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Grain feeding, or grazing lush young pastures or cereal crops, may predispose sheep to . . .

ENTEROTOXAEMIA ("PULPY KIDNEY" DISEASE)

By J. CRAIG, M.R.C.V.S., Senior Veterinary Surgeon

ENTEROTOXAEMIA, or pulpy kidney disease, is found in all areas of the world where sheep are raised and has occurred in Western Australia for many years, particularly in the great southern districts. At one time it was known as "Beverley sheep disease." The infectious nature of enterotoxaemia was first described by Bennets in 1932.

Enterotoxaemia is an acute toxaemia of the sheep which is caused by the multiplication of the organism Clostridium welchii type D within the intestines of the animal. As a consequence potent toxins are liberated and these quickly kill the animal.

Predisposing factors
The organism Clostridium welchii is a normal inhabitant of the alimentary tract but is probably present only in small numbers. It is unable to live in the ground for very long.

Under certain conditions, the organisms have the ability to multiply profusely in the intestines and in such circumstances produce lethal quantities of toxin.

The husbandry factors which predispose to outbreaks of disease include grazing lush, rapidly-growing pastures or young cereal crops, and grain feeding. Lambs whose mothers are good milk producers are particularly vulnerable.

The highest incidence of disease occurs in young animals, although older sheep are also susceptible.

It would appear that over-eating on a starch diet such as grain, or a sudden change of diet from roughage to grain, is the most important factor assisting toxin production.

A slowing of the alimentary tract movement is also thought to assist toxin absorption. Heavy infestation with tape-worms as well as damage to the bowel wall from the barbed hairs of flowering and seeding stinkwort (Inula graveolens), are also known to aid absorption of the toxin.

Symptoms
An outstanding feature of the disease is the suddenness of death; the symptoms are so transient in nature that affected sheep are seldom noticed to be sick. More often than not the carcasses are found in the paddocks distended with gas, discoloured and undergoing rapid decomposition.

VACCINATION IS ESSENTIAL
Vaccination is the only practical means of protecting sheep against enterotoxaemia. One injection should be given to lambs at marking, followed by a second from two to six weeks later. An annual booster maintains immunity.

Young lambs will have early immunity if the ewes are given a booster injection in late pregnancy.

In some cases an additional vaccination is warranted just before sheep are put into risky situations, such as grazing storm-damaged wheat crops or when they have access to flowering stinkwort in later summer.
STAGES IN THE DEATH OF A HOGGET FROM ENTEROTOXAEMIA

TOP.—The animal appears dazed and unaware of its surroundings.

BELOW.—About an hour later the animal has collapsed and is unable to stand when lifted to its feet. It lies with its head turned towards its flank.

Death can be expected soon afterwards.

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 excreta 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 towards the flank. From this stage the sheep rapidly sinks into a comatose state and dies within three to four hours of the first appearance of symptoms.

Lambs occasionally show convulsive symptoms. The affected lamb falls to the ground, lying outstretched on its side with the head turned backwards and making violent struggling movements until death supervenes.

An atypical form of the disease in which death is delayed for 18 to 24 hours and is preceded by aimless wandering, also occurs at times.

Post-mortem lesions

The carcass is usually in good condition. Few abnormalities will be seen when a post-mortem examination is carried out immediately after death. The most constant of these is the presence of blotchy haemorrhages on the inner surface of the heart as well as pin-point haemorrhages on the outer heart surface.

There is usually an excess of fluid within the heart sac. Patchy congestion of the lining of the stomach and intestines is also seen.

Decomposition proceeds rapidly after death and in a matter of a few hours the carcass becomes greatly distended with gas, the inside surfaces of the thighs and forelegs show a purplish discoloration and the wool is easily plucked.
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The liver is dark and congested, and the kidneys in particular undergo very rapid decomposition and appear as a soft pulpy mass of dark red tissue; hence comes the term "pulpy kidney" by which the disease is popularly described. It must be emphasised however that "pulpy kidney" is a post-mortem change and is not observed in the fresh carcass.

