Parturient paresis, more commonly known as “milk fever” is a common metabolic disease of dairy cattle that has been estimated by Chuck Guard, DVM at Cornell, to cost dairy producers $334 per clinical case. It is caused by low blood calcium (hypocalcemia) and is most commonly seen in dairy cows that have recently calved. Due to the rapid increase in milk production of the dairy cow after calving serum calcium may be acutely depleted. Calcium is required by muscle tissue for proper activity, therefore low levels of blood calcium (<7.5 mg/dl) will lead to loss of muscle function and the clinical signs of milk fever which include recumbency, bloat increased heart rate, and potentially death. More importantly to the dairy industry is the effect of subclinical hypocalcemia on cow performance. Subclinical hypocalcemia, while not resulting in recumbency or death, can be a major factor in the development of other diseases in dairy cows such as retained placenta, metritis, and displaced abomasum which can lead to suboptimal production and a higher risk of being culled from the herd. The condition of milk fever or hypocalcemia may be easily treated through the use of a variety of calcium preparations that may be administered intravenously, subcutaneously, or orally. While treatment is usually successful and fairly simple, producers and veterinarians would prefer to try to prevent the occurrence of the condition rather than treat affected cows.

Prevention of hypocalcemia is somewhat more complicated than treatment but can be successfully achieved. The approaches to prevention rely on nutritional intervention and the attempted manipulation of calcium metabolism in dairy cows. To understand the thought behind these nutritional interventions one must first have a basic concept of calcium metabolism in dairy cows. If a dry dairy cow is fed a diet high in calcium (>100g Ca/day) she can meet her daily calcium requirement through passive absorption of calcium from her gastrointestinal tract. Active transport of calcium from her intestines through the action of a form of vitamin D known as 1,25-dihydroxycholecalciferol (1,25-[OH]2D) or resorption of calcium from her bones due to the action of parathyroid hormone are not required and are therefore these pathways shut down. However when the cow calves and starts producing large volumes of milk her calcium requirements also increase dramatically and more calcium must be absorbed from the diet and resorbed from bone. Unfortunately, if these pathways have been shut down there will be a delay until they can start providing calcium to meet the
cow’s requirements. This delay causes excessive calcium to be removed from the bloodstream and the development of milk fever.

One nutritional intervention that may be used to prevent the shut down of active transport of calcium from the intestine or resorption of calcium from bone is feeding a low calcium diet to close-up dairy cows and then switching them to a high calcium diet post-calving. The recommended levels of calcium in the diet of the pre-fresh cows in this system is <20 g Ca/day. This low level of dietary calcium is very difficult to achieve. Fortunately, the incidence of milk fever has been demonstrated to also be decreased if dietary calcium is kept below 60g/day which is somewhat more achievable. In addition to calcium restriction, it has also been noted that phosphorus levels greater that 80g/day also increase the incidence rate of milk fever through inhibition of 1, 25-(OH)2D. Therefore, for proper cow health it is essential that dietary calcium and phosphorus levels be closely monitored.

More recently, another nutritional approach has been attempted for the prevention of hypocalcemia. This approach is known as the dietary cation-anion difference (DCAD) and involves the inclusion of anionic salts in the rations of close-up dairy cows. The theory behind the use of anionic salts is that the feeding of negatively charged anions such as chloride and sulfate will make the blood of the cow slightly acidic. The response of the cow will be to mobilize and absorb more calcium in attempt to buffer the acid in her bloodstream and therefore all of the mechanisms required for maximal calcium supply will be active at the time of calving. To achieve an effect nutritionists will attempt to increase the number of anions and decrease the number of positively charged cations (such as potassium, sodium, and calcium) to produce a diet that is higher in anions than cations. Although many slightly different formulas exist one common one used for the calculation of the DCAD is:

\[(Na + K) - (Cl + S)\]

From this equation it becomes obvious that forages high in potassium (such as alfalfa) need to be avoided as much as possible. However, the use of anionic salts makes the use of some higher potassium (and calcium) forages in close-up dairy cow diets possible. To monitor whether anionic salts are performing as hoped the urine pH of cows on anionic salts needs to be checked. The level of anionic salts in the diet should be adjusted until urine pH’s are in the range of 5.5 to 6.5 (Jerseys should be at the lower level of this range while Holsteins can be in the upper portion of the range). As most things in life there are problems in using anionic salts. The major problem encountered by producers and nutritionists is a lack of palatability. This is the last thing we want to have happen in close-up dry cows that may already be experiencing a decrease in dry matter intake. Fortunately, some of the newer proprietary anionic salt products are much more palatable than the original salts that were used in diets.

In conclusion, milk fever or hypocalcemia is large problem in the dairy industry. Dietary levels of potassium and calcium in the rations of close-up dairy cows play a major role in causing the occurrence of this medical condition. The use of anionic salts provides an option to producers with limited access to forage with low potassium or calcium levels.