

# Nutritional Risk Factors in the Etiology of Left Displaced Abomasum in Dairy Cows: A Review<sup>1</sup>

R. D. SHAVER

Department of Dairy Science,  
University of Wisconsin, Madison 53706

## ABSTRACT

The transition period occurring 2 wk prepartum through 2 to 4 wk postpartum is the major risk period in the etiology of left displaced abomasum. The prepartum depression of intake and the slow postpartum increase in intake are risk factors causing lower ruminal fill, reduced forage to concentrate ratio, and increased incidence of other postpartum disorders. Uncomplicated ketosis, retained placenta, metritis, and hypocalcemia at parturition are risk factors for left displaced abomasum. Excessive amounts of concentrate during the prepartum period increase the risk of left displaced abomasum, which may occur from the lower ruminal fill caused by greater prepartum intake depression and reduced forage to concentrate ratio, decreased ruminal motility from lower ruminal fill and higher volatile fatty acid concentration, and decreased abomasal motility and emptying from higher concentrations of volatile fatty acids. Effects of volatile fatty acids on motility may be exacerbated by low ruminal absorption of volatile fatty acids during the transition period. Minimal intake of concentrate during the prepartum period may increase the risk of left displaced abomasum through failure to increase the absorptive capacity of the ruminal papillae and failure of the microbial population of the rumen to adapt prior to the intake of high energy postpartum diets. Increased risk of left displaced abomasum in cows that are hypocalcemic at parturition may be due to decreased ruminal and abomasal motility.

(**Key words:** nutrition, displaced abomasum, peripartum disorders, transition cow)

**Abbreviation key:** BCS = body condition score, F:C = forage to concentrate ratio, LDA = left displaced abomasum, OR = odds ratio.

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## INTRODUCTION

Abomasal displacements cause economic loss in dairy herds through treatment costs, premature culling, lost production, and death. Current treatment costs range from \$100 to \$200 per case, and 10% of the cows that are diagnosed with displaced abomasum are culled or die before the next test day (13). Treated cows that remain in the herd produce 350 kg less milk the following month than cows without a displaced abomasum (13).

Eighty to 90% of all abomasal displacements are left-sided (21, 27, 34). Estimates of mean yearly incidence rates for left displaced abomasum (LDA) in lactating dairy cows range from 1.4 to 5.8% (7, 8, 12, 15, 22, 24, 25, 27). Incidence rates for individual herds within these studies ranged from 0 to 21.7%. Pehrson and Shaver (27) reported a mean incidence rate for LDA of 5% (0 to 21.7%) from a survey of 71 dairy herds with 5742 cows. Incidence rates ranged from 1% (0 to 3%) in 34 low incidence (<3% LDA) herds to 8.7% (3.2% to 21.7%) in 37 high incidence (>3% LDA) herds. Jordan and Fourdraine (22) reported a mean incidence rate for LDA of 3.3% (0 to 14%) from a survey of 61 high producing dairy herds (244 cows per herd) averaging 11,096 kg of milk per lactation.

Nutrition has been implicated as a major risk factor in the etiology of LDA (5). This review evaluates nutritional risk factors in the etiology of LDA and discusses feeding and management practices for its prevention. For a recent review of nonnutritional risk factors in the etiology of LDA, refer to the publication by Geishauser (17).

## TRANSITION PERIOD

Eighty to 90% of LDA are diagnosed within 1 mo postpartum (4, 15, 27, 28, 34); of those, the proportion of LDA that are diagnosed within 2 wk postpartum is estimated to be 52% (34), 56% (15), 57% (4), and 86% (28). This transition period, from 2 wk prepartum through 2 to 4 wk postpartum, is the major risk period in the etiology of LDA. The transition period is characterized by prepartum intake

depression (2), followed by a slow increase in postpartum intake (23). Low feed consumption during the transition period is a risk factor for LDA through lower ruminal fill, reduced forage to concentrate ratio (**F:C**) in herds that are not fed TMR, and increased incidence of other postpartum disorders. Low ruminal fill may provide greater opportunity for migration of the abomasum (4). Reduced F:C results from the overconsumption of concentrate relative to forage and contributes to lower ruminal fill. The decline of DMI is about 35% over the final week prepartum, which causes increased concentrations of liver triglycerides immediately postpartum (2). Concurrent postpartum disorders have been implicated as risk factors for LDA (4).