**Diagnosis**

When deaths have occurred suddenly, particularly during the green feed season, and this is associated with rapid decomposition of the carcass, the presence of cardiac haemorrhages and the characteristic pulpy and degenerated kidneys at post-mortem examination soon after death, a diagnosis of enterotoxaemia will usually be justified.

Veterinarians may seek to confirm their field diagnosis by assessing the presence of sugar in the urine, since a high blood sugar is typically encountered in the terminal stages of enterotoxaemia. Confirmation can also be made by taking smears of small intestinal contents for subsequent microscopic examination, or submitting samples of small bowel contents 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 must therefore be collected and despatched, suitably preserved with a few drops of chloroform, as soon after death as possible.

**Prevention**

The two major precautions against enterotoxaemia are reduction of feed intake and vaccination.

Before vaccination became available various expedients were used in an endeavour to prevent losses and it was common practice to move sheep from paddock to paddock at frequent intervals, to yard them periodically or to transfer them to a bare paddock. These measures often checked losses by reducing the intake of feed and providing the flock with exercise. It was also possible to achieve the same effect by moving sheep to a scrub paddock which provided a diet high in fibre content.

Vaccination, however, offers the only practical means of preventing enterotoxaemia. Approved vaccines available are of two types. One is an alum-precipitated trypsinised toxoid and the other an aluminium-treated purified toxoid. These vaccines must be properly stored when not in use and the manufacturer's directions carefully followed as to dosage during a vaccination programme.

Vaccination is generally highly effective, although individual sheep may, for various reasons, fail to maintain their immunity. This is most likely to happen where only a single injection of vaccine has been given.

The important point to realise is that when sheep are vaccinated against enterotoxaemia for the first time in their lives they must receive two injections. The first of these is merely a sensitising dose and only gives a transient protection. The second raises the immunity to a much higher level, which generally remains effective for about six months. The interval between the first and second injections may vary from two to six weeks without affecting the results and this allows the flock owner some latitude in arranging the vaccination programme.

Provided sheep have been given this sound basic immunity, the level of immunity following on a booster dose given 12 months after the initial injections can be expected to afford protection for a further period of one year.

The recommended procedure entails vaccination of lambs at marking time, and a second injection given one month later. Thereafter annual booster injections should be given during late autumn to ensure that the flock has a durable immunity during the subsequent green feed season.

Enterotoxaemia can also occur in lambs at a very young age and the problem may arise of having to protect them during the first few weeks of life before marking. It has been shown, however, that lambs born from ewes which have a sound basic
immunity and are given a booster injection of vaccine in late pregnancy, receive a temporary immunity via the colostrum or first milk. This immunity lasts for several weeks and is sufficiently durable to protect lambs until they can be given their first injection of toxoid at marking time.

There are a number of occasions when apparently correctly immunised sheep are not fully protected. For example sheep may be especially at risk when they are being fattened on peas or turned onto storm-damaged wheat crops, and under these circumstances an additional vaccination would be warranted immediately beforehand. A similar precaution would be worth while in sheep running on flowering stintwort in late summer.

**Control**

When an outbreak of enterotoxaemia occurs in a flock which has not been previously immunised, further deaths may be prevented by the injection of enterotoxaemia anti-serum (anti-toxin). This product, although more costly in comparison to toxoid, gives immediate but short-lasting protection, the duration of immunity only persisting for about two weeks. If it is desired to prolong the immunity the sheep should be injected at the same time on an alternative body site with enterotoxaemia toxoid and a second injection of toxoid given one month later. By this means an immunity which is both immediate and lasting will be obtained.

As an alternative procedure, probably more economic and practical, the flock may be vaccinated with enterotoxaemia toxoid and then transferred to a bare paddock or to scrub country for a period of two weeks, during which time an immunity will become established; this injection should be repeated after a period of one month.

When a breakdown of immunity has occurred in a previously inoculated flock the sheep should be re-vaccinated immediately. This will boost the immunity to an effective level and can be relied upon to prevent the occurrence of further deaths.
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