Displaced Abomasum in Dairy Cattle: Etiological Factors

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Abstract

Displacement of the abomasum in dairy cows is occurring with increasing frequency in the United States and probably in other areas of the world where dairying is practiced intensively. The primary causative factor appears to be an atony of the abomasum accompanied by dilatation and accumulation of gas. Factors contributing to atony and ultimately to displacement include stress of parturition, toxemia due to concurrent disorders such as metritis or mastitis, and metabolic disorders especially milk fever and ketosis. Genetic and mechanical effects seem to play a minor role in etiology of this disorder. Preventive measures include minimum grain and corn silage prepartum with other forage ad libitum and the best known management practices to reduce other parturient disorders.

Introduction

According to Svendsen (43) displaced abomasum in cattle was described by European writers as early as 1898, but it was not until about 1950 that several others in the United States and England (3, 15, 29, 33) rediscovered this disorder and it became recognized as a not unusual phenomenon. During the course of 580 exploratory laparotomies between 1948 and 1954, Michigan workers (33) found 33 animals with displaced abomasums (DA's) and 31 of them developed symptoms of the disorder during the late stages of gestation or within 2 wk postpartum. Since that time veterinary clinics in New York (16), Pennsylvania (38), Ohio (24), and Ontario (31) have reported a large increase in the number of cows treated for DA. Though some of this increase may reflect neo-diagnostic techniques and their application, some of the increase must be due to an increase in the occurrence of this serious disorder.

Displaced abomasum is a condition in which the abomasum becomes enlarged with fluid and/or gas with subsequent migration to the left or right and dorsally within the abdominal cavity. The rumen usually descends to trap the abomasum in the abnormal position. Right displacement (RDA) is usually accompanied by torsion (5, 43) which prevents digesta passage and presents a critical condition which requires treatment. Left displacement (LDA) is often associated with gas accumulation but little torsion so that digesta passage though reduced in volume is not blocked and a chronic condition may ensue.

The more common form in the USA is LDA with 80 to 90% of the displacements of this type (47). However, Espersen (14) reported that RDA is a disorder found more often in Denmark. Although spontaneous recovery may occur from LDA, it is less likely to occur from RDA. Consequently, highly successful surgical techniques have been developed (2, 22, 30, 39) which usually involve suturing the abomasum back in its normal position on the floor of the abdominal cavity. Non-surgical techniques which involve casting and rolling also have been advocated (6), but recurrence detracts from this less expensive procedure. Fortunately, recurrence is rare and recovery is usually rapid following surgical correction. The cost, however, including surgery and loss in milk can reach $150 per cow even under the most successful conditions.

Gregg (20) has summarized three predominant theories to explain causative factors associated with displacement of the abomasum: (a) the genetic theory explains the increased incidence of DA by attributing it to selective breeding for cows with large abdominal cavities which provide ample room for the abomasum to migrate; (b) the mechanical theory espouses the idea that the gravid uterus elevates the rumen from the abdominal floor and pushes the abomasum to the left and forward; when the fetus is expelled, the rumen descends to the abdominal floor trapping the abomasum; (c) the abomasal atony theory contends that atony is a prerequisite to displacement and that causes of atony include the stress of parturition, toxemia due to concurrent disorders such as metritis, mastitis or indigestion, and metabolic disorders especially milk fever and

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My purpose is to discuss these three theories and integrate known information into a series of suggestions to reduce occurrence of this disorder in dairy cows.

Recent reviews covering the subject of DA include those by Dirksen (11), Fox (16), Hull and Wass (25), Hartikka and Roine (21), Ide and Henry (27), Martin (31), Robertson (38), and Svendsen (43).

**Genetic and breed effects.** Pinsent et al. (36) reported that of 80 clinical cases in their practice, 30% were in the Channel Island breeds, but this group composed only 13% of cows in that region; therefore, a breed disposition or genetic effect was suggested. Subsequently, Ide and Henry (27), in addition to Blood and Henderson (4), made similar proposals. Conversely, Dirksen (11) with experience in 95 cases stated that there was no breed disposition toward displacement; support for this statement was given by Gould and Gould on 70 cases. In 1972, Martin (31) made an epidemiological study of LDA in Ontario, Canada, and on a limited number of Holstein cows with traceable sires, concluded that the chance of a cow having an LDA was 1.5 times greater if she were sired by a member of one group of bulls compared to a cow sired by another bull in that survey.

