Three Weeks Prior and Three Weeks Postpartum—What I Do To Optimize Fertility In The Next Pregnancy

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Introduction

Definition and Description

The transition period is the most important period in a cow's lactation-reproduction cycle.[11,14,17] These events set the stage for the subsequent lactation. They largely decide the production, health and reproduction of the dairy cow.

This period consists of two phases. They are (1) the three weeks before calving (i.e., close up dry cows) and (2) the three weeks after calving (i.e., early fresh cows).

This is a very dynamic time metabolically and physiologically. During the close-up dry-cow period, rapid fetal growth, allometric proliferation of mammary gland tissue, growth of rumen papillae, and declining dry matter intake (**DMI**) take place. During the early fresh-cow period, continued development of mammary tissue, further growth of rumen papillae, rising milk production, and lagging DMI occur. Also, during these phases the dairy cow confronts challenge to most infectious and metabolic diseases.[43]

Ideal Transition Cow

Reviewing the target goals of the transition program is often helpful. Ideally, each cow would do the following:

1. *Calve without any complications.* Specifically, each cow would deliver a healthy calf without assistance, provide an adequate supply of high quality colostrum, and avoid physical injury.
2. *Remain free of metabolic or infectious disease.* Each cow would not contract infectious diseases [e.g., mastitis, metritis, etc.]. She would escape metabolic diseases [e.g., ketosis, milk fever (**MF**), laminitis, displaced abomasum (**DA**), etc.].
3. *Undergo accelerated feed intake.* Healthy cows have hearty appetites. A good transition program presents cows that have good appetites. Maximum DMI would occur by 90 days in milk (**DIM**).
4. *Experience an exponential rise in milk production.* Milk production should rise rapidly, peaking by 50 to 60 DIM. Each pound of peak milk increases lactation yield 200 to 250 lb.
5. *Commence reproductive cyclicity.* Cows that have high feed intakes, return to estrus sooner and have higher fertility. The ideal cow would have her first ovulation by 21 DIM.

Objective

The basic tenet of this paper is that cows that have fewer health disorders have greater milk production and better reproductive performance. Conversely, unhealthy cows do not produce or reproduce well. Controlling metabolic diseases, then, is paramount for good lactational and reproductive performance.[43]

Also, noting that metabolic diseases in the dairy cow are complexes is important.[20,39,40,45] That is, one condition leads to another. For example, a cow that has milk fever (**MF**) is four times more likely also to have a retained placenta (**RP**) and 16.4 times more likely to develop ketosis.[45]

This paper will focus attention on controlling three metabolic diseases. They are fatty liver (**FL**), hypocalcemia (**HC**), and rumen acidosis (**RA**). It is this individual's conviction that they are key diseases hindering reproductive performance in dairy cattle today.

Fatty Liver Disease

Description

Some misconceptions exist about FL. One, it is commonly thought that FL disease develops after calving when she is mobilizing large amounts of body fat. Recent research[9,23,24] shows that FL often develops prepartum or at parturition. Thus, it is a periparturient disease. Two, it was thought that FL disease is the re-

sult of obesity. These cows are more susceptible, but some surveys show that all cows have some degree of fatty liver.[26]

Cows show depression, lack of appetite, general weakness. Also, there are generally complicating conditions such as MF, DA, RP, metritis, and mastitis. The outstanding feature of this disease is the poor response to treatment of the accompanying diseases. For example, a cow with metritis may die or recover slowly and milk poorly.

Table 1 is a summary of a case study of a Michigan dairy herd.[30] This study shows the devastating effects of FL disease.

