Hypocalcemia as a contributing causative factor in the development of abomasal displacement

by

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INTRODUCTION

Abomasal displacement in cattle was first reported in 1948 in the United States (43) and in 1950 in England (4). Since these early reports there have been reports of the disease in many countries. In some countries, usually those where feed is insufficient, the disease has not been reported (62). In the United States most abomasal displacements are to the left side, while in many of the cases seen in the Scandinavian countries the displacements are to the right side. Right sided displacements can progress to an abomasal torsion which is an acute syndrome. Also it should be noted that although there seems to be a herd incidence of left sided displacement many times, right sided displacement is usually an individual situation.

Many advances have been made in the diagnosis and treatment of the displaced abomasum. Early cases were found as a result of exploratory abdominal surgery for other reasons (other than a diagnosis of displaced abomasum) or on post mortem examination. Since that time a very common history has evolved. Paracentesis and examination of the pH of aspirated fluid have been used as an aid to diagnosis with some success. Today however, abomasal displacement is usually

diagnosed by the common history in conjunction with auscultation alone or auscultation and percussion.

Most articles written on abomasal displacement today are on the treatment of this condition. These range from spontaneous recovery to surgical intervention. Many surgical approaches have been described and all seem to be quite successful (80-90%). The choice of method of correction probably therefore would be an individual preference.

Even though there are many successful methods of correction the disease is becoming more prevalent. Part of the increasing recognition is due to the fact that there are now diagnostic methods for this syndrome. However, the rate of increasing prevalence of this syndrome would suggest that part of this increase must be a true increase.

The veterinary profession should find the cause of this condition and then try to work for a method of prevention rather than be satisfied with a good surgical correction.

Previous work as to the etiology of abomasal displacement is very limited and is based mainly on conjecture. The most commonly accepted is the theory of mechanical displacement of the rumen by the gravid uterus and subsequent trapping of the abomasum at the time of calving. The first mention of this theory goes back to Moore et al. (43) who presented it

mainly as a thought on possible cause. Since then this has been claimed to be the cause with little if any proof other than circumstantial evidence. Svendsen (62) has done some research as to the cause and has found limited evidence to incriminate high levels of volatile fatty acids in the abomasum as a result of high concentrate feeding.

The one thing common to nearly all cases of displaced abomasum is the history of recent parturition. Probably a large gravid uterus lifting a rumen plays some part in the development of displacement, however, there must be some atony of the abomasum if it is to remain displaced. It is interesting to note that although beef cattle also have gravid uteri lifting rumens the incidence of displacement in these animals is very low, perhaps even nonexistent. The argument of large calves is not necessarily valid in this case as some beef breeds particularly the Charolais have larger calves than dairy breeds yet the cows still do not become afflicted with abomasal displacement.

A clinical observation which was in part responsible for the initiation of this study is the fact that many displacements can be corrected by administering calcium preparations such as those designed for the treatment of milk fever. Calcium therapy, however, usually does not

result in permanent correction, but rather a temporary relief of the clinical signs. Many cows that have abomasal displacements also have metritis in conjunction with this displacement. Both uterus and abomasum have smooth muscle. Calcium is needed for normal smooth muscle tone. These facts coupled with the clinical observation that some of the cows with displacements also have a history of having been afflicted with milk fever led to this study.

The purpose of this investigation was to study the possibility that cows with abomasal displacement had a lower blood calcium or a more prolonged low blood calcium at parturition as compared with blood calcium levels of herd mates. This was done by taking blood samples at parturition and immediately post partum on two herds of Holstein cattle. Blood calcium levels were determined on these samples. Samples from the cattle later developing displacements were compared with their herd mates.

LITERATURE REVIEW

Abomasal displacement in cattle was reported for the first time in the United States in 1948 (43) and in 1950 in England (4). Since then the disease has been observed and reported with increasing frequency. Part of this increased incidence can be explained by the fact that the disease has now been described and a diagnostic history and procedures with which to diagnose it have been developed. However, it would seem that the increase is at least partially a true increase. Practitioners who have been diagnosing this disease for the last twenty years are seeing more of it today than they did twenty years ago. This must be a true increase as these people were able to diagnose the disease previously. This disease is a problem mainly in countries where agriculture is intensive and has seldom been described in countries without the most advanced agricultural techniques (62).

Displacement of the abomasum is usually seen in dairy cattle, especially high producing dairy cattle. This disease has been seen in heifers and bulls, (1, 29, 37) however, many cases in these animals have extenuating circumstances. For example the case seen by Albert and Ramey (1) was associated

with consumption of gasoline. Regardless of these extenuating circumstances with so few cases seen in heifers and bulls one would tend to think of these as special cases which one might consider exceptions. Therefore, it can be assumed that displacement of the abomasum as ordinarily observed is seen in adult dairy cattle. In adult dairy cattle this disease is almost always associated with parturition. Wood (68) claims to have seen many cases not associated with parturition. However, this could be questioned on the basis of the displacement being subclinical or the diagnosis having been missed earlier.

