Spinal Cord Diseases of the Horse: Relevant Examination Techniques and Illustrative Video Segments (Parts 1 & 2)
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1. ABSTRACT
The white matter of the spinal cord relays information in both directions between the body and the brain. Upper motor neurons influence posture and gait by modulating lower motor neuron activity. General proprioceptive (mostly cuneocerebellar and spinocerebellar) and pain (spinothalamic) pathways convey sensory information from the torso and limbs to the brain. Limb reflexes (flexor, patella) and long spinal reflexes (slap, cervicofacial, back, cutaneous trunci) are the final determinant of gait and posture. Careful evaluation of these reflexes along with assessments of gait and limb strength, allow accurate localization of spinal cord lesions. Localization of spinal cord lesions is an important step in the processes of diagnosis and treatment.

2. FUNCTIONS

**Upper Motor Neuron - Muscle Tone and Voluntary Movement**
Axons of the upper motor neuron (UMN) extrapyramidal and vestibular systems travel from cell bodies throughout the brain and pass predominantly in reticulospinal and vestibulospinal tracts to lower motor neurons (LMN) in the ventral and intermediate columns of the gray matter of the spinal cord. This system provides tonic support of the body against gravity and recruits spinal reflexes for the initiation of voluntary movement. Upper motor neurons act by influencing \( \alpha \) and \( \gamma \) motor neurons in the spinal cord. These LMN, in combination with afferent nerves and stretch receptors in the neuromuscular spindles and tendons, control muscle tone and movement by myotatic and anti-myotatic reflexes. Most descending UMN tracts are inhibitory to extensor motor neurons. Basic locomotor activity involves recruitment and control of these reflexes by distinct postural and voluntary UMN systems. Interruption of UMN tracts in the spinal cord causes signs of ipsilateral weakness of the trunk and limbs. Signs of paresis range in severity from slight toe-dragging and delayed protraction to recumbency and inability to rise. Because myotatic reflexes are released from inhibitory UMN influences, there may also be spasticity (stiffness) of limb movement. This is most obvious in the thoracic limbs, which may appear to “float” during walking. Interference with UMN also may manifest as delayed initiation of voluntary movement or alterations in gait cadence. For example, some horses with spinal cord disease may have a lateral “pacing” gait at walking speed. If LMN are not affected, spinal reflexes are either normal or exaggerated on the side of the spinal cord lesion and crossed extensor reflexes may be seen. In keeping with the basic principles of neuroanatomic localization, UMN lesions from C1–T2 (inclusive) may cause neurologic signs in all 4 limbs, lesions from T3–S2 can only affect the pelvic limbs, and lesions caudal to S2 do not directly affect gait. With external compression of the cervical spinal cord as in horses with cervical vertebral stenotic myelopathy (i.e., wobblers), signs typically are worse in the pelvic limbs than in the thoracic limbs.

**Lower Motor Neuron and Spinal Cord Reflexes**
The effector function of the central nervous system (CNS) is exerted entirely through the actions of LMN on skeletal and smooth muscle. LMN to skeletal muscles are found in the ventral columns of the gray matter while those of the autonomic nervous system are located in the intermediate columns. LMN form a ventral root which then exits the vertebral canal through the intervertebral foramen, usually of the vertebra of the same name. The ventral root joins with the dorsal sensory root to form the segmental spinal nerve. In the cervical vertebrae, this foramen is at the cranial end of each vertebra. For the remaining roots, the foramina are at the caudal end. The more caudal spinal cord segments have long nerve roots because the spinal cord segments are shifted cranially with respect to the vertebrae. The neurons of the afferent (sensory) component of reflexes course from receptors in the skin, muscle, or tendon, through the spinal nerve and dorsal root, into the dorsal horn of the gray matter where they
terminate on interneurons. The interneurons then complete the pathway by passing to the LMN. An exception to this is the patella reflex wherein the sensory neuron terminates directly on the LMN in the ventral horn. Long (i.e., multi-segment) spinal cord reflexes, including “slap” tests, cervicofacial, and cutaneous trunci reflexes, and caudal reflexes, including anal and tail-clamp, are routinely evaluated during neurologic examination. Limb reflexes, including withdrawal, patella, and triceps, are always evaluated in neonatal foals and in older horses that are recumbent. Abnormalities of LMN (in gray matter, ventral root, plexus, peripheral nerve, or neuromuscular junction) manifest as flaccid muscle weakness (paresis, paralysis) with hypotonia and hyporeflexia. Within 2–4 weeks, muscle atrophy is noticeable and this neurogenic muscle atrophy progresses rapidly. Ventral nerve roots contribute to multiple peripheral nerves and peripheral nerves are derived from multiple roots, so injury to gray matter of an individual segment or to a ventral nerve root produces less severe neurologic signs than does loss of function in a peripheral nerve.