Table 1. Morbidity and Mortality in a Holstein Herd with a High Incidence of Severe Fatty Liver*

CONDITION	MORBIDITY/MORTALITY (% OF CALVINGS)
Milk fever	5
Ketosis	38
Retained Placenta	62
Mastitis	6
Death	25

*Source: Data from Morrow[30]

Effect on Reproduction

Fatty liver disease adversely affects reproduction. It may be apparent at lower concentrations of FL than seen with the severe manifestations of the disease.[25] Reduced breeding efficiency results from delayed time from calving to first estrus and decreased fertility. Work by Britt[10] suggests that poor corpus luteum (**CL**) formation and reduced progesterone are the probable cause. Herdt[25] points out that cows with FL have reduced serum cholesterol concentrations, and an extracellular source of cholesterol is necessary for maximum production of progesterone by the CL.

Etiology

Fatty liver begins with mobilization of body fat. Many factors stimulate lipolysis of adipose tissue, including negative energy balance (**NEB**), hypoglycemia, low serum insulin concentrations and relatively high serum concentrations lipolytic hormones, such as growth hormone, placental lactogen, and prolactin.[8,25,26] Estrogen, a potent regulator of hepatic FA metabolism in nonruminants, may play a key role in FL development.[15,22] Mobilization of fat from adipose tissue results in an increased level of serum nonesterified fatty acids (**NEFA**) and increased uptake by the liver. Fatty acids (**FA**) in high concentrations are toxic to tissues. Once in the liver, they are subject to two metabolic pathways: oxidation or esterification.[25] Re-esterification leads to triglyceride (**TG**) synthesis. Triglyceride is a source of energy for body tissues and the mammary gland. For TG to be exported from the liver, it must be packaged into lipoprotein (**LP**) particles. When hepatic production of TG exceeds LP export, FL results.[9,26,24]

Nutrient Partitioning and Fatty Liver

Competition among organs for nutrients also affects the development of FL. Fetal needs receive top priority in advanced pregnancy. Understanding the dynamics of fetal growth is helpful to our understanding of the development of FL.

- *Gestational Nutrient Requirements.* The goal of most consultants is to feed cows during the nonlactating, pregnant cow for maintenance and gestation.[15] The debatable question is: What is the requirement for gestation? The National Research Council[31] (**NRC**) states that the gestational requirement is 30% of maintenance (e.g., 80 Kcal of NEL/BW $kg^{0.75}$). Plainly, the energy requirement for gestation is 24 Kcal of NEL/BW $kg^{0.75}$ (NRC).[29] However, some consultants think this number is low.[15]

Fetal growth during the last trimester is exponential.[8,20,45] This creates a very large increase in the gestational requirement before parturition. Providing the nutrients required during this period of rapid growth is imperative. Work by Bell[8] and Ferrell[19] showed that this is a significant nutritional requirement.

The NRC gives a range of 3 to 6 Mal of NEL per day. This compares to about 3 Mcal from the above formula for a 1400-lb cow (i.e, 24 kcal of NEL/600 kg $BW^{0.75}$). Based on a maintenance requirement of 10 Mcal of NEL, then the requirement for maintenance for gestation is 13 or 16 Mcal. Energy densities of 0.59 or 0.73 Mcal of NEL/lb. DM, respectively, are necessary to meet the energy needs of the close-up cow consuming 22 lb. DMI. Obviously, most dairy consultants are using a requirement closer to the latter figure (e.g., 0.73 Mcal of NEL/lb. DM). See Table 3 for additional information on the nutrient needs of the prepartum cow.

- *Paradoxical Decline in Feed Intake.* Unfortunately, DMI begins to decline around 3 weeks before calving. Feed intake falls about 30%.[9] It begins to decline around 5 weeks with twins. The increased gestational requirement coupled with a decline in DMI make it necessary to feed a nutrient dense ration for 3 weeks before calving. Cows carrying twins should receive a greater supply of nutrients for 5 weeks.
- *Use of Maternal Energy.* The conversion of maternal energy into energy deposited into the gravid uterus is poor. Ferrell[19] reported a 14% conversion of dietary metabolizable energy (**ME**) into energy retention of the gravid uterus. This compares with

a conversion of ME to NEL of 60%.[31]