The author has seen two such cases which were 5-7 months after parturition, however, in both of these cases the history would indicate a subclinical problem. Both of these cows had a history of recurrent ketosis and lower production than was normally expected. Also both of these animals had healed ulcers in the wall of the abomasum and had greatly thickened abomasal walls. With only a few exceptions therefore displacement of the abomasum commonly occurs in association with parturition.

Usually this syndrome occurs either just before parturition or within 2-3 weeks after parturition. Although close association with parturition is constant no one has

investigated the reason why parturition and displacement of the abomasum are so closely related.

In an article published in 1954 Moore and his associates (43) stated that the etiology of the displaced abomasum was poorly understood. They thought the uterus pushed the rumen up and pushed the abomasum to the left. At parturition the uterus empties and the abomasum is trapped on the left. Begg and Whiteford (4) in 1956 expressed the same opinion as to etiology. This theory on etiology has been repeated in many more recent articles (25, 29, 42, 47). Marr and Jarrett (38) have stated that they felt abomasal ulcers were the cause. This could be refuted on the basis that abomasal ulcers are often a sequella to displacement as a result of impaired circulation. Nilsson (47) cites careless loading and unloading of the animals as a possible cause. In support of his theory he states that in bigger and better farms there is more trucking of animals and that this explains the high incidence on these farms. He also cites one specific case where a new steep chute was installed and the incidence increased sharply only to decrease when the chute was removed. Ide and Henry (29) subscribe to the theory of the uterus lifting the rumen, but feel diet, feeding habits, exercise, housing conditions, size of calf and heredity can all be contributing factors.

Rines (51) handles all the unproven theories very nicely when he states "The original theories of uterine migration; heavy concentrate feeding; unusual or violent activity, such as jumping, rolling or falling; and even abomasal ulcers with adhesions still fit into the etiological picture, but possibly only in a contributory role." Fox (18) in 1960 and Mather and Dedrick (40) in 1966 summed up the present state of thinking quite well by stating "etiology unknown".

In the literature there are several statements on etiology which are worthy of consideration. Mason (39) states that atony and dilation of the abomasum precede displacement. Dirksen (14) feels the primary cause is a hypotony or an atony of the abomasum with more or less severe dilation in connection with an accumulation of gas in the fundic portion. He states that dislocation does not occur suddenly, but rather gradually and he feels the atony can be due to three causative factors which may act alone or together. These three groups are: 1) nutrition, 2) stress-conditions and metabolic dificiencies and 3) several organic or general diseases.

Fincher (17) concurs with the nutritional theory in that he feels high grain and low roughage predispose to a displacement and that the stomach of a cow that develops a displacement must be greatly distended through the consumption of

large amounts of either fine roughage or fine grain. This cannot be entirely true, however, because beef cattle in a feedlot do not develop displacement of the abomasum. Woelffer (66) feels heavy grain feeding especially late in pregnancy may be a factor. High grain feeding increases blood volatile fatty acids and blood amino nitrogen (32). Kesler and Spahr (32) go on to state low fiber feeding tends to cause bloat and other digestive disturbances. He suggests at least 13-14% fiber in a diet to prevent problems. Svendsen (62) feels the high blood volatile fatty acids decrease abomasal motility and contribute to the atony or hypotony.

To fully evaluate how the atony can play a role in displacement a review of the anatomy of the abomasum in both its normal state and in its displaced state would be appropriate.

Nilsson (47) feels there may normally be some displacement of the abomasum before parturition. Jones (31) quotes Habel as saying "The fundus of the abomasum may be seen on the left under the free end of the reticulum". He states that the body of the abomasum lies near the midline, more on the left than on the right. Jones conducted a controlled abattoir survey, in which the cows were always put down in the same position, and Jones concluded that 30% of the abomasums are

normally to the left of the midline, 58% are on the right and 12% lie on the midline. This study was conducted on 50 cows in various stages of pregnancy and the position of the abomasum had no correlation with the state of the uterus. In such a study the position of the cow is suddenly changed from standing to prone. In this case there is the distinct possibility that the position of the abomasum can change. Therefore, this study could be open to question. However, the cows were always put down in the same manner (restrained so they fell on their left side) and there is the possibility that the abomasum could shift either way. This study does point out that finding the abomasum on the left is not necessarily abnormal.

The largest portion of the abomasum is not held in position, but is merely suspended by omental sheets (29). Anteriorly the abomasum is attached to the rumen, reticulum and omasum by folds of peritoneum. The posterior or pyloric portion has a rather firm attachment to the liver near the attachment of the bile duct. This attachment is by the lesser omentum. Thus this leaves the major portion of the greater curvature of the abomasum hanging suspended as a loose sack. Suspended in this manner the greater curvature has a great deal of freedom of movement. As already stated this greater

curvature generally lies posterior to the omasum and very close to the midline just to the right of the rumen in most cases.

