oz. of dicalcium phosphate; 2) Grind the concentrate as coarse as possible, yet crack all corn kernals.

Response: Two DA's in the next year.

Future: Remove screen in forage harvester to ensure adequate length of cut.

Case #3

This herd of 35 cows housed in tie stalls was producing about 19,000 lbs. of milk. During the summer of 1981, there were 5 DA's, one became a downer and died. During the summer of 1982 1 more cow was operated on for a DA.

Rations: 1. Milking Cows
- Approx. 40 lbs. corn silage
- Free choice baled hay
- 10-35 lbs. of grain mix
- Protein supplement 5% added fat

2. Dry Cows
- Free choice baled hay
- 10 lbs. corn silage
- 5 lbs. commercial dry cow concentrate

DA's occurred in summer. Why?

A. Hot weather equals decreased DM intake.
B. Field work = increased corn silage and grain too fast.
C. Purchased out of date (special price) high fat protein supplement (rancid?)
D. Does high fat supplement complicate ketosis-fat cow-DA syndrome?

Case #4

This 120 cow Holstein herd producing at above 17,000 lbs. of milk housed in a free stall had a 60% incidence of DA's in the previous year. Fifty percent of the DA's were in late lactation. Sub-clinical ketosis was present in many cows. Ketosis-milk fever (?) at calving—Alert cows down that responded to calcium therapy. There were computer balanced rations for 3 production groups and the dry cows. It was basically 4 total mixed rations plus free choice hay.

Metabolic profile tests revealed anemia, increased calcium and increased phosphorus.

Ration abnormalities:

1. Excessive calcium and phosphorus in diet.
2. 20 times Vitamin D and 10 times Vitamin A.
3. Dry matter intake calculated to be 60 lbs. in high production group (decreased hay intake).
4. Odor of acetate in corn silage.
   - high acetate to propionate ration in rumen fluid.

Changes made were a reduction of abrupt ration changes, but still having some problems.

General Recommendations

If it works, don't fix it, you might break it. There are no specific recommendations that will work in every case. Standing back and looking at the facilities, feeding program and the dairyman is very important in understanding the problems that are inherent and the problems that are introduced by the people involved.

There are 2 recommendations that are always important in prevention of DA's and the periparturient disease complex.

1. Avoid abrupt ration changes.
2. Do not chop silages shorter than ¼" theordical cut and do not use a screen.

In addition, if problems are occurring, return the ration to its simplest but completely balanced form in mineral, vitamin and protein considerations.

Milk Fever Prevention Update and Vitamin D_3 Toxicity

William G. Olson, *D.V.M., Ph.D.*
College of Veterinary Medicine
University of Minnesota

Milk Fever Prevention

Discussing problems of milk fever with practicing veterinarians today reveals that in some herds there are occasionally epidemics that occur for a variable period of time in some herds. The overall incidence in the U.S. is apparently down from the 8-9% of the 1960's to about 6-7%. However, sales of milk fever treatments may be rather stable. This may also mean that the average milk fever cow may get 2 bottles per treatment.

Today, in the large herds, where caloric and calcium intake are very closely regulated to minimize overfeeding of energy and calcium, the herd incidence of milk fever may be below 1%. The major question for the medium size farm is whether or not it is worthwhile to prevent milk fever if the management upset is too great. This is a real problem for

dairymen on all legume forages.

There are some possible Vitamin D_3 metabolite products that may soon be tested for milk fever prevention. The major problem with 25 OH cholecalciferol was that when used in cows fed "high" phosphorus diets to also prevent milk fever, it was not effective. As with feeding or injecting very large amounts of Vitamin D_2 or D_3, toxicity is a real concern.

Midlactation downer (MLD) is another problem to be concerned with today. They can occur at any time and in a few herds we have investigated there have been 3-6 cases and in some cows repeat episodes. In one herd, the only nutritional change was a reduction in phosphorus due to observed higher amounts of phophorus in the forages. Addition of phosphorus up to previous amounts in the diet and paying more attention to the dry cow diet management reduced problems in this herd. Laboratory results revealed hypocalcemia, hypophosphatemia and an adequate 1,25 OH_2 Vitamin D_3 increase in plasma. In a second herd, occasional MLD's are occurring after a more serious "epidemic." This dairyman was exceeding by nearly 2 times the already quite high levels of fat soluble vitamins and minerals that were recommended.

