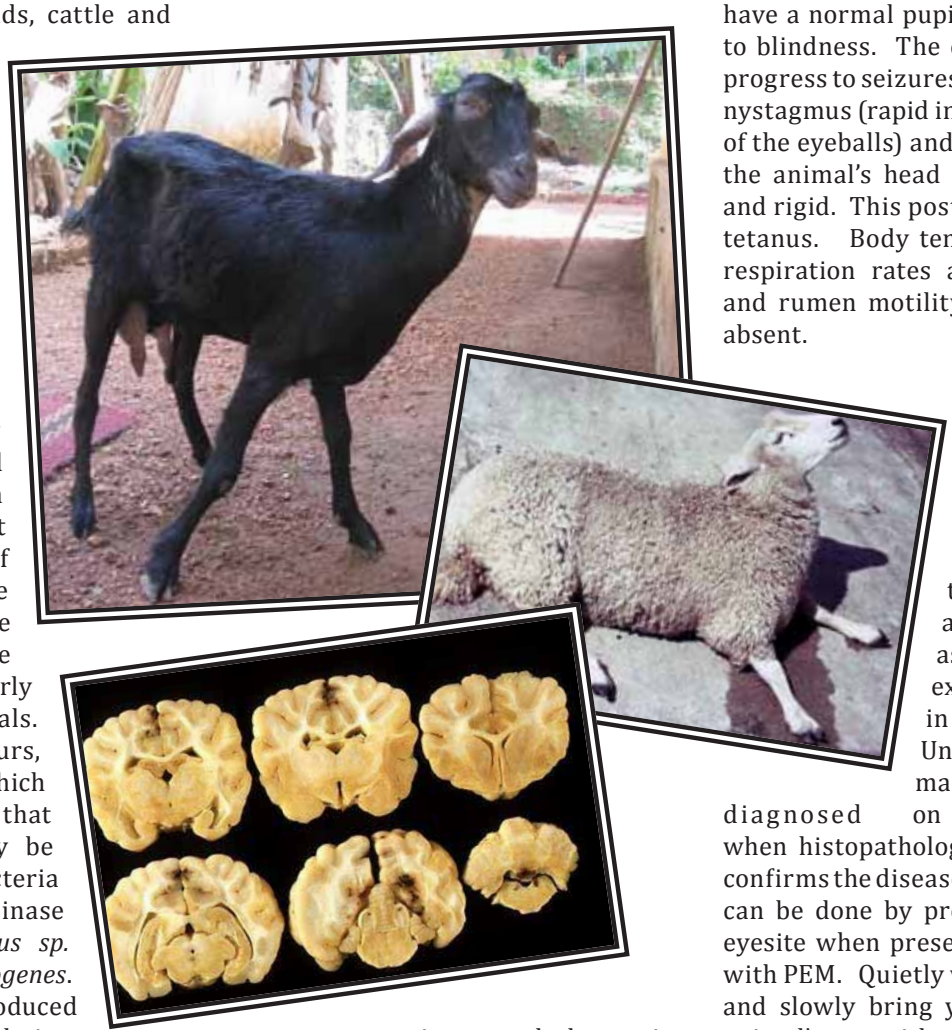
POLIOENCEPHALOMALACIA

A NUTRITIONAL DISEASE WITH MAJOR NEUROLOGICAL COMPLICATIONS

by Dr. Beth Johnson

Commonly referred to as "polio" in the ruminant world, polioencephalomalacia (PEM) is a metabolic disease resulting in neuromuscular clinical signs as a result of a lack of thiamine, Vitamin B1. Sheep, goats, camelids, cattle and other ruminants are all susceptible to this condition at any age but seen more often in young animals less than one year of age. Polio is usually found in ruminants on a high concentrate/lush pasture ration. Thiamine molecules are normally produced in the "healthy" rumen by naturally occurring bacteria and protozoa. Any change in this rumen environment may affect production of thiamine, increase the degradation of thiamine or prevent thiamine from functioning properly in ruminant animals. When this change occurs, thiaminase enzymes, which are enzymatic proteins that catabolize thiamine, may be produced by several bacteria within the rumen. Thiaminase I is produced by *Bacillus sp.* and *Clostridium sporogenes*. Thiaminase II is produced by *Bacillus aneurinolyticus*. Thiamine is a cofactor in the metabolism of carbohydrates (CHO's) and when it is not available there is a depletion of carbohydrates which causes alterations of the mechanism of action of the nervous system and neuronal death in the brain. A deficiency of CHO supply to nerve cells will cause central nervous system disorders, polioencephalomalacia and death if left untreated.