- *Fuel for the Fetus.* The fetus has very specific metabolic needs. Chandler[15] sheds light on this subject:
  1. Use of ME from the dam is exceedingly low (i.e.,14%).
  2. The fetus has a very high metabolic rate. There is a temperature gradient of 0.50 to 1.0°C over the dam. This high metabolic requirement results in 60% or greater of the energy being dissipated as heat.
  3. The placenta has an oxidative requirement equal to or exceeding that of the fetus.
  4. The primary substrates for achievement of this condition via oxidative metabolism are glucose, lactate, and amino acids. Since lactate originates from glucose, we can state that the fetus runs on glucose and amino acids.
  5. The amino acid role is significant with data showing that almost 60% of the nitrogen uptake is lost in oxidative forms.
- *Fuel for the Dam.* With the fetus dominating the use of glucose for energy, the dam must rely largely on the VFA, acetate, and long chain FA. Fatty acid mobilization occurs to supply energy for the dam. No doubt, this predisposes her to FL. Also, if there is any additional depression in feed intake or if the ration lacks proper nutrient balance, an exacerbation of FL may occur.

Treatment and Prevention

No treatment is effective. Prevention is the best course of action. Because FL develops by day 1 after calving, strategies to prevent metabolic disorders must start before calving.[24] The key is to avoid excessive FA mobilization from body fat. Plausible strategies include the following:

- *Body Condition Management.* Cows should not lose body condition (**BC**) during the dry period.[17] Dairymen should score dry cows each week. Cows losing weight may have twins. Move these cows to a higher energy ration. Increasing BC minimally is possible (i.e., 0.25- to 0.50-point) during the dry period.

Avoid getting cows overconditioned (i.e., >4.0). Fat cows have reduced appetites after calving. Though no studies have shown greater depression of DMI before calving,[24] anecdotal evidence suggests that is the case. The best way to avoid fat cows is to manage energy balance during the latter half the lactation.[15]

- *Properly Balanced Close-Up Dry Cow Rations.* Provide 6 to 10 lb. of grain daily to promote growth of the rumen papillae and allow the rumen microflora to acclimate to grains. Feed some silage or haylage to allow adaptation to fermented feeds if the milking rations contain them. Provide a protein balance of 15-16% crude protein that is 25-30% soluble protein and 35-40% rumen undegradable.[29,45] High-quality protein supplementation (e.g., blood meal, fish meal, meat meal, etc.) that provides a good supply of essential amino acids is critical. This is necessary to support gluconeogenesis.[15,25]
- *Provide Glucose Precursors.* Feed 4 to 8 oz. of propylene glycol or 8 to 12 oz. of calcium propionate if ketosis is a problem. Provide fermentable carbohydrates (e.g, corn meal, high moisture corn, barley, etc.) Corn meal is the ideal choice, because it ferments slowly.
- *Prompt Treatment of Fresh Cow Problems.* **Do not allow a fresh cow to get lost in the herd. Treat health problems promptly. Encourage fresh cows to eat. Force feed cows off-feed to prevent excessive loss of body weight.**
- *Dry Matter Intake.* Provide feed *ad libitum* to maximize feed intake during the transition period. Energy intake follows feed intake (i.e., DMI). Dry matter intake depends on many variables. They fall into three general categories: (1) environment, (2) cow, and (3) ration. Table 3 summarizes these variables.

Table 2. Variables that Influence Dry Matter Intake

ENVIRONMENT	COW	RATION
Temperature	Milk production	Physical texture
Ventilation	Body size	Palatability
Humidity	Hormonal status	Fiber content
Feedings per day	Breed	Nutrient balance
Water	Body condition	Moisture content
Sprinklers, fans	State of health	Forage quality
Social structure		

Hypocalcemia

Description and Etiology

With the initiation of lactation, most cows experience some degree of hypocalcemia (i.e., low blood Ca).[7,40] There are two types of hypocalcemia: clinical and subclinical.[40] Hypocalcemia results from the sudden flow of Ca from blood into colostrum. There is an influx of 23 g of Ca from blood during the first 24 hours after calving. This is 9x the available Ca pool in plasma.[45]