### Postpartum Disorders

Curtis et al. (8) reported that cows with uncomplicated ketosis were at increased risk [odds ratio (**OR**) = 11.9] for LDA. Cows with retained placenta [(24); **OR** = 6.8] or metritis (7, 24); **OR** = 43.7 and 4.7, respectively] were also at increased risk for LDA. Massey et al. (25) reported that cows that were hypocalcemic at parturition (total serum calcium <7.9 mg/dl) were at increased risk (**OR** = 4.9) for LDA. This result suggests that feeding and management practices that prevent other postpartum disorders reduce the risk of LDA.

Conversely, LDA has been found to increase the risk of other postpartum disorders. Cows with LDA are at increased risk for complicated ketosis (8, 24; **OR** = 53.5 and 50.4, respectively) and metritis (8; **OR** = 3.6). Thus, feeding and management practices that prevent LDA reduces the incidence of some other postpartum disorders. Incidence of ketosis and LDA are closely related postpartum disorders.

### Body Condition Score

Cows with excess body condition score (**BCS**) at parturition are at increased risk for LDA (12); incidence rates for cows ( $n = 1401$  in 95 commercial dairy herds) with low (2.75 to 3.25; thin), medium (3.25 to 4), and high ( $\geq 4$ ; obese) BCS at parturition were 3.1, 6.3, and 8.2%, respectively. The increased incidence rate for cows with high BCS may be related to increased ketosis and fatty liver, greater prepartum intake depression, and slower increases in postpartum intake for cows that are overconditioned at parturition.

Cows with excess BCS at parturition are at increased risk of ketosis (12); incidence rates for cows with low, medium, and high BCS at parturition were

8.9, 11.5, and 15.7%, respectively. The higher incidence of ketosis in cows with greater BCS at parturition may have predisposed those cows to the higher observed incidence of LDA. Further, cows with higher prepartum plasma NEFA concentrations were at increased risk of LDA; incidence rates for cows with low, medium, and high NEFA concentrations were 3.6, 5.9, and 10.2%, respectively (12).

Cows with low DMI at d 1 prepartum have reduced DMI at d 21 postpartum (20). There are few data concerning the relationship between BCS and prepartum intake depression, but reports (14, 36) support the premise that overconditioned cows have greater depression of prepartum intake. Emery (14) summarized feed intake data from the dry period of 20 multiparous cows and found DMI of 1.5 and 2.0% of BW for cows with high ( $>3.6$ ) and low ( $\leq 3.6$ ) BCS, respectively.

Garnsworthy and Topps (16) fed cows to a BCS (adjusted to a five-point scale, where 1 = thin to 5 = obese) at parturition of 2 to 3 (low), 3 to 4 (medium), and 4 to 5 (high) in two trials. During the first 16 wk postpartum, cows with higher BCS at parturition consumed less DM and reached maximum DMI later. Similar results were reported by Treacher et al. (32) using two groups of cows with BCS at parturition of 3 and 5.

### Lead Feeding

Lead feeding, the practice of increasing concentrates during the last 2 to 3 wk prior to parturition, is a common practice on commercial dairies (30). Evidence is limited that this practice reduces postpartum disorders.

Curtis et al. (8) reported that cows that were lead fed energy and protein were at lower risk of LDA (**OR** = 0.3 to 0.4) and ketosis (**OR** = 0.2 to 0.8), respectively. However, Correa et al. (7) reported that cows that were lead fed concentrates were at increased risk of LDA (**OR** = 4.4) and milk fever (**OR** = 2.4). Differences in level of lead feeding between studies may explain the conflicting results; Correa et al. (7) suggested that diets administered as part of the lead-feeding programs they surveyed had higher energy and calcium concentrations than recommended.

A retrospective case survey of 30 low incidence herds and 30 high incidence herds for LDA noted that higher grain amounts had been fed 4 to 6 wk prepartum in the high incidence herds (28). Because no within-herd correlation was found between energy and incidence of LDA, this relationship could have been coincidental rather than cause and effect.

