Dairy Section

Chairman: Dr. Samuel Hutchins, III, South Barre, Vermont

Physiology of Abomasal Displacements

Right Paralumbar Omentopexy for the Correction of Left Displaced Abomasum

Non-Surgical Treatment of Displaced Abomasum

Endocrine Control of Calcium Metabolism and Parturient Hypocalcemia in Dairy Cattle

Symposium on Economics of Therapy from Dairy Herd Owners' Viewpoint

Physiology of Abomasal Displacements

R. H. WHITLOCK, *D.V.M.*
New York State Veterinary College
Cornell University
Ithaca, New York

Abomasal displacements were reported as early as 1898 but not frequently until the middle of the twentieth century (1). Since that time, they have been reported with increasing frequency in countries where intensive farming practices are reported.

Many investigators have offered opinions as to the cause of abomasal displacements (2,3,4,5). Esperson (6) stated that stricture of the duodenum may be associated with the right abomasal displacement. Sand or so-called geosediment has been associated with right abomasal displacements (7), but forced feeding of large amounts of sediment (sand) did not cause right abomasal displacements (8). Many writers have indicated that during parturition, the pregnant uterus displaces the rumen dorsally to allow the displacement to occur. Localized peritonitis (9), ascites (10) and inappropriate transportation of pregnant cattle (11) have all been suggested etiologic factors of abomasal displacements.

It seems unlikely to cite pregnancy and parturition as direct causes of displacements as displacements have occurred in adult dairy bulls, cows in the middle of lactation, and in heifers (13). Certainly the gravid uterus and parturition must be considered as predisposing factors as nearly 80% of abomasal displacements occur in the first month postpartum (14).

Most of us agree with Pinsent (13) and Dirksen (5) who proposed early that atony with gas distention must be prime requisites for abomasal displacements; however, until recently, the genesis of the atonic state was only speculation.

Recently, Svendsen (16) has shown that abomasal motility is directly related to the feed or concentrate consumption by the cow. The normal abomasum has 1.2 to 2.2 contractions per minute. These contractions are not continuous but cyclical. Nor are the contractions peristaltic but occur in two regions, the fundus and the pylorus. These contractions are increased in rate and strength during the feeding of hay and concentrates to the cow. However, after feeding concentrates, the rate of contractions and strength of contractions are diminshed for

about one to two hours. They indicated that the motility was directly related to the concentration of fatty acids concentration (VFA) in the abomasum. The increased VFA concentration was associated with increased concentrate feeding.

Svendsen also measured the amount of gas produced by the normal abomasum when the cow was not eating good. This amounted to about 500 milliliters per hour. When the cow consumed hay actively, 800 liters of gas were produced per hour in the abomasum. When three pounds of concentrate were fed, this increased to 1100 milliliters per hour. When 15 pounds of concentrate were fed, this increased to 2200 milliliters per hour. Again, a direct relationship existed between the amount of abomasal gas formed and the amount of concentrate fed. The gas was primarily carbon dioxide, methane and nitrogen. The amount of each of these varied considerably with each experiment. Now that we have covered the factors relating to the cause of displacements, let us investigate the consequences of displacement of the abomasum.

Characteristic fluid and electrolyte changes are associated with right or left abomasal displacements (13). These include a hypochloremia, an elevated blood bicarbonate, a decreased blood potassium, dehydration as indicated by an increase in total protein and packed cell volume. The typical cow will show a normal chloride of about 100 to 105 with a severe abomasal torsion which may go as low as 60 meq per liter. Conversely, the bicarbonate will increase from a normal of 25 meq to 45 or 50 with a severe alkalosis. The potassium tends to decrease from a normal of about 4 meg. to 2 meg./l. The PCV will increase from 33% up to 40 to 45%.

