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ENTEROTOXAEMIA ("PULPY KIDNEY") OF SHEEP

By C. R. TOOP, B.V.Sc.,
Chief Veterinary Surgeon



Fig. 1.—The outfit shown here is ideal for vaccinating against enterotoxaemia. It consists of a hypodermic syringe connected to a bottle containing the vaccine. The syringe is automatically re-charged after each injection.

INFECTIONOUS enterotoxaemia or the so-called "pulpy kidney" disease of sheep has been known to occur in Western Australia for many years and has been responsible for heavy though largely preventable losses. The earliest record of its occurrence dates back to 1915 when it appears to have been confined to the Great Southern districts and was known as "Beverley disease." With the cultivation and cropping of farm lands, it has since spread from that centre and, at the present time, the whole of the agricultural areas are involved.

The true nature of the disease was not determined until 1931 when it was shown by Bennetts to be caused by a microbe, commonly present in the soil, which multiplies in the small bowel of the sheep under certain conditions favourable to its growth and produces a potent toxin.

A vaccine prepared from cultures of this organism has proved effective for the prevention of the disease.

CONDITIONS UNDER WHICH THE DISEASE MAY OCCUR

Enterotoxaemia is mainly a disease of the flush season. The majority of outbreaks occur between April and October when green feed is abundant, and losses are heaviest during favourable seasons. Deaths may commence at any time during this period and may continue until the mortality is checked by the drying off of

the feed in late spring. Losses are usually intermittent, a few sheep being found dead at a time at irregular intervals, but in the aggregate the mortality may reach serious proportions and has been known, during the course of a season, to account for 30 per cent. of a flock.

This is the usual pattern but occasionally sudden and severe mortalities may be experienced when a large number of sheep may be lost within a short space of time.

LOSSES UNDER DRY FEED CONDITIONS

Losses from enterotoxaemia are not confined to the flush season; they may also occur during the dry period of the year. Mortalities associated with the consumption of flowering stinkwort during the late summer months when feed is scarce are not uncommon and outbreaks have often been reported amongst flocks running on peas or on wheat crops left unharvested because of storm damage or poorness of yield, and where a supplementary ration of concentrates has been fed.

THE CAUSE OF THE DISEASE

Enterotoxaemia is caused by the microbe *Clostridium welchii* Type D, which is frequently present in the soil of farmlands where it is able to persist for long periods by the formation of spores. Because of its wide distribution this organism must frequently be ingested by sheep while grazing, but it normally passes through the digestive tract without producing any ill effects. When, however, conditions are favourable to its growth, it multiplies in the small intestine producing a powerful toxin the absorption of which results in the death of the sheep usually within the space of a few hours. It has been shown that a condition which favours this abnormal upgrowth of the organism is a temporary stasis or sluggishness of the bowel but just how it is brought about does not yet appear to be clearly understood.

THE PREDISPOSING FACTORS

Fibre and Exercise.

Green feed is the principal predisposing factor. When young and luxuriant it is low in fibre and high in moisture content

and, in addition, the sheep is able to obtain its food requirements with a minimum of effort.

It is well known that both fibre and exercise stimulate bowel activity and when these factors are lacking or not fully operative, it would be reasonable to expect that a slowing down of the movements of the bowel predisposing to enterotoxaemia would occur. Similar effects could also be expected from the consumption of dry peas, fallen grain or a ration of concentrates, all of which are low in fibre content.

Injury to the Bowel Wall.

When stinkwort is consumed by sheep in the flowering and seeding stage the small barbed hairs of the pappus (developing fruit) penetrate and adhere to the bowel wall and are often so numerous in the small intestine as to form a feltwork covering a large area of the lining membrane. The damage inflicted by these hairs predisposes to enterotoxaemia and the outbreaks which occur in sheep having access to the plant in late summer are a sequel to their penetration.