Proprioception

Through a system of receptors in muscles, tendons, and joints, the general proprioceptive system is able to monitor the position of the body or limbs in space. Proprioceptive information is passed centrally in sensory nerves that terminate in the dorsal gray column on cell bodies of neurons in the spinocerebellar (pelvic limbs) or cuneocerebellar (thoracic limbs) tracts. These tracts pass cranially and provide information for the cerebellum to use in its role of regulating posture, muscle tone, locomotion, and equilibrium. Other proprioceptive pathways that serve in conscious proprioception pass from the spinal cord to the somesthetic sensory cortex via relay nuclei in the midbrain and thalamus. Interruption of spinal cord proprioceptive pathways interferes with recognition by the brain of the positions in space of the body and limbs. This is manifest as ataxia. Signs of proprioceptive deficit in the horse include base-wide or base-narrow limb placement, swaying of the trunk and torso during walking (but not usually at rest), and overstriding, especially in the pelvic limbs. During circling, limb ataxia is evident as circumduction in the pelvic limbs and interference in the thoracic limbs.

Urination

Parasympathetic LMN to the bladder’s smooth muscle (detrusor) originate in the intermediate column of the gray matter of S2–4. These neurons exit in ventral roots and contribute to the pelvic plexus, a network that supplies autonomic innervation to the smooth muscle of the bladder and rectum. Sympathetic LMN to the bladder begin in the gray matter of L1–4, exit the vertebral canal, and course caudally to the pelvic plexus. Post-ganglionic sympathetic neurons terminate on smooth muscle in the body and neck of the bladder and proximal urethra. These autonomic LMN function in local reflexes. Afferent neurons pass from stretch receptors in the bladder wall and enter the spinal cord in dorsal sacral nerve roots to exert inhibitory influences on parasympathetic and sympathetic LMN. Striated muscle of the urethra is innervated by somatic LMN in the pudendal nerve. Urination occurs when there is stimulation of parasympathetic nerves to the detrusor muscle, inhibition of sympathetic nerves to the detrusor muscle, and inhibition of sympathetic and somatic nerves to the urethra. The net effect of this activity is contraction of the smooth muscle of the body of the bladder and relaxation of the proximal urethra. Centers in the midbrain and hindbrain receive sensory information from the bladder and modulate reflex activity via UMN passing caudally in the spinal cord. Forebrain influence on these centers is responsible for initiation of voluntary voiding. In horses with severe spinal cord disease cranial to S2, there may be loss of voluntary control of urination. Within 2 weeks, “spinal reflex bladder” function develops which results in intermittent voiding with retention of small amounts of urine. When there is a spinal cord lesion between L4 and S2, reflex pathways for inhibition of sympathetic activity may be interrupted, resulting in increased urethral tone and functional obstruction of urinary outflow. With injury to the sacral spinal cord segments or nerves, the bladder and urethra are atonic and distended and there is overflow incontinence. Although a small amount of intrinsic reflex bladder contraction (“automatic bladder”) may occur, it is ineffectual and large volumes of urine and sediment remain. In cases of LMN urinary incontinence, additional non-urinary signs of sacral nerve injury are expected. These are the signs of cauda equina syndrome and include fecal incontinence, paralysis of the anus, tail, and penis, and perineal analgesia.
Defecation
The smooth muscle of the rectum and anus is innervated by post-ganglionic parasympathetic neurons in a way that parallels that described for the bladder. Somatic innervation of striated muscle of the anus is provided by the pudendal nerve. Spinal cord lesions cranial to S2 are unlikely to affect defecation; however, involvement of the sacral segments results in rectal obstipation and may cause colic. Diseases causing rectal paralysis are the same as those described under the section on Urination.

