As winter approaches and producers start to increase grain and forage quality decreases, I typically see more cases of polioencephalomalacia in developing ruminants. Whether as a refresher on knowledge you already have or adding to your growing level of wisdom, hopefully you find this information useful.

Polioencephalomalacia, which I'll simply refer to as “polio” for the rest of this article, is the scientific term for damage caused to the brain due to a metabolic disorder. You may be familiar with the term “thiamine deficiency” as well. This is essentially what causes the signs associated with polio because thiamine is responsible for carbohydrate metabolism, which ultimately provides glucose for the brain and other parts of the body. Goats are particularly reliant on one of the thiamine-dependent methods of glucose metabolism. The brain is usually the first noticeable organ to suffer from a shortage of glucose.

Signs of polio vary. Animals can show a slow progression that starts with being off-feed or a mild diarrhea. Because of the brain's reliance on thiamine, the central nervous system is affected and symptoms such as staggering, muscle tremors, apparent blindness and/or other eye abnormalities, and odd-posturing can be seen. In severe cases, animals will be lying on their sides with their heads pulled back and rigid limbs. Seizures, coma and eventually death in 1-2 days follow if treatment is not given. Because these signs can be seen with other diseases such as rabies, listeriosis, over-eating, tetanus, pregnancy toxemia, a host of toxicities, and others, it is difficult to observe an animal and say definitively that it does or does not have polio. Lab diagnosis is possible (speak with your veterinarian), but since treatment should be initiated immediately, response to treatment often is the best means of diagnosing polio.

Healthy ruminants do not typically need any thiamine added to their diet. This is because the rumen microbes make enough thiamine (also known as Vitamin B1) for the body to function properly. Problems most typically arise when something the animal eats causes the rumen pH to drop. Sudden ration changes, over-feeding concentrates, underfeeding forages or using poor quality forages, moldy feed, stress such as weaning, feed high in molasses (often horse feeds), and certain plants can do this. As the rumen pH drops, the population of microbes in the rumen is altered, essentially leading to a loss in the “good bugs” and an overgrowth of the “bad bugs.” The helpful microbes are those that make thiamine. The harmful microbes produce byproducts that degrade thiamine before it can be used by the body. The lack of thiamine leads to a glucose shortage in the brain, eventually causing death of certain brain cells. The speed and severity of the signs depends on how quickly brain cell metabolism stops and cells begin to die.
It has been well-documented that amprolium (Corid®) used to treat coccidiosis can lead to an increase in polio cases if it is not dosed properly. Amprolium does not necessarily drop the rumen pH, but it can prevent thiamine from being used by the body. Also, oral antibiotics are believed to increase the likelihood of an animal getting polio. It is thought that the mechanism is a direct alteration in the population of bugs in the rumen (the antibiotics don’t discriminate between killing good bugs or bad bugs) leading to decreased thiamine production. Finally, high levels of sulfate in the diet cause polio-like symptoms and polio lesions in the brain. Mis-feeding, high-sulfur water sources, and some urinary acidifiers (NOT ammonium chloride) can be the culprits of high-sulfate diets. Testing the feed or water is always a good idea when polio is seen in many animals in a short period of time.

Thiamine is the only treatment, and it must be prompt. The recommended dose for thiamine is 10 mg/kg of body weight. The 200 mg/ml thiamine that I carry in my truck is quite common, and this dose would equate to ½ cc per 20 pounds of body weight, or 2.5cc per 100 pounds. If possible, the first dose should be given IV. Follow-up doses can be given every 6 to 12 hours for a day or two, and may be given IM or SQ. If you can’t get thiamine, you can substitute a higher dose of Vitamin B complex (make sure it’s not Vitamin B12). The high level Vitamin B complex has 100 mg/ml, so the dose would be 1cc per 20 pounds or 5cc per 100 pounds of body weight. Dexamethasone may help improve signs by reducing inflammation in the brain, and can be given in the same syringe with thiamine (and the same volume of thiamine if using the 2 mg/ml dexamethasone). If the thiamine is given IV, response can be seen in a few hours. Most of the symptoms can be completely reversed, although blindness can be permanent.

Prevention should be focused on management, not dietary supplements. There is some literature that mentions thiamine before stressful events or in herds where polio is a problem. Dietary supplements are available, but if management does not change, the causes of the pH drop are not addressed, and there will simply be more thiamine for the bad bugs to degrade. Supplements may slow the progression or help some of the subclinical animals that have poor appetites and diarrhea, but they aren’t meant to cure sick animals or completely prevent this disorder.

The bottom line is that polio is not an infectious disease. Due to the array of causes, it is almost impossible to completely prevent all polio cases. Good management will limit the number of times you have to deal with it, and prompt treatment will greatly increase your odds of success.