**New York survey.** To obtain data on DA occurrences and sire identification that would permit estimation of heritability and provide an indication of the incidence of this disorder, survey forms were mailed to the 4,680 Holstein herd owners in New York who are members of the DHI Record System (8). The front of the form was a letter explaining the rationale for the study including a request that all dairymen who were fortunate enough not to have had the problem to please return the blank form so that the incidence study would not be biased by lack of representative returns from both those with and without the disorder in their herds. On the back of the form were three columns to list the cows by DHI index number that had DA's during the 3 calendar yr included in the survey. A check-off box was provided for those who wished to receive results of the study. Of 4,680 forms sent out, 1,864 were returned — a response of 39.8%. Twenty-one herds were not on the computer tapes, which indicates they had not been enrolled in the system long enough to have completed lactations. Of the 1,843 herds with available data, 446 (24.2%) reported at least one DA during the 3 yr and 1,397 herds (75.8%) reported no occurrences during 1970, 1971, and 1972. Within the 446 affected herds, 944 cows had a DA. This represents an incidence of 1.16% among the affected herds of the total lactations (944/81,662 lactations during 3 yr). Expressed over the total lactations from all herds that reported in the survey, the incidence was .35% (944/267,844 total lactations). A search of the records for the affected cows revealed only 481 of the 944 reported. This difference reflects cows not part of the DHI system long enough to have completed records, misidentification (use of a barn number instead of an index number), and the same cow listed twice in separate years. If 481 cows affected is taken as the true occurrence, the incidence is approximately one-half that above. I believe the former number, 944, the more accurate of the two.

Incidence increased sharply during the 3 yr of the survey with 63 cows in 1970, 151 in 1971, and 267 in 1972. Some of this increase may be more apparent than real, a result of greater recall by respondents in recent years.

Production data in Table 1 show that non-DA cows in DA herds produced 334 kg more milk than cows in non-DA herds. But affected cows in the year of the DA produced 230 kg less milk than unaffected cows in the DA herds. For a more precise comparison, DA cows produced 346 kg less milk and 3.4 kg more milk fat than their herdmates in the year of the DA. And, in the lactation preceding the DA, affected cows produced 241 kg more milk and 8.6 kg more fat than their herdmates. Therefore, the cows affected by DA's were higher producers than their herdmates, and they were from higher producing herds than herds without DA’s. The affected cows were also older and heavier than the average of cows reported in this survey, a characteristic reported by other workers (31, 38).

A comparison of management factors (Table 2) between the DA affected herds and the unaffected herds revealed that DA herds were characterized by higher production, a larger number of cows, slightly heavier cows, more concentrate fed per cow, more succulent roughage fed per cow, less dry roughage fed per cow, and fewer days on pasture.

Of the 481 DA cows identified in the record system, 110 had no sire identification; the 371 remaining cows were daughters of 194 different sires. The six most widely represented sires had from 7 to 14 daughters each. These six sires were AI proven sires that were widely used and consequently have many
Table 1. Production, weight, and age of displaced abomasum (DA) cows and non-DA cows in New York survey (8).

<table>
<thead>
<tr>
<th></th>
<th>DA cows in DA herds</th>
<th>Non-DA cows in DA herds</th>
<th>Cows in non-DA herds</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Year of DA</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Milk production (305 day, ME), kg</td>
<td>6,784</td>
<td>7,014</td>
<td>6,680</td>
</tr>
<tr>
<td>Milk fat (305 day, ME), kg</td>
<td>258.8</td>
<td>252.9</td>
<td>239.2</td>
</tr>
<tr>
<td>Deviation from herd-mates, kg milk</td>
<td>-346</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>Deviation from herd-mates, kg fat</td>
<td>3.4</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td><strong>Lactation preceding the DA</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Deviation from herd-mates, kg milk</td>
<td>+242</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>Deviation from herd-mates, kg fat</td>
<td>+8.6</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>Body weight, kg</td>
<td>624.7</td>
<td>575.2</td>
<td>565.7</td>
</tr>
<tr>
<td>Age, mo</td>
<td>69</td>
<td>54</td>
<td>55</td>
</tr>
<tr>
<td>No. lactations</td>
<td>481</td>
<td>81,181</td>
<td>186,182</td>
</tr>
</tbody>
</table>

Table 2. A comparison of production management factors in DA and non-DA herds (8).