The reasons for the acid-base and electrolyte changes include a reflux of hydrochloric acid from the abomasum into the rumen. This creates a massive vat with increased concentrations of HC1. This leaves a relative excess of bicarbonate in the extracellular fluid thus a metabolic alkalosis. So far we have been able to explain the increase in blood bicarbonate and the decrease in the plasma chloride. The potassium tends to decrease with a shift of potassium into the cell as the hydrogen ion comes out of the cell trying to neutralize the excess bicarbonate. As the alkalosis is corrected, potassium again will tend to come back out of the cells to help restore the extracellular potassium level. However, cows with abomasal displacements tend to be anorexic and as a result of this, consume less potassium than normal. The kidneys usually excrete a large amount of potassium, with a decreased dietary intake the kidneys are unable to decrease the potassium excretion proportionally and we have excess potassium loss through the kidneys in anorexic cows.

Thus, when considering therapy for abomasal displacements, one should use solutions of sodium chloride and potassium chloride. If the conditions are not too advanced, potassium chloride (60 to 80 grams per day per os) is of value. In severe metabolic alkalosis, sodium chloride or ammonium chloride intravenously are indicated. We use 180 grams of potassium chloride and 80 grams of ammonium chloride in 20 liters of water. This provides about 75 milliequivalents per liter of ammonia, 75 milliequivalents of potassium and 150 milliequivalents of chloride. To this we often add 500 ccs of 40% dextrose to provide energy. This combination of electrolytes seems to be very beneficial for correcting the severe metabolic alkalosis associated with abomasal torsion or abomasal impactions.

References

1. *Corougeau and Prestat.* Torsion de la caillette chez un veau. J. Med. Vet. 2: 340, 1898. – 2. *Moore, G. R., W. F. Riley, R. W. Westcott and G. H. Conner.* Displacement of the Bovine Abomasum. Vet. Med. 49: 49, 1954. – 3. *Hansen, A. G., E. P. Elefson, H. E. Warsinske, O. Hjort and R. Schornberg.* Displaced Abomasum: A Relatively Common Bovine Syndrome. North Am. Vet. 38: 129, 1957. – 4. *Begg, H. and W. A. Whiteford.* Displacement of the Abomasum in the Cow. Vet. Rec. 68: 122, 1956. – 5. *Dirksen, G.* Die Erweiterung, Verlagerung and Drehung des Labmagens beim Rind. Paul Parey, Berlin and Hamburg, 1962. – 6. *Jones, W.* Abomasal Displacement in Cattle. Cornell Vet. 42: 53, 1952. – 7. *Esperson, G.* Dilatatio et Dislocatio ad Dextram Abomasi Bovis. Nord. Vet. Med. 13, Suppl. I, 1961. – 8. *Svendsen, P.* Geosedimentum abomasi bovis. Nord. Vet. Med. 17: 500, 1965. – 9. *Marr, A. and W. F. Jarrett.* Displacement of the Abomasum Associated with Peptic Ulceration in a Cow. Vet. Rec. 67: 332, 1955. – 10. *Albert, T. F. and D. B. Ramey.* Abomasal Displacement Associated with Ingestion of Gasoline. J.A.V.M.A. 145: 460, 1964. – 11. *Nilsson, L. S.* Etiology of Abomasal Displacement. Mod. Vet. Pract. 43: 68, 1962. – 12. *Rosenberger, G. and G. Dirksen.* Uber die Labmagenverlagerung des Rindes. Deutsche Tierartzl. Wshnschr. 64: 2, 1957. – 13. *Whitlock, R. H.* Unpublished observations. – 14. *Whitlock, R. H.* Diseases of the Abomasum Associated with Current Feeding Practices. J. Am. Vet. Med. Assoc. 154: 1203, 1969. – 15. *Pinsent, P. J. N., P. A. Neal and H. E. Ritchie.* Displacement of the Bovine Abomasum: A Review of 80 Clinical Cases. Vet. Res. 73: 729, 1962. – 16. *Svendsen, P.* Abomasal Displacement in Cattle. Nord. Vet. Med. 22: 571, 1970.