Gross Pathology of Abdominal Viscera

Herschel Giles, *D.V.M., Ph.D.*
Department of Pathology
School of Veterinary Medicine
Auburn University
Auburn, Alabama 36830

Ulceration in the rumen of cattle, I think, commonly or more frequently represents a rapid change in diet, producing rumen acidosis which is followed by necrosis of the epithelium, and ulceration. These ulcers frequently can lead to rumen abscessation. Bacteria can get into the submucosa and then metastasize to the liver. *Spherophorus necrophorus* is most commonly in the textbooks associated with these lesions, but in our particular locality at Auburn we very seldom see it. Other organisms can cause it. Many fungal agents also get in the submucosa of the rumen, but the primary problem is acidosis. The lesion would be typical of a fungal infection starting out in the rumen. But rumen acidosis following a rapid change in diet is the primary cause of rumen ulceration. Other causes that should not be neglected are infectious, such as virus diarrhea.

Another type of ulceration that we have is *ulceration in the abomasum.* In large ulcers there is often hemorrhage in the submucosa. Abomasal ulceration should lead you down a line more of a toxic substance initially. Of course infectious diseases can produce abomasal ulceration, again like virus diarrhea, but certainly think of toxic substances such as arsenic, or other heavy metals.

Another factor that can cause ulcers in the abomasum is malignant lymphoma, infiltrating the wall of the stomach. When this happens, the neoplasm can grow through the submucosa and into the mucosa, producing ulcers and even lead on to a perforation.

Impacted abomasum. If I tried to tell you I knew what caused this, I would be lying to you. I don't understand this but sometimes we see it. I suppose it is a combination of the possibility of de-innervation or indigestion problems. Occasionally we encounter it. Some people from South Alabama and South Florida have told me that they have seen abomasal impactions associated with animals grazing on pastures that have a lot of sand.

Abomasal edema. The primary thing to see in this condition is the thickening of the folds. They are glistening or shining. I think that edema in cattle can best be demonstrated in the folds of the abomasum. This is a variable item in different species of animals. In pigs you want to look under the eyelids and the skin. In horses, subcutaneous edema in the abdomen and the legs. In cattle, regardless of the cause of the edema, a good place to find generalized edema is in the folds of the abomasum, whether the cow has heart failure, or parasites or hypoprotein anemia from any cause.

Hemorrhage of the abomasum. Hemorrhage of the abomasum as you think of it similarly to ulcers in the abomasum. It may simply be a preceding phase of ulceration so you should think of caustic agents first and then followed by infectious agents, arsenic in our particular locality; bracken fern.

The condition that I think currently is one of the most important economic diseases in the Southeast is *ostertagiosis.* I want you to look for the small nodules in the mucosa, very small nodules. The reason why this disease is so economically important is because protein has become so expensive. Ostertagia, when it gets in the submucosa, changes the pH of the abomasum. It changes it from an acid environment to an alkaline environment and in order for the pepsin and pepsinogen to make the initial breakdown of protein, you have to have an acid environment. A lot of these animals with severe ostertagiosis, you can take a little dip stick and just drop it in the abomasum, and it will be alkaline instead of acid.

Hemorrhage in the intestinal tract should be thought of similarly to abomasal ulceration and abomasal hemorrhages, first of all caustic. If they manage to get through the stomach, they still can be caustic when they get into the intestine. Also it is an example of many acute infectious conditions, like salmonellosis, or it could be anthrax. Any infectious septicemic disease could produce hemorrhages. The hemorrhages are frequently visible from the serosa. There are hemorrhages throughout the intestinal tract here. This happens in bracken fern poisoning or arsenic also.

Johne's disease or paratuberculosis, a condition that results in a prolonged diarrhea, is characterized by many corrugations, with thickening of the intestinal tract. The location of the lesion of paratuberculosis is important. It occurs primarily in the ileum and the cecum.

