Diseases of the Peripheral Nerves, Neuromuscular Junction, or Uncertain Sites: Relevant Examination Techniques and Illustrative Video Segments

Robert J. MacKay, BVSc (Dist), PhD, Diplomate ACVIM

1. Introduction
Mechanical injuries to peripheral nerves occur because of compression, entrapment, transection, laceration, ischemia, crushing, stretching, or chemical or burn damage.1,2 Neurapractic lesions are characterized by a failure of conduction of the action potential across the injured axonal segment.3 Axonotmesis is axonal interruption caused by axon and myelin sheath injury, whereas neurotmesis reflects complete disruption of endoneurium, perineurium, and/or epineurium. Recovery by axonal regrowth is unlikely after neurotmesis.4 After axonotmesis of motor nerves, muscle reinnervation is to be expected and occurs by 2 separate mechanisms: collateral sprouting and axonal regrowth.1 If there is incomplete loss of axons, reinnervation of muscle units by sprouting occurs in days to weeks. Reinnervation by axonal regrowth occurs at a rate of 1 millimeter per day (approximately 1 inch per month). Skeletal muscle cells deprived of nerve supply ultimately undergo fibroblastic transformation; thus, reinnervation may not be possible after more than 12 months, although this remains a controversial issue. Peripheral nerve injuries are characterized by weakness of the innervated muscle accompanied within 2 to 4 weeks by appreciable atrophy. When the injured nerve supplies important extensor muscles of the limbs (e.g., as is the case for radial, femoral, sciatic, and peroneal nerves), there is obvious alteration of gait. Areas of cutaneous anesthesia occasionally accompany peripheral nerve injuries. Over the neck and trunk, cutaneous sensory innervation occurs in defined bands associated with segmental dermatomes. Damage to a peripheral spinal nerve or dorsal nerve root results in cutaneous anesthesia/hypalgesia over the supplied dermatome. With damage to the pudendal nerve or its sacral nerve roots, there is anesthesia/hypalgesia of the perineal area. In contrast, relatively small autonomous zones have been defined for sensory components of the ulnar, musculocutaneous, median, femoral, tibial, and peroneal nerves.5,6 Sympathetic fibers are distributed with peripheral nerves, so denervated skin also may be evident as circumscribed spontaneous sweating. The most common and important syndromes of me-
chanical injury to peripheral nerves of the limbs are described below.3–8

2. Suprascapular Nerve

The suprascapular nerve arises from C6 and C7 spinal cord segments. Injury occurs most commonly when a horse’s shoulder is impacted at speed such that the nerve is injured as it curls around the front of the neck of the scapula. There immediately is laxity and lateral instability of the shoulder joint, which bows out or “pops” as the affected limb bears weight. Within 2 to 4 weeks of injury, there is obvious atrophy of the supraspinatus and infraspinatus muscles (Sweeney). At least 50% of such injuries are predominantly neurapractic, and recovery of function (and muscle mass as there is atrophy) is evident within 30 days of injury and is complete within 60 days. In more severe injuries with morbid axonal injury, successful reinnervation is evident as recovery of muscle bulk in the ventral part of the supraspinatus muscle beginning within 3 months of injury. Maximal recovery takes an additional 3 to 12 months. Regrowth of the suprascapular nerve can be facilitated by surgery to resect scar tissue and a constricting band (external neuralysis) and reduce tension on the nerve as it crosses the front of the scapula (often performed 3 to 6 months after injury). Removal of a piece of bone from the neck of the scapula may further relax the injured nerve, but the scapular notch created by this procedure creates a potential nidus for scapular fractures during recovery from anesthesia.

3. Radial Nerve

The radial nerve innervates a flexor of the shoulder and the extensors of the elbow, carpal, and digital joints. It arises from T1. The nerve root may be lacerated by fractures of the C7 or T1 vertebrae or first rib. The nerve is commonly damaged as a result of humeral fracture9 and can be injured as it curls around the front of the neck of the scapula. Ischemic damage may occur in horses anesthetized in lateral recumbency.10 The lower part of the nerve may be injured by dislocation or fractures that involve the elbow.

Horses with complete radial paralysis stand with the shoulder extended, the elbow “dropped,” and the dorsum of the hoof resting on the ground. When forced to walk, the horse may partially protract the limb by exaggerated extension of the shoulder; however, the toe drags and the horse collapses on the limb during the weight-bearing phase of the stride. If the site of damage is distal, the shoulder and elbow are normal. Although the radial nerve has numerous cutaneous sensory branches, injury to this nerve does not result in any consistent area of cutaneous anesthesia.

