FACTORS TO CONCENTRATE ON TO PREVENT PERIPARTURIENT DISEASE IN THE DAIRY COW

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Most of the veterinary care for sick cows is needed within 3 weeks of calving. Cows that develop one disease around the time of calving tend to develop other problems as well. If we can prevent a disease like milk fever (**MF**) we automatically reduce the risk of ketosis and mastitis in that herd as well.

I think that 90% of sick cow problems can be prevented, if we can successfully prepare the cow for the stress of calving and the onset of lactation by focusing on these three areas.

PREVENTION

I. Prepare the rumen so that high energy feeds can be fed early in lactation to meet the energy needs of the cow.

♦ Stimulate the growth of *good* bacterial species in the rumen.
♦ Stimulate growth of the rumen wall so absorption of nutrients is maximized.

How do we do this?

- Introduce grain into the ration of the cow for at least 3 weeks before due date. Heifers especially may need to be on this diet for 5 weeks. Protein content of the transition ration should be 16% CP.

  a. In total mixed ration herds, feed a ration that has from .71 - .73 Mcal NE_L / lb feed for the last three weeks of pregnancy, last 5 weeks for heifers.

  b. In herds fed hay and a concentrate mix separately, introduce grain 4 weeks before calving and increase slowly over a period of two weeks so that during the last 2 weeks before calving the cows are eating .75 - 1% of their body weight as concentrate (8-12 lbs/ day). If corn silage comprises a majority of the forage, this number can be reduced. FEED HAY BEFORE GRAIN!

- Maximize feed intake by drying cows off at body condition scores of 3.5. Above 3.75 is too fat. Depressed feed intake at calving leads to fatty liver and ketosis.

Payoff - less ketosis, fewer displaced abomasums, less rumen acidosis and less lameness due to laminitis in early lactation.

II. Prevent major decrease in blood clcium concentration at calving.

Because a tremendous amount of calcium is being put into colostrum and milk, the cow's blood can become deficient in calcium. Severe cases result in MF. Less severe cases result in feed intake depression and poor muscle tone which in turn cause retained placenta, displaced abomasum, and environmental mastitis (especially because the teat end won't close properly after milking).

How do we do this?

- Control cation-anion balance - MF is usually caused by the presence of high potassium (and in some cases sodium in heavily irrigated parts of N. America) cations in the diet. To some extent potassium can be counteracted by adding anionic salts to the diet, such as calcium chloride, ammonium chloride, or magnesium sulfate.

- Provide adequate magnesium - a lack of magnesium will prevent the hormones that defend against a drop in blood calcium from working properly. I recommend dietary magnesium levels

that are much higher than current NRC recommendations.

A good mineral profile for a transition cow (last 3-4 weeks of gestation) diet:

-calcium	1-1.2%
-phosphorus	0.4 - 0.5 %
-magnesium	0.4 %
-sodium	as close to 0.1% as possible
-potassium	as close to 0.7% as possible (This is a problem - most diets will be workable if you can get down to 1.5-1.8% potassium)
-sulfur	0.3- 0.4%
-chloride	high enough to bring urine pH between 6 and 6.8 for Holsteins and 5.8 and 6.5 for Jerseys.

My current philosophy is to formulate the ration using forages with the lowest potassium content that I can find, that are still reasonably well digestible. Corn silage is excellent. Beet pulp without molasses, some distillers grains or brewers grains, and corn gluten feed can often be used as well in the diet. First cutting of hays or alfalfa are generally higher in potassium than later cuttings grown under dry conditions. DO NOT TRUST POTASSIUM VALUES DETERMINED BY NEAR INFRARED ANALYSIS.

Next I add magnesium sulfate or magnesium chloride to the diet to bring magnesium content to 0.4%. Then, if needed, I add dicalcium phosphate to bring phosphorus to .45% and calcium chloride to bring chloride to 0.55%. Add calcium carbonate to bring calcium to 1%. In some cases a small amount of calcium propionate (0.25 lbs/day) can also be used to help increase dietary calcium and at the same time supply propionate, which the cow will convert to glucose (problem = cost).

This is where I start. If urine pH is not low enough I will add more calcium chloride to the ration. Add as little as possible to get the job done - too much risks knocking the cows off feed as anionic salts are generally unpalatable.

FUTURE - Hydrochloric acid may be available as a cheap and more palatable source of anions to prevent MF. I would then use it in place of calcium chloride. Would likely add some calcium carbonate to diets to get to 1% calcium, though some calcium could come from calcium propionate .

- Oral calcium supplements (Table 1) the day of calving

  a. Boost blood calcium for 6-10 hrs at time the cow needs them most.

  b. Drenches are more effective than gels or pastes but have greater chance of causing aspiration pneumonia if they go into windpipe instead of stomach when administered incorrectly.

