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The Art and Science of Equine Neurology – Peripheral Nerves & Stringhalt

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Peripheral Nerve Injury

Mechanical injuries to peripheral nerves occur because of compression, entrapment, transection, laceration, ischemia, crushing, stretching, or chemical or burn damage. Neurapractic lesions are characterized by a failure of conduction of the action potential across the injured axonal segment. Axonotmesis is axonal interruption, while neurotmesis reflects disruption of endoneurium, perineurium, and/or epineurium. Recovery by axonal regrowth is unlikely after neurotmesis. After axonotmesis of motor nerves, muscle reinnervation occurs by 2 separate mechanisms: collateral sprouting and axonal regrowth. 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 mm per day (approximately 1 inch/month). 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 2-4 weeks by appreciable atrophy. When the injured nerve supplies important extensor muscles of the limbs (e.g., radial, femoral, sciatic, peroneal), 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. 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 mechanical injury to peripheral nerves of the limbs are described below.

Suprascapular Nerve

Arises from C6 and C7. 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 2-4 weeks of injury, there is obvious atrophy of the supraspinatus and infraspinatus muscles (sweeney). 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 3-12 months. Regrowth of the suprascapular nerve can be facilitated by surgery to resect scar tissue (external neurolysis) and reduce tension on the nerve as it crosses the front of the scapula (often performed 3-6 months after injury)
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 fracture and can be injured at the brachial plexus by trauma to the shoulder region. Ischemic damage may occur in horses anesthetized in lateral recumbency. The lower part of the nerve may be damaged 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 rest 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.

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/anesthesia over the dorsomedial aspect of the knee and proximal metacarpus and atrophy of the biceps and brachialis muscles.

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 at the brachial plexus or along the medial aspect of the upper limb. There is a “tin soldier” gait, with decreased flexion and dragging of the toe and hypalgiesia/analgesia of the skin of the caudal forearm, lateral metacarpus, and medial pastern areas and atrophy of the carpal and digital flexors.

Femoral Nerve

Arises from L3 to L5. The femoral nerve innervates muscles that flex the hip and extend the stifle. The nerve can be damaged by ilial, femoral or vertebral fractures. Ischemic injury caused by prolonged stretch or increased tissue pressure during anesthesia in dorsal recumbency or after severe prolonged dystocia. 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.

Sciatic Nerve
Arises from L5-S1. The sciatic nerve supplies important extensors of the hip and flexors of the stifle. Damage usually is a result of deep injections into the caudal thigh. 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 while 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/anesthesia over most of the limb except for the medial thigh.

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 pulled caudally as the horse attempts to bear weight. There is atrophy of the anterior tibial and long and lateral digital extensors and immediate cutaneous hypalgesia/anesthesia over the lateral metatarsus.

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.

Prognosis

With suprascapular neuropathy secondary to confirmed or suspected shoulder trauma, restoration of shoulder stability occurred in 3-12 months in 7/8 horses treated only with stall rest. 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, 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) usually resolves in days.

Neurologic Testing of Recumbent Horses

Examination
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.

Check long spinal reflexes (cervicofacial, cutaneous trunci, slap) and test cutaneous sensation systematically over the limbs and torso. 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 response. Sensory fields for some peripheral nerves of horses have been described.

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 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 (Fig. 50-9). 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 just behind the greater trochanter; (2) the gastocnemius reflex – strike the Achilles tendon close to its insertion; and (3) the anterior tibial reflex – strike the middle of the anterior 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 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 likely 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.

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 obstipation may be the presenting sign of rectal paralysis and urinary overflow incontinence is a sign of bladder paralysis.