4. Musculocutaneous Nerve

The musculocutaneous nerve arises from C7 and C8 and supplies flexors of the elbow. Injury is uncommon and causes only transient toe-dragging. The shoulder may be held in a flexed position and the elbow in an extended position. There is hypalgesia/analgesia over the dorsomedial aspect of the knee and proximal metacarpus and atrophy of the biceps and brachialis muscles.

5. Median and Ulnar Nerves

The median nerve arises from C8 and T1 and the ulnar nerve from T1 and T2. Injury to either can be caused by injury to the brachial plexus or along the medial aspect of the upper limb. Clinical findings include a “tin soldier” gait, with decreased flexion and dragging of the toe during protraction of the limb and hypalgesia/analgesia of the skin of the caudal forearm, lateral metacarpus, and medial posteri- 

areas and atrophy of the carpal and digital flexors.

6. Femoral Nerve

The femoral nerve arises from L3 to L5 and innervates muscles that flex the hip and extend the stifle. The nerve can be damaged by ilial, femoral, or vertebral fractures. Ischemic injury is caused by prolonged stretch or increased tissue pressure during anesthesia in dorsal recumbency or after severe prolonged dystocia.11 With unilateral paralysis, the pelvic limb is abnormally flexed, usually with the foot flat on the ground, and buckles when the limb bears weight. In the case of bilateral involvement, the horse is either unable to rise or stands uncomfortably in a crouched position. If the nerve injury occurs proximal to the saphenous branch, there is anesthesia/hypalgesia of the skin over the medial surface of the thigh and atrophy of the quadriceps muscle.

7. Sciatic Nerve

The sciatic nerve arises from L5-S1 and supplies important extensors of the hip and flexors of the stifle. Damage usually is a result of deep injections into the caudal thigh.4 The nerve also may be injured by fractures of the ilium or ischium or sacroiliac or coxofemoral dislocations. The limb is held slightly caudal with the dorsum of the hoof resting on the ground. The stifle and hock are extended, whereas the distal joints are flexed. The leg is dragged forward by the actions of the quadriceps and biceps femoris muscles. These muscles, in concert with the reciprocal apparatus, allow the horse to bear some weight on the limb if the foot is first placed in normal position. There is cutaneous hypalgesia/analgesia over most of the limb except for the medial thigh.

8. Peroneal Nerve

The peroneal nerve arises from the sciatic trunk deep to the biceps femoris and is motor to the flexors of the tarsus and the extensors of the digit. Paralysis results in extension of the tarsus and flexion of the distal joints of the pelvic limb. At rest, the limb
is held slightly caudally with the distal joints in flexed position and the dorsum of the hoof contacting the ground. During walking, the limb is moved erratically. The toe is dragged along the ground during the weak protraction phase, then is pulled caudally as the horse attempts to bear weight. There is atrophy of the cranial tibial and long and lateral digital extensors and immediate cutaneous hypalgesia/anesthesia over the lateral metatarsus.

9. Tibial Nerve

The tibial nerve is the direct continuation of the sciatic nerve and innervates the gastrocnemius (extensor of the hock) and digital flexors. The limb is held flexed and the foot contacts the ground in normal position, the fetlock often partially collapses into a flexed position (i.e., “knuckles”). The foot is moved in stringhalt-like fashion, with exaggerated flexion of the hock and stifle during protraction followed by sudden extension to the weight-bearing phase of the stride. There is atrophy of the gastrocnemius and cutaneous hypalgesia/anesthesia of the caudal metatarsal region and bulbs of the heels. The flexor reflex is tested by pinching the skin over the dorsal aspect of the fetlock. The reflex is present but weaker than on the normal side.

10. Prognosis for Peripheral Nerve Injury

With suprascapular neuropathy secondary to confirmed or suspected shoulder trauma, restoration of shoulder stability occurred in 3 to 12 months in 7 of 8 horses treated only with stall rest.12 Atrophy of the infraspinatus and supraspinatus resolved in only 2 of these horses. Although there is no comparative study to show any additional effect of procedures to relieve entrapment of the suprascapular nerve,13,14 it is expected that these procedures should improve recovery of muscle mass. Data for outcome of radial nerve neuropathy are not published; however, it is reasonable to assume that the prognosis for complete recovery from signs of radial paralysis induced by positioning in lateral recumbency, soft-tissue trauma, and humeral fracture is good (>80%), fair, and poor, respectively. Injection-associated neuropathy (usually sciatic) typically resolves in days.

11. Neurologic Testing of Recumbent Horses

Examination

The examiner should carefully note whether or not the horse moves its limbs voluntarily without stimulation. If possible, assist and stimulate the horse in such a way as to assess which of the following best describes the horse’s maximal voluntary motor function: (1) lifts head off the ground; (2) rolls shoulders and chest into a sternal position, or; (3) straightens thoracic limbs and assumes a “dog-sitting” position.