In most cows, activation of Ca homeorhetic mechanisms restores normal blood Ca early in the postpartum period.[21,38] If the system malfunctions, however, it increases the severity and duration of hypocalcemia. This

predisposes the cow to periparturient disorders.[7,40]

Milk fever is the clinical manifestation of hypocalcemia. There is an accentuation of the degree and duration of hypocalcemia in milk fever. Clinical symptoms reflect changes in neuromuscular function.[34] Initial symptoms are tremors. Subsequent neromuscular dysfunction leads to sternal recumbency and lateral recumbency. Death generally results if not treated. It occurs in about 5 to 10% of the cows.[34] Calcium homeostasis functions to maintain normal Ca concentration. These mechanisms maintain blood Ca by adjusting the supply and loss of Ca. The supply side of Ca is gut absorption and bone resorption. When compared with normal cows, milk-fever cows are the result of a breakdown on the supply side. The problem is inefficient Ca absorption from the gut and poor Ca resorption from bone.[36]

Relation to Other Diseases

Parturient hypocalcemia is a risk factor for several metabolic diseases that negatively affect post-partum health and performance.[2-5] Cows with milk fever are 3 - 9x more likely to develop other calving disorders (e.g., dystocia, RFM, ketosis, DA's, mastitis, and uterine prolapse).[39,45] Risco[39,40] showed a significant relationship between PP, Dystocia, and RFM.

Subclinical hypocalcemia is generally a greater problem than clinical hypocalcemia. Therefore, greater economic losses occur due to the obvious form of hypocalcemia. The uterus, rumen, and abomasum have significant smooth muscle function. Subclinical hypocalcemia can adversely affect their performance. Beede[7] described a hypocalcemia cascade that illustrates the association of hypocalcemia to other health disorders. Several studies confirm the relation of subclinical hypocalcemia to metabolic disease.[2-5,37,38,93,41]

Treatment and Prevention

The aim of treatment is to correct the paresis and to sustain the cow until the reestablishment of normal Ca levels in the blood. Calcium deficits are generally in the amount of 8 g. Giving 500 mL of 23% calcium gluconate intravenously provides 10.8 g of calcium.[40] An additional 500 mL subcutaneously is commonly given to reduce the incidence of relapses. This commonly alleviated clinical signs, but hypocalcemia remains for 2-3 additional days.

Cows generally respond favorably to a single Ca treatment. Other results include the following:[34]

- Incidence of relapse 12 to 48 hours after treatment ranges from 25 - 40%.
- Ten percent will remain recumbent for 24 hours but eventually return.
- Ten percent will die or become "downer" cows.

Programs for prevention of clinical hypocalcemia and correction of subclinical hypocalcemia usually revolve around: (1) manipulating the Ca and P content of the diet, or (2) manipulating the ionic balance of the diet. Four programs commonly encountered in the field are as follows:

- *Animal Nutrition, Incorporated (ANI).* This scheme recommends high daily levels of Ca and P, 200 g and 100 g, respectively. The promoted diet is a bulky diet of alfalfa hay, oats, and wheat bran.
- *Dietary Electrolyte Deprivation (DED).* This is a very controversial dry cow feeding scheme. It calls for acclimation to all of the milk cow feed ingredients. The aim is to restart the blood electrolyte transport mechanism by depriving cows of all major minerals the last 2 to 3 weeks before calving. No supplemental limestone, dicalcium phosphate, salt, sodium bicarbonate, magnesium oxide, etc., are fed. This method does call for vitamin and trace mineral supplementation. Some nutrients (e.g., vitamin E) are fed at increased levels.
- *National Research Council (NRC).* The NRC[31] recommendation for a mature dry cow during the prepartum period is 36 to 43 g per day (i.e.,0.39% of ration DM). This is below maintenance requirements. This recommendation assumes a positive Ca balance at the beginning of the dry period. (NRC, 1989) Field experience reveals two basic problems with this approach: (1) This level is not low enough commonly to reduce the incidence of milk fever; (2) achieving low levels of Ca from the available feedstuffs desirable to feed dry cows is not possible. Therefore, the frequently suggested compromise is to feed <100 g of Ca and <50 g of P daily. Typically this approach is effective in preventing clinical hypocalcemia; however, in other incidences it has been completely ineffective.[20]
- *Dietary Cation-Anion Difference (DCAD).* This scheme is a more reliable method of preventing milk fever when the Ca intake exceeds NRC requirements.[2-6,11-13,20,21,28,40] It is a method that balances rations for cations and anions. Synonymous names are dietary cation-anion balance, dietary electrolyte balance, cation-anion balance, strong ion balance, and fixed ion balance. It has become popular in recent years. Cows are fed an anionic ration (i.e., a negative DCAD) the last 2 to 3 weeks before calving. A negative DCAD causes a mild acidosis, increases mobilization of Ca from bone and possible gut absorption of Ca from the gut.[7,28] This increases the cow's ability to maintain normal blood Ca concentrations and reduces the incidence of clinical and subclinical hypocalcemia.[7] This method also calls for Ca, 120 - 180 g/day.[6,11,12]

The formula for DCAD is as follows:

DCAD= mEq [(%Na/0.023) + (%K/0.039)] - [(%Cl/0.0355) + (%S/0.016)]/100 g DM

For example, if the dry-matter content of a ration is 0.10%, 1.2%, 1.0% and 0.45% of sodium, potassium, chlorine, and sulfur, respectively. The DCAD calculations are as follows:

DCAD=[(0.10/0.023) + (1.5/0.039)] - [(1.0/0.0355) + (0.45/0.016)] mEq/100 g DM
[(4.35) + (38.46)] - [(28.17) + (28.13) mEq/100 g DM
-13.49 mEq/100 g DM

Dry Cow Feeding: Practical Implications and Strategies

This paper will primarily deal with dietary cation-anion balance (DCAB) in dairy nutrition. Achievement of this idea is not always successful.[7,13,27] It is the purpose of this paper to describe some ration formulation principles and feeding management guidelines necessary to use this nutritional concept.

Phase Feeding. Two groups are necessary to properly feed dry cows.[7,13] They are: far-off dry cows and close-up dry cows. The basic reason for this is a paradoxical decline in DMI prepartum. This decline occurs 2 to 3 weeks before calving for single births and 5 to 6 weeks for twins. This has serious consequences because there is not a reduction in nutrient demands. Contrarily, there is additional need for energy to maintain body condition and protein for fetal growth and mammary development. As previously stated, one of our greatest challenges is to prevent body condition loss in dry cows.

For example, if the daily nutrient requirements are 16 mcal NEL and 3.5 lb. protein. A 1400-lb cow eating 2% of BW will consume 28 lb. DMI. A ration density of 0.57 mcal NEL/lb and 12.5% crude protein will provide the amounts needed. However, if the DMI declines to 22 lb. as happens with the cow as she approaches parturition, then a ration density of 0.73 mcal NEL and 15.91% crude protein is necessary. Table 3 is a summary of these two phases.

Table 3. Phase Feeding of Dry Cows to Provide 16 Mcal NEL and 3.5 lb. Crude Protein.

Item	Far-Off Cows	Close-up Cows
Dry matter intake (lb/day)	28	22
Net Energy Lactation (Mcal/lb)	0.57	0.73
Crude Protein (% of DM)	12.5	15.91
Soluble Protein (% of CP)	40-50	26-30
Rumen Undegradable Protein (% of CP)	26-30	36-40

Ration Formulation. This writer previously recommended a method for balancing cations and anions in dry cow rations.[11,12] An updated version is as follows:

1. *Balance Mg at 0.40% of DM.* The mineral of choice is magnesium sulfate. Do NOT use magnesium oxide.
2. *Balance S at 0.40% of DM.* Use calcium sulfate to provide any additional sulfur. Avoid using ammonium sulfate.
3. *Counterbalance K with Cl to achieve a DCAD of -10 to -15 mEq(Na + K) - (Cl + S) / 100 g of DM.* Follow these steps. First, use calcium chloride up to a daily inclusion of 0.25 lb./head. Second, use ammonium chloride if supplying an additional chloride is necessary. Third, limit total dietary chloride to 1%.
4. *Provide 150 g of Ca and 30 g of P per cow / day.* Use traditional sources of Ca and P (i.e., calcium carbonate, dicalcium phosphate, etc.).
5. *Balance protein fractions.* Supply 15 to 16% crude protein. Restrict protein solubility to 25 to 30% of CP. Balance rumen undegradable protein (**RUP**) at 36 to 40% of CP. Not only should this diet be in bypass protein, but it should come from high-quality protein sources. Animal-marine protein byproducts (e.g., blood meal, fish meal, meat meal, etc.) are excellent sources. Remember: the growing fetus has a large requirement for essential amino acids during the last 2 - 3 weeks before calving.[15] This also points out why reducing the use of the ammoniated salts is best.

Balance Forage Base. Accurate knowledge of the forages included in the ration is essential for the dairy consultant. Test forages for crude protein, soluble protein, acid detergent fiber, neutral detergent fiber, nonfiber carbohydrates, Ca, P, Mg, K, Na, S, and Cl. If a change of forage is necessary, the dairy advisor should receive immediate notification. The consultant can, then, reformulate another ration. Failure to maintain a consistent forage base will result in animals doing poorly. Herds that cannot conform to these standards are NOT good candidates for feeding anionic salts.

Feeding Management. Feeding anion diets to prepartum cows is a challenge. Anionic rations are easy to formulate, but they can be difficult to manage. Manipulation of the DCAD provides a means to fine-tune rations. It does, however, require excellent management of nutrition and feeding. Consequently, there have been failures in the field.[7,13] Adherence to specific feeding principles is important for success. Field experience shows that feeding management is more important than ration formulation. Key feeding rules are as follows:

- *Minimize forage preference.* Feeding programs that allow preferential selection of feedstuffs invite problems. Three rations may exist on a farm: the ration formulated, the ration fed, and the ration consumed. The goal of ration delivery is for the cow to consume the ration formulated. Problems are common if cows are not fed a TMR and, consequently,

when they can express forage preference. This advisor has had success hand-feeding anionic salts in well managed herds. Incorporate the anionic salts into 6 to 8 lb. of grain. This grain mix is fed as a topdress onto approximately 10 lb. corn silage. Feedbunk space should be 2.5 to 3.0 ft per head to be sure that all animals consume their portion of the feed. Hay is fed free choice. Ideally, anionic rations are fed as a TMR. Total mixed rations offer many advantages.

- *Optimize dry matter intake.* Maintain DMI at 22 lb. or greater. Field experience has taught that if DMI falls below this key level, serious metabolic and disease problems will follow.[13] Generally, there will be an increased incidence of RP, FL, ketosis, and DA.

These ingredients are not palatable. This can lead to problems with consumption. Incorporate anionic salts into a TMR, which has some moist, highly palatable feeds (e.g., wet brewers grains, brewers condensed solubles, wet distillers grains, etc.). If this is not possible, then combine them in a grain mix with some appetizing ingredients (e.g., distillers grains, molasses, etc.). Pelleting may improve consumption,[35,36] and it helps prevent the separating out of the anionic minerals. Formulate the pellet for an inclusion of 6 to 8 lb. per head per day.

Anionic salts will negatively affect DMI when 3 or more equivalents are required for a negative DCAD of -10 to -15 mEq/100 g DM. In such cases, forage substitutions should be made to reduce the amount of anionic salts.

Emphasis of other basic factors affecting DMI is also essential. See Table 3 for a summary of these key determinants.

