Clinical Considerations of Bovine Coccidiosis

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As bovine practitioners we probably have a poorer understanding of the protozoan diseases than of diseases of bacterial or viral etiology. This, despite the fact that coccidiosis, the most common of the protozoan diseases, is one of the major diseases of feedlot cattle. One survey indicated that coccidiosis costs an average of $54.25 per infected animal. In addition to the economic losses associated with weight loss, treatment cost and death of the acutely involved animal there are significant, more subtle costs associated with the oft unrecognized subclinical form of the disease. This paper will address both forms of the disease.

There are fourteen species of bovine coccidia. Of these, only two, *Eimeria bovis* and *Eimeria zurnii* are frequently of clinical significance. The host specificity of all coccidial species is notable. In the food animals only sheep and goats have some species of coccidia which cross infect. Although a disease primarily of animals from three weeks to one year of age, older animals appear to be increasingly affected. In most cases the older animals are symptomless carriers of the disease providing a constant source of infection for the younger animals with which they comingle.

The life cycle of coccidia is somewhat complex, comprising both an asexual and sexual phase within the host plus a required incubation period outside the host. An understanding of the life cycle is of value in establishing a control program. Intestinal epithelial cells are destroyed at each stage of the asexual as well as sexual portion of the life cycle.

Briefly the life style is as follows: Oocysts in feces ➔ sporulate in environment to infective state containing 2 or 4 sporocysts ➔ ingested, releasing sporozoites into the intestinal lumen ➔ penetrate mucosal cells to form trophozoites ➔ divide to form schizonts (schizogomy) contain many maturing merozoites ➔ released into lumen (destroying host cell) ➔ form a new generation of schizonts *or* procede to sexual phase of reproduction (gametogomy) ➔ fertilization ➔ zygote ➔ oocyst ➔ release of oocysts causing further host cell destruction ➔ oocysts in feces.

Each species of coccidia is programmed as to the location of sexual and asexual stages within the G.I. tract, the number of asexual stages, the number of merozoites which can be produced, the overall length of the life cycle and the incubation period. Host cells are destroyed by schizogomy, gametogomy and finally by the release of oocysts. Each schizont contains hundreds to thousands of merozoites and there may be one or several generations of schizonts. Thus it can be seen that each ingested oocyst has the predetermined ability to destroy thousands to millions of intestinal cells. This predetermined ability, which is characteristic of the species, helps dictate the type and severity of clinical signs observed. Each oocyst observed in a fecal sample represents more than one intestinal cell destroyed and in severe infection there may be thousands of oocysts passed per gram of feces. Although coccidiosis is said to be a self limiting disease, it can be seen that much damage can be done before the disease self limits. Animals which survive the initial infection of coccidiosis are usually immune to that species. There is little cross immunity between species so animals infected with *E. bovis,* for example, may later, if exposed at a later time to an infective dose of *E. zurnii,* again develop clinical coccidiosis. The severity of infection is determined not only by the species of coccidia involved and the immune status of the host but also by the number of infective oocysts ingested. An animal may be a symptomless carrier, develop a subclinical case or a severe, even fatal case, depending on the numbers of oocysts initially ingested. The importance of sanitation and its role in preventing ingestion of massive numbers of oocysts is obvious.

Other factors such as nutritional status and stress exposure may also influence the severity of infection in an animal or a group of animals. Stress as a predisposing factor is difficult to objectively evaluate. Explosive outbreaks of clinical coccidiosis frequently accompany such stress factors as crowding, shipping and especially severely cold weather. Experimentally it has been shown that the administration of corticosteroids will allow subclinical infected animals to develop clinical signs. Administration of dexamethasone has been demonstrated to stimulate a ten fold increase in oocyst output. From this it would appear that stress precipitated coccidiosis is mediated by increased corticosteroid output.

Well nourished animals appear to be as susceptible as animals receiving marginal diet but those animals in good physical condition are able to withstand the effects of clinical disease more successfully than animals being fed marginal diets. Vitamin A is especially important because of its role in maintaining epithelial health. Adequate hepatic stores of vitamin A also insure that there will be sufficient amounts available during subclinical or clinical infection when absorption of vitamin A precursors may be impaired by mucosal damage.

