What's New in our Knowledge of the Ruminant Digestive Tract?

Chairman: Dr. L. Mac Cropsey

What's New in the Lower Digestive Tract?

The Biochemistry of Ruminant Nutrition

Relationship of Energy, Cow Size and Sire to Calving Difficulty

Polioencephalomalacia

Mineral Sources for Bovines

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Abomasal Ulcers

There are four syndromes associated with abomasal ulcers (1). The first type is that of slight ulceration or erosion with only mild or no clinical signs. These ulcers are often found associated with cases of septic metritis or mastitis in the early post-partum period. These ulcers or erosions of mucosa are multiple and manifested by dark, fetid feces associated with toxic states. They respond to supportive therapy aimed at the primary disease.

The second syndrome is that of marked hemorrhage or anemia. These are usually single ulcers in the mucosa that erode a vessel associated with the greater curvature of the abomasum. Where there is sufficient hemorrhage into the lumen of the gut the animals become anemic. These cases are manifested by a pounding heart, increased pulse rate, and pallor of the mucous membranes. Dark, tarry feces are often the first clinical sign noted. These animals should be treated with supportive therapy—blood and fluids—aimed at the correction of the anemia. Gastric protectants are indicated.

The third syndrome is that of abomasal perforation with acute circumscribed peritonitis. The clinical signs in this syndrome are similar to those of acute traumatic gastritis. The primary difference is the site of localized pain. The pain in traumatic gastritis is in the left side of the midline just behind the costal condral arch. However, that of acute circumscribed peritonitis with abomasal perforation is on the right side. Treatment is similar in both cases.

Abomasal perforation with diffuse peritonitis is a very fulminating syndrome with death resulting in 24 or 48 hours after the onset of clinical signs. We have attempted surgical repair but without success to date. Again, these are usually single large ulcers ranging up to two inches in diameter.

Whenever presented with a cow showing evidence of abomasal ulceration with hemorrhage and anemia, one should consider lymphosarcoma, as lymphosarcoma has a predilection for the uterus,

heart and abomasum. Often the first presenting signs are that of a bleeding abomasal ulcer. Additional clinical tests are in order to eliminate the possibility of lymphosarcomatous invasion of the abomasum. This would include a complete blood count, serum lactic dehydrogenase, a rectal examination, and palpation of external lymph nodes.

Abomasal ulcers are certainly multicausal in origin. They can be visualized as a balance of the *defensive* factors versus the *aggressive* factors (2). The defensive factors include *normal local blood flow to the mucosa.* As evident here, when lymphosarcoma invades the abomasal wall it will interfere with the normal blood flow. *Mucous cover* is constantly secreted by the abomasal lining that prevents autodigestion by acid and enzymes. *The mucosal cell resistance* is an inherent factor in the mucosa that prevents autodigestion. This has been shown to be related to the level of protein in the diet. Rats with low protein diet will have a greater incidence of gastric ulcers than those in a higher protein diet (2). This has been shown also in beef cattle (3). *Duodenal break* is the situation where excessive gastric contents enter the duodenum and a reflex occurs with a diminished gastric acid secretion (2).

The aggressive factors include *hyperacidity* which we can all understand as the cause of abomasal ulcers. *Pepsin excess* may be related to gastric ulcers in man. *Trauma* to the abomasal mucosa is certainly a factor in milk fed calves when converted to roughage diet (4). More than 95% of calves over 12 weeks of age had evidence of abomasal ulcers when examined at slaughter. *Hormonal factors*, especially steroids, are very important in the genesis of abomasal ulceration. Another hormonal factor to be considered is progesterone, although its effects seem to be protective rather than aggressive. It is interesting that both gastric ulcers in women and abomasal ulcers in cattle are very rare during gestation. This would suggest that progesterone may play a defensive role in ulcer formation.

Gastric hyperacidity, as indicated, is an important factor in the genesis of abomasal ulcers. Hypocalcemia in the early postpartum period will promote gastric secretion. This has been well documented in other species (2). Hypocalcemia, as we are all aware, is common in the early postpartum period as reported by Mayer (5), among others. *Stress* also may cause hyperacidity and is nearly maximal in the early postpartum period, as these cows are being fed increasing amounts of concentrate. Thus, we have ulcers being most common in this early postpartum period (5,6,7,8).

"Stress" may be manifest by two methods: 1. Via the anterior hypothalamus, vagal nucleus and vagal nerve resulting in increased gastric acid secretion; and 2. Via the posterior hypothalmus, pituitary, and adrenal cortex, resulting in excess corticosteroids with increased gastric acid secretion.