The long spinal reflexes (cervicofacial, cutaneous trunci, slap) and cutaneous sensation over the limbs and torso should be evaluated systematically. Test sensation at each site by grasping a fold of skin between the jaws of the hemostat then firmly squeezing the skin and watching for evidence of a conscious response by the horse. This is a behavioral reaction and must be distinguished from a reflex that may occur without cognitive acknowledgement. Sensory fields for some peripheral nerves of horses have been described.6

Test pelvic limb reflexes and function: First, assess extensor tone in the limb by testing resistance to passive flexion. Next, perform the flexion test by pinching skin on the distal limb with a hemostat. If there is no response, try pinching skin elsewhere on the leg. A normal response is flexion of the limb, usually with some behavioral evidence that the horse can feel the skin pinch. When abnormal, the flexor response may be reduced or absent and may be accompanied by reflex extension of the contralateral digit (crossed extensor reflex). To test the patellar reflex, hold the pelvic limb in a moderately flexed position, and strike the skin over the middle patellar ligament. A twitch handle works well for this purpose in full-sized horses; a patellar hammer or reversed hemostat can be used in foals. The expected response is brisk extension of the stifle. If the reflex is absent, move the leg into different positions and retest. Classify the response as absent, normal, or increased. Other extension reflexes in the pelvic limb cannot be elicited reliably but should be tested for comparison with the opposite leg. These include (1) the tibial reflex: tap the biceps femoris just behind the greater trochanter; (2) the gastrocnemius reflex: strike the Achilles tendon close to its insertion; and (3) the cranial tibial reflex: strike the middle of the cranial tibial muscle.

Test thoracic limb reflexes and function: Assess extensor tone and evaluate the flexor reflex as described for the pelvic limb. No other reflex can be obtained consistently in the thoracic limbs but, for comparison with the opposite limb, test the following: (1) triceps reflex: with the limb in flexed position, strike the triceps muscle and watch for elbow extension; (2) biceps reflex: strike the front of the elbow and watch for extension of the shoulder and flexion of the elbow joint; and (3) strike the middle of the extensor carpi radialis muscle and look for extension of the carpus.

Lesion Location

If a horse can dog-sit, the principal spinal cord lesion probably is behind T2. Inability of a horse to roll from lateral into sternal recumbency is associated with severe lesions (usually involving gray matter) of the caudal cervical and/or cranial thoracic segments, whereas serious injury to the spinal cord in the rostral part of the neck may also prevent a horse from raising its head off the ground.

With involvement of lower motor neurons to limb muscles, there is reduced or absent extensor tone. Limb reflexes are reduced or abolished if the sensory nerves, motor nerves, or central components of the
Reflexes are affected. In contrast, extensor tone and limb reflexes may be exaggerated beginning several days after injury to descending upper motor neurons. For example, after trauma to the spinal cord at the T13 segment, patellar reflexes and pelvic extensor muscle tone may be exaggerated.

If there is no response to strong pinching of the skin over caudal regions of the body, there probably is catastrophic damage to the spinal cord cranial to the anesthetic area. If there is no response to deep pain for more than 24 hours, there is at least functional transection of the spinal cord.

**Associated Clinical Signs**

Large horses that remain recumbent for more than a few hours often have reduced skin sensation in the distal limbs secondary to pressure-induced injury of superficial sensory nerves. This complicates interpretation of tests for flexor reflexes and for presence of deep pain.

**12. Tail and Anus**

**Examination**

Assess tail strength by lifting (extending) the tail. Prod or pinch the skin adjacent to the anus and observe the anal contraction and tail-clamp reflexes. If these reflexes are abnormal or if the history suggests possible cauda equina syndrome, perform a rectal examination to assess rectal tone and bladder size and tone. Assess muscular symmetry of the tail and test cutaneous sensation over the tail and caudal structures.

**Lesion Location**

Anesthesia and areflexia of the tail, penis, and perineum and paralysis of the anus, rectum, bladder, and penis are signs of **cauda equina syndrome**. Lesions of the spinal cord or nerve roots caudal to the S2 spinal cord segment cause some to all of the signs of cauda equina syndrome. The bundle of roots forming the cauda equina are vulnerable as they pass through the sacrum and proximal coccygeal vertebrae.

**Associated Clinical Signs**

With involvement of S2 and more cranial nerve roots, expect to see signs of pelvic limb weakness in addition to cauda equina syndrome. Colic caused by obstruction may be the presenting sign of rectal paralysis, and urinary overflow incontinence is a sign of bladder paralysis.

**References**