Sensation
Sensory information from pain, thermal, and touch receptors is conveyed to the spinal cord by peripheral nerves and dorsal roots. Branches pass several segments both cranially and caudally from the site of spinal cord penetration and terminate on spinthalamic neurons or interneurons involved in spinal reflexes. Spinthalamic pathways servicing a single dorsal nerve root course cranially on both sides of the spinal cord. Pain perception requires interpretation of afferent information by the forebrain and has been described in the Forebrain Diseases lecture. The flexor (withdrawal) reflexes require only sensory nerves, contiguous spinal cord segments, and LMN. In the thoracic limbs, spinal cord segments C6–T2 are required and L6–S2 are involved in the pelvic limb reflex. Evaluation of sensation over the trunk and limbs requires knowledge of the common autonomous zones for sensory nerves.

3. CLINICAL EXAMINATION OF SPINAL CORD FUNCTION1,3,4

General Examination of the Neck, Trunk, and Limbs
With the horse standing squarely, assess muscle mass, paying particular attention to asymmetries. Firmly press the cranial edge of each of the cervical transverse processes from C3 to C6 on each side to test for a pain response. Put pressure on the C6–C7 intervertebral joints by pushing medial to the deep pectoral muscle in front of the shoulder on each side. Test lateral neck flexion by enticing the horse to move its head toward feed held at the point of the elbow. Severe or rapidly developing muscle atrophy indicates denervation and is a localizing sign. Neurogenic muscle atrophy is caused by damage to the LMN in the ventral column of the gray matter, nerve roots, or peripheral nerves supplying that muscle. Neurogenic atrophy of thoracic limb musculature results from lesions of the C6–T2 spinal cord segments, brachial plexus, or peripheral nerves, while atrophy of pelvic limb muscles reflects involvement of L3–S2 or associated ventral roots or peripheral nerves. Anesthesia of a strip of skin is caused by loss of the segmental sensory nerve, dorsal nerve root, or connections in the spinal cord. Because sympathetic fibers are distributed with spinal nerves, spontaneous sweating may occur over denervated skin.

Slap Test
While standing on the left side, reach under the horse’s neck and hook the index and middle fingers of the left hand over the highest palpable point of the larynx - the muscular process of the arytenoid. Have the handler move the head slightly to the left of midline, then gently strike the horse behind the withers several times with the palm of the right hand. The expected response is slight palpable movement (adduction) of the arytenoid in response to each slap. Repeat the procedure from the right side. Sensory input to this reflex is from pleural stretch receptors to the sensory nerves and roots under the area that is slapped (~T7–T11). Central pathways are thought to cross to the other side at this level, pass cranially to the nucleus ambiguus, then efferent fibers pass out in the vagus nerve via the recurrent laryngeal nerve to innervate the contralateral laryngeal adductor muscles. Severe cervical spinal cord disease often affects this test bilaterally, and the vagus and recurrent laryngeal nerves may be affected at the guttural pouch or within the jugular groove. It is important to note that variations in neck anatomy make this reflex easy to palpate in some horses and difficult or impossible in others. The examiner must take into account the relatively common finding of a negative right to left slap test result, reflecting idiopathic left recurrent laryngeal neuropathy, even in horses with no history of respiratory noise or exercise intolerance. Negative left to right tests are thus usually accorded more significance in examination of horses with suspected spinal cord disease.
Cervicofacial Reflex
Place the left index and middle fingers at the commissure of the left lip, then strike the skin over the brachiocephalicus muscle with the closed tip of the hemostat. Begin at the cranial end of the neck and continue back to the shoulder. The expected response is facial contraction, detected by the examiner’s finger as retraction of the commissure of the lip, and contraction of the brachiocephalicus and cutaneous colli, observed as shrugging of the shoulder, lateral jerking of the head, and twitching of the skin of the neck. Interruption of reflex components in the facial nerve, cervical nerves or roots (sensory or motor), or local cervical spinal cord segments can affect the cervicofacial reflex. This reflex typically is reduced at the level of a cervical spinal cord lesion, but is normal cranial and caudal to the lesion.