Polio is often seen when there is a sudden diet or environmental change, but can also be seen in ruminants that have consumed poisonous plants, animals which have been treated with antiprotozoal drugs, i.e. Amprolium, been recently dewormed, animals grazing lush highly fertilized pastures and/or animals exposed to high levels of sulfur.



CLINICAL SIGNS:

Clinical signs seen in ruminants affected with polio are a result of damage to the cerebral cortex of the brain. In the very early stages, a producer may notice that the animal is dull, lethargic, depressed, grinding its teeth and exhibiting a base wide stance or reluctance to move. This is due to gastrointestinal upset and bilateral blindness. The affected animal does not have a normal pupillary light reflex due to blindness. The clinical signs quickly progress to seizures, lateral recumbency, nystagmus (rapid involuntary movement of the eyeballs) and opisthotonus, where the animal's head is thrown backward and rigid. This posture is also seen with tetanus. Body temperature, pulse and respiration rates are usually elevated and rumen motility may be present or absent.

DIAGNOSIS:

As with any other disease, a thorough physical examination including body temperature and neurological assessment is extremely important in the diagnosis of PEM. Unfortunately, too many of these cases are diagnosed on the autopsy table when histopathology of cerebral tissue confirms the disease. A simple procedure can be done by producers to check for eyesite when presented with an animal with PEM. Quietly walk up to the animal and slowly bring your hand up to the animal's eye without touching the head. If the animal does not blink on either side then there is a high probability of PEM.

Some diseases with similar symptoms that should be ruled out are listeriosis, enterotoxemia, pregnancy toxemia, rabies, tetanus, or poisoning by chemicals or toxic plants.

TREATMENT AND PREVENTION:

As with many diseases, the sooner treatment intervention occurs the better prognosis for complete recovery. Response to treatment is highly correlated with the extent of brain lesions. If treatment is initiated in the early stages of thiamine deficiency, i.e. animal is still ambulatory with or without blindness, then response to treatment occurs relatively quickly. Thiamine is administered IV or IM at the dose of 4.5-10mg/lb body weight. I highly recommend that only veterinarians administer thiamine IV since if administered improperly can result in death. An IM dose can be repeated every 3-6 hours as needed for treatment. If treatment is delayed, a full recovery may not occur so it is imperative that treatment be initiated as soon as signs develop. In non-pregnant animals, a dose of dexamethasone may be beneficial to reduce the edema/inflammation occurring in the brain. In pregnant animals, use nonsteroidal anti-inflammatory medicines. Also dextrose should be administered orally or subcutaneously to provide carbohydrates to the brain tissue as soon as possible. A good broad spectrum antibiotic should also be administered to prevent septicemia.

One should also evaluate the diet of the ruminant animals affected. Consider increasing the amount of roughage the animal is receiving and provide a good quality pasture or hay. Evaluate the amount of sulfur in the diet, i.e. does the water source contain high sulfur levels. What about the forages being consumed-are they high in sulfur content? Also be sure to monitor ruminants that are being treated for coccidiosis and recently dewormed. If necessary a diet with levels of thiamine at 1.5-4.5 mg/kg of feed can be fed to animals at risk of developing PEM.

Hopefully you will never have to experience this metabolic disease in your herd. If there was one drug to have on hand at all times, it would be a bottle of thiamine. You will never know when you will need it, but one thing is for certain in livestock production, you sure want to be prepared. Always consult with your veterinarian for treatment, control and prevention!

References:

Polioencephalomalacia (Goat Polio), Alabama Cooperative Extension Service bulletin #UNP-65, Alabama A&M and Auburn Universities. http://www.aces.edu/pubs/docs/U/UNP-0065/UNP-0065.pdf

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