Sack (57) has done a very good study on the anatomy of a cow with a displaced abomasum. To accomplish this study he embalmed a cow in the standing position. The displaced abomasum in addition to being dislocated also caused a dislocation of the omasum, reticulum, and duodenum. The abnormal position of these organs in turn caused a change in position of the rumen and liver. The greater omentum became folded between the abomasum and the rumen. The other significant finding of this study is that in a displacement the greater curvature of the abomasum is greatly distended.

The normal abomasum lies to the right of the midline. Nilsson (47) believes that in the case of the cow that later develops a displaced abomasum that the uterus lies more anterior and ventral than normal. He feels this lifts the rumen and reticulum creating a void into which the abomasum can slip. His reasoning again brings up the same old theory but again offers no proof. Moore and his associates (43) concur with this reasoning.

Robertson (53) analyzed the records at the University of Pennsylvania and found several interesting facts. The mean

age of cattle with displacements is higher than the population average. Pinsent and his associates (50) in their study involving 80 clinical cases have stated that all their cases were in high quality dairy cattle from 2 to 10 years of age. However, they concluded that there is no age incidence. The age incidence found by Robertson could be a true variation or it could be related to the fact that cows with displacements tend to be better cows and therefore might be kept in the herd longer than the average animal. However, it is a well known fact that displacement of the abomasum rarely occurs in first calf heifers. Robertson also found that 72% of his 202 displacements occurred during the stabling period. There is also a peak during the month of August. This is in agreement with others (45, 50) who feel the most common time for displacements to occur is from October to April. This seasonal variation cannot be fully explained by the seasonal incidence of calving.

Robertson (53) found there was no evidence of any breed predisposition. Neal (45) agrees with this finding, although he found that Jersey cows in his study tended to have a higher incidence. When he analyzed his figures, however, he found that this higher incidence in Jerseys was accounted for by one herd with an especially high incidence. When he

eliminated this one herd from his study he found that all breeds had equal prevalence. After eliminating this one herd these may not be valid conclusions. Possibly there is a breed incidence. Pinsent and his colleagues (50) feel there is a breed incidence. They point out that 30% of their cases are Channel Island breeds while only 12.7% of the cattle in England are Channel Island breeds. Thus there seems to be some difference of opinion as to whether there is a breed incidence or not.

Pinsent and his colleagues (50) state that since there is a breed incidence that this may possibly indicate a hereditary tendency. They simply presented this as a postulation and did not offer proof. Neal (45) concludes after examining breeding records that there is no hereditary tendency. There continue to be reports of displacements in related animals. These reports, however, are rare and it must be remembered that there is a herd predilection and these related animals are usually in the same herd.

Robertson (53) and others (29, 50) state that the incidence of abomasal displacement is higher in herds that have high production records. He draws some parallels between this and the feeding of rations with high grain content, and high ratio of concentrate to roughage. However, the best fed

animals within these high producing herds are not the most susceptible. He did find a high correlation between the incidence of abomasal displacement and lead feeding of grain in the dry period. Morrow (44) found a significantly higher incidence of displacements in cows on a high grain diet with restricted roughage than grain diets without restricted roughage. In Neal's (45) study, the farm with the highest incidence, limited roughage feeding in order to increase concentrate consumption. He found no common feed among concentrates fed in the various herds.

How this high concentrate and low roughage contribute to the abomasal displacement is poorly understood. Pinsent et al. (50) point out that the products of protein and fat digestion inhibit gastric motility. Svendsen (62) based his entire research on this fact and reproduced right sided displacement by infusing fatty acids directly into the abomasum. Svendsen also pointed out that lowering the pH of the stomach contents decreases abomasal motility as does histamine. Mather and Dedrick (40) state that histamines will inhibit or stop abomasal contractions and that these histamines can result from systemic diseases such as metritis or mastitis or could come from the consumption of high protein concentrate feeds. Neal (45) feels diet is very important in abomasal motility.

Neal (45) believes that the reason high concentrate and low roughage feeding causes displacement is because the decreased volume of the rumen allows for more mobility of the abdominal viscera. It should be pointed out that many beef cattle are maintained entirely on concentrate feeds and rarely if ever exhibit a displaced abomasum.

In view of these facts it would seem that although high concentrate feeding definitely seems to play a role in abomasal displacement, parturition must also play a role. The cause may well be multifactorial with the actual physical lifting of the rumen by the pregnant uterus being only the triggering mechanism.

