Veterinarians treating dairy cattle commonly diagnose displacement of the abomasum. However, displacement of the abomasum (DA) is a relatively recent phenomenon with respect to the development of the veterinary profession. The earliest reported cases of DA were by Carougeau and Prestat in 1898 and Fincher in 1927, but this disease did not become commonly reported until after the 1940’s. ¹⁻⁷ Prior to that time, DA’s either did not occur, were not diagnosed, or were not described as such. Begg reported three cases of left sided displacement of the abomasum (LDA) of which one cow died of peritonitis following surgical reduction of the abomasum and two cows returned to normal after withholding all food for 48 hours.³ Jones described manual correction of LDA without stabilization of the abomasum in two cows.⁴ The incidence of diagnosis of displacement of the abomasum increased greatly after the mid-1960’s and is now an internationally recognized problem of dairy cows. Displacement of the abomasum is commonly referred to as a “disease of high milk production”. This statement may be justified in that as dairy cows have been selected for genetic improvement based primarily on milk production, these same cows may have been selected into a high-risk group for development of DA. Presumably, displacement of the abomasum has existed for many centuries and was simply not recognized. The justification for this supposition is based on the fact that DA’s are observed in calves, young stock, adult bulls, and beef cows. Only recently has research been directed more toward identification of risk factors and institution of prevention strategies rather than comparisons of specific treatment techniques. This article will focus on the clinical syndrome of DA and will summarize available information regarding risk factors and prediction variables associated with development of displacement of the abomasum.

**Clinical Syndrome**

Displacement of the abomasum may occur to the left (LDA ~ 90 %), right (right displaced abomasum (RDA) or right abomasal volvulus (RAV) ~ 10 %), or, rarely, medially displacing the omental sling. In 1971, Wallace reported that LDA occurs most commonly in 4 to 6 year old Holstein cows during the first 6 weeks postpartum.⁸ Historical factors found to be common among cows with LDA included hypocalcemia (12.4%), ketosis (42%), metritis (41.2%), retained fetal membranes (30.1%), and various stressors (11.7%). Also, LDA appeared to be more common among cows having twins. Physical examination findings common among cows with LDA included metritis (43.5%), mastitis (19%), enteritis (7.3%), and retained fetal membranes (4.8%). Research documented since this study generally has concurred with these findings. Left displacement of the abomasum causes a displacement of the abomasum along its long axis (e.g. partial torsion) without volvulus. The torsion is caused by rotation of the abomasum along its long axis ventral and to the left of the rumen. Little or no outflow obstruction of the abomasum occurs and life-threatening abomasal obstruction is rare with LDA. LDA does result in decreased appetite and milk production possibly as a result of pain, increased forestomach transit time, and stress. Left displacement rarely causes ischemia to the abomasum, but abomasal ulcers are not uncommon among cows having long-standing LDA.⁹,¹⁰ Right displacement causes a similar clinical syndrome as LDA, but is potentially life threatening because the displacement also may rotate about the mesenteric axis and cause abomasal
volvulus. Abomasal volvulus causes an outflow obstruction of the abomasum and ischemia ensues if the gastric arteries or veins become obstructed (abomasal arterial blood is supplied by the right and left gastric arteries along the lesser curvature and the right and left gastroepiploic arteries along the greater curvature; venous drainage occurs via veins of the same name). Abomasal outflow obstruction causes progressive forestomach dilation (e.g., fluid bloat) which causes eventual respiratory and cardiovascular embarrassment. Rumen distention is most severe if the omasum becomes involved as a result of the volvulus of the abomasum (e.g., failure of eructation causing fluid and free gas bloat). Abomasal-omasal torsion and volvulus has a greater risk for development of ischemia to the forestomachs because occlusion of the venous drainage and arterial supply is more likely. Fortunately, relatively few cattle with RAV have concurrent omasal torsion.\(^8\) Medial displacement of the abomasum is rare and causes identical clinical signs as LDA. In this type of displacement, the greater curvature of the abomasum rotates (torsion) medially and dorsally along the medial wall of the rumen (does not pass under the rumen). This results in the abomasum displacing the omentum dorsally so that it protrudes into the supr omental recess. Definitive diagnosis of abomasal displacement is based on surgical findings or necropsy. However, clinical diagnosis based on simultaneous auscultation and percussion of the abdomen has been shown to be a reliable tool for diagnosis of DA. Smith et al performed a retrospective study of 366 cows with right-sided abdominal resonance (“ping”).\(^11\) Of 366 cattle, 137 had DA, 157 had intestinal gas involving the cecum, spiral colon, or small intestine, and 2 cattle had peritoneal gas. A definitive diagnosis was available for 151 cattle and yielded a positive predictive value of 96% for abomasal displacement and a positive predictive value of 87% for cecum or ascending colon distention.

