

2019 TVMDL Amarillo



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NEUROLOGICAL EXAMINATION OF THE RUMINANT AND LESION LOCALIZATION

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INTRODUCTION

When presented with neurologic signs in the bovid, my approach begins with two basic questions: is it rostral or caudal to foramen magnum and is it a primary neurologic disease? Etiologies that can cause neurologic signs include almost every category and consist of bacterial, viral, toxic, metabolic/nutritional, traumatic, neoplastic, congenital or hereditary and degenerative disease, therefore, a thorough, relevant history can be vital to an efficient arrival of a diagnosis.

Investigative historical questions should include;

- Environment?
 - i.e. other species of animals nearby?, junkyard?, plants?, feeding practices?, silage?
- Past disease?
 - i.e.: pneumonia?, diarrhea?, navel infection?, BVD?
- Age of onset?
 - Congenital
 - Breeds i.e.: Brown Swiss, Charolais, Saler
 - Later or adult onset
- Length of illness?
- Therapy and response?
- Vaccinations, dehorning, castrations

Organization of the CNS

- Sensory – afferent nerves
- Brain and Spinal Cord – integration centers
- Motor – efferent nerves
 - Autonomic – Sympathetic, Parasympathetic
 - Somatic – Upper Motor Neuron
 - Initiates movement
 - Signs – normal to hyper-reflexic, hypertonic muscles
 - Somatic – Lower Motor Neuron
 - Brain stem- ventral horns of the spinal cord
 - Signs – loss of reflexes, hypotonic muscle tone, atrophy



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NEUROLOGIC EXAMINATION

Exam starts at a distance. Needs to be in a new environment with unfamiliar obstacles/pens/alleyways if possible. Starts when they get off the trailer, or when they are observed “over the fence” on a farm visit. One should evaluate the following:

Gait
Posture
Mentation

Overall assessment from a distance

- Gait – focusing on coordination and strength
 - Ataxia
 - Vestibular ataxia – lean, circle, head tilt, maintains strength but “uncoordinated”
 - Cerebellar ataxia – maintains strength, NO proprioception deficits (knuckling/buckling)
 - Proprioceptive ataxia – weakness, knuckles, lower motor neuron signs
- Posture – i.e.: head, body, limbs – animals with postural abnormalities may have normal gaits, but animals with abnormal gaits will always have abnormal postural reactions
- Mentation – is animal responding appropriately to environmental stimuli?

Closer Examination (hands-on, or kind of!)

- Cranial nerve examination:
 - Practically 2-12 and 11 is questionable. I would argue that almost all of these can be evaluated from a distance, but to get the detailed examination you do have to lay hands on them.
- Ocular exam
 - Palpebral, menace, papillary light reflex, corneal reflex
 - Fundic examination
- Postural responses:
 - Proprioception, (adults), placing, hemi-standing/walking (young or small ruminants)
- Spinal reflexes:
 - Panniculus, perineal, patellar and withdrawal (flexor)
- Palpation:
 - Localized areas of pain, sweating, atrophy
- Peripheral nerves:
 - Obturator, sciatic, femoral, peroneal, tibial, suprascapular, radial



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ANCILLARY DIAGNOSTICS

Ancillary diagnostics that can be commonly employed such as advanced imaging are most of the time not practical in cattle medicine. However, examination of CSF can be rewarding. We typically NEVER take this from the cisterna magna. Lumbosacral taps are the most practical to perform and the safest! As a matter of interest, here are some reference values for normal bovine CSF:

- **Protein:** < 40 mg/dl
- **Nucleated cells:** < 10/microliter – monocytes
- **Pandy:** neg. for globulin
- **Glucose:** 60-80% of blood
- **CPK:** < or = 20 IU/dl
- **Sodium:** 134-144 mEq/L

DISEASES IN WHICH CSF RESULTS CAN REALLY HELP!

Meningeal worm – eosinophilic pleocytosis

Listeria suspect – mononuclear pleocytosis

Meningitis suspect calf – neutrophilic pleocytosis

Obviously, other diagnostic tools such as a CBC and chemistry panel can help to some degree answer the question, is it primary neurologic disease?

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1. de Lahunta A, Glass E, Kent M. Veterinary neuroanatomy and clinical neurology. 4th edition, St. Louis: Elsevier-Saunders; 2015
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NEUROLOGICAL SIGNS LOCALIZABLE ROSTRAL TO THE FORAMEN MAGNUM

CEREBRUM

Opisthotonus

Opisthotonus is defined as dorsoflexion of the head and neck. If the animal is able to sit sternal, this is sometimes referred to as “stargazing”. However, in the author’s experience, ruminants with opisthotonus more frequently lay in lateral recumbency and are unable to right themselves. Ruminants with advanced tetanus or hypomagnesemia may appear to have opisthotonus as well, therefore it is important to assess the complete neurological and physical examination findings in order to determine its origin.