Stringhalt (Classical Stringhalt, Bilateral or Australian Stringhalt)

Stringhalt, also known as springhalt (Shakespeare; Henry VIII, I, iii, 11-13) or, in German, hahnentritt, is a condition of horses characterized by sudden exaggerated flexion of one or both pelvic limbs during the swing phase of locomotion. Two distinct syndromes are recognized: classical stringhalt, a persistent condition of individual horses involving only one limb, and bilateral or Australian stringhalt which involves both pelvic limbs, occurs in outbreaks, and usually resolves without treatment. Classical stringhalt is seldom reported in the veterinary
literature but probably occurs worldwide. Bilateral or Australian stringhalt is well documented and, in addition to its eponymous occurrence,\textsuperscript{31,33,35} has been reported in New Zealand, the United States, Chile, Brazil, Japan, Italy, and France.\textsuperscript{15,16,20,25,26,40,41} In recognition of the syndrome’s worldwide occurrence in pastured horses, the terms bilateral, plant-associated stringhalt\textsuperscript{34} and acquired bilateral stringhalt\textsuperscript{41} have been used as alternatives to Australian stringhalt.

\textit{Etiology}

The cause of classical, “true” or “conventional” stringhalt is unknown. Foot conditions and articular lesions of the hock or stifle are possible risk factors.\textsuperscript{18,20,23,29} Individual cases have been associated with recent hoof trimming, hoof abscess, thigh muscle hematoma, and arthritis of the distal tarsal joints.\textsuperscript{29,31} Trauma to the proximal dorsal metatarsus, particularly over the digital extensor tendons, often precedes classical stringhalt and likely is an important factor in the development of this form of stringhalt.\textsuperscript{18,20,31} Months may elapse between injury and the onset of stringhalt.\textsuperscript{22}

In 1884, Kendall\textsuperscript{33} reported that bilateral stringhalt was associated with consumption of the common pasture weed \textit{Hypochoeris radicata} (also known as flatweed, castear, cat’s ear, or false dandelion). Most reported outbreaks of bilateral stringhalt occur in late summer or autumn among horses grazing drought-damaged pastures heavily infested with \textit{H. radicata}. Cases often occur following weeks to months of grazing on suspect pastures.\textsuperscript{16,25,27,31,35,36,41} Interestingly, some affected horses are thought to have developed a taste for flatweed, to the exclusion of other forage.\textsuperscript{25,27} Outbreaks in New Zealand and Chile involved horses that were grazing pastures overgrown with “true” dandelion (\textit{Taraxacum officinale}), another member of the family Asteraceae and a close relative of \textit{H. radicata}.\textsuperscript{15,20} Attack rates during bilateral stringhalt outbreaks are usually less than 50\% but vary widely.\textsuperscript{15,31} There are occasional reports in stabled horses with minimal paddock exposure {Barry 1956} and \textit{H. radicata} plants were not found in the pasture of 4 horses that developed bilateral stringhalt in the early summer in Japan.\textsuperscript{40} Mallow (\textit{Marva parviflora}) was implicated in a single outbreak in Tasmania.\textsuperscript{35} A bilateral stringhalt-like syndrome in horses in California, suspected to be a form of neurolathyrism, was reproduced by feeding the legume sweet pea (\textit{Lathyrus odoratus}).\textsuperscript{34}

Some attempts at producing bilateral stringhalt by feeding cut \textit{H. radicata} to horses have been unsuccessful.\textsuperscript{37} Concentrated extracts of \textit{H. radicata} caused no toxic changes when injected into laboratory animals.\textsuperscript{35} Recently, stringhalt was induced in a 6-month-old colt fed a daily average amount of nearly 10 kg \textit{H. radicata} harvested from a paddock where the disease had recently occurred. Clinical signs typical of bilateral stringhalt first appeared 19 days after feeding began. Interestingly, signs waned when an alternative source of \textit{H. radicata} was provided but appeared again once feeding of plants from the original paddock resumed. Fifteen days after cessation of flatweed consumption, the colt was clinically normal.\textsuperscript{16} Overall, this indicates that the concentration of toxin in \textit{H. radicata} is variable and often fails to reach clinically important levels.