Payoff – less MF, maintain DMI, less retained placenta, less displaced abomassum, less environmental mastitis.

III. Maintain a strong immune system

At calving white blood cells of all cows show a decreased ability to fight off infections, which increases the susceptibility to mastitis and uterine infections. In part the immune suppression is thought

Table 1: Comparison of oral calcium supplements

Calcium chloride based supplements	Calcium propionate based
Advantages	Advantages
-cheaper	-not as irritating
-less volume to give	-rapidly absorbed
-rapidly absorbed	-supplies energy and calcium
Disadvantages	Disadvantages
- caustic	- requires more volume
	- slightly more expensive

to be due to changes in hormones at calving. However better nutrition can strengthen the immune system at this time.

How do we do this?

- Prevent MF

  a. MF causes tremendous release of cortisol which inactivates the immune cells

- Feed adequate selenium

  a. 0.3 ppm is legal limit in USA. In some situations this is not enough.

  b. Injectable selenium may be an option.

- Feed vitamin E to animals without access to pasture

  a. Recent work suggesting that adequate vitamin E requires 2000 IU/day for the 2 weeks before and after calving. This is much higher than NRC suggests. Expensive, but worth it, if it prevents just one case of mastitis / 100 cows.

  b. Injectable vitamin E is an option also. Give 5 g intramuscularly 30 days before calving and again within a week of calving. Occasional abscesses occur at injection site.

- Prevent energy and protein deficiency (See I above)

- Supply trace minerals at 20 - 50% above NRC recommendations to account for decline in dry matter intake (**DMI**) that accompanies calving

  a. Copper and zinc deficiency seem to be the problems I see most in Midwest, often caused by too much iron in the ration and the water.

Payoff - Less mastitis, retained placenta (enhancement of neutrophil attack on fetal tissues) and uterine infection.

For more details on the physiology of periparturient disease readers are referred to Goff and Horst (1997).

As can be seen from the above, hypocalcemia can be a major factor setting cows up for development of many other secondary diseases. The following section will concentrate on the theory behind cation- anion balancing to prevent MF and practical steps to prevent MF.

DIETARY EFFECTS ON ACID-BASE METABOLISM AND CLINICAL IMPLICATIONS OF THESE EFFECTS ON MILK FEVER RISK IN DAIRY CATTLE

Since Stewart (1985) proposed the strong-ion difference theory, our understanding of the factors that determine the pH of blood has increased greatly. Put simply, the basic tenet of this theory is that the electrical charge of a solution, whether it be a glass of water or extracellular fluids, must always be neutral. When cations exceed anions in a solution the pH is increased (positively). When anions exceed cations the pH decreases.

Blood pH is ultimately determined by the number of positive and negative charges entering the blood from the diet. The major cations present in feeds and the charge they carry are sodium (+1), potassium (+1), calcium (+2), and magnesium (+2). The major anions and their charges, found in feeds, are chloride (-1), sulfate (-2), and phosphate (-3). The difference between the number of cation and anion particles absorbed from the diet determines the pH of the blood. The cation-anion difference of a diet is commonly described in terms of mEq/kg of just sodium, potassium, chloride, and sulfate as follows:

Dietary Cation-Anion Difference (**DCAD**) = $(Na^+ + K^+) - (Cl^- + S^{--})$.

This equation is useful, although it must be remembered that calcium, magnesium, and phosphorus absorbed from the diet also influence blood pH. Any positively or negatively charged ion that enters the blood will change the blood pH. A more complete equation, proposed earlier by our laboratory, assigns a relative strength to each dietary cation or anion, based on the average absorption efficiency as listed in NRC (1988) and other publications, determined primarily in lactating cows.

Thus, DCAD = $(0.38\ Ca^{++} + 0.3\ Mg^{++} + Na^{+} + K^{+}) - (Cl^{-} + 0.6\ S^{--} + 0.5\ P^{---})$ where Na, K, and Cl are considered to be absorbed with 100% efficiency. From this equation an equivalent of chloride will cause a greater change in blood pH than an equivalent of sulfate. In recent months we have evaluated the relative acidifying activity of various anionic salts by feeding them to dry cows and evaluating their ability to reduce urine pH (which reflects the changes in blood pH). These data lead us to believe the DCAD of a diet and its acidifying activity is more accurately described by the following equation:

$(0.15\ Ca^{++} + 0.15\ Mg^{++} + Na^{+} + K^{+}) - (Cl^{-} + 0.2\ S^{--} + 0.3\ P^{---})$.