- *Acclimatize to anionic salts.* How long should anionic salts be fed? They are expensive, costing about 30 cents per cow per day. So the length of time is an important consideration.

Research trials have ranged from 21 to 45 days.[2-7,35] Based on these studies, anionic salts need to be fed 10 to 14 days to produce the desired physiological response. The inaccuracy of freshening dates makes this too restrictive and impractical in the field. Therefore, feeding these minerals for 3 to 5 weeks is best.

Herds wishing to feed anionic rations need to keep good reproductive records. Accurate prognostication of calving dates in bull-bred herds is necessary. Otherwise, cows are in the springer group either too long or too little.[27]

Also, herd size can be a factor. Smaller herds, for example, may need a longer period that will allow adequate group size for proper mixing and delivery of the ration.

Rumen Acidosis

Description

Rumen acidosis is the result of acids accumulating in the rumen.[32,33,42] It generally occurs early in lactation with a shift to high-grain rations. There are few symptoms in mild cases and the condition often goes undiagnosed. Severe overfeeding causes overt clinical signs.

There are several causes of rumen acidosis. Basically, they all relate to excessive grain feeding.[32,33,42] It can result from feeding too much grain too quickly to fresh cows. Another cause is feeding grain before forage in component herds. It can develop from feeding forages that are cut so fine that they lack sufficient effective fiber (i.e., the ability to stimulate cud chewing).

The basic course is that grain ferments into volatile fatty acids (**VFA**) in the rumen. When production exceeds absorption of VFA, these acids accumulate in the rumen causing the pH to drop to 6 or lower.[33]

There are three times based on the degree of acidosis.[42] They are subacute, acute, and peracute.

- *Subacute (mild) acidosis.* Indigestion and off-feed problems characterize the mild form. It is a common underlying factor for ketosis and displaced abomasum. It may also produce laminitis and foot problems. Consequently, it results in fresh cows with reduced feed intake, accentuated body condition loss, and delayed return to estrus.
- *Acute (moderate) acidosis.* Weight loss, poor milk production, chronic lameness, and inferior reproduction characterize this form. Also, there is damage to the rumen lining (i.e., ulceration). This results in bacteremia and bacterial localization in the liver, lungs, and other target tissues (e.g., heart valves, joints, and kidneys). Liver and lung abscesses are common findings in cows that suffered an attack of acute acidosis.
- *Peracute (severe) acidosis.* This form results from extreme overconsumption of grain. Death is common without prompt veterinary intervention.

Treatment and Prevention

The difficulty of diagnosing the subacute and acute forms at the time of the insult make treatment enigmatic. Peracute acidosis demands immediate and drastic action. Rumenotomy and removal of rumen contents, followed by intensive fluid therapy is a common practice.

Prevention is always better than treatment. It should go like this:

1. *Prepare the rumen.* The transition period is a time to equip the rumen for moderate grain feeding after parturition. Feeding 6 to 10 lb. grain during the close-up dry phase facilitates growth of the rumen papillae (i.e.,finger-like projections

of rumen epithelium). This increases the ability of the rumen to take up VFA. Also, feeding a modest amount of grain allows the rumen microflora to adapt to highly fermentable feeds (e.g, high moisture shelled corn, ground shelled corn, barley, etc.). In addition, introducing a small amount of any silages or haylages that are being fed to the milk cows is advisable. Remember: during the transition period we are trying to feed the feeds that will help the dairy cow to make the transition as smoothly as possible into the milking herd.