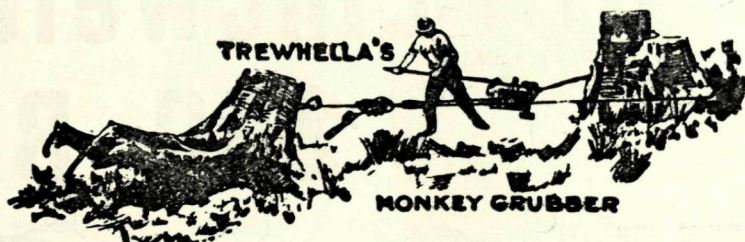
SYMPTOMS

Sheep of all breeds and of all ages are susceptible. Losses are usually distributed fairly evenly throughout the flock, but in some outbreaks the mortality may be largely confined to grown sheep while in others it occurs mainly in lambs.

In Western Australia lambs seldom become affected before the age of 10 to 12 weeks whereas in New Zealand and parts of Eastern Australia mortalities are heaviest in lambs during the first few weeks of life.

It has often been remarked that sheep in fat condition most frequently become affected and, while this may be so during the flush season, it is by no means always the case. Sheep dying of enterotoxaemia in late summer are often in store condition.

An outstanding feature of the disease is the suddenness of death and the symptoms are of such short duration that affected sheep are seldom noticed ailing. More often their carcasses are found in the paddocks highly distended with gas, discoloured and undergoing rapid decomposition.



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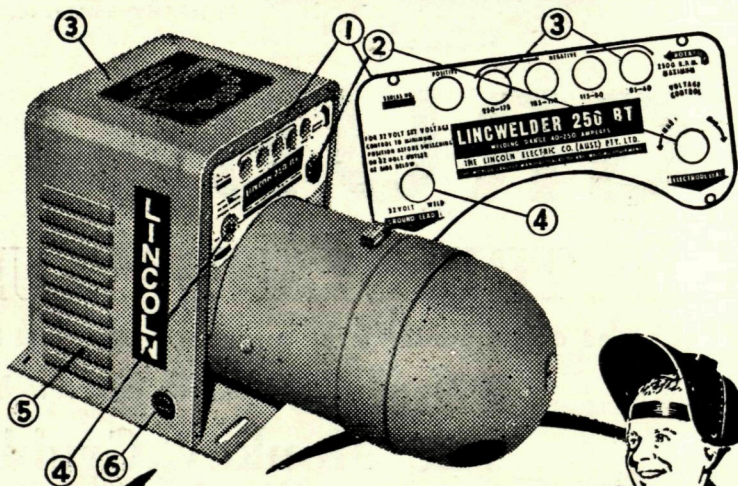
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Fig. 2.—If the injection is made on the side of the brisket behind the elbow any resulting carcass blemish can be readily trimmed off



When symptoms are observed the affected sheep will be noticed to lag behind the flock moving about with its nose to the ground and often chewing excitedly at sticks, stones and other small objects. Champing of the jaws is usual and there is sometimes a frothy discharge of saliva from the mouth. Scouring with the passage of liquid excretions which soil the breech may also occur, but this is by no means a constant symptom.

The gait is weak and unsteady and the animal may knuckle over at the fetlocks or go down on its knees. Soon it is unable to stand and lies on its side with the head turned in towards the flank. From this stage it rapidly sinks into a condition of coma and dies often without shifting its position in three to four hours of the first appearance of symptoms.

Occasionally in lambs, less often in grown sheep, there are convulsive symptoms. The animal, apparently in normal health, is suddenly seized by convulsions. It falls to the ground lying outstretched on its side with the head turned backwards making violent struggling movements until death supervenes—usually within two to three hours.

An atypical form of the disease has been described in which death is delayed for 18 to 24 hours and is preceded by aimless wandering, but this must be regarded as unusual.