This equation suggests that the major dietary factors determining blood and urine pH are Na, K and Cl. This equation offers a more rational approach to use of DCAD in MF prevention.

Milk Fever

The onset of lactation incurs a sudden and large demand for calcium from the blood of the dairy cow. In order to prevent blood calcium from decreasing, the cow must replace calcium lost to milk by withdrawing calcium from bone or by increasing the efficient absorbtion of dietary calcium. Bone calcium mobilization is regulated by parathyroid hormone (**PTH**) produced by the parathyroid glands located in the neck. Whenever there is a drop in blood calcium, blood PTH levels increase dramatically. A second hormone, 1,25-dihydroxyvitamin D, is required to stimulate the intestine to efficiently absorb dietary calcium. This hormone is made within the kidney from vitamin D in response to an increase in blood PTH. Put simply, MF occurs when cattle do not remove enough Ca from their bones and the diet to replace Ca lost to milk. This occurs because a key hormone involved in Ca metabolism, PTH, acts only poorly on bone or kidney tissues when the blood pH is high (Goff et al., 1991). Blood pH of cattle is often alkaline because forage K is excessively high. To avoid MF the blood pH needs to be decreased. The best way to do this is to reduce the K content (and in some areas of the country, the Na content) of the diet fed to the prepartum cow. Removing potassium from the ration can present a problem. All plants, must have access to a certain amount of K to obtain maximal growth. However alfalfa, other legumes, and at least some grasses accumulate K within their tissues to concentrations that are well above that required for optimal growth of the plant if soil potassium is high. Optimal growth of alfalfa occurs when the plant K concentration is 1.7-2.0%. Alfalfa often contains much higher levels. Lanyon (1980) reported that the K concentration of alfalfa samples submitted by Pennsylvania producers averaged 3.1% K, ranging from 1.42 to 4.05%. Many producers fertilize alfalfa heavily with potassium to increase the plant's resistance to winter kill. However it is unlikely that any benefit is seen by increasing plant potassium beyond 2.5%. It appears that current agronomic practices encourage overfertilization with K, resulting in luxury consumption of K by plants, which can be detrimental to the health of the periparturient dairy cow. What practices can be instituted by the producer so that a low K forage crop can be obtained for the transition cow ration?

Low Potassium Forages

Grasses

Corn is actually a warm season grass. Corn silage tends to be 1.1-1.5% potassium. It is difficult to find any other forage this low in potassium. Some other warm season grasses, such as switchgrass, big bluestem, and indiangrass tend to be low in potassium also, but they are low in protein and digestibility.

Cool season grasses such as bluegrass, orchardgrass, and bromegrass tested lower in potassium than alfalfa did 20 years ago. At that time these hayfields were unlikely to receive fertilizer. The tremendous increase in the number of cows on each farm has not been accompanied by an increase in the amount of land available for spreading manure. As a result, hayfields that were not fertilized in the past are now being used extensively as a place to dispose of animal wastes. Cool season grasses have a fibrous root system, which makes them very efficient utilizers of soil potassium. They will actually out compete alfalfa for potassium - this is why alfalfa stands eventually becomes grassy. Research at the Miner Institute (Thomas, 1996) indicates that timothy accumulates potassium to a lesser extent than other grasses and the second cutting of grass hays generally contain less potassium than the first cuttings.

Legumes

In the past alfalfa and other legumes were left out of dry cow rations because they were high in calcium. However we now know that dietary calcium has little effect on the alkalinity of the cow's

blood under practical conditions, so it does not induce MF. By restricting potassium application to the soil, it is possible to grow alfalfa that is as low in potassium as many of the grass hays. However, this eventually allows grasses to take over the stand and increases winterkill. One option may be to withhold potassium fertilization from a field that is in its last year of production and harvest that field specifically for the dry cows. However it can take several years to deplete soil potassium reserves if plant potassium values have been high. Some other rules of thumb:

- Potassium content is highest in alfalfa harvested in the early vegetative stage.
- Full bloom alfalfa may be more suitable for the dry cow.
- Potassium is released from wet soil more readily than from dry soil.
- Most years the first cutting of alfalfa will have a higher potassium content than later cuttings.

The key to MF prevention is to find a low potassium hay source and combine it with corn silage to form the basis for your dry cow ration. Try to formulate a total ration with less than 2% potassium. Limit access to pasture and watch to see if cows are eating bedding. Oat straw bedding is particularly high in potassium.