**Mechanisms for Displacement of the Abomasum**

By-products of fermentation seem important to the development of DA, but the management and animal factors, which culminate in DA, are not clear. Petty et al suggested that altered exercise and feeding patterns caused by management practices might contribute to development of LDA.\(^25\) The reason for displacement to the left side rather than to the right side is elusive. Mulville and Curran (1993) described the abomasum as being positioned along ventral midline medial and ventral to the omasum.\(^26\) Presumably, displacement of the abomasum to the left side is caused by pressure from the omasum located dorsal, lateral, and to the ride side of the abomasum. This theory is supported by an anatomy text in which the abomasum is shown lying medial and ventral to the rumenoreticular groove with the omasum located dorsal to it.\(^27\) This orientation would cause an anatomic predisposition to left-sided displacement through the space created by the confluence of the rumino-recticular groove and the cranial groove of the rumen. Terms commonly used to evaluate risk factors include relative risk (RR), odds ratio (OR), and likelihood ratio. Relative risk and odds ratios are measures of the odds of an animal with a given factor having the disease compared with the odds of an animal without that factor having the disease. Likelihood ratios are a means to express the odds that a certain value of a diagnostic test would be expected in a subject with the disease compared to a subject without the disease. Harman et al evaluated the effects of season, parity, and concurrent disease on parturition-to-conception interval in 44,450 Finnish Ayrshires.\(^28\) In this study, 148 multiparous cows were diagnosed with abomasal disorders (0.5% lactation incidence) at a median of 29 days-in-milk and 30 primiparous cows (0.2% lactation incidence) had abomasal disorders a median of 41 days-in-milk. Constable et al (1992) found that age, breed, gender, and season were risk factors for abomasal volvulus and LDA.\(^29\) Cattle at the greatest risk of developing LDA or RAV were 4
to 7 year old dairy cows in January (for RAV) or March (for LDA) and during the first 2 weeks of lactation. Among dairy breeds, Guernsey cattle had greater odds of LDA and Brown Swiss cattle had lower odds of RAV compared with Holstein cattle. Markusfeld reported that cows in their 5th or higher lactation had the highest risk of developing DA (RR 2.41). Periparturient diseases are common among cattle with DA. Oikawa et al (1997) reported that ketosis and LDA were linked to fatty liver disease. Interestingly, cows in Oikawa’s study with LDA alone had higher NEFA’s but similar BHB concentration compared with cows in mid-lactation. Cows with LDA and ketosis had BHB concentrations similar to cows with ketosis alone. Rehage et al (1996) reported that the severity of fatty liver disease, determined histologically, in cows having a DA was severe in 32 %, moderate in 40 %, and mild or absent in 28 %. Of these cattle with DA, 55 % had mastitis, endometritis, or lameness. Itoh et al (1997) found that cows with ketosis, DA, and fatty liver disease had decreased concentrations of apolipoprotein B (normal for mature cows, 259 ± 63 µg/ml). Geishauser et al (1997 and 1998) found that cows having an AST 180 U/L and those having a BHB of 1600 µmol/L had greater odds ratio for subsequent diagnosis of LDA. Time of the year, animal factors, and feed factors are important to the development of DA, but these effects are difficult to quantitate. Of 15 management factors entered into a risk factor model for LDA by Correa et al, only lead feeding was preserved in the model after statistical analysis. Cows that had lead feeding had an odds ratio of 4.4 for development of LDA. In that study, cows having metritis had an odds ratio of 43.7. Markusfeld (1986) reported the relative risk for abomasal displacement associated with periparturient factors which included twinning (RR 2.29), retained placenta (RR 6.62), metritis (RR 4.26), aciduria (RR 6.17), and ketonuria (RR 33). Hypocalcemia has long been recognized as a risk factor for LDA because of abomasal atony. Massey et al (1993) found that cows with hypocalcemia at parturition had 4.8 times greater risk of developing LDA. Oetzel (1996) prophylactically administered calcium chloride gel to cows 12 hours before calving, at calving, and 12 and 24 hours after calving. Significantly fewer cases of parturient paresis, parturient hypocalcemia, and DA occurred in treated cows. Goff and Horst reported that the incidence of post-partum hypocalcemia greatly increased when potassium was added to the diet at 2.1 or 3.1 % (sodium was present at 0.12 %). When a diet of 1.3 % sodium and 1.5 % calcium was fed, a similar effect was observed.