POST-MORTEM APPEARANCES

A post-mortem examination carried out immediately after death will reveal few abnormalities. Slight changes, however, are usually present, the most constant of which are subendocardial haemorrhages which appear as rather large bluish blotches beneath the lining membrane of the inner surface of the heart. Similar, though much smaller haemorrhages may also be observed on the epicardium or outer surface of the heart and there is often an excess of fluid in the heart sac. The small intestine may be somewhat distended with gas and its contents reduced in quantity and the blood vessels which supply it may be congested and appear more prominent than usual.

Post-Mortem Decomposition of the Carcass—Pulpy Kidney.

Putrefaction commences immediately after death and thereafter proceeds at an extremely rapid rate. After lying a few

hours the carcass will usually have become greatly bloated with gas and the wool may be plucked from it without difficulty, while the inside surfaces of the thighs and forearms, which are bare of wool, may show a green or purplish discolouration. Upon opening the abdominal cavity the intestines will be found to be highly distended with gas and the extent of the putrefactive changes, which have already occurred in the internal organs, will be apparent. The kidneys in particular undergo very rapid decomposition and will be observed to have broken down to a soft pulpy mass of dark red tissue and to have lost their well-defined shape—hence the name “pulpy kidney” by which the disease is popularly described. It must be emphasised, however, that “pulpy kidney” is a post mortem change and that it is not observed in the fresh carcass.

DIAGNOSIS

When deaths are occurring suddenly and irregularly, particularly during the flush season, and this is followed by abnormally rapid bloating and decomposition of the carcass and a post mortem examination carried out a few hours after death has revealed the kidneys to have become characteristically pulpy and degenerated, a diagnosis of enterotoxaemia will, in most cases, be justified. But where it is desired to confirm the diagnosis a sample of the small bowel contents should be submitted for laboratory examination. Filtrates from the small bowel contents of a sheep affected by enterotoxaemia, because of their toxin content, are rapidly fatal when injected into laboratory animals, whereas similar material from healthy sheep or from sheep dying of other causes produces no ill effects.

Small bowel contents are of no value for diagnostic purposes unless obtained from a freshly dead sheep and should therefore be collected and despatched as soon after death as possible. The intestine should be tied off at its junction with the fourth stomach and again where it joins the caecum or blind gut and then freed of its attachments. By drawing it between the thumb and forefinger, the contents are forced to one end and may then be transferred to a wide mouthed

specimen bottle. A few drops of chloroform (0.5 cc. per 100 cc. of contents) should be added as a preservative and the specimen despatched to the laboratory without delay. Specimen bottles, together with preservative, may be obtained on application at the Animal Health and Nutrition Laboratories, Hollywood, Western Australia.

PREVENTION—INOCULATION WITH ENTEROTOXAEMIA VACCINE

Before the advent of vaccines, various expedients were employed to prevent mortality. It was a common practice to move the sheep from paddock to paddock at frequent intervals; to yard them periodically, or to transfer them to a bare paddock and these measures, by reducing the intake of food and providing the flock with exercise, often checked the losses. It was also possible, by moving the sheep to a scrub paddock, which provided a diet high in fibre content, to arrest the mortality completely.

Inoculation with enterotoxaemia vaccine, however, provides the only means of prevention which is both practical and effective. A sheep injected with this product will, after an interval of 10 to 14 days, develop an immunity which will enable it to resist the disease if subsequently exposed.

The use of an alum-precipitated, trypsinised vaccine of high potency is recommended and it should be given at the maximum dosage advised by the manufacturer. The immunity derived from a single injection is not of a high order and, in order to obtain a safe and satisfactory degree of protection, a second injection after an interval of not less than 30 days is necessary. The first of these injections is to be regarded as a sensitising dose; the second boosts the immunity to a much higher level which reaches its peak in a fortnight and remains effective for about six months.

It has been the custom in Western Australia to rely on a single injection and the results have generally been found satisfactory. Breakdowns have, however, occurred, and in favourable seasons in which serious mortalities may be anticipated a second inoculation after an interval of 30 days is

strongly recommended and this, under any circumstances, is the safest procedure to adopt.