Coppock et al. (6) fed TMR containing 75, 60, 45, and 30% forage (DM basis) to 40 Holstein cows from 4 wk prepartum through 4 wk postpartum. No LDA was observed in cows fed the high forage diet. Incidence rates for LDA in cows fed the 60, 45, and 30% forage diets were 16.7, 40, and 36%, respectively. This study involved an abrupt switch to TMR, with higher percentages of concentrate at 4 wk prepartum, rather than a gradual increase in the amount of concentrate fed during the last few weeks prior to calving. This abrupt dietary switch may have aggravated the LDA response to higher concentrate feeding, but reflects lead feeding practices for commercial dairy herds fed TMR (30). It was impossible to separate effects of lead feeding from effects of high postpartum concentrate feeding in this study because the same TMR were fed prepartum and postpartum. However, prepartum lead feeding was probably a factor in the high incidence of LDA because the earliest LDA were diagnosed 3 d postpartum (mean = 10.6 d; range = 3 to 25 d). Mean DMI during the prepartum period was not affected by treatment, but DMI at parturition was highest for the high forage diet (1.6 vs. 1.2% of BW), and prepartum intake depression increased as the percentage of concentrate increased. This result may partially explain the high incidence of LDA observed in this study.

For herds that are not fed TMR, high amounts of concentrates may result in very low prepartum F:C. Coppock et al. (6) defined lead feeding as the practice of increasing the amount of concentrate fed over the 2 to 3 wk prior to parturition to 1 to 1.5% of BW. With a total DMI of 1.25% of BW at 1 d prior to parturition (2), dietary concentrate at 1% of BW would result in a 20:80 F:C. This ratio assumes that the concentrates offered are consumed at the expense of forage DM. A guideline for prepartum concentrate lead feeding of 0.5 to 0.75% of BW restricts F:C between 60:40 and 40:60 after acknowledging prepartum intake depression.

The increased risk of LDA from excessive amounts of concentrates fed 2 to 4 wk prior to parturition may be related to lower ruminal fill from greater prepartum intake depression and reduced F:C, decreased ruminal motility from lower ruminal fill (35) and higher VFA concentration (18), and decreased abomasal motility and emptying from higher VFA concentration (3, 19). Effects of VFA on motility may be exacerbated by low absorption of VFA during the transition period (11).

Dirksen et al. (11) reported that the cross-sectional area of ruminal papillae declined when cows were placed on a low energy dry cow diet; the lowest point was attained 1 to 2 wk prior to parturition. The cross-sectional area of ruminal papillae increased

gradually after cows were placed on a high energy lactation diet starting 2 wk prior to parturition, but area was not maximized until 6 to 8 wk postpartum. This result suggests that the capacity for ruminal VFA absorption is lowest during the transition period. In support of this conclusion, an absorption experiment (11) showed that relative VFA absorption of cows was higher after a high energy diet was fed than after a low energy diet (100 vs. 30 and 46%) was fed.

Minimal lead feeding may increase the risk of acidosis and LDA through a failure to increase the absorptive capacity of the ruminal papillae prior to the intake of high energy postpartum diets. Prepartum adaptation of the microbial population in the rumen prior to the intake of high energy postpartum diets may also be important. Further, lead feeding of concentrate during the prepartum period may increase energy intake of cows and reduce fatty acid mobilization from adipose tissue, which may reduce the incidence of fatty liver and ketosis (2).

The practice of lead feeding is equivocal. Intake of excessive or minimal amounts of dietary concentrates prepartum may increase the risk of LDA. More research is needed on lead feeding strategies. Until more data are available, a recommendation for prepartum concentrate lead feeding of 0.5% of BW with an upper limit of 0.75% of BW appears to be reasonable.

### Postpartum Concentrate Feeding

Because of the slow increase in intake during the postpartum period (23), very low F:C may occur in herds that are not fed TMR. Using DMI for wk 1 through 4 postpartum of 16.6, 19.3, 21.1, and 22.3 kg/d [(23); multiparous cows] and assuming that the amount of concentrate formulated for wk 4 with 50:50 F:C is fed during early lactation, the F:C for wk 1, 2, and 3 are 30:70, 40:60, and 45:55, respectively. These rations assume that the concentrates offered are consumed at the expense of forage DM.

It is recommended that concentrates be held to the lead feeding amount for 3 to 4 d following parturition. Concentrate DM can then be increased at the rate of 0.20 to 0.25 kg/d until peak lactation is reached. Concentrates should be fed at least three to four times daily. Feeding a TMR to control F:C is recommended. A transition group TMR for early postpartum cows is also recommended.