\textit{Pathogenesis}
Bilateral stringhalt is a distal neuropathy primarily affecting large myelinated axons of the peripheral nervous system. In accordance with the principle that severity of axonal injury in distal “dying back” axonopathy is proportional to distance from the neuronal cell body, the longest nerves in the body - namely, the recurrent laryngeal nerves and the peroneal and tibial branches of the sciatic nerve - appear to be most severely affected. This also explains why tall horses are more prone to the condition than young horses, ponies, or small native Chilean breeds. The lesions of bilateral stringhalt are consistent with ingestion of a neurotoxin produced either by pasture weeds such as *H. radicata* or, less likely, by associated fungi. Many members of the plant family Asteraceae are known either to be poisonous or to produce pharmacologically active substances. For example, repin, a sesquiterpene alkaloid neurotoxin produced by the asters yellow-star thistle and Russian knapweed, causes equine nigropallidal encephalomalacia.

Normal gait in the pelvic limbs requires rhythmic alternating activation and inhibition of the myotatic anti-gravity reflexes. Sensory information regarding muscle length and position, obtained from muscle spindles and Golgi tendon organs, is used to regulate extensor and flexor muscle tone during locomotion. It is likely that bilateral stringhalt and some cases of classical stringhalt result from interference with myotatic reflexes. The immediate cause of pelvic limb hyperflexion in bilateral stringhalt presumably is progressive degeneration of large myelinated axons in tibial and peroneal nerve branches including: α motor neurons to skeletal muscle, 1A and 1B sensory neurons from muscle spindles and Golgi tendon organs, and γ efferents to muscle spindles. Classical stringhalt may be the final common sign for a variety of insults: besides interference with limb reflexes, mechanical effects of adhesions involving the muscles and tendons of the digital extensors of the pelvic limb may be important as well as painful conditions of the hock or distal limb.

**Clinical Signs**

The defining clinical sign of stringhalt is abrupt hyperflexion of the hock and stifle during attempted movement (Figure 1). In classical stringhalt only one limb is affected; by contrast, bilateral stringhalt is almost always bilateral although signs may be asymmetric. Stringhalt is most evident during backing, sharp turning, when going down a slope, after a sudden stop, and during the transition from standing still to walking forward. Signs may be exacerbated by excitement, cold weather, or hard exercise. The clinical presentation of stringhalt varies greatly, even among horses with bilateral stringhalt grazing the same pasture and can be graded using a 5 point scale. In mild cases flexion is only slightly exaggerated and pelvic limb action may be completely normal at gaits above the walk. Such horses are able to perform without impairment. At the other end of the spectrum, the upper joints of the limb flex so violently that the dorsum of the fetlock slaps against the horse’s abdomen with each stride. In such cases the extension phase of protraction is delayed and often features one or two small downward jerks of the hoof before the limb finally snaps into full extension and forcefully strikes the ground. When the condition is bilateral, extension of one pelvic limb may be sufficiently delayed that the swing phase is still in progress when the opposite limb begins to flex, leaving both hooves off the ground during much of the stride cycle. The result is a bouncing bunny-hopping gait in which “progression can only be accomplished with a series of bounds and plunges extremely painful to witness”. In the most severe cases, movement is impossible. An atypical bilateral stringhalt
syndrome is described where the forelimbs also are involved and there are signs of generalized weakness. There is carpal flexion or stiffness and toe-scuffing and knuckling in the fetlocks of all four feet.17,20,31,34,35 Some horses with atypical bilateral stringhalt are anxious and a few become recumbent and unable to rise. In a series of cases reported in New Zealand respiratory stridor was a common presenting sign in horses that later developed limb signs of stringhalt.17,20 More typically, a variable proportion of horses with bilateral stringhalt are shown by endoscopic examination to have abnormal laryngeal movement.20,31,40,41