Clinical Comparison of *Eimeria bovis & Eimeria zurnii*

These are the two most significant coccidial species infecting cattle. It is possible that some of the other species may cause disease if animals are exposed to an

overwhelming dose of infective oocysts. This does not often occur under field conditions. The characteristic signs of bloody diarrhea, tenesmus and obvious weight loss are usually a manifestation of *E. zurnii.* The frank hemorrhage seen accompanies an infection which usually occurs somewhat lower in the G.I. tract than with *E. bovis. E. zurnii,* more than *E. bovis* is likely to be associated with clinical signs seen in older feedlot calves and yearlings and is more likely to be incriminated in nervous coccidiosis which is occasionally seen accompanying the enteric form of the disease. *E. bovis* is more likely to cause subclinical or mild clinical disease and the insidious losses associated therewith. The slightly different sites of infection of the two species may account for an apparent difference in response to prophylactic and therepeutic agents used to combat the disease, Inophores such as lasalocid and monensin have been demonstrated to have value as anticoccidial drugs in some species. They have not been approved for this use in cattle and there have been varying and conflicting reports as to the anticoccidial properties of these compounds. A survey of large Kansas feedlots indicated a significant reduction in feedlot deaths due to coccidiosis coinciding with the advent of the routine use of monensin as a feed additive in 1976. The ionophores have been demonstrated to be more likely to have value in controlling *E. bovis* than *E. zurnii.* Decoquinate and oral sulfonomides appear to be equally effective against both strains whereas amprolium appears to be more effective against *E. zurnii.* Immunity to one species does not confer immunity to another.

Nervous Coccidiosis

A confusing and poorly understood manifestation is the occasional observation of central nervous signs in animals with coccidiosis. A majority of these are animals weighing less than 500 pounds. The clinical signs observed are uniquely characteristic of the disorder and resemble a grand mal type of epilepti-form seisure. The seizure may occur spontaneously or be precipitated by excitement or overactivity. The typical animal becomes apprehensive and exhibits a stilted gait or stance. The muscles of the face and neck begin to twitch and the animal usually falls on its side and a tonic clonic involvement of the entire body ensues. Opisthotonous and nystagmus are frequently observed. The seizure often terminates in a final motion in which the animal draws its hind legs up against the abdomen following which the animal begins to relax. Within thirty minutes the animal may be back on its feet and appear normal until another seizure occurs. The duration of each seizure is usually about five minutes but the duration increases and the interval between decreases as the disease progresses. Death is caused by respiratory arrest during a seizure. The death rate is normally high, probably over 75% regardless of treatment. Iowa practitioners in a 1984 survey reported death in 133 of 183 treated cases of the nervous form of the disease. Fortunately less than 1% of the animals with enteric coccidiosis develop nervous signs. *E. zurnii* has been incriminated in over 90% of the cases in which the responsible agent has been identified.

In the midwest there appeared to be a dramatic increase in reported cases of nervous coccidiosis in the winter of 1983-84. This may have been tied to an increase in the incidence of enteric form of the disease. 73% of the veterinarians responding reported an increase in enteric coccidiosis during that time. There is no totally acceptable explanation for the signs observed in nervous coccidiosis.

Some suggested causes have included the following:

1. Anemia—associated with fecal blood loss.
2. Hypomagnesemia
3. Hypoglycemia
4. Avitaminosis A

} associated with malabsorption of these nutrients.

5. Toxemia—due to toxins released from the coccidia.
6. Hyponatremia and Hypochloremia } due to increased loss of sodium ions through damaged intestinal mucosa and failure of conservation (reabsorption) of these ions.

The latter explanation is probably the most feasible in light of recent studies. Although the vitamin A theory has merit in some respects, it has been shown that hepatic reserves of vitamin A were normal in several studies of animals showing nervous coccidiosis signs. The clinical signs observed are nearly identical to those seen as a result of increased CSF pressure associated with hypovitaminosis A.

Treatment of the disorder should include, in addition to the use of anticoccidial drugs, fluid therapy with emphasis on sodium and chloride. Supportive therapy and nursing care including B vitamins, aminoacids and the provision of a dry, quiet recovery area.

Control of Coccidiosis

It is obvious that sanitation and minimization of stress are objectives vital to the minimizing losses due to this disease. Under feedlot conditions these objectives cannot always be achieved. Most feedlot operations are, therefore, high risk operations as regards to coccidiosis. In light of the hidden and apparent costs associated with this disease, it is advisable to routinely use a prophylactic anticoccidial preparation, especially in calf feeding operations. Monensin cannot be relied upon as an effective coccidiostat in cattle under normal usage conditions. Amprolium and decoquinate would both appear to be cost effective medicants under most conditions. One concern pertaining to the use of these drugs involves possible interference with host immunity. As animals develop an immunity only through being infected clinically or subclinically, one might expect to see not a total prevention, but rather a delay of onset of infection when these products are used. In light of this, it might be advisable to delay the start of a preventative program for one to two weeks following arrival in a feed yard. This should allow natural infection to procede far

enough to allow immunity to develop, but still intercept the infection before clinical signs develop.

