

SPINAL COLUMN AND CORD ASSESSMENT AND DYSFUNCTION



BASICS

OVERVIEW

- Causative lesions can be localized using the principles outlined below
- Diagnostic rule-out lists can be generated based on neuroanatomic localization, history, and signalment.

SYSTEMS AFFECTED

- Nervous

PATHOPHYSIOLOGY

- Neuroanatomic divisions of the spinal cord are described, with abbreviations of spinal cord segments as C, cervical; T, thoracic; L, lumbar; S, sacral; Ca, caudal.
 - *C1 to C5*
 - *C6 to T2* – Nerve supply of thoracic limbs
 - *T3 to L3*
 - *L4 to S1* – Nerve supply of pelvic limbs
 - *S2 to Cd*
- Selected functional units relating to the spinal cord are described below:
 - *Upper motor neurons (UMN)* – Act entirely within the CNS to regulate the function of other neurons. *UMN pathways* originating from brainstem nuclei pass to spinal cord lower motor neurons (LMN) either directly or via networks of interneurons to regulate gait, muscle tone, and posture of the trunk and limbs. Lesions cause spastic weakness of limbs.
 - *General proprioceptive tracts* – Bring proprioceptive information through the spinal cord from muscles, tendons, and joints, predominantly to the cerebellum (*spinocerebellar tracts*). Additional, relatively minor conscious GP pathways relay GP information to the forebrain, specifically, to the somesthetic (sensory) area of the cerebral cortex. Lesions of GP tracts cause ataxia.
 - *Lower motor neurons (LMN)* – Provide the anatomic connection between the CNS and muscle. Extend from a neuronal cell body in the gray matter of the spinal cord via a single continuous axon to striated muscle (*somatic LMN*) or, after synapse on a second (post-ganglionic) neuron, to smooth muscle (*autonomic LMN*). Lesions cause flaccid weakness of limbs.
 - *Spinothalamic tracts (ST)* – Conduct sensory information from spinal nerves through the spinal cord and brainstem to the somesthetic (sensory) cerebral cortex in the forebrain to allow perception of touch, heat, and noxious (painful) stimuli. Highly redundant system;

tracts mostly cross to the opposite side of the spinal cord. Lesions diminish sensation to touch and noxious stimuli, especially on opposite side.

PHYSICAL EXAMINATION FINDINGS

- *Spastic weakness* – Lesions of UMN tracts remove inhibition of reflex “anti-gravity” extensor tone in limbs on same side resulting in stiff (spastic) gait. In thoracic limbs, sometimes termed “tin soldier” gait. If severe, results in collapse and buckling of joints and recumbency. In recumbency, tend to struggle and thrash. Because of proximity of UMN to GP (spinocerebellar) tracts, spastic weakness and ataxia typically occur together
- *Ataxia* – Impaired awareness of body position at rest and motion. Manifests as delayed initiation of forward strides, overly long strides, base-wide or -narrow postures, excessive swaying and swiveling of hindquarters, circumduction of pelvic limbs, and interference between pairs of limbs during tight circling
- *Flaccid weakness* – In contrast to UMN weakness, limbs of animals with LMN lesions are floppy (hypotonic) and tend to collapse when weight-bearing. Strides are short and shuffling, and muscles may tremor. Limb reflexes are diminished or absent. Within two weeks, there is obvious muscle atrophy.

Signs related specifically to injury within each anatomic division of the spinal cord