Therapy for abomasal ulcers is certainly symptomatic. One should change the diet to one high in roughage. In severe anemias, blood

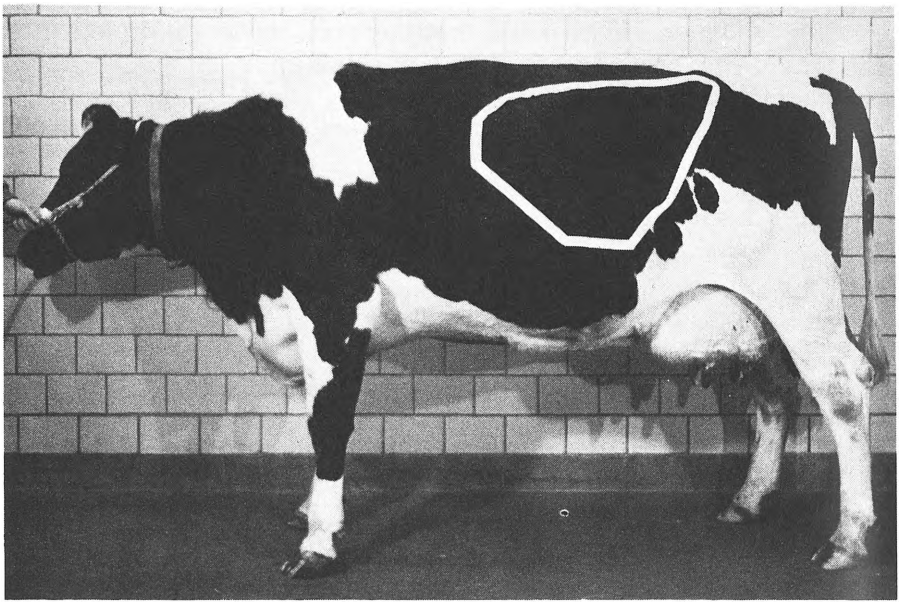
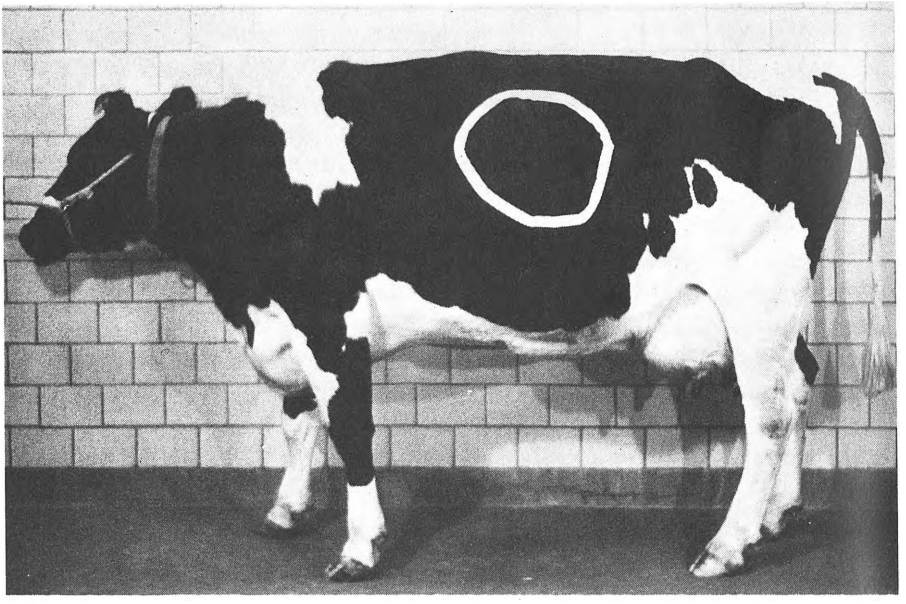


Figure A. A "ping" may be elicited by percussion in the area outlined by white. This large area extending from the tuber coxae to the tenth rib is compatible with air in the peritoneal cavity or gas in the rumen.

Figure B. A "ping" may be elicited by the snap finger technique in the circular area. This is compatible with a "typical" left abomasal displacement. If the ping can be detected back to the tuber coxa then one must consider gas in the rumen or peritoneal cavity. If gas is present in the peritoneal cavity, one should be able to elicit a ping on both sides of the cow's abdomen.