Blindness

Vision can, in part, be assessed from a distance as the animal is asked to navigate unfamiliar surroundings. It is important, especially with small ruminants, to assess the animal as an individual as their strong “herd” instincts allow them to use other heightened senses and their herd mates to navigate their environment. A complete ocular neurologic examination should follow and will aid the clinician to determine whether the lack of vision is of cerebral cortical origin. Lack of vision with an intact pupillary light reflex (PLR) is a hallmark of cerebral cortical disease. If the PLR is absent unilaterally or bilaterally, the clinician should consider dysfunction of the ocular pathways or retina (vitamin A deficiency).

Abnormal Mentation

While abnormal mentation in a ruminant can range from stupor and depression to excitement and mania, the primary determinant is how the animal is responding to its environment. While some may consider this to be “behavior”, the natural temperament of the animal can mimic abnormalities in mentation in some cases. It is fairly straightforward to recognize the extreme in abnormal mentation, however, the author would argue that often times the alteration from the normal temperament of the animal is subtle. Consequently, abnormal mentation assessment should be coupled, if possible, with the caveat of whether a change has occurred.

Change in Behavior

Due to the fact abnormal mentation can often be “normal” for some individual animals due to temperament (i.e. a normally fractious animal when confined may also be manic from an altered mentation) the author always quizzes the owner about changes from “normal” for that particular animal. Former exhibition animals are most often calm when handled, however, if the owner now notes that they are unusually unruly, it could be indicative of a change in behavior due to cerebral disease.

Aimless Wandering or Compulsive Circling

Without cerebral function, the brain stem takes over in locomotion resulting in a slow, forward movement with no guidance from the cerebrum. When the animal reaches an obstacle or enclosure, if visual, they may press into it or circle away from it. If blind, the animal may circle an enclosure, however, if diffuse cerebral disease is present, this circling is in no particular direction.



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Seizures

Seizures are involuntary episodes of muscular activity during which the animal becomes laterally recumbent with an altered mentation. The involuntary muscular activity usually results in paddling of the limbs. Time between episodes may be hours (early nervous coccidiosis) or minutes (polioencephalomalacia) depending upon the etiology. Ruminants in lateral recumbency with other conditions such as advanced tetanus and hypomagnesemia may appear to be having seizures, however, these animals do not have an altered mentation and are usually visual.

Abnormal Vocalization

Vocalization that would be considered abnormal would include instances where vocalization is continuous, involuntary and unstoppable by external stimuli. Further, changes in pitch or volume during these episodes may indicate not only cerebral disease, but also disease of the innervation to the pharyngeal region (rabies).

CEREBELLUM

Ataxia Without Weakness

Ataxia is abnormal gait with accompanying incoordination. The gait is controlled by the cerebellum, brain stem, spinal cord and peripheral nerves. Determining whether the ataxia is of cerebellar dysfunction requires one to evaluate for the presence or absence of muscle strength. Strength can be assessed by pulling on the tail while the animal is moving in an attempt to pull it off course. The ability of the animal to resist this motion and apply strength to keep itself on course is indicative of normal muscle tone. Obviously, this procedure may be impossible to perform safely on all cases. Observing the gait from a distance for proprioceptive deficits during ataxia can be helpful as well. Ruminants with cerebellar disease do not have proprioceptive deficits during ataxia.

Truncal Sway

Truncal sway is simply a side to side swaying of the body during forward locomotion. This can also be observed in animals with lesions in the cervical spinal cord.

Hypermetria

Hypermetria is an exaggerated movement of the limbs during forward movement. In ruminants, hypermetria is most easily and readily appreciated in the forelimbs.

Wide Base Stance

Although not specific for cerebellar disease, positioning the limbs further away from their normal axial plane can be indicative of cerebellar dysfunction. The loss of muscle coordination leads the animal to position itself such that it may maintain balance. In the opinion of the author, these animals appear to be “holding on” to the ground for fear of falling over.

Intention Tremors

Small, rapid muscle contractions in various muscle groups that occur when the animal is moving are referred to as intention tremors. A “bobbing” of the head up and down as the animal moves to drink or eat is an example of an intension tremor as is diffuse areas of small, rapid contractions occurring when the animal initiates movement. Tremors that occur at rest are much less likely to be of cerebellar origin.



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VESTIBULAR SYSTEM

Localizing lesions of the vestibular system to either a peripheral or central lesion is very important for determining the prognosis of some cases. In the author's experience, peripheral vestibular disease responds more favorably to treatment than does a central lesion.

Head Tilt to the Side of the Lesion

A head tilt is defined as a continuous positioning of the head so that the eyes are off the horizontal plane and the muzzle of the animal is tilted away from the side of the lesion. This condition is present with both peripheral and central cranial nerve VIII dysfunction, however, it is more severe and often accompanied by recumbency with central lesions.

Falling/Leaning/Circling to the Side of the Lesion

Falling, leaning and circling to the side of the lesion occur with both peripheral and central lesions. However, central lesions most often result in smaller diameter circles ultimately resulting in recumbency with their head turned into the flank on the side of the lesion.

