmental stressor. Our study was undertaken in order to describe under controlled experimental conditions the effects of cold stress on pregnant ewes and their progeny. Further, the coldest temperature conditions imposed on the animals in our study were no different, and in some cases warmer, than those often found under natural conditions. Recommendations for flock management designed to help producers prevent environmental stress of their lambs must be made on the basis of sound background information and scientific evidence such as was developed in our study rather than on casual field observations alone. The results obtained by our work provide direct evidence for use by veterinary practitioners when justifying to their clients the importance of management practices designed to prevent environmental stress of their farm flocks. I invite Dr. Olson to review again the statements made in the introduction and discussions sections of the paper as a measure of the intent of our work.

Respectfully,

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Editor's note:
The reviewers and editors failed to note this lapse. The CVJ routinely rejects manuscripts which do not adhere to the Canadian Council on Animal Care Guidelines.

Ivermectin Toxicity in Small Animals

Dear Sir:

It is with some interest that I read the dialogue which took place regarding ivermectin toxicity in small animals (Can Vet J 1987; 28:18, 299, 399). On behalf of MSD AGVET, Division of Merck Frosst Canada Inc.,

I would like to make several comments regarding this matter.

Firstly, the dose of ivermectin administered to the 13 year old Siamese cat was unknown. The dose given the mixed breed dog was approximately 150 μg/kg bodyweight, and, since it was intended to be used as an "all-purpose dewormer", one can only assume that the dose rate selected was an extrapolation of that used in cattle and horses.

The problem of off-label use of ivermectin in small animals was reported in 1983 and it was this practice which first shed light on ivermectin toxicity in dogs, most commonly in, but not restricted to, those of the Collie breed (1, 2). Subsequent testing done in the development of a commercial ivermectin preparation for dogs showed that in nonsusceptible dogs, a dose of 2500 μg/kg bodyweight once per os was necessary to evoke signs of ivermectin toxicity (3).

In more specific testing of ivermectin toxicity in Collie breeds and its possible relationship to Collie eye anomaly, it was found that susceptible dogs given ivermectin at 200 μg/kg bodyweight once per os will show signs of toxicity (4). A further study revealed that mild signs of toxicosis may be observed in susceptible dogs given doses as low as 100 μg/kg bodyweight once per os and this problem may be seen in both smooth coated and rough coated Collies (5).

Extensive laboratory and field testing of the commercially available product, Heartgard 30*, indicated that, at the recommended dose level of 6 μg/kg bodyweight once per os on a monthly basis, the product is safe in dogs including susceptible and nonsusceptible dogs of all breeds. Veterinarians in Canada are reminded that at the present time, the package insert for Heartgard 30* contains a cautionary statement that the product is not to be used in Collies less than 4.5 kg bodyweight and that all collies should be observed closely for signs of toxicity.

I trust that this information will help to clarify concerns which may have been raised regarding the reported cases. Further questions or inquiries in this matter should be directed to MSD AGVET, Division of Merck Frosst Canada Inc., P.O. Box 1005, Pointe Claire/Dorval, Quebec H9R 4P8.

S. Bauck, D.V.M., M.Sc.
Manager, Veterinary Services

References

1. SEWARD RL. Reactions in dogs given ivermectin. J Am Vet Med Assoc 1983; 183: 493.
2. PRESTON JM. Adverse reactions to unapproved applications. Vet Rec 1983; 112: 286.
3. SEWARD RL, BROKKEN ES, PLUE RE. Ivermectin vs heartworm — a status update. Proc Am Heartworm Symp, 1986: 1-8.
4. PULLIAM JD, SEWARD RL, HENRY RT, STEINBERG SA. Investigating ivermectin toxicity in Collies. Vet Med 1985: 80: 36-40.
5. PAUL AJ, TRANQUILLI WJ, SEWARD RL, TODD US Jr, DiPIETRO JA. Clinical observations in Collies given ivermectin orally. Am J Vet Res 1987; 48: 684-685.

Feline Dilated Cardiomyopathy

Dear Sir:

Some recent developments in the field of feline dilated (congestive) cardiomyopathy have prompted me to submit this preliminary update to the Journal.

At the fifth annual meeting of the American College of Veterinary Internal Medicine held in San Diego this past May, Drs. Paul Pion and Mark Kittleson, in conjunction with Drs. Quinton Rogers and James Morris, presented preliminary data on the etiology of feline dilated cardiomyopathy. Their work indicates that a deficiency of taurine, an essential amino acid in cats, causes dilated cardiomyopathy in some cats.

Since December 1986, they have investigated about forty consecutive cats with dilated cardiomyopathy. These cats were highly symptomatic with this disorder on presentation. Assays for taurine revealed the plasma levels to be less than 25% of the normal plasma levels for cats. Furthermore, when these cats were supported for their heart failure with captopril and furosemide while receiving supplementation with 0.5 grams of taurine orally twice daily, signs of cardiac failure resolved rapidly. Within five weeks it was possible to "wean" these cats off all therapy except taurine. In addition, echocardiograms normalized (a clinical cure) within 16 weeks of beginning taurine supplementation.

It must be emphasized that this is preliminary information; nevertheless, it does represent a major impact on the prognosis of feline dilated cardiomyopathy. Until now, this disorder generally carried a very grave prognosis with most individuals succumbing to this disease within two months with only a few exceptions. However, the work of Drs. Pion, Kittleson, Rogers, and Morris suggest that a significant, if not major percentage of cases of feline dilated cardiomyopathy may be clinically cured if the symptoms of heart failure can be controlled for one to four weeks with cardiotonic agents while plasma and tissue levels of taurine are augmented with the therapy.

Taurine is an essential amino acid in the cat and therefore is required in the diet, whereas dogs do not require it in their diet. Cats possess a significant taurine "sink", in that hepatic bile acid conjugation occurs via taurine in the cat instead of via glycine in the dog. High fiber diets and diets low in other sulfur-containing amino acids increase the dietary taurine requirement of cats.

A number of commercial cat foods have been investigated by Drs. Rogers, Morris, Pion, and Kittleson. All have concentrations of taurine that meet or exceed the NRC requirements. Nevertheless, these same diets have resulted in severely-depleted plasma taurine concentrations. This may represent an increased loss of dietary taurine in some individuals via the stool or urine, or perhaps a failure to absorb dietary taurine. The mechanisms for this phenomenon still need to be investigated. However, supplementation of these severely taurine-depleted individuals did increase their plasma taurine concentrations, reverse their symptomatology and appear to resolve their cardiac dysfunction. A number of commercial cat food companies have responded by increasing the level of taurine in their diets.

Drs. Pion and Kittleson have also had a preliminary look at a number of cats with hypertrophic cardiomyopathy and found their plasma taurine levels to be normal. Similarly, it would appear that a taurine disorder is not a significant part of canine giant breed cardiomyopathy.

In conclusion, a large percentage of cases of feline dilated cardiomyopathy may be controlled or even cured with taurine supplementation if the heart failure can be controlled for several weeks while plasma and/or tissue levels of taurine are increased. Echocardiography (cardiac ultrasound) continues to remain the best non-invasive means of distinguishing be-