Enteric Pathology

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This presentation was intended to be a series of photographs of enteric lesions. This objective was not fully achieved, partly because enteric pathology is quite variable and frequently unremarkable on gross observation. The following is an attempt to describe some opinions formed as a result of recent experience, primarily in feedyards in the southwestern United States.

Enteric Pathology may result in a variety of effects on the victim. These are frequently, but not always, observed as diarrhea and its consequences.

Results of Enteric Pathology

1. *Fluid Loss:* Associated with diarrhea and manifested clinically as dehydration. Keep in mind that dehydration is very significant when it becomes part of a combination of imbalances including altered electrolyte levels.

2. *Electrolyte Imbalance:* Clinically measurable critical factor in survival. Rapid loss of fluid and electrolytes into the intestine can lead quickly to life-threatening imbalances such as hyperkalemia.

3. *Malabsorption:* Failure to assimilate ingested nutrients. This may vary considerably in degree and result in dramatic malnutrition or prolonged subtle poor feed conversion without clinical signs. Mild lesions in the small intestine may prevent absorption of fluid and nutrients. The large intestine may continue to absorb fluids. Therefore, malabsorption may not be clinically visible as diarrhea.

4. *Septicemia:* Host damage extending beyond failure of enteric function. This may result from absorption of toxins produced within the intestine with or without visible enteric lesions. Systemic extensions of infectious agents may also be a serious sequelae.

5. *Recovery:* Rapid repair of damaged bichemical or tissue systems. The intestine is uniquely capable of very rapid repair when offending pathogens are removed. Turnover of epithelial cells is normally rapid and accelerates if injury occurs. The entire enteric epithelium can be replaced in a very few days.

Discussion

The following discussion deals with causes of enteric malfunction in feedyard cattle. It is far from all-inclusive and deals with those identifiable conditions that are of major significance.

Bovine virus Diarrhea (BVD)

This major and common cause of enteric damage is recognized in individuals or groups of feeder cattle as persistent, unresponsive diarrhea. Classical lesions include discrete ulcers of the entire digestive epithelium. Intense inflammation or ulceration of Peyer's Patches are frequent findings. The occasional animal with the mucosal form may have diffuse inflammatory lesions with exudation and ulceration.

Unfortunately, the actual clinical manifestations may be quite nondescript. Diarrhea may occur without the lesions described above. Debililation may occur as the only clinical sign. This may be due to the ability of BVD virus to invade various tissues with manifestation as a predisposing factor in shipping fever or as an immunosuppressive agent. The subtle forms are by far the most common making the role of BVD and the need for control programs very difficult to evaluate in many situations. For instance, there are unexplained regional influences. It is quite rare for BVD to be identifiable as a clinical or necropsy entity in Arizona feedyards. That doesn't mean it is not significant but vaccination programs usually cannot be **demonstrated** to be beneficial. This most certainly is not the case in other areas of the country where immunization programs appear to be essential.

Other viruses may be factors in enteric pathology but agents such as rotavirus, coronavirus, adenovirus and others simply are not routinely recognized.

Coccidiosis

This is a common problem in recently arrived feedyard cattle and regional influences are again obvious. It is a major but sporadic disease in southwestern feedyards but is more significant in areas with colder and more moist weather. It is by far the most common cause of fresh red blood in feces of feedyard cattle. It is also the most common cause of swelling, inflammation, hemorrhage, and exudation in the lower small intestine and the large intestine. It is not fully controlled by levels of ionophores commonly used in rations of incoming cattle.

Diagnosis may be quite easy if numerous animals have typical bloody feces and numerous oocysts can be identified on flotation. Keep in mind that most cattle have a few coccidia and may shed quite a few oocysts without clinical significance. Unfortunately, the disease may be more subtle and require necropsy observation and elimination of other causative agents. The life cycle of coccidia may complicate diagnosis and control. It appears that clinical enteritis may persist in a chronic form, perhaps involving secondary agents, after a wave of coccidia have damaged the intestine,

matured and passed in the feces. We frequently look for coccidia by flotation or histapathology too late in the course of the disease. The life cycle is also an important factor in therapy because most coccidiastats are effective only in certain stages of the life cycle, often before infection is clinically obvious. This necessitates a well-designed control program. Treatment of clinically affected animals is generally directed at secondary pathogens.