The most important thing to remember before you diagnose enteritis is to make sure the animal has diarrhea. Diarrhea is a clinical sign of enteritis. In the absence of diarrhea, 95% of the time you will not have enteritis. It is difficult to go through the intestinal tract and determine whether or not an inflammatory

process is present. The reason why it is difficult is because of the fecal material and mucus that are normally present. The amount of mucus that is present in the GI tract can be influenced very readily by whether or not an animal has been eating. An animal that has been off feed for a few days will have a tremendous amount of mucus in the intestinal tract. This leads to a diagnosis erroneously of catarrhal enteritis. I think it simply means the animal has been off feed. To diagnose enteritis you need to have an exudate that adheres after you run water over it sufficiently to wash the fecal content off. You need to have an exudate that adheres.

Another condition that affects cattle is *coccidiosis.* Coccidiosis can look similar to Johne's disease because the intestinal tract becomes thickened and reddened, but the location is different. Coccidiosis, when it kills animals, usually involves the cecum and the colon. With Johne's disease you've got the terminal part of the small intestine and the cecum and there is also a difference in the course of the disease. Coccidiosis is a peracute disease and it can be so peracute that the animals may die in convulsions before diarrhea is observed. I think this is especially true in the western parts of the United States. Another condition is caused by *esophagostoma.* On occasion we have samples sent in, in which there is some intestinal wall and someone wants to know what the caseous lesions are in the intestinal wall that produce nodules. The majority of cases of peritonitis that we see are associated with perforation some place in the intestinal tract or in the genital tract. There are a few infectious diseases that can produce peritonitis in cattle. You can even see peritonitis in some instances with blackleg. Any of the clostridial diseases that have toxins that are capable of increasing the permeability of blood vessels potentially could result in a fibrinous peritonitis.

When an animal dies, I think the permeability of the vessels in the intestinal tract open up, the heart beats a couple more times and bacteria can rapidly be carried to the liver. If the animal sits around and cooks for five or six hours, the bacteria along with the normal post-mortem degeneration can produce focal areas which I think can mimic focal areas of necrosis. These may also accompany many septicemic diseases. Housewives don't like to buy livers with black spots. In case of fatty infiltration of the liver, the texture will be considerably softer, it will be more fryable. One of the most common causes of fatty change in cattle is gestation. During the later stages of pregnancy, cattle will develop a certain degree of fatty infiltration of the liver. Crotolaria poisoning produces fibrosis of the liver. Feel it and determine whether it is hard or soft.

Liver abscesses most commonly come from rumen ulceration or abomasal ulceration. They could come from other localities–metastatic abscesses. In some parts of the country the majority of the these abscesses are caused by *spherophorus spp.* but in our locality we seldom see spherophorus. Another condition that we are seeing in South Alabama and some parts of Florida is liver flukes problems. Liver flukes produce a dark pigment in the liver. Another condition that occurs in the peritoneal cavity of cattle is referred to as fat necrosis or lypomatosis, which at times can result in death of an individual by producing a constriction in the GI tract.

Most of the time congenital cysts in the kidney occur singly or possibly as three or four. The only thing that it could be confused with would be hydronephrosis. To eliminate the confusion you should look at the ureter to see if it has undergone dilatation.

A random distribution of lesions in all lobes of the kidney indicates that probably the route of infection was hematogenous, so the importance of this would be to go back and look at the heart to see if you have a lesion on the left AV valve. *Pyelonephritis* traditionally in this country has been associated with *C. renale* cattle but it doesn't have to be *corynebacterium renale.* Almost any enteric organism when presented to the urinary tract, after obstruction, can produce pyelonephritis, and ascending infection. If a kidney is slightly more icteric than it should be and there are small dark spots representing hemoglobin pigment, it is somewhat typical of what you would expect with an acute hemolytic disease such as leptospirosis. Another condition that we occasionally see is referred to as *bovine enzootic hematuria.* There are pedunculated lesions in the urinary bladder and these lesions bleed. This condition is currently associated with poor pastures and low levels of bracken fern.

(Prepared from a tape recording of Dr. Giles' presentation.)