Ide and Henry (29) believe an abomasum with normal muscle tone will not slip into the space between the rumen and the abdominal floor and that a certain amount of atony therefore must preclude displacement. In reference to initiating circumstances Neal (45) states that for a displacement of the abomasum to occur the abomasum must be atonic to a significant degree and that a route must be available for the abomasum to move to the left. He further states that abomasal tone is affected by: 1) diet, 2) metabolic disease, 3) abomasal ulceration, and 4) systemic disease. Rines (51) states that atony is an essential factor because in an atonic abomasum

the normally formed gas is not expelled. Svendsen (62) is of the opinion that this gas is produced when the bicarbonate of the rumen contents comes in contact with the acid media of the abomasum and that by normal abomasal motility this gas is forced back into the rumen and is eructated. Pinsent et al. (50) reports that the anatomical changes of parturition are insignificant because the abomasum is a powerful contractile organ which should be able to escape from under the rumen. He continues to state that the abomasum must therefore be atonic for a displacement to occur but does not postulate on the cause of the atony.

Rines (51) presents a list of factors which may decrease abomasal tone: "Other disease conditions which authors have considered as possible factors in decreasing abomasal tone are hypocalcemia, ketosis, metritis, mastitis, abomasal ulceration and vagal paralysis." Robertson (52) found in his study that the most frequent previous illnesses encountered in histories of cows with displaced abomasums were metritis and post-parturient hypocalcemia. Neal (45) states that many of his cases had a history of recurrent hypolcalcemia, hypomagnesemia or endometritis. Pinsent et al. (50) had similar findings in their cases. Moore et al. (42) found that 53 of their 55 cases were associated with parturition and the other two cases

occurred immediately after treating the animal for milk fever. Many have seen this association of metritis and abomasal displacement and there is general consensus (19, 21) that these two conditions are associated in about one out of three cases of displacement. Considering this it should be noted that the uterus has little involution during milk fever (51). Rines (51) points out that parturient paresis will relax the abomasum and Braun (12) gives 500 ml. of calcium borogluconate to increase the tone of the abomasum when repositioning it by rolling the cow. He feels this increases the tone and helps improve the rate of recovery.

The only blood calcium levels on cows with displacements which have been reported in the literature were reported by Robertson (54) on cows with clinical cases of displacement. In this study he found that 35% of his clinical cases had a hypocalcemia. There have been no reported studies of blood calcium levels taken immediately before the occurrence of a displacement.

A review of calcium levels at parturition may be helpful in associating the incidence of abomasal displacement with low calcium levels at parturition. There is a decrease in serum calcium in all cows at calving time and this decrease is significantly greater in those cows which develop milk

fever (6). There seem to be gradations between the seemingly normal cow and the cow that is down and comatose. The onset of paralysis is generally associated with calcium levels of 5 mg. % or less (41), although as with all biological systems there is some variation. In an experiment Mayer et al. (41) found that prolonged hypocalcemia (above 5 mg. %) did not produce paresis and concluded, therefore, that the degree was more important than the duration.

In a study on normal and mastectomized cows Niedermeier et al. (46) showed that of the factors which cause depression of total serum calcium levels, the onset of profuse lactation is probably the most important factor. At parturition an increased loss of calcium in the urine or feces does not occur (7). Payne (49) states that calcium mobilization rates and the immediately available calcium reserves were not significantly affected by lactation, but were lower in advanced pregnancy and were insufficient to compensate for the expected loss of calcium in milk at the onset of lactation.

To quote Kuribayashi (35), "In smooth muscle the excitation of the cell membrane is greatly influenced by calcium ions and there is a possibility that the action potential is generated by the influx of calcium ions. The mode of the interaction between calcium ions and the smooth

muscle myofilament is still unknown." There have been many hypotheses on the action of calcium on the contraction of smooth muscle. Kuribayashi (35) proposes the hypothesis that in contraction an actinomycin-ATP-calcium bond may form. Van Breemen (64) believes that the calcium ions activate the actinomysin during excitation with probably both intracellular and extracellular calcium contributing although it is probably mainly intracellular. Goodford (22) proposes that it is possible that the rhythm of contraction and relaxation in smooth muscle may be associated with a tidal movement of calcium from sites at the cell membrane to other sites inside the cells. Such a flow of ions could be associated with a flow of electrical charges. Robertson (56) believes that although calcium ions are essential for smooth muscle contraction, ions may be needed only intracellularly.

Bennett (5) has shown in the smooth muscle cells of the guinea pig vas deferens that increasing the calcium concentration will increase the amplitude and rate of rise of active response. He postulates that calcium probably exerts its effect on the membrane potential by altering the sodium permeability and that smooth muscle membranes are depolarized in low calcium solutions. Bozler (9) supports this postulate in his work with cardiac and smooth muscle. He found that the

tension produced by cardiac and smooth muscle in a solution of ATP was proportional to the amount of calcium in the solution. Takeuchi (63) supports this in his work proving that increasing the calcium concentration reduces the sodium conductance of the end plate membrane produced by acetylcholine with little or no effect on potassium conductance. Durbin and Jenkinson (15) believe the magnitude of response of smooth muscle to acetylcholine is dependent on extracellular calcium content.

