avoided. Intake should not be cut sharply. Care should be taken that the sheep do not go unfed during periods of storm or while being shipped. A moderate amount of exercise is important. An adequate water supply should be provided. A constant supply of salt is desirable.

The theory of treatment is simple: It is necessary to raise and maintain the sugar level of the blood. In practice, however, treatment on an economically feasible basis is difficult.

Loss of appetite is a constant symptom, and force feeding is necessary until the animal again begins to eat. That may be done by injections of dextrose (in a 10 percent solution) intravenously, intraperitoneally, or subcutaneously. Dextrose or molasses in water may be given by mouth, but, since a comatose sheep may be unable to swallow, it should be administered by a stomach tube. Injections must be given daily or, if treatment is by stomach tube, 2 or 3 times a day.

It is evident that the treatment of affected sheep involves considerable time, labor, and expense, and the results generally have not been encouraging. Most of the ewes that are down and comatose eventually die, but a reasonable proportion of those showing symptoms but still on their feet may be expected to recover. Probably the chief benefit of adding sugar to the ration derives from tiding over ewes that are starting to show symptoms.

Other treatments that have been used on an experimental basis include injections of adrenocorticotropic hormone (ACTH) and cortisone. The value of concentrated vitamins, injectable amino acids, and rumen inoculation is also being investigated. Those treatments, alone or in combination, show promise, but they are costly.

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White Muscle Disease in Lambs and Calves

O. H. Muth

WHITE MUSCLE disease was recognized in this country as a serious disease of lambs in the early 1920's. Some called it "stiff lamb disease." In later years it has been recognized also as a killer of calves. It is marked by a degeneration of muscle tissue.

It is known to occur in lambs and calves across the northern half of the United States. Probably it is most serious in some of the fertile irrigated districts of the western intermountain area. It varies considerably in extent from year to year, but no correlation has been noted between climatic differences and the extent of the trouble.

It may occur at any time of year. Although it does occur in calves whose dams have spent the previous 6 months on good irrigated pasture, most cases occur in the spring, when calves and lambs are most numerous.

Losses range from a few to a high percentage of the young in the flocks,
bands, and herds. Many farmers have stopped keeping breeding animals because of the losses, which in small herds in irrigated districts may reach 100 percent.

Animals from birth (or earlier) to several months of age and otherwise in good physical condition are affected. The finding of the young affected at birth suggests that the disease at times may be the cause of stillbirths.

Injuries are seen oftenest in lambs 3 or 4 weeks old and calves 4 to 6 weeks old. We have no evidence that any breed is more susceptible than others.

The injuries and losses result from the deterioration of muscles. An animal whose muscles are severely affected moves with difficulty and is generally weak. Some animals cannot rise and nurse, although their appetites remain good. The leg muscles may contract in time.

When the heart muscle is affected, breathing is labored, and death may occur a few hours after the symptoms appear. Calves that die in this way often discharge blood-tinged foam from the nostrils just before death.

A degeneration of the muscles of movement is most frequent in lambs. Heart injury is most common in calves. The heart and other muscles may be affected in either. This is not an inflammatory disease, such as might result from an infection, and rises in body temperature are not constant.

Many affected animals recover without change in feeding or management. A considerable number (especially among lambs) may show some injury, yet there may be no losses. The evaluation of a treatment therefore is apt to be difficult. Animals showing signs of injury of the skeletal muscles may die suddenly from heart failure due to heart injury. Those with heart injury usually live several hours after symptoms develop, but some die without outstanding symptoms.

The heart and other muscles, when they are examined after death, are seen to be affected in varying degree. Most of the damage in some animals will be in the heart. The damaged tissues appear as discolored areas from which the normal pigment is gone. They may be slightly lighter than normal or markedly bleached, with white streaks. The lining of the lamb’s heart may contain white patches suggestive of white enamel. In cases where damage impaired the function of the heart and the breathing is labored for some time before death, the lungs are congested with blood and fluid—a condition that a person not trained in pathology might mistake for a sudden and severe pneumonia.

Under a microscope, the damaged heart and muscle tissues are revealed to have lost their normal substance, which in some instances has been partly replaced with calcium salts.

Because other diseases occur in lambs and calves that have white muscle disease, diagnosis may be uncertain if the animals are not carefully examined by a qualified person. Symptoms of pneumonia and various types of arthritis might be confused with those of white muscle disease.

The cause of white muscle disease is not understood fully. As I said, it often occurs in lambs and calves in western irrigated districts, where dry climate, productive soil, and controlled irrigation favor the production of feed, especially excellent hay. The dams of the affected young are usually well managed.

The disease has been associated with the feeding of legume hays during the winter feeding period. Some cases occur after the dams of the young have been maintained on irrigated legume pastures for as long as 6 months. A similar, if not identical, disease occurs in New Zealand in lambs from ewes maintained on irrigated clover pastures.

The disease has occurred where alsike, ladino, sweetclover, native clovers, alfalfa, and vetch have formed a large part of the feed—with and without supplements of grain and molasses. It has occurred also where clover silage has been fed.

The disease is not generally a prob-
White muscle disease seems to have become more common following improved forage production. Many producers have related the disease to fertilization of their meadows, although the kinds of fertilizer have varied greatly. A similar or identical disease occurs in Sweden in years of heavy rainfall—which also stimulates plant growth.

When animals are maintained experimentally on rations deficient in vitamin E, which was first linked with reproductive functions in rats, muscle injury similar to that of naturally occurring white muscle disease occurs. In some parts of Scotland where beef cattle are normally maintained on a ration consisting largely of rutabagas, mangolds, fodder beets, and oat straw—feeds that are deficient in vitamin E—muscular degeneration occurs in the calves. The condition responds to supplements of vitamin E or disappears when the animals are turned on grass. The situation thus is different from the one found in the irrigated areas of this country where the dams are wintered on liberal rations of excellent feed and calves may get the ailment after the dams have been on pasture for a long time.

Experimental supplementation of ewes' rations with vitamin E has failed to prevent white muscle disease in their lambs. Vitamin E has also been used in lambs and calves as a preventive and treatment, but the results have not been wholly satisfactory.

Although investigations point to the cause of white muscle disease as being nutritional or metabolic in origin, the disease does not appear in the light of present information to be an uncomplicated vitamin E deficiency. Biochemical investigations of the problem were under way at several agricultural experiment stations in 1956.

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