<table>
<thead>
<tr>
<th></th>
<th>DA herds</th>
<th>Non-DA herds</th>
<th>All Holstein NYDHI herds 1972</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Production</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Milk, kg</td>
<td>6,547b</td>
<td>6,246b</td>
<td>6,271</td>
</tr>
<tr>
<td>Milk fat, kg</td>
<td>236</td>
<td>224</td>
<td>228</td>
</tr>
<tr>
<td><strong>Herd</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Size, no. cows</td>
<td>63.2</td>
<td>49.6</td>
<td>61.3</td>
</tr>
<tr>
<td>Avg body wt, kg</td>
<td>572.0</td>
<td>563.0</td>
<td>572.0</td>
</tr>
<tr>
<td><strong>Nutrition</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Concentrate/cow per year, kg</td>
<td>2,441</td>
<td>2,267</td>
<td>2,375</td>
</tr>
<tr>
<td>Succulcent forage/cow per year, kg</td>
<td>5,919</td>
<td>5,174</td>
<td>6,073</td>
</tr>
<tr>
<td>Dry forage/cow per year, kg</td>
<td>1,436</td>
<td>1,688</td>
<td>1,510</td>
</tr>
<tr>
<td>Cow days on pasture</td>
<td>121</td>
<td>126</td>
<td>115</td>
</tr>
</tbody>
</table>

* Actual production.
* Based on herd averages reported in this survey for 3 yr, 1970, 1971, 1972.
why some cows encounter this disorder and others do not.

Abomasal atony. Dirksen (11) felt that mechanical factors were of minor importance in the development of DA. He considered the primary cause to be a hypotony or atony of the abomasum which was accompanied by some dilation and accumulation of gas in the fundic region. The question arises as to what causes atony of the abomasum to develop in some cows and not in others. Dirksen attributed abomasal atony to three causal groups acting singly or in concert: (1) nutrition, (2) metabolic and deficiency disease, (3) several organic or general diseases. Nutrition and metabolic disturbances were considered the principal causes which led Dirksen (11) to call DA a civilization disease. He felt that the most effective treatment would be one which induced a return of motility and tone to the abomasum.

Mather and Dedrick (32) consider abomasal atony a predisposing element to displacement. They attribute one cause of atony to histamine which reduces abomasal motility. Increased histamine can arise from tissue breakdown associated with diseases such as metritis and mastitis. Additional histamine can come from large amounts of high protein concentrates. Others (4, 36) support the contention that for displacement to occur the abomasum must first become atonic and that subsequent accumulation of gas and ingesta make spontaneous recovery unusual. Espersen (14) too noted that heavy beet feeding, common in Denmark, often led to toxic indigestion and atony of the forestomachs.

Goats with abomasal and duodenal cannulas were used by Singleton (41) to show that proteins, their breakdown products, and fats inserted into the duodenum reduced abomasal motility which confirmed that contents of the duodenum influenced gastric emptying and abomasal activity in ruminants. These effects provided a basis for the suggestion of Neal (34) that diets high in protein and fat are predisposing to displacement.

Over 10 yr ago a practicing clinician (48) observed that the highest incidence of DA occurs in herds where high grain is fed, especially in late pregnancy. Since then high grain near parturition has been implicated by a number of writers (9, 34, 36, 38, 42, 45, 47). Present et al. (36) emphasized the rapid change in diet which occurs as grain feeding is increased before and after parturition. Neal (34) observed high incidence in a herd fed limited forage and one in which dry cows were fed 5.4 kg/day of a high protein cake. Based on experience with over 200 cases, he concluded that dietary factors provided major predisposing elements, especially rations high in protein, fat, and low in bulk. Supporting the idea of limited forage involvement with DA is a report by Trimberger et al. (45) in which cows fed 5.4 kg of corn silage and 3.6 kg of alfalfa grass hay/day as forage had more DA's during a three lactation study than cows fed 16.3 kg of corn silage/day plus alfalfa-grass hay ad libitum. A summary of these DA's by treatment is shown in Table 3. Treatment group 3 received the limited forage, and groups 1 and 2, the combination of forages with hay ad libitum. Group 4 received corn silage ad libitum as the only forage and also encountered a high percentage of displacements. If corn silage is thought of as a combination of equal parts forage and grain, corn silage is a high concentrate diet.