Brading et al. (10) in their work with guinea pig Taenia coli found that excessive calcium caused hyperpolarization and increased membrane conductance. Excessive calcium reduces the membrane resistance. He also concluded that sodium influences muscle contraction by competing with calcium in controlling membrane potential, but that calcium is the main ion involved in the spike mechanism in Taenia.

Clegg and his associates (13) found that a high calcium concentration increases the spontaneous activity of certain smooth muscles in vitro whereas a high magnesium concentration decreases it. However, he is quick to add that the effect of these ions on the response to stimulants may vary. Harkness and his associates (26) agree that the tonus of smooth muscles is greater in higher concentrations of calcium. Kobayashi (33) found this to be true and also found that excesses of calcium

in the solutions caused a marked increase in amplitude when stimulated. He feels this is due to the calcium ions which carry some of the current responsible for the rising phase of the action potential.

It is generally accepted that spike height is related functionally to the influx of the cation which carries the positive charge inward (61). However, there is evidence to indicate that sodium, potassium and calcium ions may compete with one another for the negatively charged sites in smooth muscle.

There seems to be no definite answer to the role played by calcium in the contraction of smooth muscle. Our present knowledge of the role of calcium is summed up quite well by Kuribayashi (34). He states that calcium ions are necessary for the genreation of action potential and also for the contraction of smooth muscle.

In addition to this work on the action of calcium in the muscle it should be remembered that calcium is necessary for proper nerve irritability (30) and that in smooth muscle calcium enhances the transmitter output during stimulation (36).

Since calcium in most of the above experiments was shown to cause its action over a wide range of concentrations we should consider that the lowered calcium levels at

parturition may be of significance in muscle function even though they may not be of low enough concentration to cause parturient paresis.

MATERIALS AND METHODS

This study covered the time from 1 October 1969 to 1 June 1970. Two herds of Holstein cattle were used for this study. Both herds were commercial herds of above average production. Venous blood samples were obtained from puncture of the jugular vein with a 20 gauge 1½ inch needle. The blood samples were collected in 10 ml. silicone coated vacuum tubes.* The blood was allowed to clot and was brought to the laboratory where it was centrifuged. The serum was pipetted off and was frozen until a sufficient number of samples were collected for analysis.

Experimental Animals

Herd A is a state owned herd of about 85 milking cows. These cows are housed in a stanchion barn and are milked in a milking parlor.

This herd was picked for the study because during the winter prior to the study (1 October 1968 - 1 May 1969) this herd had 5 cases of abomasal displacement out of 43 cows which calved during that time. The ration for the animals in this herd was 20-25 lbs. of alfalfa hay, 22-25 lbs. of corn silage

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*B-D Vacutainer (red stopper) Becton-Dickinson and Company, Rutherford, New Jersey.

and approximately 25 lbs. of concentrate. The concentrate consisted of 1400 lbs. of shelled corn, 400 lbs. of oats and 200 lbs. of protein supplement. In addition these cows had access to a mineral-mix free choice. This mineral-mix was 52% trace mineralized salt, 40% dicalcium phosphate and 8% monosodium phosphate. The 85 cows in the herd consumed approximately 50 lbs. per week of this mineral mix.

Samples were taken on these cows on day three and day seven postpartum. If any of the cows had clinical hypocalcemia an additional sample was taken immediately prior to treatment for the hypocalcemia. Also if any of the cows developed an abomasal displacement a sample was taken at the time the displacement was diagnosed. During the period of the study herd A had 17 cows calve. One of these cows died before day three when the first sample would have been taken.

Herd B is a privately owned herd of 32 milking cows. These cows are housed in a loose housing arrangement and are milked in a milking parlor.

This herd was chosen for the study because of its excellent management and the cooperation of the owner. In the previous winter this herd had one case of abomasal displacement. The animals in herd B are fed 27-30 lbs. of good quality alfalfa hay and 20-25 lbs. of concentrate depending

on production. The concentrate consists of 1600 lbs. of shelled corn, 300 lbs. of 30% protein premix, 80 lbs. of molasses and 20 lbs. of dairy mineral premix. They also receive free choice trace mineralized salt.

Samples were taken on the cows in herd B between 8 and 12 hours after calving. Samples were also taken on day 3 and day 7 postpartum. If any of these cows had clinical hypocalcemia an additional sample was taken immediately prior to treatment for the hypocalcemia. During the period of the study herd B had 23 cows calve.

Analysis of Samples

The serum samples were analyzed for calcium content with an atomic absorption spectrophotometer.* The flame utilized in this process was fed by acetylene and compressed air. The lamp used in the spectrophotometer was a hollow cathode lamp sprayed with calcium oxide.