### Hypocalcemia

From a study of 510 Holstein cows in a commercial dairy herd, Massey et al. (25) reported that cows that

were hypocalcemic at parturition (total serum calcium < 7.9 mg/dl and serum-ionized calcium < 4.0 mg/dl) were at increased risk (OR = 4.9) of LDA. Using i.v. infusions of sodium EDTA to induce hypocalcemia in cows and ewes, Daniel (9) observed reduced ruminal and abomasal motility. That result may explain why cows that are hypocalcemic at parturition are at increased risk of LDA. Strategies to prevent hypocalcemia at parturition, such as formulation of prepartum diets for dietary cation-anion difference, may be useful for the prevention of LDA.

### RATION PHYSICAL FORM

Dawson et al. (10) reported that cows fed ground alfalfa hay (0.64-cm hammer mill screen) and concentrate in a pelleted (0.48 cm) experimental TMR starting at parturition were at higher risk for LDA (17.4 vs. 1.6%; OR = 10.8) than were cows fed the standard herd ration of sorghum silage (1.27-cm theoretical length of cut) and concentrate mixed plus loose alfalfa hay. Cows that developed LDA were diagnosed within 8 to 18 d postpartum.

These results demonstrate that an extreme alteration in ration physical form (pelleted TMR) during the early postpartum period increases the incidence of LDA. Data are lacking with regard to the impact on the incidence of LDA of variations of the physical form of silage and TMR within typical field ranges. Shaver (29) recommended that haycrop silages be chopped to contain 15 to 20% of the particles (weight basis) over 4 cm long, but this recommendation was based solely on milk fat test considerations.

Research is needed to determine the critical physical form of the forage and TMR for preventing LDA. The lack of physical form reduces chewing activity (1, 31), ruminal fill (31), motility (1, 35), and fiber mat formation (33) and increases ruminal VFA concentration (31), all of which may affect the etiology of LDA (21). The importance of physical form as a risk factor for LDA is likely to be greatest during the early postpartum period because of the physiologic and metabolic changes in the transition period.

### DRY COW FORAGE PROGRAM

A myriad of forage programs are used for dry cows on commercial dairies, but data are limited regarding their impact on the incidence of LDA. Zamet et al. (36) reported LDA incidence rates of 3.5, 10, and 10% for dry cow forage programs of chopped hay, haycrop silage, and corn silage. Nocek et al. (26) evaluated dry cow forage programs consisting of long hay, 50% long hay and 50% corn silage (DM basis), and corn silage DM restricted to 1% of BW plus 1.1 kg of liquid protein supplement/d per cow. Incidence rates for

LDA were 3.0, 4.3, and 6.3% for hay, hay and corn silage, and corn silage, respectively. Incidence rates for ketosis were highest for hay (9.1% vs. 6.3 to 6.4%). The higher incidence of LDA for corn silage may have been due to low ruminal fill being related to restricted amounts of dietary energy and lack of physical form. The higher incidence of ketosis for hay may have been due to lack of dietary energy. The lowest incidence of LDA plus ketosis was observed for hay and corn silage (10.6% vs. 12.1 to 12.7%). Rations of all corn silage should not be fed to dry cows. If cows are fed restricted amounts, ruminal fill may not be sufficient to prevent LDA, but excess energy consumption may cause overconditioning and associated metabolic disorders. However, the controlled use of corn silage as a component of forage programs for dry cows may be beneficial.

### BUNK MANAGEMENT

Feed bunk management is a risk factor for LDA through effects on feed consumption and actual nutrient densities of the consumed ration. Inadequate bunk space and high competition at the feed bunk may limit feed intake. Also, restricted bunk access time and feed availability may limit intake. Poor environmental and social adaptation of transition cows may limit feed intake. Low feed intake may lower ruminal fill, providing a greater opportunity for migration of the abomasum (4). The importance of bunk management practices that limit feed intake in the etiology of LDA is likely to be greatest during the early postpartum period, because of the coinciding events of the transition period. The TMR mixing process can alter the actual nutrient densities of the consumed ration relative to nutrient specifications of the formulated ration. Sorting of the TMR in the feed bunk can also cause this problem. Fiber densities of the consumed ration may result that are below minimum recommended allowances. Excess TMR mixing may grind coarse particles and cause a lack of fiber physical form.

### CONCLUSIONS

Because of low feed consumption, the transition period is the major risk period in the etiology of LDA. Feeding and management practices that prevent other postpartum disorders reduce the risk of LDA. Ketosis and LDA are closely related postpartum disorders, and cows that have excess BCS at parturition are at increased risk of ketosis and LDA.