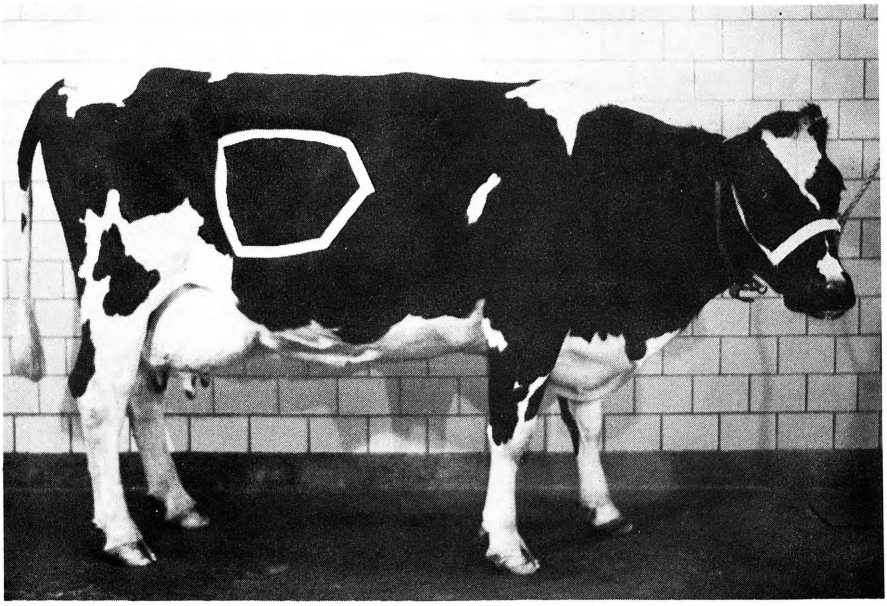
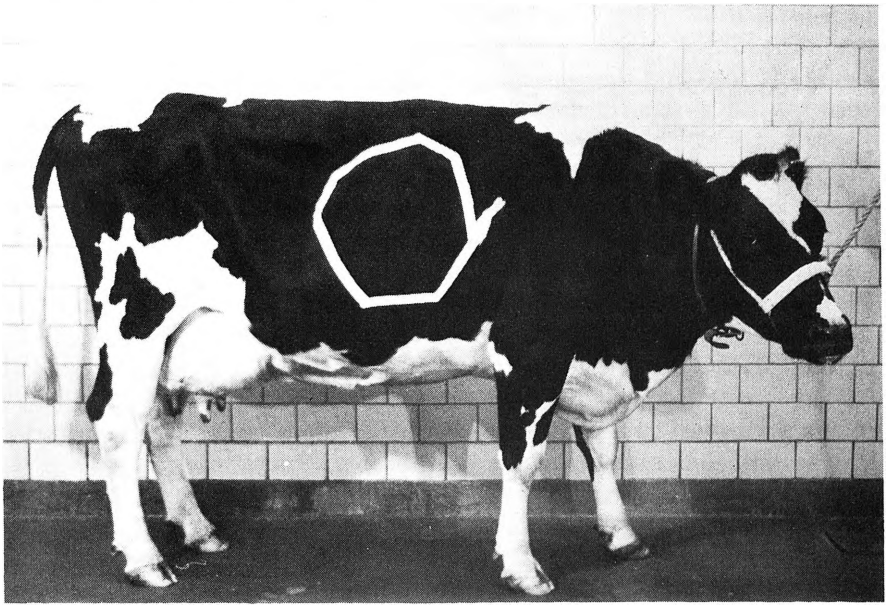


Figure C. A "ping" may be elicited from the enscribed area. This is compatible with a dilation or displacement of the cecum and proximal colon. A rectal examination will confirm this in most cases. The cecum is often within the pelvic inlet or just anterior to it.

Figure D. A "ping" is elicited within the white circular area and is compatible with a right abomasal displacement. Rarely does the abomasal extend more than half way back to the tuber coxae from the last rib. An abomasal torsion would enscribe a similar but larger area.