Grain feeding prepartum has been associated specifically with DA. Robertson (38) in 1968 reported a comparison of management practices in 30 herds in Pennsylvania with a history of DA and closely adjacent herds that had no history of DA. The primary difference between the two groups was that affected herds were fed significantly more grain the month prepartum and throughout the winter feeding season. Grain was probably fed separately from forage in these herds.

High grain in complete feeds also has been associated with a high incidence of DA. Smith (42) observed 11 cases of DA in 46 cows fed a complete feed of 40% chopped alfalfa hay (3.2 cm hammermill screen) and 60% concentrate ad libitum beginning 4 wk prepartum. Production stratification did not reveal a direct relationship to DA. Using equal parts forage dry matter from alfalfa grass silage and corn silage in complete feeds, Purdue workers (9) assigned 40 Holstein cows 28 days prepartum to four complete feeds with: (a) 75:25, (b) 60:40, (c) 45:55, and (d) 30:70 forage to concentrate ratios expressed on a dry basis. During the period 3 to 25 days postpartum, Table 3. Effect of dietary regime on occurrence of DA (45).

<table>
<thead>
<tr>
<th>Ration</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of cows</td>
<td>15</td>
<td>10</td>
<td>15</td>
<td>10</td>
</tr>
<tr>
<td>DA's, this study</td>
<td>2</td>
<td>2</td>
<td>5</td>
<td>4</td>
</tr>
<tr>
<td>DA's, previous to this study</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>2</td>
</tr>
</tbody>
</table>
there were zero, two, four, and four cases of displacement in the four ration groups, respectively.

A recent Cornell study involved three forage groups (not fed as complete feeds) of 16 cows each fed either: (a) alfalfa-grass hay plus corn silage, (b) alfalfa-grass silage plus corn silage, or (c) corn silage ad libitum. Each forage group was divided equally with one subgroup lead fed and the other fed constant low grain until parturition. Lead feeding is defined as the practice of gradually increasing grain beginning 3 wk prepartum to 1 to 1.5% of body weight by the predicted parturition date. The association between treatment and DA is in Table 4. Although all seven displacements occurred in the corn silage forage group, there is little reflection of lead feeding. However, the high grain content of the corn silage may have masked this potential effect. The combination of high grain plus corn silage prepartum was also recently associated with 10 DA’s in 18 cows in one treatment group (7).

High grain feeding pre- and postpartum appears to predispose cows to DA. This seems true whether a constant ratio of grain to forage is used as in complete feeds or increasing grain as in lead feeding. Lead feeding depresses forage intake drastically at parturition, and complete feeds high in grain depress total dry matter intake prepartum (9).

Physiological studies by Svendsen (43, 44) provide some explanation for an association between high grain feeding near parturition and DA occurrence. He showed that high concentrate in the diet increased volatile fatty acids entering the abomasum, which decreased abomasal contraction. The same type of reduction in motility was achieved by injection of 300 ml of a fatty acid (VFA) mixture similar to that in grain fed cattle. It was also demonstrated that grain feeding increased the amount and changed the proportion of gases released from the abomasum into the reticulo-rumen.

By passing a catheter through a rumen fistula and through the omasal orifice, Svendsen (43) infused the abomasum with VFA and nitrogen and succeeded in producing a right displacement. He further demonstrated that high grain feeding (compared to hay feeding) produced a much higher concentration of VFA in the abomasal contents and that the flow of digesta to the abomasum was increased from 1 to 2 h after feeding, but the flow from the abomasum was reduced during this time. One may interpret this work to mean that a reduction in abomasal motility and an increase in abomasal gas production provide a strong predisposing condition for abomasal displacement in cows fed high concentrate diets near parturition. However, the highest grain intakes by dairy cows usually occur between 8 and 12 wk postpartum (23), subsequent to the highest incidence of displacement. Moreover, DA’s in steers fed all concentrate diets are rare, so the proximity to parturition and high grain feeding is an important one.