Nystagmus

Lesions involving the vestibulocochlear nuclei often result in a sustained nystagmus. The nystagmus is typically horizontal, but may change to vertical or rotary when the head position is changed. The fast phase is away from the side of the lesion.

Proprioceptive Deficits on the Side of the Lesion

Cattle with severe central lesions typically display proprioceptive deficits on the side of the lesion most readily observed in the forelimbs. The result of these deficits in a standing animal is extension of the forelimb opposite the side of the lesion.

Depression/Anorexia

Animals with signs of vestibular dysfunction that are also depressed and anorexic are most typical of a central lesion involving the brain stem nuclei and reticular activating system. These animals also are most likely to be recumbent and moribund.

CRANIAL NERVES

As one examines the cranial nerves it is important to note that these nerves have either sensory, motor or both modes of function. It is possible, therefore, to lose one functional aspect of a cranial nerve while the other remains intact.

I through IV

Cranial nerve I is difficult to evaluate effectively and will not be discussed. Deficits in cranial nerve II are noted by blindness and absence of the pupillary light reflex. An absence of the pupillary light reflex, ventrolateral strabismus and dilation of the pupils are typical of cranial nerve III dysfunction. Cranial nerve IV deficits are manifested as dorsomedial strabismus.



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V through VIII

Loss of sensation to the head and corneal surface is indicative of damage to cranial nerve V, as is a dropped, slack jaw. Deficits in cranial nerve VI are manifested as a ventromedial strabismus and an inability to retract the globe following a stimulus. Cranial nerve VII is motor to the muscles of the face, therefore, droopy ears, eyelids and lips, deviation of the nasal philtrum (cattle do not display this sign) and inability to blink are signs of dysfunction. Cranial nerve VIII peripheral disorders create head tilt, circling and leaning/falling to the side of the lesion.

IX through XII

Cranial nerves IX and X disorders cause an inability to swallow. A flaccid protruding tongue with decreased or absent ability to retract it is indicative of cranial nerve XII disease. Cranial nerve XI is difficult to evaluate, however, an inability to turn the head and neck from side to side away from a noxious stimulus is suggestive of dysfunction.

RETICULAR ACTIVATING SYSTEM, THALAMUS, HYPOTHALAMUS

Depression/Altered Mentation

Lesions of the reticular activating system in the brainstem cause profound depression. It should be noted that sometimes due to the normal demeanor of the animal, this may be a subjective assessment, however, when a change has occurred, determination of neurologically derived depression becomes more reliable.

Difficulty Regulating Body Temperature

Large variations in core body temperature without any evidence of an infectious cause should be considered as having lesions in the thalamus and/or hypothalamus. Following heat stress, camelids and young calves can experience prolonged periods of decreased ability to regulate body temperature.

Depressed Respiration

The respiratory centers in the brainstem are controlled by the reticular activating system. Therefore, respiratory rate decreases in response to disease conditions.

NEUROLOGICAL SIGNS LOCALIZABLE CAUDAL TO THE FORAMEN MAGNUM

SPINAL CORD

C1 to C5

Altered head and neck movements with no cranial nerve abnormalities are often noted with lesions within this region of the spinal cord. Further, all reflexes of the fore and hind limbs are exaggerated (hyper-reflexia). The increased extensor tone noted in all limbs also results in ataxia with or without truncal sway. When pivoting, animals may display proprioceptive deficits in the pivot limb (inside limb). Severe cervical lesions cranial to C4 often result in lateral recumbency.



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C6 to T2

Ruminants with lesions from C6 to T2 display depressed to absent reflexes with decreased muscle tone in the forelimbs and exaggerated hind limb reflexes with normal muscle tone in the hind limbs. Knuckling, stumbling and collapse of the forelimbs are indicative of lower motor neuron disease.

T2 to L3

Reflexes in the forelimbs are normal, while hind limb reflexes are exaggerated. Proprioceptive deficits are noted in the hind limbs in addition to ataxia. Larger ruminants may “dog sit”, even though muscle tone and extensor tone is increased.

L4 to L6

The most notable clinical difference in ruminants with lesions further caudal in the lumbar portions of the spinal cord is the absence of hind limb reflexes and decreased muscle tone. In larger ruminants, this clinically may be difficult to distinguish between lesions here and between T2 and L3. However, if practical, performing hind limb spinal reflexes and evaluating muscle tone should be differentiating.

S1 to S3

Decreased anal tone, tail tone and loss of sensation to the perineal region are typical of lesions in this region. Further, a distended urinary bladder that “dribbles” constantly is indicative of a “lower motor neuron” urinary bladder lesion.

SUMMARY

Overall, with a proper history, physical and neurologic examination, signs of neurologic dysfunction can be localized to a primary or secondary origin. Once localized, the clinician can then answer the following questions “Is it primary neurologic disease, and is it rostral or caudal to the foramen magnum?” Determining the most likely origin greatly narrows the differential list, streamlines the number of ancillary testing necessary, greatly improves the accuracy of prognosis and guides treatment regimens.

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