Neurologic disease is not an uncommon complaint in small ruminants, particularly goats. There are several key diseases that can result in signs of neurologic disease. Unfortunately, definitive diagnoses usually require samples from a post-mortem examination, however a thorough physical examination, history and a few diagnostic tests that can be performed on a farm setting can often times at least reorder your list of differential diagnoses and provide substantial support for one differential over the others. Listeriosis, is caused invasion of *Listeria monocytogenes*, a small motile gram-positive bacteria, into the Central Nervous System (CNS). Small ruminants appear to be more susceptible than cattle and often times have a history of eating silage. Clinical signs of disease occur when the bacteria travels to the brain via the trigeminal nerve. A key finding on neurologic evaluation will usually result in unilateral facial nerve paralysis, often times noted by only one-nostril flaring with respirations or a drooped ear. Additionally, these animals are often ataxic and may be circling, recumbent or head pressing. Performing a cerebral spinal tap to collect cerebral spinal fluid can be helpful in forming a tentative diagnosis. Cerebral fluid is usually inflammatory in nature, although the causative organism is rarely seen. The prognosis is generally poor, however prompt and aggressive treatment can be rewarding in some cases. Treatment usually involves antimicrobial therapy (Penicillin G 44,000 iu/kg once daily for 7-14 days), supportive care (fluid therapy if they are unable to drink and nursing care for those that are recumbent) and thiamine to help prevent the development of thiamine deficiency secondary to rumen dysfunction during their course of illness. Generally speaking those that are recumbent at the time of presentation carry a grave prognosis. Thiamine deficiency (polioencephalomalacia) is another common cause of neurologic disease. Thiamine deficiency in ruminants is usually secondary to rumen dysfunction and not a lack of intake. Protozoan in the rumen are responsible for producing and when there is a disruption in their health, thiamine deficiency can result. Clinically, these animals often have cortical blindness and may also experience seizures, coma and acute death. Treatment includes supplementation with thiamine (10 mg/kg SQ initially 2-3x/day then once daily or in intravenous fluids) and supportive care. With treatment some animals will regain sight but all may not return to normal. Additionally, addressing the underlying cause of the rumen dysfunction is crucial in treatment and transfaunation with healthy protozoan may be warranted if microscopic evaluation of a sample of rumen fluid reveals decreased protozoal activity. Meningeal Worms (*Paralaphostrongylus tenius*) result in aberrant migration to the central nervous system in goats. Clinical signs usually involve progressive
weakness and ataxia with the hind limbs more commonly affected than the forelimbs, but ultimately resulting in recumbency. Recumbency carries a very poor prognosis, however treatment at earlier stages involves anti-helminthic administration (often ivermectin and fenbendazole at maximum dosages), supportive care and occasionally steroid administration has been helpful in reducing inflammation while treatment is being instituted. Regular and appropriate deworming may reduce the risk of contracting meningeal worm, however regular deworming can lead to further problems with anti-helminthic resistance with other gastrointestinal parasites.

Trauma to the spinal cord can also result in damage to the spinal cord and/or musculoskeletal trauma that can present similarly to neurologic disease. Treatment for spinal cord trauma involves supportive care and anti-inflammatory treatment (corticosteroids and/or NSAIDs). Prognosis may be more favorable if initial response to treatment is positive. Unfortunately, treatment of neurologic diseases of the small ruminant are often unrewarding, particularly when the patient is presented later in the course of disease. Even “successful” treatment may not result in complete resolution of neurologic deficiencies.