Cutaneous Trunci Reflex (“Panniculus”)
When examining the reflex on the left side of the horse, use the extended right thumb to firmly prod the skin of the sensory field, beginning cranially just behind the shoulder and extending caudally to the last intercostal space. Check every intercostal space both ventrally and dorsally. For safety, firmly grasp the back of the mane with the left hand and face backwards when testing the reflex, because horses that resent this test will try to kick the examiner. A normal response is twitching of the skin, with or without indication of conscious perception of the stimulus. Regardless of the site of stimulus, the twitch response is the same. The reflex pathway is input from sensory thoracic nerves to the ipsilateral spinal cord, from where it courses cranially via interneurons to end in the C8 and T1 segments, and thence via the brachial plexus to the lateral thoracic nerve and the cutaneous trunci muscle. Interruption of this pathway in the spinal cord white matter results in loss of the reflex from approximately the point of the lesion caudally. A lesion of the sensory nerve will only affect the reflex within the same dermatome (skin strip) while loss of lateral thoracic nerve function ablates the entire ipsilateral reflex.

Back Reflexes
Make sure that the pelvic limbs are positioned equally and squarely, then stroke the closed tip of the hemostat caudally along the skin over the longissimus muscle, from mid-thorax caudally to the level of the tuber coxa. For safety, hold the back part of the mane with the other hand. The expected response is brisk extension of the back and flexion of the pelvis followed quickly by return to normal posture. Next, stroke the hemostat caudally along the skin over the gluteal muscles. This should elicit flexion, followed by relaxation of the lumbar spine and pelvis. Common abnormal reactions to these tests include 1) partial collapse in the pelvic limbs, associated with unlocking of patella ligaments from the lateral trochlear ridge of the tibia, 2) wobbling of the pelvis from side to side, and 3) bouncing up and down of the pelvis; 4) delayed or slow return of the flexed pelvis to normal position, and; 5) no response.

Tests for Limb Strength
During the passive part of the examination, it is difficult to detect mild thoracic limb weakness. Only very weak horses buckle a thoracic limb in response to downward pressure exerted over the withers. The hopping test is more sensitive. From the left side, hold the halter and lead rope with the left hand. Pick up the left leg then push the head toward the right side while leaning against the left shoulder. Normal horses use the right limb to hop briskly around a circle centered on the pelvic limbs. With limb weakness, the response may be delayed so that the horse leans markedly before hopping or the limb may buckle after landing. Strength of the pelvic limbs is evaluated by resistance to pressure over the pelvis and to sideways pull on the tail. Additional insight is provided by the back reflexes described earlier in this section. To test resistance to dorsal pressure, stand on one side of the horse and hook the fingers over the opposite tuber sacrale. Use maximal effort to try to collapse the near limb. Only weak horses buckle in response to such pressure.