In the treatment of clinically involved animals, the choice of an anticoccidial preparation is probably less important than the provision of supportive therapy and adequate nutrition for recovery. These animals absorptive capabilities have been severely impaired and will be compromised for some time. By the time clinical signs are observed, the life cycle of the parasites is sufficiently advanced that severe damage has already occurred and the majority of the forms are beyond the developmental stages that will be affected by treatment.

Early observation of and veterinary response to clinical signs is important. Where animals are not receiving preventative medication, the first clinical cases observed should serve as a flag signaling the immediate implementation of a preventative program. In most cases there will still be a significant reduction in clinical cases and subclinical damage if herd medication is started immediately after the first cases appear.

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Questions & Answers:

Question: (inaudible)

Dr. McElroy: The comment was the fact that Montford in Colorado has done some work where they deliberately dropped one ovary into the peritoneal cavity and left it there to become attached. I did read some of that work. They did get some additional gains, but I think they did have some attaching and I think they had some heifers that did cycle. Occasionally you'll leave one in there unintentionally!

Question: Do you turn them directly on to grass?

Dr. McElroy: The comment there was that in Montana they are finding that if they spay the heifers early and do them while they are still receiving hay and everything, do it awhile before they go to grass. they get better gains than if they go in directly to grass. I think I would concur with that. This last year in southern Iowa we did them all. We did what we didn't want to do. We unloaded heifers off the truck one day and spayed them the next day and went to grass, because we had fed every drop of feed that we had in southern Iowa. But I concur with you entirely. Dr. Bohlender, did you have a comment?

Dr. Bohlender: Regarding this study at Montford, I think you might pay a lot of attention to that. The cost of pregnancy in feedlots, on 10,000 head in the study from Montford's, they found they had 16½% bred. The liability of each pregnant heifer came to about $115, for every one calving within the yard $157. If they did nothing in the way of aborting or reselling at the point of purchase, it cost them $19 for every head that they had in at the 16½% pregnancy rate. So I think pregnancy liability in the feedlot is definitely something that we need to address for our clients.

Ouestion: Dr. Arthur's comments here are that Washington State is doing something on chemical spaying. I've just seen it mentioned, but I haven't seen anything further on it. Is anybody aware of that or have any answers?

Dr. McElroy: Yes, I have. The comment there is of a brand in the shape of a spade, and I think that it is recognized by the federal and state people. If I am going to do that, I'm going to put that on before I spay her. I don't want her stirred up after I get through. And it's another procedure. In our area we are not as good at reading brands. It is one method. But it still does not identify it back to the man that did the job. And I think as in the preconditioning program we found it very important. I'd still like some identification.

Dr. Bohlender: I think we need to be aggressive about what we're going to do on this thing. In Nebraska, they had a resolution come up before the membership asked for an S brand. jaw brand, on spayed heifers. This is going to be very disruptive to our procedure of doing it. I don't know how many of you are old enough to remember back when we used to jaw brand B's on the jaw. Those are not legible. They are not a useable area for a brand and the government is already wanting to use this thing. But if we don't take a hold of it for the proper ID, the cattlemen are going to do this. It was even suggested that we tattoo them. So we've had a lot of discussion. Dr. Johnson is handling the tags, as I understand. Remember we're changing the brucellosis status of these animals as soon as we spay them. Like with us, where we have cattle coming in for grazing, they're going to be moving across state lines, unless you want to run those through and start putting tags in them, you might as well have a tag in them right away when you do it. You can write a health paper, you have the individual identification on it. So if you use a tag number it better have enough numbers and a state ID on it.

Dr. McElroy: We found this same thing. We shipped 3,000 or so heifers out of the state without any difficulties at all on this type identification.

I would like to see the AABP adopt something to this effect. In Wyoming they are using a blue tag. It has the state and then an SP and five numbers and spayed on the back. I think we can use the same type of numbers, the same state designations that they use for pass tags and that type of thing, with a color for easy identification.