Anionic Salts

Adding anions to the diet of the cow can counteract the effects that dietary potassium and sodium have on the blood pH. Commonly used anion sources are calcium chloride, ammonium chloride, magnesium sulfate, ammonium sulfate, and calcium sulfate. All anionic salts are unpalatable as they give a strong salty taste to the diet. Sulfate salts are somewhat more palatable than chloride salts - but since they are much less effective acidifiers of the blood their use is not highly recommended. If used inappropriately they will cause inappetance and actually exacerbate fresh cow problems. Therefore, they should be used sparingly. The pH of the urine of the close-up dry cow can tell you if the blood of the cow remains too alkaline or if you have added too many anionic salts. In herds experiencing a MF problem the urine of close-up dry cows will be very alkaline with a pH above 8.0. For successful control of MF the average pH of the urine of the cows (Holstein) should be between 6.0 and 6.5. In Jersey cows the average urine pH of the close-up cows has to be reduced to between 5.8 and 6.2 for effective control of MF. If the average urine pH is between 5.0 and 5.5 you have probably added too many anions to the diet and the cows will suffer from a decline in DMI.

Various formulas exist to tell you how much of an anionic salt to add to the diet. Most nutritionists using the equation $(Na^{+} + K^{+}) - (Cl^{-}+S^{--})$ have a target DCAD for MF prevention of about -50 mEq/kg. Using the more physiologically relevant equation, $(0.15\ Ca^{++} + 0.15\ Mg^{++} + Na^{+} + K^{+})- (Cl^{-} + 0.2\ S^{--} + 0.3\ P^{---})$, the target DCAD should be between +200 and +300 mEq/kg. These are simply guidelines and are based on the setting of certain parameters at constant values as outlined below. Urine pH of the cows will be the better gauge of the appropriate diet DCAD than any formula. Some of the variables in the above formulas are somewhat fixed. Dietary magnesium should be set at 0.4% (higher than NRC recommendations). We like to use magnesium sulfate in our close-up rations to supply magnesium in a readily soluble form, not because it is an effective source of anions to prevent MF. Magnesium chloride, where available, would be another good method of raising diet magnesium to 0.4% and would give a stronger acidifying effect. The diet should supply between 35 and 50 g phosphorus daily so diet phosphorus will be set at about 0.4 %. More than 80 g phosphorus / day will inhibit renal synthesis of 1,25-dihydroxyvitamin D, which can induce MF. Dietary S should not exceed 0.4%. Some studies have reported a polioencephalomalacia-like syndrome (non-responsive to thiamine) when dietary sulfate is raised above 0.4%. In addition our results suggest that adding more sulfate is a poor choice because it is a fairly ineffective acidifying agent.

Dietary Cl can nearly always be raised to 0.5% with little effect on DMI. Most diets will require closer to 0.6% Cl for effective prevention of hypocalcemia. Getting ration Cl above 0.8% risks inappetance in the animals. Dietary Ca remains somewhat difficult to set. In a controlled trial there has been no advantage in keeping dietary calcium low (less than 40 g /day; Goff and Horst, 1996). Anecdotal evidence and at least two published trials suggest that high dietary calcium concentrations (>1.5 % Ca) are desirable when coupled with anionic salts and help prevent hypocalcemia (Beede, 1992; Oetzel et al., 1988; Oetzel, 1991). Good results have

been achieved by feeding as high as 180 g calcium per day. However when limestone is used to achieve these high dietary calcium levels the alkalinizing effect of the added calcium carbonate can be a factor. More importantly the limestone is taking up room in the ration that might better be used for energy sources. We currently bring calcium to 1.2%, which is fairly easily achieved, especially if calcium chloride is used as one of the anionic salts. More work needs to be done on availability of the different calcium sources and the role of dietary calcium during the periparturient period. The bottom line to balancing DCAD in dry cow rations is to concentrate on the minerals that actually vary in the diet; Na and K coming from forages and C1 which we will add to counteract the Na and K. Perhaps the most useful DCAD equation should be Na + K - C1 = DCAD, as described by the poultry people years ago.

Anionic salts generally add between $5 and $9 to feed costs for a close-up dry cow. We are currently investigating the use of hydrochloric acid preparations as a source of anions for the dry cow. These have proved more palatable, in our hands, than traditional anionic salts as they impart an acidic taste rather than a salty taste to the ration and should be less expensive as well.

Anionic diets prepartum may enhance milk production and health in the subsequent lactation, simply because hypocalcemia is decreased and the animal does not have the secondary problems associated with MF (Beede et al., 1991; Block, 1984; Curtis et al., 1983). It is difficult to assess the economic impact of subclinical hypocalcemia. It seems likely that if MF is associated with loss of muscle tone (i.e., abomasum, teat sphincters) and ruminal stasis, subclinical hypocalcemia will be associated with these same problems to a lesser degree. The impact of subclinical hypocalcemia on herd health may be nearly as great as MF because it is much more common than MF.

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