2. *Introduce Grain Slowly in Early Lactation.* This is a time when DMI is lagging. Also, the rumen papillae are continuing to elongate. The transition fresh-cow feeding program should not hinder either of these. There is, however, the tendency to get as much grain in the cow as quickly as possible to minimize NEB in early lactation.[33] This usually results in decreased DMI and even greater NEB. Consequently, we must strive for a balance between adequate fiber and energy. With TMR herds this is just simply a matter of a properly balanced ration. Component fed herds present a difficult task. My recommendation is to increase grain slowly, 1 lb. per day until the cow reaches peak grain level. Some recommend a more conservative approach.[42] They advocate that the cow be fed no additional grain the 1st week after calving. Afterwards, increase grain 0.50 to 0.75 lb. per day until peak grain level. This results in weekly grain increases of 3 to 5 lb. Anecdotal experience is that this is a very conservative approach and may allow for excessive weight loss, ketosis, and fatty liver.
3. *Reduce the acid produced after each meal.* The properly formulated TMR is the best way to accomplish this.[16,42,44] This permits a constant ratio of forage to grain. However, even with a TMR, providing adequate effective fiber is absolutely necessary. That is, fiber that promotes cud chewing. This requires forages that have adequate particle length. Fifteen to 20 percent of the pieces should be greater than 1.5 inches long. With component-fed herds, avoid "slug feeding" of grain. Divide grain into three or four daily feedings. Always feed forage before grain. Buffers may also help to maintain pH. Use buffers as aids, not as substitutes for good nutritional management.

Monitoring Transition Performance

Monitoring occurrence of health disorders and overall performance is a critical aspect of successful dairy cattle nutrition and management. Specifically, monitoring the three metabolic diseases discussed is possible (i.e., FL, HC and RA).

- *Fatty Liver.* A liver biopsy is a direct way of testing for fatty liver. Indirectly, measurement of serum NEFA is a good indicator of potential problems.
- *Hypocalcemia.* Serum Ca is the most explicit way to check for hypocalcemia. Urine pH analysis is a vague method of determination.
- *Rumen Acidosis.* Determining the pH of rumen contents is the most direct way. Analysis of individual cow records for butterfat test depression and protein-fat inversion is a roundabout way.

Besides monitoring for the incidence of these diseases, monitoring the occurrence of related health problems is useful. Since we are dealing with disease complexes, monitoring the incidences of these related disorders is revealing. Field experience reveals this to be a practical alternative in many cases to specifically testing for FL, HC, and RA. Table 4 is an example of some parameters that are helpful to watch.

Table 4. Sample of Some Key Items to Monitor Monthly

PARAMETER	JAN	FEB.	MAR	APR
Cows calved, 1st lactation	50	45		
Cows calved, 2nd lactation	40	20		
Cows calved, 3+ lactation	35	35		
Total cows calved	125	100		
Total cows in herd	500	530		
Daily milk production, lb.	80	84		
Cows >100 lb. daily milk, lb.	50	60		
150-day corrected milk, lb.	81	84		
Mastitis, clinical cases	20	30		
Mastitis, acute cases	0	0		
Somatic cell count (SCC), 1000s	200	180		
Bacteria count	2000	1200		
P I count	7500	9000		
Milk fever	5	10		
Retained placenta	25	22		
Ketosis	15	20		
Displaced abomasum	10	4		
Foot rot	0	0		
Sole ulcers	5	5		
Subsolar abscesses	15	20		
Died	5	7		
Cows sold <90 DIM	5	10		
Total cows sold	15	20		

Summary

The transition period is from three weeks before to three weeks after calving. It is a critical time in the life of the dairy cow. It is during this time that the mod-

ern dairy animal makes a great metamorphosis. She moves from a dry cow with marginal nutrient requirements to a lactating cow with massive metabolic needs. How well she makes this transition affects her health, production and reproduction.

Our challenge is to feed and manage the modern dairy cow so that she makes this progression smoothly. Ideally, each cow should (1) calve without any complications, (2) remain free of any infectious or metabolic diseases, (3) undergo an accelerated feed intake, (4) experience an exponential rise in milk production, and (5) commence reproductive cyclicity by 21 DIM.

In short, a healthy cow will give more milk and breed back sooner. Fatty liver, hypocalcemia, andrumen acidosis are metabolic diseases that adversely affect reproduction. Transition management and feeding programs that successfully reduce their incidence will generate greater reproductive efficiency.

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