The crisis in calcium homeostasis here is unknown, but off feed to some degree certainly will compromise intestinal absorption. Stress or estrogenic activity if in estrous may affect bone mobilization, however this remains speculation. These cases respond readily to calcium therapy.

Until a product is available that can be used with an all legume dry cow roughage program, maintaining calcium to between 60-100 grams certainly is effective in reducing the incidence. The other critical factor is keeping the cow on feed through the periparturient period. Here the very old advice of gradual feed changes to minimize rapid changes or death of ruminal microflora is often ignored. Many dairymen may get by with major feed ingredient changes but many do not.

Vitamin D_3 Toxicity

Vitamin D supplementation is required for all animals housed in absence of sunlight. Exposure to sunshine 1-2 hours per day will prevent rickets. The requirements for dairy cattle range from 600 IU per day of Vitamin D in calves to 6000 in lactating dairy cattle. Common recommendations are for 2 to 4 times the NRC requirements, 12-25,000 IU of Vitamin D per day. There is no data available to show that feeding over 12,000 IU per cow day will improve calcium availability or improve milk production.

The rationale for supplementing higher or "mega" levels comes from at least 5 different reasons.

1. *Milk fever prevention*

   Oral injection of 20 to 30 million IU of Vitamin D_2 or 10 million IU of D_3 administered in a single intramuscular injection will reduce the incidence of milk fever. Soft tissue calcification became a serious side effect unless oral dosage was discontinued after 7 days or a balance in calcium, phosphorus and magnesium intake was controlled. Hibbs and Conrad reported that feeding 35,000 IU of Vitamin D_2 per pound of concentrate year around reduced the incidence of milk fever in problem herds, however cows with no previous history of milk fever had an increased incidence of milk fever.

2. *Overage in supplements to insure meeting label claims*

   Of unknown importance is the excess Vitamin D concentration formulated into various feeds in order to meet label claims. This was a possible source because of studies by Waibel and others that some sources of Vitamin D were of lower biological activity than the chemical assay indicated. Current practices are unknown, however we do not know of any instances where this is a serious problem.

3. *Several supplemental sources or accidental toxicity*

   On a few occasions in field problem investigation, several sources of supplemental vitamins were present, resulting in Vitamin D levels over 300,000 IU per day based on label claims.

4. *To increase milk production*

   There is the "claim" by a few nutritionists and veterinarians that when high amounts of Vitamin D are supplemented (200,000 IU per day or more) there is an increase in blood levels of calcium and that this in turn will increase milk production. The data to substantiate this claim are not known.

5. *"If a little is good, a lot is better"*

   Shotgun and excessive dosages of vitamins and drugs are rarely critically evaluated other than clinical impressions and often are accompanied by other changes which makes scientific evaluation impossible.

Over the past 8 years, poor production, sudden deaths, emaciation and possible increased susceptibility to common diseases were observed in several herds on an unusual feeding program. The feeding program provided a balancer to be fed with corn silage to make a complete ration. It is evident that the ration is deficient in energy, protein and phosphorus for 70 lbs. of milk production. In addition, Vitamin D_3 and Vitamin A were fed at 200 and 50 times respectively of the NRC recommended allowances. All of these herds were assessed by metabolic profile testing (Stevens, *et al*). Every herd was anemic. Statistically significant hypercalcemia and hyperphosphatemia was present in individual herds. All calcium, phosphorus, magnesium, and hematocrit values from 5 herds were combined according to milk production and analyzed. The increased calcium and phosphorus concentrations were highly significant. Magnesium was slightly lower and anemia was highly significant in cows over 40 lbs. of milk production.