The tail-pull test is done both at rest and while the horse is walking in a straight line. With the horse standing squarely, take the tail and pull sideways with gradually increasing force. After initial slight movement in the direction of pull, normal horses usually cannot be moved sideways, even with strong pressure. Next, pull sideways on the tail while the horse is walking in a straight line. Always work on the same side as the handler. Exert moderate lateral pressure during all phases of the stride, then
rhythmically increase pressure as the horse supports weight on the near leg. Normal horses of 450-kg bodyweight can only be moved slightly to the side. Response to this test varies according to the size and strength of the examiner and the bodyweight, strength, and temperament of the horse. Excited anxious horses cannot be accurately examined and even weak horses may test as normal if not relaxed. Perform the test in mirror-image fashion from the right side (i.e., with the horse being led from the right side). Interpretation of this test requires practice and experience on the part of the examiner. If a full-sized adult horse can easily be pulled sideways at rest, there likely is interruption of the anti-gravity myotatic reflex pathway. Practically, such a lesion most likely is located either in the ventral gray matter or roots (L3–L5) that form the femoral nerve or in the femoral nerve itself. If cutaneous sensation over the saphenous vein is absent or reduced, the lesion is central to the ventral aspect of the shaft of the ilium (the site at which the sensory saphenous nerve joints the femoral nerve). The much more common finding - namely, lack of resistance to tail-pull only during walking - is consistent with an ipsilateral spinal cord lesion affecting descending upper motor neurons anywhere between and including C1 to S2.

**Gait**

Have the handler lead alternately from left and right sides and walk the horse in straight lines. The handler should be instructed to keep the horse’s head and neck as straight as possible during walking. When the horse turns its head to one side there is reciprocal and potentially confusing movement of the pelvis toward the same side. Follow directly behind the horse. From this vantage point evaluate leg position and stride symmetry. Also, watch for excessive 1) side to side (wobbling) movement of the pelvis, 2) up-and-down movement of the tuber coxae (pelvic roll around the z axis), and 3) side-to-side rotation of an imaginary line from the tailhead to the skin between the tuber sacrale (pelvic “yaw” around the y axis). Next, watch the gait from the side while walking in stride with the pelvic and then thoracic limbs. Note any toe-dragging, knuckling, stride-length asymmetries, and abnormal protractive movements such as hyperflexion, stiffness (hypometria), or excessive range of movement (hypermetria). Often, these signs are most obvious as the horse transitions from standing still to walking. Repeat this part of the examination with the horse’s chin lifted and with the horse walking up and down a modest slope. These maneuvers exacerbate most gait abnormalities, especially stiffness of the thoracic limbs. Take the horse in hand for the next part of the examination. Hold the lead rope with the left hand and, by walking backwards, lead the horse in counterclockwise circles. It is very important that the horse is always walking **forward** in these circles. Vary the diameter, making the circles alternately small and large. Observe carefully the motion of the right (outside) pelvic limb by looking under the horse’s torso. This limb will often arc out widely on the outside of the circle (i.e., circumduct) in horses with spinal cord disease. In mirror-image fashion, lead the horse from the right side in clockwise circles. Next, pull the horse sideways in **tight** circles in either direction. This is done from a position slightly behind the shoulder by pulling the leadrope at an angle sideways and caudally. If done correctly in normal horses, the opposite thoracic limb should cross in front of the supporting limb and the pelvic limbs should move reciprocally causing the horse to pivot around a point midway between the thoracic and pelvic limbs. Horses that are weak and ataxic tend to sag backwards in the hindquarters before they start to move, then pivot the front part of the body around one or both pelvic limbs. There is often also interference between or otherwise inappropriate **inconsistent** placement of thoracic limbs. Use the leadrope to push the horse backwards in a straight line. A normal horse should move backwards in 2-beat fashion, with simultaneous movement of diagonally opposite pairs of limbs (e.g., left thoracic and right pelvic limbs). A horse with spinal cord disease may sag backwards before moving and slide its hooves along the ground rather than picking them up and placing them.

**References**