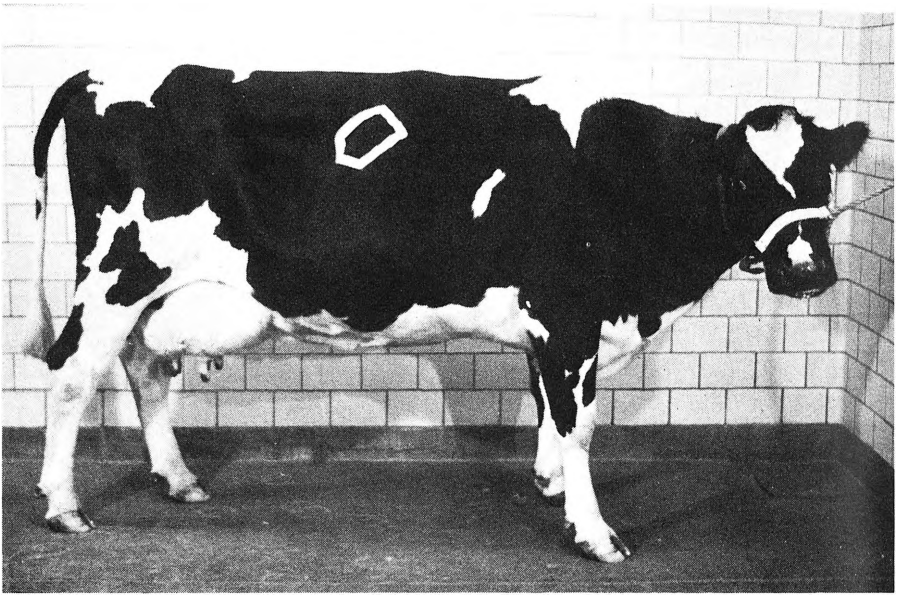


Figure E. A "ping" in this area is often confused for a right abomasal displacement or cecal torsion. Actually, many anorexic cows can have a gas pocket in the cecum and coiled colon. The pitch is variable as is the size. A "ping" ventral to the last two ribs should be considered as physiological until proven otherwise. This area where gas collects in the coiled colon may be much larger under certain circumstances.

transfusions are certainly indicated as are intravenous fluids and electrolytes. Surgical intervention is of value in abomasal perforations. Gastric protectants and antibiotics also provide supportive therapy.

Cecal Torsion or Dilation of the Cecum and Coiled Colon

The clinical signs are similar to abomasal displacement with a cow being off feed and decreased milk production. However, there does not seem to be a relationship between cecal torsion and the early post-partum period. The temperature and pulse are normal in most cases. The diagnostic feature is the ellicitation of a ping in the right paralumbar fossa. This ping is fairly high pitched and extends caudally from the tenth rib back to the tuber coxae. One must differentiate a ping in this area from the ping of the atonic cecum and coiled colon and from air in the peritoneal cavity or a displaced abomasum to the right. With a right displaced abomasum, the ping area is usually anterior to that of the cecum. If the gas is in the coiled atonic portion of the cecum, it is usually under the last rib or one or two inches in front of it. If the air is in the peritoneal cavity, it extends over a much larger area and usually can be detected on both sides of the cow. Rectal examination is the clincher in the diagnosis of cecal torsion. Almost always the cecum can be palpated in or just anterior to the pelvic canal. It seems to feel like a Vienna loaf of bread or a flexed human knee. It is usually filled with fluid and gas. Therapy is surgical intervention with opening of the tip of

the cecum and draining the contents. Fixation of the replaced cecum is not necessary.

The causative factors related to cecal torsion are very similar to those of abomasal displacement. Increased concentrate feeding results in an increased VFA production in the cecum. The increased volatile fatty acids decrease the motility of the cecum and increase gas production and incorporation in the cecum, allowing for a displacement and eventual torsion.

The Fatty Liver Syndrome

The *fatty liver syndrome* is most common in the early postpartum period and occurs in cows that were very fat prior to parturition. Cows fed high-grain diets, especially high moisture corn and corn silage, are predisposed. These obese cows have the stress of calving and high production in the early postpartum period and incur fatty metamorphosis in the liver to the point where eventually the process becomes pathologic. Clinical signs are those of chronic anorexia, depressed milk yield with chronic ketosis. Treatment with dextrose and steroids are only temporary. We have had best results with rumen transplantation. However, some cows will continue to deteriorate and lose condition despite therapy. Occasionally these cows will show vague CNS signs: holding their head high with an odd stare in their eyes. The primary postmortem lesion is an extensive fatty metamorphosis of the liver. Several of these cows died with an associated septic mastitis or salmonellosis. It seems that the fatty liver predisposes them to other infectious diseases.

References

1. *Fox, F. H.* in Bovine Medicine and Surgery, Chapter 10, p. 426, American Veterinary Publications, Inc. (1970). – 2. *Bockus, H. L.* Gastroenterology. Vol. I, 2nd Ed., W. B. Saunders Co., 421-613, 1963. – 3. *Todd, G. C. and L. Krook.* Nutritional Hepatic Necrosis in Beef Cattle. Path. Vet. 3: 379, 1966. – 4. *Rooney, J. R., D. F. Watson and W. C. Hoag.* Abomasal Ulceration and Perforation. North Am. Vet. 37: 750, 1956. – 5. *Mayer, G. P., C. F. Ramberg and D. S. Koonfeld.* Calcium Homeostasis in the Cow. Clinical Orthopaedics 62: 79, 1969. – 6. *Pinsent, P. J. N. and H. E. Ritchie.* Ulceration of the Abomasum in a Guernsey Cow. Vet. Rec. 67: 769, 1955. – 7. *Fox, F. H.* Cattle Have Ulcers Too! Form Research 29: 14, 1963. – 8. *Begg, H.* Diseases of the Stomach of the Adult Ruminant. Vet. Rec. 62: 797, 1950. – 9. *Svendsen, P.* Cecal Dilation in Cattle. Nord. Vet. Med. 22: 578, 1970.