Histopathology can be an effective diagnostic aid. Several sections of lower small intestine, cecum, and colon should be submitted. Again, tissues harvested from too late in the course of the disease may be misleading because coccidial forms may be infrequent or absent.

Salmonellosis

This common cause of enteric damage may be subtle and sporadic or it may produce explosive outbreaks. Diagnosis is based on clinical signs including poor performance, poor response to therapy and often diarrheal disease. Isolation of *Salmonella typhimurium, S. dublin,* and other species from feces may be helpful but keep in mind that *Salmonella spp.* may be shed by animals without clinical salmonellosis. Necropsy findings may be nondescript or enteric pathology may be noted as gaseous, flacid intestines. Some animals have congested intestinal walls. Hemorrhage is rare. Fibrinonecrotic enteritis may be observed in a few animals. Systemic invasion shortly before death may result in a variety of lesions including swollen or mottled liver, cardiac hemorrhages, and petechiation of various surfaces. Histologic lesions in the intestine and liver may be helpful but are not definitive and must be supported by bactirial isolation. Salmonellosis is a frequent cause of death in animals debilitated by some other malady.

Nematodes

These are a common cause of diarrhea, debilitation, and decreased resistance. Evaluation of clinical significance must incorporate history (did the animals originate in a moist, humid area or in a desert?), symptoms, fecal flotations and necropsy. Flotations are helpful but must be interpreted with some professional judgement because no definitive translation can be based on numbers of eggs per gram of feces. A definitive diagnosis of severe parasitism can be based on necropsy findings of emaciation, often anemia, abomasal mucosal swelling or irregularity, and observation of numerous nematodes with the aid of some magnification. The diagnostic dilemma involves situations where infection levels are low or moderate.

Veterinarians frequently are negligent in developing specific anthelmintic programs for feedyards based on the needs of specific groups of incoming animals simply because the guidelines are unclear and the decisions may be difficult. We rarely recognize type II ostertagiasis as a significant event. This may be because it is of low incidence in southwestern feedyards or perhaps because of failure of diagnostic methods to recognize it. Most incoming calves are from a wide variety of source herds so the sporadic type II affected animal may simply go unnoticed.

Feed Related Deaths

Bloat, acidosis, and enterotoxemia are all related to feeding practices and eating habits of cattle. These conditions are particularly distressing because the animal may simply be found dead without therapeutic opportunity and because more valuable animals may die late in the feeding period.

Bloat is diagnosed by a distended rumen in the fresh carcass and the resultant ischemia of viscera due to pressure. As the carcass ages, diagnosis becomes more difficult and may depend on a process of eliminating other causes of death.

Acidosis may cause some hyperemia and edema of the rumen wall but some animals die without observable lesions. Diagnosis depends on history and rumen pH. The following useful rumen pH table was presented by Dr. W. E. Lloyd at a previous AABP meeting:

Acute Urea Toxicity	7.0—9.2
Protein Excess	7.0—8.6
Fasting	6.8—7.3
High Forage Ration	6.0—6.8
High Grain Ration	4.5—6.2
Excess Grain	4.0—5.0
Grain Engorgement	3.0—4.0

Absolute pH is less significant than a sudden reduction in a specific animal usually resulting from a change of ration, poor bunk management, or individual irregularity in eating.

Enterotoxemia may cause hyperemia of short segments of intestine but hemorrhagic lesions are very rare. The most common finding is a lack of significant gross lesions. Diagnosis is frequently a professional guess based on history and lack of any other obvious cause of death. Laboratory confirmation may be helpful if the carcass is fresh but the diagnostic laboratory should be contacted for specific instructions that apply to that laboratory before any submissions are attempted.

Questions & Answers:

Question: Have you encountered nervous coccidiosis?

Dr. Glock: That's how we usually find coccidiosis in a pen. Somebody notices an animal going crazy which is identified as a "brainer", and they send the head to me for examination. I find no leisons so if they did not look at the animal and the pen we are in trouble. There is nothing new on its pathogenesis and control. Maybe a lot of electrolytes can help, assuming the nervous signs are due to an electrolyte imbalance. Use a coccidiostat for the rest of the pen.

Question: What products are we going to have in the near future?

Dr. Clymer: The ear tag market is a big one and there are good things on the horizon — application devices and methods of delivery.

Question: Body condition scoring and passive transfer can someone speak on this?

Answer: There is a strong relationship between body condition scores in two year old heifers and calf serum immunoglobulin level. In cows three years or over there was no relationship.