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Polioencephalomalacia — an increasing problem with sheep in the Great Southern

By D.P. McKenzie and P. Steele*

Polioencephalomalacia (P.E.M.) or cerebrocortical necrosis (C.C.N.) is an increasingly serious disease of sheep in the Great Southern. It affects the nervous system, and is caused by a deficiency of the vitamin thiamin (Vitamin B1). An alternative name is cerebrocortical necrosis.

Normally the microbes of the rumen supply thiamin, but in animals affected by P.E.M., thiamin is rapidly destroyed by abnormally high levels of the enzyme thiaminase. The disease is not caused by an infectious agent, and cannot be transmitted from one animal to another.

Occurrence

P.E.M. occurs in adult sheep grazing pasture under low stocking rates, or more commonly during prolonged supplementary grain feeding on stubbles. Stud flocks are therefore especially susceptible. Outbreaks on pasture occur mainly in high rainfall areas of the southwest, in September and October with fewer cases extending to the end of April.

In grain fed sheep, weaners are mainly affected early in the dry season, whereas ewes are mainly affected towards the end of pregnancy — in late autumn or early winter.

During drought years, P.E.M. is likely to affect ewes fed grain for long periods on bare paddocks. It has been encountered in sheep being fed oats, wheat, barley and occasionally on lupin seed and clover burs.

Although P.E.M. may appear suddenly, it is commonly seen six to eight weeks after a change in feeding. If not treated, the sudden outbreak may be protracted.

In affected mobs, the incidence may vary from 1 to 25 per cent. It is rare in sheep in poor condition.

The disease occurs elsewhere in the world, and cattle and goats are also affected. However, the incidence in grazing sheep is seldom high in comparison with recent outbreaks in Western Australia. In New South Wales outbreaks occur in sheep grazing the fern Nardoo, so when this fern appears in pasture, it provides a warning of stock losses. Bracken fern has a similar effect in laboratory feeding experiments.

However in Western Australia, the disease does not seem to be related to pasture species, as feed samples taken at outbreak sites have negligible effect on the breakdown of thiamin.

Signs of P.E.M.

When the animals are upright, the main signs of P.E.M. are blindness, "star gazing", circling and head pressing due to damage of the cerebral cortex.

Once the animal goes down, it may show mild tetany, intermittent convulsions and grinding of the teeth. The rigidity of the affected animal may progress to a flaccid state, but death may occur first.

In spring, sheep showing these symptoms may live for up to five days but in heat wave conditions they usually last only a few hours.

With a post mortem examination, it may be possible to see depressed areas on both sides of the cerebral cortex, and in severely affected brains, the whole cortex may be yellow. Usually cerebro-spinal fluid builds up, and pressure effects may be visible on the cerebral cortex and the cerebellum.

Usually the sheep are in very good condition and breakdown of fat will be related to the length of illness. Under Western Australian conditions, the breakdown of thiamin which causes a deficiency is apparently due to micro-organisms not normally abundant in the rumen. In other places, this breakdown has also been caused by certain types of plant material in the rumen.

The change in feeding which initiates P.E.M. in Western Australian sheep, is believed to favour the development of organisms which break down thiamin. The presence of these organisms has been confirmed in samples from affected sheep.

Treatment

When the condition is first recognisable, it can be successfully treated with 200 mg of thiamin injected intravenously or subcutaneously. This will stop further brain damage, although it will not repair damaged tissue, so early treatment is essential. The subcutaneous injection also will provide up to six weeks protection for sheep at risk.

The thiamin used may be either injectable pharmaceutical grade or a solution prepared from the thiamin powder before injection. A convenient concentration is 200 mg per ml.

Due to the sporadic nature of P.E.M., it is unnecessary to treat all mobs at first appearance. Most outbreaks occur on a high energy ration, and a change to a lower energy ration will commonly halt an outbreak.

Further work

Because of the unique nature of P.E.M. in Western Australian sheep, attempts are underway to define the changes in the biochemistry and physiology of the rumen which cause the thiamin deficiency. Better knowledge of the biochemistry may help to define paddock conditions which start an outbreak.

A field experiment is also underway to evaluate methods of preventing P.E.M. in grain-supplemented sheep. Possible methods include the normal thiamin subcutaneous injection, intramuscular injection of a modified thiamin compound to prolong the effect of thiamin treatment, and a sustained release bolus to modify the fermentation pattern of the rumen in grazing animals.

Outbreaks of the disease are also being studied to relate it more specifically to feeding and management.

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