

## Metabolic diseases

### **Hypocalcaemia (Parturient Paresis)**

Parturient paresis is caused by a decrease in calcium intake under conditions of increased calcium requirements, usually during late gestation. This results in a low serum calcium concentration, particularly in animals pregnant with multiple fetuses. Parturient paresis can occur at any time from 6 week before to 10 week after parturition. Low blood calcium concentration usually 3rd to 4th day after lambing → mobilization of stored calcium can be inadequate to meet the animal's needs, especially, in older ewes. Some cases are complicated by concurrent pregnancy toxemia.

ETIOLOGY: Reduced feed intake, Reduced intestinal motility, Vitamin D deficiency, which occurs in housed ruminants during winter months, also depresses calcium absorption from the GI tract. Low content of phosphorus in the ration, Incorrect Ca: P ratio.

CLINICAL SIGNS: the most commonly signs are stiff gait, ataxia, salivation, constipation, and depressed rumen motility, progressing to hyposensitivity, bloat, recumbency, loss of anal reflex and, if untreated, death. Tachycardia may be present. Often when recumbent, ewes are in a sternal frog-lying position, with the hindlegs extended behind.

DIAGNOSIS: based on the history and clinical signs, can be confirmed by testing serum calcium levels before treatment. Urine ketone or serum  $\beta$ -hydroxybutyrate levels should always be evaluated at the same time. The pH of urine  $>8.0$  indication of alkalization.

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### **Pregnancy Toxemia in Ewes/ Pregnancy ketosis**

Pregnancy toxemia affects ewes during late gestation and is characterized by partial anorexia and depression, often with neurologic signs, progressing to recumbency and death. Most cases develop 1–3 week before parturition.

The primary predisposing cause of pregnancy toxemia is inadequate nutrition during late gestation, usually because of insufficient energy density of the ration and decreased rumen capacity as a result of fetal growth.

CLINICAL SIGNS: Animals spend more time lying and more frequent bouts of lying than their healthy herdmates. As the disease advances, signs of listlessness, aimless walking, muscle twitching or fine muscle tremors, opisthotonos, and grinding of the teeth. This progresses to blindness, ataxia, and finally sternal recumbency, coma, and death.

PREVENTION-MANAGEMENT: Body score condition should be managed in late lactation, provide suitable-balanced rations 4-6 weeks before gestation, adequate feeder space for pregnant animals.

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### **RUMINAL ACIDOSIS**

Ruminal acidosis is caused by ingestion of diets high in rapidly fermentable carbohydrates and/or deficient in physically active fiber, reduced saliva production, high concentration of lactic acid → depression of the ruminal pH, microflora disorder.

CLINICAL SIGNS: Anorexia, depression, bloating, diarrhea, cases of lameness

PREVENTION: Balanced rations, use of buffers (control of ruminal pH), providing adequate fiber intakes, gradual changes in the ration (allowing the rumen to adapt to high-grain diets, as well as limiting intake of readily fermentable carbohydrates).

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### **BLOAT**

The result of an excessive accumulation of gas in the rumen. Bloat is generally associated with cows grazing pastures with a high legume content (clover or lucerne) in spring and autumn. Occasionally young grasses can cause bloat if they contain large amounts of soluble protein. Intensive system of beef fattening, which contains high amounts of concentrates, low amounts of forages.

### CLINICAL SIGNS:

no longer grazing, a reluctance to move, distended left abdomen, appear distressed, bellowing, eyes bulging, strain to urinate and defaecate, rapid breathing — mouth may be open with tongue protruding, staggering.

Death can occur within 15 minutes after the development of bloat

Gaseous bloat is usually seen in one or two animals. Frothy bloat can affect up to 25% of cases. In some cases sudden death may be the first sign seen by the stockman.

PREVENTION: Balanced rations, use of buffers (control of ruminal pH), avoid using high-risk pastures at high-risk times, providing adequate fiber intakes at risk periods, gradual changes in the ration.