To prepare the samples for processing 0.6 ml. of serum was added to a small amount of distilled water. The amount of distilled water was of no importance as more distilled water was added later to bring the sample up to volume (25 ml.). To

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*Techtron, Manufactured by Techtron Pty. Ltd., Melbourne, Aust.

the 0.6 ml. of serum was added 2.5 ml. of lanthanum chloride (10% solution) stock solution. Distilled water was added to bring the total volume up to 25 ml. The samples were then mixed by inverting several times.

The serum was added to a small amount of water (several ml.) to prevent the lanthanum chloride from precipitating the serum as it seemed to do if the two were added directly to each other. The lanthanum chloride was added as it competes with phosphates and sulfates to free all available calcium.

When analyzing the samples in the spectrophotometer they were compared to a standard made up of 1.5019 gm. of calcium carbonate in 250 ml. of distilled water. This 1.5019 gm. of calcium carbonate yielded 0.613 gm. of calcium. This gave a standard stock which contained 2.405 mg. of calcium per milliliter. To prepare the working stock, 0.1 ml. of the above standard stock was added to 10 ml. of lanthanum chloride solution and brought up to 100 ml. with distilled water. Thus both the sample and the standard contained 1.0% lanthanum chloride. The lanthanum chloride used in preparing both the samples and the standard was a 10% solution of lanthanum chloride.

Additional Data

In addition to the above data collected by the author, data was also available on 45 Jersey and Aryshire cows on a milk fever experiment. This experiment was designed to determine if ration (specifically acid-base balance of the ration) had any bearing on incidence of milk fever. Of these 45 cows in the above experiment 3 were affected with abomasal displacements after calving (these three cattle will be mentioned later in the discussion). This work was done by Larry Schroeder of the Animal Science Department at Iowa State University. He was willing to share the calcium levels on the cases with abomasal displacement.

In addition to the documented cattle with calcium levels determined from the two herds worked with during the study, each of these herds has had one abomasal displacement since the end of the sampling period.

RESULTS

For the purpose of this study any cow with a calcium level of 6.5 mg. % or below is considered to be a milk fever candidate, and definitely to be lower than normal. Most cows have lowered serum calcium at calving, but do not go as low as 6.5 mg. %. There is also biological variation as to the level at which an animal will develop clinical milk fever. As can be seen from tables #1 and 2 one cow in this study which had a serum calcium level of 6.22 mg. % developed clinical milk fever, while another cow with a level of 5.8 mg. % showed no clinical evidence of milk fever. Also after calving, calcium mobilization returns to normal and normal blood calcium levels are usually attained within 4-5 days after calving. Normal calcium levels in the bovine are approximately 10 mg. %. Therefore, for the purpose of this study any cow having a serum calcium level of 8.5 mg. % or below at the end of one week was considered to be abnormal.

With these guidelines it is observed that herd A had two cows with abnormal calcium levels. In addition they had one cow which died of milk fever before any samples were taken. One of these cows developed clinical milk fever (cow A 2), but did not develop an abomasal displacement. Another cow (cow A

10) did not have calcium level sufficiently low to develop clinical milk fever, but did have a prolonged lower than normal calcium level. This cow did develop an abomasal displacement 6 days after calving. A second cow in herd A did develop an abomasal displacement, (cow A 5). However, her calcium levels were always within the normal range on the days this cow was sampled.

Herd B had 2 cows with abnormal calcium patterns at calving. One of these cows showed no clinical evidence of milk fever, but did develop mastitis 3 days after calving. The other cow (cow B 24) did not have clinical milk fever, although her calcium levels were low (5.8 mg. %) and stayed low for an abnormal length of time. This cow developed an abomasal displacement 5 days after calving. Another cow in herd B had milk fever before calving. Unfortunately no blood sample was taken by the attending veterinarian before treating this cow, therefore we do not know the calcium level at that time. When this cow calved her calcium level was again in the normal range. Even though this cow had milk fever she did not develop an abomasal displacement.

Since the conclusion of sampling, each herd has had one abomasal displacement. In both cases this displacement was preceded by clinical milk fever. The exact calcium levels on

these two cows are not known, however, clinical milk fever is evidence of hypocalcemia.

Thus out of 45 parturitions in the 2 herds studied 5 cows later developed abomasal displacements. Of these 5 cows 4 had abnormal calcium levels. Three of these four had calcium levels sufficiently low to be considered milk fever candidates, although one did not develop the clinical syndrome. One cow (A 10) did not have a lowered calcium level. It is interesting to note that at least two of these cows had prolonged lower than normal calcium levels. Both were 8.5 mg. % or below one week after calving.

