

EVALUATION OF THE NEUROLOGICAL RUMINANT

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Neurological exam

The goals of physical and neurological examination in animals, in which neurological disease is suspected, should focus on differentiation of primary and secondary neurological disease as well as the localization of suspected lesions within the peripheral or central nervous systems (CNS or PNS). A detailed description of examination procedures has been published previously (Constable, 2004).

In farm animal species, neurological signs most commonly result from an underlying metabolic disease, such as pregnancy toxemia or hypocalcemia. In neonatal patients, sepsis or hypoglycemia are common causes of clinical signs associated with the nervous system. The goal of physical examination in conjunction with blood work should therefore be the exclusion of these common diseases from the list of differential diagnoses. Once primary neurologic disease has been determined to be the most likely cause of clinical signs, localization of lesions to one of four functionally different anatomical regions of the nervous system is imperative. These four neuroanatomical regions are: (1) cerebrum, (2) cerebellum, (3) brainstem and cranial nerves, and (4) spinal cord and peripheral nerves. Keeping in mind the three main functions of the nervous system in farm animals (mental state, sensory perceptivity, posture and gait), the localization of lesions by physical and detailed neurological examination is possible (see Table 1).

Clinical signs associated with cerebral disease are mainly those of altered mentation and may include somnolence, depression, stupor, coma, or nervousness and excitation. Central blindness is also common and may result in slow forward locomotion and, at least in a new environment, walking into objects. Many metabolic diseases, especially alterations of the glucose, electrolyte, and acid-base homeostasis may lead to impairment of cerebral function. Evaluation of serum chemistry is helpful in ruling out underlying causes of cerebral disease. Primary causes of cerebral disease in ruminants include polioencephalomalacia, bacterial meningitis, infection with lentiviruses (CAE and Maedi/Visna), herpesvirus encephalitis, rabies, pseudorabies, and plant toxicoses.

Cerebellar disease causes abnormalities of posture and gait with clinical signs of upper motor neuron disease. Typical clinical signs include ataxia, dysmetria, and truncal sway. The presence of weakness should be assessed, which can be performed by pushing on the rump of the animal at a walk or in the standing patient. While normal strength is present in animals with cerebellar disease, signs of weakness may result from vestibular, spinal, or peripheral nerve ataxia, and from muscle disease. Causes of cerebellar disease include transplacental infections with pestiviruses (e.g. cerebellar hypoplasia) or grass staggers.

Lesions associated with brainstem and cranial nerves may result in a variety of clinical signs, depending on the region of the brain stem affected and dysfunctional cranial nerves. Mentation is commonly abnormal in patients with listeriosis and depression and stupor may be observed. Posture and gait may also be affected when the function of cranial nerve (CN) VIII (vestibulocochlear) is impaired. Cranial nerves are assessed as functional units. Lesions of CN II (peripheral blindness) and CN III, IV, V and VII may be assessed with pupillary light reflex (presence in blind animals suggests central blindness), menace response, palpebral reflex, and corneal reflex evaluations. In addition, lesions affecting CN VII result in drooping of ears, lips, and eyelids, and facial deviation. Lesions of CN VIII result in head tilt, nystagmus, and circling to the affected site. Nystagmus may also result from cerebellar disease but will be present on the opposite site of the lesion. Dysphagia and decreased tongue tone are seen in patients with impaired CN IX, X, and XII. In ruminants, listeriosis is the most common cause of brain stem and cranial nerve deficits, but otitis media/ interna, pituitary abscess syndrome, and aberrant parasite migration (mostly spinal cord defects) should also be considered.

Spinal cord and peripheral nerve lesions can result in unilateral or bilateral deficits. When gait and postural abnormalities are present, differentiation into upper or lower motor neuron disease is helpful, if

possible. Lower motor neuron disease always results in decreased muscle tone (paresis or paralysis), while decreased or increased muscle tone may be seen in upper motor neuron diseases.

Neuroanatomic location	Gait	Mentation	Posture	Spinal reflexes
Cerebrum	Normal	Abnormal	Normal	Normal
Cerebellum	Abnormal	Normal	Abnormal	Abnormal or normal
Brainstem and Cranial Nerves	Abnormal or normal	Abnormal or normal	Abnormal or normal	Abnormal or normal
Spinal cord and peripheral nerves	Abnormal	Normal	Abnormal	Abnormal or normal

Table 1 Neuroanatomic regions and abnormalities associated with neurologic disease (From: Constable, 2004)

Cerebrospinal fluid (CSF) analysis

Analysis of the cerebrospinal fluid is one of the most reliable diagnostic tests for determining the presence of infectious neurological diseases of ruminants. Different from other species the collection of CSF is relatively easy and inexpensive in ruminants. These are step-by-step directions to collect CSF in different ruminant species:

- Lumbosacral collection
 - Few variations compared to atlanto-occipital space
 - Slightly higher protein and cell count
- 1 ml/ 5 kg BW is considered safe
- 3 – 5 ml necessary for analysis

Animal	Needle length	Needle gauge
Lambs < 30 kg	1 inch	21 gauge
Ewes 40 – 80 kg	1.5 inch	20 gauge
Rams > 80kg	2 inch	20 gauge
Calves < 100 kg	1 inch	19 gauge
Calves 100 – 250 kg	2 inch	19 gauge
Cattle > 250 kg	4 inch	18+ gauge