Both excessive and minimal amounts of dietary concentrates during the prepartum period may increase the risk of LDA. More research is needed on lead feeding strategies. Prepartum concentrate lead

feeding of 0.5% of BW with an upper limit of 0.75% of BW is recommended. For herds that are not fed TMR, postpartum concentrate DM can be increased at the rate of 0.20 to 0.25 kg/d until peak concentrate intakes are reached; concentrates should be fed at least three to four times daily. A TMR that has been formulated to control F:C and to consider nutritional needs of early postpartum cows is recommended.

There is increased risk of LDA in hypocalcemic cows at parturition, suggesting a role for the formulation of prepartum diets for dietary cation-anion difference in the prevention of LDA. Although a pelleted TMR increased the incidence of LDA, research is needed to determine the critical forage and TMR physical form for preventing LDA. Rations composed entirely of corn silage should not be fed to dry cows. Feed bunk management is an important risk factor for LDA that should be monitored closely on commercial dairies.

## REFERENCES

- 1 Beauchemin, K. A. 1991. Ingestion and mastication of feed by dairy cattle. *Vet. Clin. North Am. Food Anim. Pract.* 7:439.
- 2 Bertics, S. J., R. R. Grummer, C. Cadorniga-Valino, and E. E. Stoddard. 1992. Effect of prepartum dry matter intake on liver triglyceride concentration and early lactation. *J. Dairy Sci.* 75:1914.
- 3 Bolton, J. R., A. M. Merritt, G. M. Carlson, and W. J. Donawick. 1976. Normal abomasal electromyography and emptying in sheep and the effects of intraabomasal volatile fatty acid infusion. *Am. J. Vet. Res.* 37:1387.
- 4 Constable, P. D., G. Y. Miller, G. F. Hoffsis, B. L. Hull, and D. M. Rings. Risk factors for abomasal volvulus and left abomasal displacement in cattle. *Am. J. Vet. Res.* 53:1184.
- 5 Coppock, C. E. 1974. Displaced abomasum in dairy cattle: etiological factors. *J. Dairy Sci.* 57:926.
- 6 Coppock, C. E., C. H. Noller, S. A. Wolfe, C. J. Callahan, and J. S. Baker. 1972. Effect of forage:concentrate ratio in complete feeds fed ad libitum on feed intake prepartum and the occurrence of abomasal displacement in dairy cows. *J. Dairy Sci.* 55:783.
- 7 Correa, M. T., C. R. Curtis, H. N. Erb, J. M. Scarlett, and R. D. Smith. 1990. Ecological analysis of risk factors for postpartum disorders of Holstein-Friesian cows from thirty-two New York farms. *J. Dairy Sci.* 73:1515.
- 8 Curtis, C. R., H. N. Erb, C. J. Sniffen, R. D. Smith, and D. S. Kronfeld. 1985. Path analysis of dry period nutrition, postpartum metabolic and reproductive disorders, and mastitis in Holstein cows. *J. Dairy Sci.* 68:2347.
- 9 Daniel, R.C.W. 1983. Motility of the rumen and abomasum during hypocalcemia. *Can. J. Comp. Med.* 47:276.
- 10 Dawson, L. J., E. P. Aalseth, L. E. Rice, and G. D. Adams. 1992. Influence of fiber form in a complete mixed ration on incidence of left displaced abomasum in postpartum dairy cows. *JAVMA* 200:1989.
- 11 Dirksen, G. U., H. G. Liebich, and E. Mayer. 1985. Adaptive changes of the ruminal mucosa and their functional and clinical significance. *Bovine Pract.* 20:116.
- 12 Dyk, P. B. 1995. The association of prepartum non-esterified fatty acids and body condition with peripartum health problems on 95 Michigan dairy farms. M.S. Thesis, Michigan State Univ., East Lansing.
- 13 Eicker, S. W. 1995. Milk production loss after displaced abomasum disease in New York Holsteins. *J. Dairy Sci.* 78(Suppl. 1):169.(Abstr.)
- 14 Emery, R. S. 1993. Energy needs of dry cows. Page 35 in *Proc. Tri-State Dairy Nutr. Conf. Ft. Wayne, IN. Ohio Coop. Ext. Serv., Columbus.*
- 15 Erb, H. N., R. D. Smith, R. B. Hillman, P. A. Powers, M. C. Smith, M. E. White, and E. G. Pearson. 1984. Rates of diagnosis of six diseases of Holstein cows during 15-day and 21-day intervals. *Am. J. Vet. Res.* 45:333.