Of the 45 cows on the milk fever experiment three later developed abomasal displacement. Please refer to table #3. Cow number 1 had milk fever on the day of calving with a calcium level of 6.7 mg. % and had a prolonged low calcium level (still only 8.1 mg. % at the end of one week). Cow number 2 was treated for milk fever twice (once 9 hours before calving with a calcium level below 5 mg. % and again 6 hours after calving with a calcium level of 4.0 mg. %). This cow's calcium level did return to normal within 5 days of calving. Cow number 3 calved on 13 March and had a lower than normal calcium level for 6 days, but did not develop milk fever until 6 days after calving at which time her calcium level was 5.8

mg. %. The serum calcium of this cow was still abnormally low one week after calving.

Of the 45 cows in the milk fever experiment a total of 18 cows developed milk fever. This number included the 3 with abomasal displacement.

DISCUSSION

Abomasal displacement has been recognized for the last twenty years, but as yet the cause of this syndrome is unknown. The previously held theory of abomasal displacement being caused by a pregnant uterus lifting the rumen is without proof. Once the cause of this syndrome is elucidated a method of prevention may be possible. Prevention would be preferable to surgical correction as there would be much less loss of milk production and it should be more economical.

Cows which have clinical abomasal displacement often have a history of previous hypocalcemia. The incidence of hypocalcemia in cows with abomasal displacements seem to be greater than that in the cow population. Calcium plays a role in smooth muscle tone and is essential for its contraction. Both the uterus and the abomasum have smooth muscle in their walls and therefore would be affected by hypocalcemia. The high correlation of abomasal displacement and concurrent metritis has often been observed, but has not been explained.

This study was designed to try to correlate low blood calcium levels or prolonged lower than normal blood calcium levels with later occurrence of abomasal displacement. If a cow with an abomasal displacement is given 500 ml. of any of

the calcium preparations used in treating milk fever the tone of the abomasum often increases sufficiently to make the clinical signs of the abomasal displacement disappear. Whether the displacement actually corrects itself or whether the tone is only increased sufficiently to cause a partial correction and the disappearance of the gas in the abomasum is not known. It is known that many abomasal displacements so treated will recur within 12-24 hours.

Studying blood calcium levels on clinical cases of abomasal displacement on admission to the Iowa State University Veterinary Clinic proved unrewarding. It was concluded that if there was a relationship between calcium levels and abomasal displacements it must be a predisposing relationship rather than a concomitant clinical relationship.

Five cases of abomasal displacement were found in this study of two herds. Blood calcium values were obtained from 3 additional cases in a third herd. Seven of these eight had abnormal calcium patterns. Of the seven with abnormal calcium patterns six had calcium levels sufficiently low to cause milk fever and at least four of these seven had prolonged lower than normal calcium levels. Of the 90 cows in the three herds 22 developed milk fever. Abomasal displacement did not develop in all of the cows with hypocalcemia.

However, six out of the eight cows which had abomasal displacements had a previous hypocalcemia.

A cow may be hypocalcemic and still not show clinical evidence of milk fever. Cow B 24 is an example of this. This cow's blood calcium level dropped to 5.8 mg. %, yet this cow at no time showed signs of clinical milk fever. There are undoubtedly many cows similar to cow B 24 which get extremely low levels of blood calcium and show no outward signs. Considering this it is possible that most if not all cows which have an abomasal displacement have had a previous hypocalcemia.

Hypocalcemia decreases the motility of the abomasum and allows the naturally produced gas to accumulate in the abomasum. As this gas is produced the abomasum becomes more buoyant than the surrounding intestines and floats to the top. At this point the abomasum is stretched to many times its normal size and is far from its normal position. When calcium levels again normalize this greatly distended organ is incapable of expelling the large accumulation of gas and returning to its original size and position. The clinical entity of an abomasal displacement is now diagnosed. However, at this time if blood calcium levels are measured they will often be found normal as the predisposing factor (hypocalcemia)

has been corrected.

Why certain herds have a higher incidence of abomasal displacement could be explained by the fact that certain herds tend to have more problem with hypocalcemia than other herds. These are the herds in which a high incidence of milk fever is reported. However, cows can be hypocalcemic without having clinical milk fever. There are undoubtedly herds which have a high incidence of hypocalcemia, although this may be unknown if the herd does not exhibit the outward sign of milk fever.

Neal (45) found a breed incidence of abomasal displacement. However, this breed incidence was due to one herd with an exceptionally high incidence. His study may point up two things. First the breed incidence (Jersey) and secondly the herd incidence. Pinsent and his colleagues (50) also found a higher incidence in the Channel Island breeds. Channel Island breeds tend to have a higher incidence of milk fever. The two syndromes certainly could be produced by the same mechanism, possibly heredity.

Certain herds of cattle have higher incidence of abomasal displacement than do other herds. This can be seen in this study. Herd A freshened 17 cows and had 2 abomasal displacements, while herd B freshened 26 cows and had one

abomasal displacement. Herd A had 5 cases of abomasal displacement in the preceding year, while herd B had only one. This observation has also been made by other authors in other herds.