When to Inoculate.

Since the greatest losses from enterotoxaemia occur during the winter and spring months, it is recommended that the inoculation of the flock be completed in April or May before the advent of the opening rains so that the sheep may enjoy the highest measure of protection during the period of greatest risk.

Where sheep are to be fattened on peas or turned on to storm-damaged wheat crops they should be immunised immediately beforehand and a similar precaution should be taken against the risk of mortality in the case of sheep running on flowering stinkwort in late summer.

Immunisation of Lambs.

Lambs should be inoculated at the time of marking and to ensure that a satisfactory level of immunity is established and maintained, a second injection should be given a month later. The vaccine should be used in the same dosage recommended for grown sheep and the injection should be made just beneath the skin on the side of the brisket behind the elbow where any blemish that may result may be readily trimmed from the dressed carcass. Care should be taken to avoid penetration of the under-lying muscles with the tip of the needle.

Young Lambs.

In those parts of Australia where heavy losses occur in lambs during the first few weeks of life, immunisation presents a special problem. Because of the very early age at which deaths occur and the interval which elapses prior to the development of a serviceable immunity, the use of enterotoxaemia vaccine is precluded and resort must be had to the immunisation of the young lamb through its mother. The ewe is inoculated with enterotoxaemia vaccine at the time of mating and a second injection is given 10 to 14 days before lambing. By this time the immunity conferred upon the ewe will have

reached its peak and it is transmitted to the lamb through the colostrum or first milk sucked. It is a passive immunity of short duration but it persists long enough to protect the lambs during the first four to six weeks of life.

Since lambs seldom become affected in this State before the age of about three months this practice appears to be unnecessary in Western Australia but, should unexpected mortalities occur in young lambs, losses may be stopped immediately with pulpy kidney antitoxin.

MEASURES NECESSARY TO CONTROL AN OUTBREAK

When an outbreak of enterotoxaemia occurs in a flock which has not been immunised, further deaths may be prevented by the injection of pulpy kidney antitoxin in a dosage of 5 cc. for young lambs and 10 cc. for older lambs and grown sheep. This product confers an immediate protection but it is of very short duration and lasts only for about two weeks but it may nevertheless tide the flock over a difficult period. The treatment of grown sheep with antitoxin is costly and amounts to about 4s. per head. If it is desired to prolong the immunity the sheep should be injected at the same time, but on the opposite side of the body with enterotoxaemia vaccine and a second injection of the vaccine should be given a month later. By this means an immunity which is both immediate and lasting will be obtained.

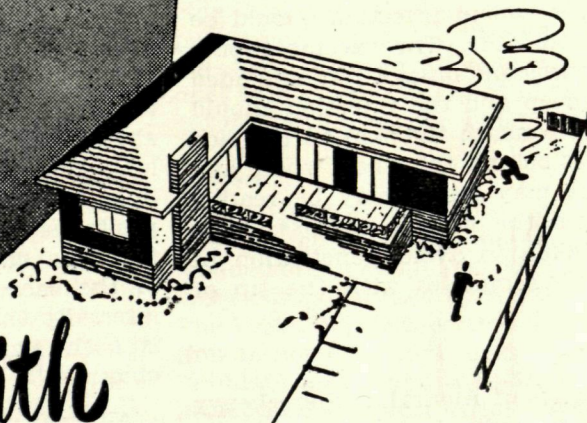
Alternatively—and this will be found much cheaper and perhaps a more practicable procedure—the flock may be inoculated with enterotoxaemia vaccine and then transferred to a bare paddock or to scrub country for 14 days to enable an immunity to become established and the injection should be repeated after 30 days.

Where a breakdown of immunity has occurred in a previously inoculated flock, the sheep should be re-vaccinated without delay. This will boost the immunity to an effective level and may be relied upon to prevent further deaths.



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