- 16 Garnsworthy, P. C., and J. H. Topps. 1982. The effect of body condition of dairy cows at calving on their food intake and performance when given complete diets. *Anim. Prod.* 35:113.
- 17 Geishauser, T. 1995. Abomasal displacement in the bovine: a review on character, occurrence, aetiology and pathogenesis. *JAVMA* 42:229.
- 18 Gregory, P. C. 1987. Inhibition of reticulo-ruminal motility by volatile fatty acids and lactic acid in sheep. *J. Physiol. (Camb.)* 382:355.
- 19 Gregory, P. C., and S. J. Miller. 1989. Influence of duodenal digesta composition on abomasal outflow, motility and small intestinal transit time in sheep. *J. Physiol. (Camb.)* 413:415.
- 20 Grummer, R. R. 1995. Impact of changes in organic nutrient metabolism on feeding the transition dairy cow. *J. Anim. Sci.* 73:2820.
- 21 Guard, C. 1990. Abomasal displacement and volvulus. Page 792 in *Large Animal Internal Medicine*. B. P. Smith, ed. C.V. Mosby Co., St. Louis, MO.
- 22 Jordan, E. R., and R. H. Fourdraine. 1993. Characterization of the management practices of the top producing herds in the country. *J. Dairy Sci.* 76:3247.
- 23 Kertz, A. F., L. F. Reutzel, and G. M. Thomson. 1991. Dry matter intake from parturition to midlactation. *J. Dairy Sci.* 74:2290.
- 24 Markusfeld, O. 1987. Periparturient traits in seven high dairy herds. Incidence rates, association with parity, and interrelationships among traits. *J. Dairy Sci.* 70:158.
- 25 Massey, C. D., C. Wang, G. A. Donovan, and D. K. Beede. 1993. Hypocalcemia at parturition as a risk factor for left displaced abomasum in dairy cows. *JAVMA* 203:852.
- 26 Nocek, J. E., J. E. English, and D. G. Braund. 1983. Effects of various forage feeding programs during the dry period on body condition and subsequent lactation health, production, and reproduction. *J. Dairy Sci.* 66:1108.
- 27 Pehrson, B. G., and R. D. Shaver. 1992. Displaced abomasum: clinical data and effects of periparturient feeding and management on incidence. Page 116 in *Proc. Am. Assoc. Bovine Pract. World Buiatrics Congress*. St. Paul, MN. Am. Assoc. Bovine Pract., Stillwater, OK.
- 28 Robertson, J. 1968. Left displacement of the bovine abomasum: epizootiologic factors. *Am. J. Vet. Res.* 29:421.
- 29 Shaver, R. D. 1990. Forage particle length in dairy rations. Page 58 in *Proc. Northeast Reg. Agric. Eng. Serv. Dairy Feeding Sys. Symp.*, Harrisburg, PA. Northeast Reg. Agric. Eng. Serv., Ithaca, NY.
- 30 Shaver, R. D. 1993. TMR strategies for transition feeding of dairy cows. Page 163 in *Proc. 54th Minnesota Nutr. Conf.*, Bloomington. Minnesota Coop. Ext. Serv., St. Paul.
- 31 Shaver, R. D., A. J. Nytes, L. D. Satter, and N. A. Jorgensen. 1986. Influence of amount of feed intake and forage physical form on digestion and passage of prebloom alfalfa hay in dairy cows. *J. Dairy Sci.* 69:1545.
- 32 Treacher, R. J., I. M. Reid, and C. J. Roberts. 1986. Effect of body condition at calving on the health and performance of dairy cows. *Anim. Prod.* 43:1.
- 33 Welch, J. G. 1982. Rumination, particle size and passage from the rumen. *J. Anim. Sci.* 54:885.
- 34 Whitlock, R. H. 1969. Diseases of the abomasum associated with current feeding practices. *JAVMA* 154:1203.
- 35 Wyburn, R. S. 1980. The mixing and propulsion of the stomach contents of ruminants. Page 35 in *Digestive Physiology and Metabolism in Ruminants*. Y. Ruckebusch and P. Thivend, ed. AVI Publ. Co., Inc., Westport, CT.
- 36 Zamet, C. N., V. F. Colenbrander, R. E. Erb, C. J. Callahan, B. P. Chew, and N. J. Moeller. 1979. Variables associated with peripartum traits in dairy cows. II. Interrelationships among disorders and their effects on intake of feed and on reproductive efficiency. *Theriogenology* 11:245.