With this herd incidence it would seem that management or feeding practices would play a role in abomasal displacement. Possibly heredity could play a role as various herds tend to have certain cow lines or cow families. Heredity could therefore explain the higher incidence in some herds than in other herds.

An interesting side light to this study is the fact that cow B 24 has had a clinical abomasal displacement for the last three years. Each time this has corrected on a truck ride. Could this hint at the hereditary tendency of this disease? At the present time this cow has no milking daughters, however, when her first daughter freshens it will be interesting to follow her disease problems in the first few lactations.

The problems of dairy cows and hypocalcemia have just begun to be elucidated and from the evidence we have found in this study it would seem very likely that one of the ramifications of hypocalcemia would be the predisposition to abomasal displacements.

SUMMARY AND CONCLUSIONS

Blood calcium levels on 90 cows in 3 herds were randomly sampled during the first week postpartum. Blood calcium levels on the eight cows later developing abomasal displacement were compared to the calcium levels of their herd mates. Seven of the eight cows with abomasal displacement had abnormal blood calcium patterns preceding the actual displacement.

Low calcium levels do not necessarily mean a cow will later develop an abomasal displacement as is evidenced by the 18 cows with clinical hypocalcemia (milk fever) that did not later develop abomasal displacement.

Blood calcium levels vary greatly the first five days after parturition and are especially erratic the first 24 hours. Studies to further elucidate the role of blood calcium levels in the development of abomasal displacement should include more frequent collections of calcium samples.

Although the numbers in this study are not sufficient to positively say that abomasal displacement is predisposed to by low calcium levels at calving, the evidence indicates that low calcium levels in the blood may predispose to later abomasal displacement. In this study no evidence was found to indicate that blood calcium levels are not related to the

occurrence of abomasal displacement.

Abomasal displacement may well have a multifactorial etiology. From the data obtained in this study hypocalcemia would certainly seem to be an important contributing factor.

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APPENDIX

Table 1. Blood calcium levels on cows from herd A samples taken on day 3 and day 7 postpartum

Cows #	Calcium Levels (mg. %) Sample 1	Sample 2	Comments
A1	9.7	9.9	
A2	8.4	8.9	had milk fever at calving, calcium level at that time 6.22
A3	8.4	8.6	
A4	9.5	9.6	
A5	10.7	10.7	displaced abomasum 10 days postpartum
A6	---	---	had milk fever at calving and died
A7	10.6	---	
A8	10.2	10.7	
A9	7.2	11.1	Septic Mastitis
A10	8.4	8.3	displaced abomasum 6 days postpartum
A11	8.9	---	
A12	8.8	9.4	
A13	9.4	9.9	
A14	8.6	9.0	
A15	8.2	8.9	Mastitis
A16	8.6	9.5	
A17	7.0	10.8	

Table 2. Blood calcium levels on cows from herd B samples taken on day 1, day 3 and day 7 postpartum

Cows #	Calcium Levels (mg. %)			Comments
	Sample 1	Sample 2	Sample 3	
B1	---	---	10.7	
B2	---	9.8	10.9	
B3	8.0	8.2	9.9	
B4	7.3	8.9	11.2	treated for milk fever 3 hours before calving
B5	9.3	9.6	11.4	twins
B6	8.5	11.4	11.1	metritis 10 days post-partum
B7	11.2	9.0	10.6	
B8	11.3	11.1	9.9	
B9	8.3	9.6	9.5	
B10	9.5	9.5	9.9	
B11	9.8	9.7	9.6	
B12	9.5	9.6	---	
B13	9.2	10.2	9.9	
B14	10.3	9.2	10.8	
B15	9.3	8.8	9.3	primary ketosis, responded to treatment
B16	9.4	9.7	9.2	
B17	6.3	8.6	9.1	
B18	8.1	9.4	10.0	
B19	9.7	9.8	---	shipped to another farm no third sample
B20	7.8	8.2	9.4	
B21	8.6	9.8	10.6	
B22	7.9	8.6	9.1	
B23	8.0	9.3	9.3	
B24	5.8	8.1	8.5	displaced abomasum 5 days postpartum
B25	8.1	8.2	---	
B26	8.2	---	9.8	

Table 3. Blood calcium levels on cases of abomasal displacement from dairy science herd

Cows #	Calcium Levels (mg. %)			Comments
	Sample 1	Sample 2	Sample 3	
1	6.7	8.9	8.1	treated for milk fever at calving. displaced abomasum 10 days post-partum
2	4.0	5.8	10.1	treated for milk fever 7 hours before calving and again 8 hours after calving. displaced abomasum 18 days post-partum
3	6.2	8.8	8.5	treated for milk fever 6 days postpartum (calcium level of 5.8 mg. %). displaced abomasum 17 days postpartum