MILK FEVER is a disease of high-producing dairy cows. It is marked by paralysis, inability to rise, and partial or total loss of consciousness in cows that have recently calved. It occasionally affects cows during calving but occurs oftenest in the first few days after calving or when the cow is coming into full production. It occurs after the first lactation and is not common in 3-year-olds. Most cases occur in cows 4 to 8 years old. The condition may return at subsequent calvings in good cows.

Milk fever is also called parturient pariesis and parturient hypocalcemia. The condition is produced by a rapid lowering of the calcium, or lime, in the blood—hence the name “hypocalcemia.” The name “milk fever” is a misnomer, as there is no fever. The temperature is normal or subnormal.

Milk fever used to cause collapse and rapid death in as many as 90 percent of the affected dairy cows. Now, thanks to more complete knowledge about the disease and modern methods of treatment, only a small percentage of dairy cattle die of uncomplicated milk fever.

A major advance in our knowledge came in 1925, when H. Dryere and J. R. Greig, of England, concluded from their extensive research that milk fever was linked to an acute deficiency of calcium in the blood and that the injection of calcium into the blood effected a cure.

Other research revealed that there is also a decrease in phosphates and sometimes magnesium. Usually there is an increase of blood sugar.

Earlier investigators had cured milk
fever by inflating the udder with air, but they could not explain why. The cure, it was learned later, had a mechanical basis. The new milk, or colostrum, is rich in calcium. Udder distention prevents further draining of calcium from the blood and the calcium already in the udder is reabsorbed. The body of the cow is able to restore the calcium level in the blood in 3 to 5 hours, and recovery takes place.

Research on the cause of milk fever has continued because of two circumstances. Rarely does a cow fail to show response to calcium therapy and die if there are no complications. Most research has been directed to a study of relapsing milk fever cases and the prevention of the disease. As the parathyroid gland is closely connected with calcium metabolism, it has been postulated that this internal gland becomes disturbed to provide the basic cause of the breakdown of the mechanism of calcium metabolism. The theory has not been completely substantiated, but we have some evidence that the basic cause may be associated with the parathyroid gland.

Most cases of milk fever occur within 72 hours after calving.

Odd cases that occur a long time before calving or several weeks afterwards are apt to be complicated infections or other conditions that resemble milk fever. A positive diagnosis can only be made by laboratory tests. Occasional conditions may arise in which blood calcium drops; they may respond to calcium treatment.

The owner may not notice or recognize the earlier stages of milk fever. Dullness and reluctance to move may appear a few hours or—more commonly—24 and 48 hours after calving. The first symptoms sometimes include excitement and tetany or spasms of the hind legs.

The early symptoms pass off quickly. Then paralysis begins to affect the hind legs. The cow staggers and finally goes down. She becomes unconscious and her head and neck are kinked laterally or turned back along the side. The pulse may be fast. She may groan and breathe heavily. Bloating occurs quickly if she becomes stretched out.

Many cases of milk fever are complicated by metritis (infection of the womb); pneumonia, caused by inhaling stomach contents; acetonemia; paralysis of the nerves of the hind legs; and sometimes dislocations and fractures.

Various ways to prevent milk fever have been suggested. None has had much success.

Delayed or partial milking may help for some cows but may be ineffective in high producers and may predispose to mastitis.

Giving calcium immediately after calving may have some merit with cows that have had milk fever. As soon as the lack of calcium was discovered as producing the symptoms of milk fever, the feeding of lime in various forms during the dry period was tried as a means of prevention. The results were nil.

J. M. Boda and H. H. Cole, of the University of California College of Agriculture, conducted more than 3 years of field trials on the prevention of milk fever. They accepted the theory that a disturbance of the parathyroid was the basic cause of milk fever. They began with several assumptions: If the cow were on a diet adequate in calcium during the dry period, very little calcium would be needed during the pre-calving period, when calcium metabolism (regulated by the parathyroid gland) became dormant. When the cow calves, the demand for calcium becomes enormous and, if the parathyroid gland is out of condition, calcium metabolism breaks down, calcium becomes deficient in the blood, and milk fever results. Then, they theorized, the parathyroid of a cow placed on a low-calcium diet during the dry period would be in good shape to meet the increased calcium demands after freshening because the parathy-
roid had been active to maintain calcium due to low intake.

They proposed the following formula for use during the dry period (in pounds): Ground barley, 800; rolled barley, 600; wheat bran, 500; cottonseed meal, 100; monosodium phosphate, 40; and salt, 10. Each cow should receive 8 pounds a day for 6 weeks before freshening plus 8 pounds of oat hay or poor hay.

In several herds where the incidence of milk fever was very high, feeding of the low-calcium diet reduced the number of cases from 30 to 3 percent. Production was not reduced noticeably.

Research conducted by J. W. Hibbs and H. D. Pounden, of the Ohio Agricultural Experiment Station, showed that the addition of vitamin D to the diet of the cow 5 days before calving reduces the incidence of milk fever.

The cow down with milk fever should be kept up on her sternum and not allowed to get out flat. Pneumonia caused by bloating and inhalation can be prevented by keeping the cow upright. The cow should be placed in a well-bedded stall to avoid injury. If she is in the pasture, she should be watched so that she does not roll or struggle into ditches or over embankments.

Air inflation of the udder will cure milk fever, but the danger of mastitis is so great that the practice has been abandoned except in dire emergency. It should be done with great care and strict antisepsis.

The procedure preferred by veterinarians is the intravenous injection of calcium gluconate in a 20-percent solution. Because acetonemia, or a lack of sugar in the blood, often accompanies milk fever, dextrose is added to the calcium solution. Calcium injections should be made slowly to avoid heart block, which is dangerous. Immediately after treatment the cow brightens up and usually makes a good recovery in 1 to 2 hours.

Relapsing cases may need one or more additional treatments. The cause of relapses has yet to be solved. Some authorities believe the addition of phosphates and magnesium to the calcium solution prevents relapses. When treatments fail, very likely the diagnosis was wrong or complications developed.

W. J. Gibbons is professor of medicine and infectious diseases in the Alabama Polytechnic Institute, in which he formerly was professor of large-animal surgery and medicine. For more than 20 years he was an instructor and professor in the New York State Veterinary College.

**Bovine Mastitis**

R. W. Brown, Jr.

MASTITIS means inflammation of the udder. It results principally from infection with micro-organisms.

Many kinds of bacteria and some yeasts can produce mastitis. The streptococci (mainly *Streptococcus agalactiae*, *S. dysgalactiae*, and *S. uberis*) and the staphylococci (*Micrococcus pyogenes*) are the chief causative agents, but mastitis due to the bacteria *Escherichia coli*, *Aerobacter aerogenes*, and *Pseudomonas aeruginosa* has been occurring with greater frequency.

Other organisms, such as *Pasteurella multocida*, yeasts, and acid-fast bacilli, also have caused outbreaks of mastitis,