Ehrlein and Hill (12) working with goats did not completely support the work of Svendsen (43). Animals with wide-bore rumen fistula and duodenal or abomasal cannula were used. Abomasal injection of short chain VFA’s had no effect on the motility of the abomasum or forestomach, but similar infusions in the duodenum reduced motility of both abomasum and forestomachs.

Twisselman (46) used cows with rumen and abomasal fistulas to define further the role of VFA’s as the causative factors in displaced abomasums. Although rumen VFA concentration increased following feeding, abomasal VFA concentration remained relatively constant following feeding. Several differences in technique make the experiments of Svendsen (43), Ehrlein and Hill (12), and Twisselman (46) difficult to compare. Svendsen (43) used dry cows and sampled the abomasal contents near the pylorus; Twisselman (46) used lactating cows and sampled abomasal contents through a cannula in the greater curvature of the abomasum. More studies of this type under carefully standardized conditions should help to reveal causative factors for this disorder. Although the basic mechanism(s) are incom-

### Table 4. Effect of forage regime and prepartum grain feeding on DA (10).

<table>
<thead>
<tr>
<th>Forage</th>
<th>No. cows</th>
<th>Lactation number</th>
</tr>
</thead>
<tbody>
<tr>
<td>Corn silage, LF</td>
<td>8</td>
<td>1 2 3 (DA's)</td>
</tr>
<tr>
<td>Corn silage, NLF</td>
<td>8</td>
<td>1 1 1</td>
</tr>
<tr>
<td>Corn silage + hay-LF crop silage</td>
<td>8</td>
<td>0 0 0</td>
</tr>
<tr>
<td>Corn silage + hay-NLF crop silage</td>
<td>8</td>
<td>0 0 0</td>
</tr>
<tr>
<td>Corn silage + hay-LF</td>
<td>8</td>
<td>0 0 0</td>
</tr>
<tr>
<td>Corn silage + hay-NLF</td>
<td>8</td>
<td>0 0 0</td>
</tr>
</tbody>
</table>

*a Lead fed.

*b Not lead fed.
pletely understood, DA’s occur primarily in cows fed high grain near parturition. Fortunately, several experiments (13, 17) have shown no response in production to lead feeding when cows were in good condition at drying off and were fed well following parturition. Consequently, there seems little reason to continue this practice. Although it is not clear whether high grain feeding prepartum or postpartum is more responsible, we do have a choice in feeding high grain prepartum but probably no reasonable alternative postpartum.

Vitamin E and selenium. Because of the involvement of these two nutrients in muscle function, it has been postulated that a deficiency of either may be contributory to the occurrence of DA. Analyses of the feeds for selenium in the Purdue study (9) and in the recent Cornell corn silage study (10) did not reveal lower selenium in those diets characterized by occurrence of DA. Any involvement by these two nutrients needs further clarification.

DA related to concurrent disorders. Pinsent et al. (36) believe that for a clinical condition of DA to occur the abomasum must become atonic. In addition to diet, abomasal ulceration and vagal paralysis, postparturient diseases were considered responsible for atonicity of the abomasum. Atony is thought necessary because gas is not expelled from the atonic abomasum.

From a study of clinical findings on 86 cases of LDA, Robertson (37) noted that 15% had a history of illness immediately prior to the DA diagnosis and 21% had concurrent diseases. Metritis and parturient paresis were most frequently involved. The primary clinical finding in nearly all cases was ketonuria and partial but chronic anorexia. An Ohio report (24) showed one-half the cows with LDA had metritis. At the Michigan State Veterinary Clinic, one-third of the cows with DA also had metritis (18). In the Purdue study (9), 9 of 10 cows with LDA had metritis. Based on experience with over 200 cases, Neal (34) stated that although the initial diagnosis was one of ketosis in a high proportion of the cases, in many other cases there was a history of recurrent hypocalcemia, hypomagnesemia, or endometritis.

A mechanism of systemic disease effect on abomasal tone has been proposed. Histamine has reduced abomasal contraction, and Mather and Dedrick (32) note that high histamine can arise from tissue breakdown associated with systemic diseases such as metritis and mastitis.