- *C1 to C5* – Spastic weakness and ataxia of all limbs or recumbency; abnormal hopping tests all limbs; hyperactive patella reflexes, crossed extensor of flexor reflexes; reduced sensation caudal to lesion (rare)
- *C6 to T2* – Weakness and ataxia of all limbs; there may be flaccid weakness of thoracic and spastic weakness of pelvic limbs (i.e., thoracic weaker than pelvic); ataxia and abnormal hopping responses in all limbs or recumbency; hyperactive patella reflexes, crossed extensor of flexor reflexes in pelvic limbs; reduced sensation over thoracic limbs; reduced flexor reflexes in thoracic limbs; Horner’s syndrome; muscle atrophy after two weeks
- *T3 to L3* – Spastic weakness and ataxia of pelvic limbs; “dog-sitting”; reduced cutaneous trunci reflexes caudal to lesion; hyperactive patella reflexes; crossed extension of flexor reflexes in pelvic limbs; abnormal hopping responses in pelvic limbs; reduced sensation caudal to lesion; urinary incontinence
- *L4 to S1* – Flaccid or spastic weakness and ataxia of pelvic limbs; “dog-sitting”; reduced or absent patella and flexor reflexes in pelvic limbs; reduced sensation caudal to lesion; urinary incontinence; muscle atrophy after two weeks
- *S2 to Cd* – Flaccid paralysis of tail, anus, rectum, bladder, penis; loss of sensation to penis and caudal structures; urinary incontinence; fecal obstipation; muscle atrophy after two weeks

CAUSES AND RISK FACTORS

A listing of the more important causes of spinal cord disorders by category follows. The species that are affected – C, cattle; S, sheep; G, goat; Ca, alpaca or llama are indicated in parentheses. If all ruminants can be affected there is no species indication.

- *Malformations* – myelodysplasia +/- vertebral anomaly (e.g., spina bifida), vertebral malformation/malarticulation
- *Injuries* – Vertebral fracture/dislocation (+/- associated nutritional osteoporosis, septic destruction), pelvic fracture, sacral fracture, spinal cord soft tissue trauma, fibrocartilaginous emboli
- *Inflammation* – Tetanus, tail docking-associated meningitis (S), parelaphostrongylosis, elaphostrongylosis (S,G,Ca), setariasis (S,G), vertebral osteomyelitis, discospondylitis, epidural abscess, viral myelitis (CAE virus, G; visna-maedi virus, S), Border disease (S,G), *Hypoderma bovis* (C), nervous coccidiosis (C), polyradiculoneuritis (G)

- *Degeneration (metabolic and toxic)* – hypocalcemia, hypomagnesemia (C), acute organophosphate toxicity, degenerative myeloencephalopathy (C,Ca), enzootic ataxia (copper deficiency) (S,G), motor neuron disease (C), post-anesthetic poliomyelomalacia, delayed OP toxicity, heat stress (Ca), tremorgenic mycotoxins
- *Neoplasms* – extradural lymphosarcoma (BLV infection; C), other neoplasms rare
- *Idiopathic* – spastic paresis (Elsie Heel; C,G)



DIAGNOSIS

To minimize handling-induced stress, ruminants with spinal cord disorders are often observed initially “over the fence” then, after the animal has been restrained, further examined during walking, while standing (beginning at the tail), and finally while recumbent. Structures that, if injured, may cause the indicated abnormal signs, are listed within parentheses. The abbreviation “SC” is used to refer to spinal cord structures not otherwise specified.

- Initially watch animal walk and/or trot unrestrained. Pay particular attention to the accuracy and strength of movements during sudden changes in direction (e.g., cutting away from fence, trotting over broken ground). Note if head is held above or below withers.
- Move the animal in straight lines on a level surface at walking speed, ideally while being lead. Watch from behind and each side. Walking should be a 4-beat gait except in camelids which usually have a lateral “pacing” gait.
- Compare stride length side-to-side and look for movement abnormalities such as swaying and swiveling of the trunk and hindquarters, scuffing of hooves, excessive flexion or extension of joints during limb protraction, or overreaching, adduction, or abduction of limbs.
- Grasp the tail and repeatedly (but smoothly) pull the hindquarters sideways with sufficient power to move the hindquarters of a normal walking animal slightly to the side. Apply this force both at rest (LMN) and during weight-bearing phases of the walk (UMN, GP tracts).
- Move the animal in circles both ways. Look for circumduction of outside pelvic limbs and interference between limbs (GP tracts, UMN).
- Attempt basic postural tests (GP tracts, UMN, LMN). Hold up a limb and push the animal laterally to hop sideways on the opposite limb. Repeat for all limbs. Move the animal into unusual postures (e.g., narrow or wide-based stance, knuckled onto dorsum of fetlock) and look for replacement of limb into correct position
- Perform rectal or external palpation to assess bladder/rectal tone. Note tight non-compressible bladder (UMN, sensory tracts), distended bladder that leaks urine when compressed (LMN).

