MEDICINE AND MANAGEMENT

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Fundamentals of milk fever

Milk fever, or, more appropriately, 'parturient paresis' (parturition-giving birth) (paresis-partial paralysis) is a problem confronted by every dairyman, and perplexing in that the complete understanding of its cause and prevention still escapes both dairymen and scientists. Indeed, there is a practical limit which one can go in an effort to prevent it.

Parturient paresis usually occurs within 72 hours after calving, occasionally shortly before calving, and infrequently weeks or even months after calving. Average incident is about 10 per cent although problem herds can be as high as 80 per cent, usually with no simple explanation for the difference.

Milk fever is associated with low blood calcium or hypocalcemia. Noraml blood plasma calcium is at a level of 10-11 mg per cent. As the plasma calcium level drops at calving time the initial signs of milk fever appear. At first the cow may appear overly excitable or hyperirritable, with a nervous, stiff gait and overextended hocks. They will shift weight on their rear legs and become wobbly. Appetite will usually be poor and constipation often occurs.

As the plasma calcium dips to around 6-7 per cent, second stage or sternal recumbancy occurs. The cow lays on her sternum, unable to rise, usually with her head turned back to her flank resting on the floor. As plasma calcium drops below five mg per cent, the third stage or lateral recumbancy deyelops where the cow is laying on her side in apparent flaccid paralysis, unable to set up without assistance and props. If untreated at this stage the cow usually goes into a coma and dies.

The temperature of a milk fever cow is usually subnormal but may be increased if exposed to heat or sunshine, as there seems to be an inability to regulate body heat.

Milk fever is usually readily recognized by the dairyman and fairly easy to diagnose. There are, however, conditions which can be confused with milk fever, the most common and important one being acute or septic mastitis. It is very important to examine the milk of a cow near calving and immediately after calving. A delay of only 12 hours could be the difference between a treatable case and one that is beyond hope. A cow with acute or septic mastitis can also be down in sternal recumbancy with low body temperature; however their eyes are often sunken, skin quite tacky, and with a mucoid diarrhea as opposed to milk fever.

Complications of milk fever are many and varied. Metritis (infection of the uterus) can exist along with milk fever, even if the placental membranes have been expelled, and will limit the response to milk fever therapy. Another problem is damage to muscles and nerves, either from thrashing around in the third stage or simply from lying for prolonged periods on concrete surfaces. A cow in terminal stages of milk fever will often regurgitate rumen contents and then inhale them into the lungs resulting in aspiration pneumonia. Such a cow will recover from milk fever only to die from aspiration pneumonia a few days later. This is why it is so important not to give any cow suspected of having milk fever anyhting my mouth as the pharynxz muscles used for swallowing are also paralyzed and whatever is given could go straight into the lungs.

The calcium balance in the animal body is a dynamic system, a state of constant change. Calcium ingested in the food is being absorbed from the gut. Some is lost in the feces, some in the urine. The storehouse of calcium is the bones where it is deposited, while during times of need some is resorbed from the bone to replenish the blood plasma supply. Much calcium is used in the milk, and also for the fetus. In fact, right before parturition a pregnant cow is losing calcium to the fetus at the rate of five grams per day.

It is currently thought that the sudden large requirement of calcium for the manufacture of milk is what precipitates the sudden drop in plasma calcium, thus milk fever. Factors which affect movement and levels of calcium and phosphorus levels, vitamin D, and a hormone produced by the parathyroid gland called parathyroid hormone or PTH. High phosporus blood levels result in decreased calcium blood levels. Vitamin D is essential for proper absorption of calcium from the gut and bone, and PTH regulates resorbtion from the bone. With all this that is known about what regulates calcium balance one would think prevention and treatment should be quite easy.

Earliest successful efforts at treatment occured in 1897 when a veterinarian in Denmark found, to his amazement, that injection of a solution of potassium iodide into the udder alleviated the signs with eventually recovery. He later found that forcing air into the udder along with the iodide gave better results. Later, another veterinarian found that inflation or insufflation of the udder with air along was equally effective. What was happening was that the pressure in the udder was

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