Hypocalcemia can be involved in abomasal motility because calcium is essential for smooth muscle contraction. Moreover, several practitioners (1, 6, 47) reported favorable response in treating DA with calcium solutions. The possibility that cows which develop DA have lower blood calcium at parturition or prolonged low blood calcium was investigated by Hull and Wass (26). Serum calcium was determined on 90 cows on days 1, 3, and 7 postpartum. Some individuals demonstrated low serum calcium without clinical signs of milk fever. Of 8 cows that developed DA, 7 had abnormal calcium patterns and 6 had clinical milk fever. But, not nearly all cows that developed milk fever suffered DA. And, blood calcium at the time of displacement was often normal because the predisposing hypocalcemia had been corrected. The conclusion from this work is that hypocalcemia can be another predisposing factor to DA.

Other associations with DA – season and parturition. The appearance of DA has been associated frequently with parturition in the cow. Forty-seven of 73 cases occurred from 0 to 1 wk postpartum (36), 88 of 106 cases within 2 wk postpartum (32), over 80% within 30 days postpartum (47), 134 of 156 cases from 2 wk before to 2 wk after calving (39), and days from calving to diagnosis were 13.0 ± 11.4 (31). Martin (31) postulated that “parturition predisposes all cows to LDA, but only certain individuals possess other equally important factors which favor development of LDA”.

Several authors have noted a seasonal variation that does not coincide closely with the number of parturitions (27, 31, 32, 36, 38). A large majority of DA’s occur during winter and spring, with few during summer.

Other writers (31, 38) have noted the DA’s occur more often in higher producing cows and older cows than the population average.

Conclusions

There is a large array of predisposing elements to displacement of the abomasum. Among the ruminants these include dairy cows that are (a) older, (b) larger, and (c) higher producers than the population average, (d) in close proximity to parturition, (e) in the winter and spring months, (f) fed high grain (perhaps high protein grain), and (g) fed high corn silage. If dairy cows are unfortunate enough to experience one or more of the common maladies, metritis, mastitis, or milk fever near parturition, the probability is increased that a displacement will occur. Although indi-
individual differences in susceptibility to this disorder exist, genetic factors do not appear to exert a primary role in the cause of abomasal displacement. It is a simplistic concept to attribute this complex disorder to a simple, single causative agent. Rather, displacement of the abomasum occurs because of the coincidence of many predisposing factors acting in concert. Each additional factor increases the probability of the disorder occurring.

Prevention. Obviously, we want to keep the dairy cow long enough for her to become older and larger, especially if she is a high producer, and to have her calve regularly. However, where occurrence of DA is high, the amount of grain and corn silage should be reduced parma but other forage offered ad libitum; feeding regimes should be used which minimize milk fever. Clean calving quarters should be provided to minimize parturient infections, and teat dipping plus dry-off treatments should be used to reduce mastitis.

References

(11) Dirksen, G. 1961. Vorkommen, ursachen und entwicklung der linksseitigen labmagenver-
SYMPOSIUM


Combating Milk Fever

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Abstract

Parturient paresis is a metabolic disease which is associated with parturition and initiation of lactation. It is characterized by low total serum calcium and inorganic phosphorus. Failure of the calcium homeostatic mechanism at parturition is associated with: (1) advanced age of cattle—older cattle absorb less dietary calcium and may have less exchangeable bone calcium; (2) too great an intake of calcium, over 100 to 125 g/day; (3) reduced intake at parturition, greater in older cattle; (4) overconditioned cows which appear to go off-feed easily; and (5) increased hormones, estrogen, and glucocorticoids at parturition which may reduce serum calcium.

Attempts to prevent failure of this mechanism include: (1) feeding prepardal diets low in calcium; (2) adjustment of the dietary calcium-to-phosphorus ratio; (3) feeding acidic diets, mineral acids, or ammonium chloride prepardum; (4) short-term administration of 90 to 100 g of calcium chloride daily; (5) feeding massive doses of vitamin D prepardum; (6) prepardum administration of 25-hydroxycholecaliciferol, a metabolite of vitamin D$_3$. 

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2 Contribution from the College of Agricultural and Life Sciences, University of Wisconsin, Madison.