Perform passive examination of standing animal. If the patient cannot stand, consider propping over bale (small ruminant) or raising with padded hip lifters (cattle) for this part of the examination.

- Observe overall conformation and symmetry of trunk and limbs. Note marked muscle atrophy (LMN)
- Assess tail tone and musculature (LMN), tail/anal reflex (spinal sensory nerves, SC, LMN), and response to squeezing of skin over the perineum (spinal sensory nerves, ST tracts, forebrain).
- Palpate spine and limbs for structure and sensitivity to pressure - i.e., pain responses, strength
- Attempt to elicit cutaneous trunci reflex (SC at front of defective reflex). This reflex is unreliable in ruminants but should be symmetric if found.
- Assess responses to noxious stimuli (typically prodding or pinching skin) applied over sides of body and limbs and neck (spinal sensory nerves, SC, forebrain).

Examine limb reflexes with patient in lateral recumbency. If safe, rope cast adult cattle for this purpose.

- Assess tone of limbs as resistance to forced flexion and extension (LMN).
- *Patella reflex*. With upper pelvic limb held in partial flexion, strike the skin over the middle patella ligament and look for reflex extension (*reduced* - sensory spinal nerve, SC, LMN; *hyper* - UMN)
- *Flexor reflex*. Squeeze the skin on the distal limb and look for flexion (spinal sensory nerve, SC, LMN) and a conscious response from the patient (spinal sensory nerve, ST tracts, forebrain). Also look for extension of the fetlock of the opposite limb (“crossed extension”; UMN).
- Assess ability of patient to voluntarily roll from lateral to sternal recumbency, then repeat reflexes on opposite side



DIAGNOSIS

DIFFERENTIAL DIAGNOSIS

- Cranial nerve disorders may be classified according to the modified MIIND system - malformation/genetic, injury, inflammation, neoplasia, degeneration, idiopathic

CBC/BIOCHEMISTRY/URINALYSIS

- Important for screening for metabolic diseases (e.g., hypocalcemia), but generally nonspecific

OTHER LABORATORY TESTS

- CSF analysis - described in “Brain Assessment and Dysfunction” chapter

IMAGING

- *Radiography* – Conventional radiography +/- contrast myelography is widely applied for diagnosis of spinal disorders
- *Cross-sectional imaging (CT, MRI)* - Impractical in most cases but excellent tool for diagnosis of vertebral abscesses
- *Ultrasonography* – Limited application but can be used in pelvic fractures, vertebral malformations.

OTHER DIAGNOSTIC PROCEDURES

- Needle electromyography (EMG) is a specialized procedure wherein spontaneous abnormal electrical activity is recorded via an electrode needle inserted into muscle. Abnormal potentials begin about seven days after the onset of LMN disease.



TREATMENT

- See specific disorders



MISCELLANEOUS

ABBREVIATIONS

- CSF – cerebrospinal fluid
- UMN – Upper motor neurons
- LMN –Lower motor neurons

Suggested Reading

Constable, P.D. Clinical examination of the ruminant nervous system. Vet. Clin. Food Anim. 20, 2004:185:214.

de Lahunta, A., Divers, T.J., Neurologic diseases. In: Divers, T.J., Peek, S.F., eds. Rebhun's Diseases of Dairy Cattle, 2nd ed. St. Louis, Saunders Elsevier, 2007:504-560

Mayhew, I.G. Large Animal Neurology. 2nd ed. Oxford, Wiley-Blackwell, 2009:3-76

Smith, M.C., Sherman, D.M. Goat Medicine. 2nd ed. Oxford, Wiley, 2005: 163-256

Cebra, C., Gemensky-Metzler, A. Disorders of the neurologic system and special senses. In: Cebra, C., Anderson, D.E., Tibary, A., van Saun, R.J., Johnson, L.W., eds. Llama and Alpaca Care: Medicine, Surgery, Reproduction, Nutrition, and Herd Health. 1st ed. St Louis, Elsevier, 2014.

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Acknowledgment The author and editors acknowledge the prior contribution of Lisa Nashold.