Plasma Vitamin D_3 and metabolite concentrations were highly elevated in these herds receiving over 1×10^6 IU of Vitamin D_3 per cow per day. It is apparent that chronic feeding of excess levels of Vitamin D_3 for 1 year or more results in elevated plasma levels of all of the metabolites except for 1,25 OH_2 D_3, when compared to control and

acute toxicity studies by Littledyke and Horst. The consistency of the data between herds and comparing the composite data to the acute toxicity is evidence for chronic toxicity. The lack of soft tissue calcification may be explained by the presence of 2.4 x 10^6 IU of Vitamin A and the deficient supplementation of minerals and protein. The severe anemia may be due to either Vitamin D or Vitamin A toxicity or protein and/or phosphorus deficiency. Iron deficiency was determined not to be a problem because of adequate iron content in bone marrow specimens.

The immediate and obvious question because of widespread Vitamin D_3 supplementation is "how much is too much." Continuing studies in field problem herds have revealed that when 80,000 IU of Vitamin D_3 are fed daily and injections of Vitamin D_3 are administered, variable hypercalcemia, hyperphosphatemia and anemia have been observed along with feet and leg problems and above average mortality rates. The problems have as a clinical observation been relieved when vitamin supplementation was reduced to recommended levels.

Salt Deficiency, Iodine Toxicity and When You Say "It Must Be The Feed" You May Have To Say It In Court

William G. Olson, *D.V.M., Ph.D.*
College of Veterinary Medicine
University of Minnesota
St. Paul, Minnesota 55108

Salt deficiency is a problem that should not occur because it is the cheapest ingredient in the ration, costing about 1 cent/cow/day. We have encountered several herd complaints which have had salt deficiency as the primary cause of production decline or loss. The reasons given include:

1) Salt will corrode the mixer grinder.
2) The water supply contains enough salt.
3) I thought a tablespoon of salt was adequate if I fed salt free choice.
4) To avoid udder edema.
5) I was told there is adequate salt and mineral in the protein supplement.
6) Errors in mixing or deletion of a portion of the amount recommended.

The major problem in these instances is to convince the individual that salt deficiency will affect milk production. The NRC requirement for salt for lactation is .46% of total dry matter. The thumb rules that have been used for decades are: 1) 1% of the concentrate (a bit less in high producers, over 50% concentrate); 2) 3-4 oz./head/day in total mixed rations; and 3) 4 oz. is ½ cup of salt (1 tablespoon is 0.5 oz.).

In several herd investigations we have measured both serum using the metabolic profile test and urinalysis including sodium determinations and creatinine for estimation of fractional excretion. Serum sodium concentration be up to 8 times more than the human requirement in 1 quart of milk. Most fluid milk markets have a self-imposed restriction on iodine content, however, it is not readily known to what extent it is monitored. Cases of iodism in man due to high dietary iodine have not been identified in the United States.

When You Say It Must Be in the Feed, You May Have to Say It in Court

Litigation is one way for farmers to recover losses from disease or nutritional imbalance. There are many ways to approach the issue, but one way is to avoid quick statements about problems unless the situation is absolutely unequivocal. This means that you *must* rule out all of the other possible causes of the problem encountered or the logic must be absolute. The approach I have generally taken is to contact the feed companies or farmer's nutritionist as soon as there is a hint of a problem involving them. If there is a specific recommendation made by an individual a frank but courteous discussion confrontation should be made immediately to discuss or correct any erroneous advice. We are all capable of making mistakes! Since production loss is usually the bottom line, immediate correction should be made to minimize continuing losses. Mostly the legal profession comes out ahead in these cases, however, there is a great deal of preparation time in lawsuits that no one sees unless you have been involved directly. It would certainly be better if the parties involved could somehow solve their own disagreement, however, phoney or unfounded claims must be sorted out too. This is when you as the farmer's advisor are the most vulnerable (want to be overly helpful). Sometimes there is no answer or the problem has too many plausible etiologies. Poor management, mud, defective equipment or cows, and oversold or overpromoted products or concepts are a few of the sources of misunderstandings that lead to litigation.