Onset of stringhalt is usually sudden. In some cases of bilateral stringhalt, exaggerated hock flexion is preceded by a short period of toe-dragging and knuckling of the fetlocks that may also involve the thoracic limbs.27 Signs may fluctuate but tend to worsen over several weeks in horses with bilateral stringhalt, before plateauing, often for several months, then beginning to improve.31,36 Within 2 weeks after onset of severe bilateral stringhalt, atrophy of the muscles of the gaskin and, less obviously, the thigh and other parts of the body, becomes apparent. Muscles are affected in approximately the following order of severity: digital extensors of the pelvic limb, cranial tibial, dorsal cricoarytenoid > biceps femoris, gastrocnemius > gracilis > semitendinosus/semimembranosus > other muscles of pelvic and thoracic limbs.31,36,38 Horses with severe or atypical bilateral stringhalt may develop profound generalized neurogenic muscle atrophy that results in marked weight loss despite normal appetite and food consumption.20,27,32

**Diagnosis/Clinical Pathology**

The diagnosis of stringhalt is made clinically. Differentiation between classical stringhalt and bilateral/Australian stringhalt on clinical and epidemiologic grounds is usually straightforward especially when bilateral stringhalt occurs as an outbreak. Routine laboratory results, including hemograms and plasma chemistry tests, are normal.25,27,40 Electromyography of the long digital extensors of horses with bilateral stringhalt both at rest and during flexion reveals increased spontaneous electrical activity e.g. fibrillation potentials, positive sharp waves.31,32,40 This is consistent with neurogenic atrophy. Nerve conduction velocities of horses with bilateral stringhalt were greatly reduced compared with a normal horse31 and a recovering horse had less severe compromise. Histologic evidence of denervation can be seen in portions of lateral digital extensor removed for treatment purposes.

Differential diagnoses for horses with hyperflexion of one or both pelvic limbs, suspected of having stringhalt, include fibrotic myopathy, upward fixation of the patella, shivers, unclassified abduction syndromes, and spinal cord disease (for example, equine protozoal myeloencephalitis). For respiratory stridor obstructive mass, liver failure and chronic lead or organophosphate toxicity are other diagnostic considerations. The stumbling, knuckling phase that may precede bilateral stringhalt could be confused with spinal cord disease such as equine protozoal myelitis, botulism, or other peripheral neuropathy such as Scandinavian knuckling horse disease.28 The profound atrophy seen in some horses with chronic severe bilateral stringhalt is similar to that observed in equine motor neuron disease.

**Post-Mortem Findings**
Abnormal findings are restricted to peripheral nerves and skeletal muscles. Not surprisingly for a non-fatal condition, there are very few descriptions of post-mortem findings for horses in the early stages of bilateral stringhalt and none of horses with classical stringhalt.

At the onset of signs of bilateral stringhalt there likely is Wallerian degeneration and secondary demyelination of the distal portions of peroneal and tibial nerve branches and the recurrent laryngeal nerves.

In longstanding cases of bilateral stringhalt there is variable, often severe, atrophy of the muscles of the pelvic limbs, especially distally, and atrophy of the muscles of the larynx supplied by the recurrent laryngeal nerves. These muscles have histologic evidence of denervation including bundles of atrophic muscle fibres with prominent sarcolemmal nuclei interspersed with normal or hypertrophic fibres. Histochemistry reveals a reduction in type II fibres. The most severely affected muscles are diffusely fibrotic. In peripheral nerves there are decreased numbers of myelinated fibres, perineural fibrosis, and accumulation of myelin debris. Evidence of nerve regeneration is usually found including regenerating nerve clusters, onion bulbs, and Schwann cell proliferation. Recurrent laryngeal nerves, branches of the sciatic nerves, and variably other nerves are affected with a proximal to distal gradient of severity.

Treatment

Care should be taken to identify sources of pain (e.g. arthritis, hoof abscess, recent hoof trimming) that might account for stringhalt like signs. Signs of stringhalt were blocked in one horse by intraarticular anaesthesia of the distal tarsal joints; use of intraarticular corticosteroids permanently resolved the problem.

Several skeletal muscle relaxants have been investigated in horses with bilateral stringhalt. Phenytoin, an inhibitor of voltage-gated sodium channels, gives partial to complete remission of signs in horses given 10-15 mg/kg orally, once or twice daily. Mephenesin, which acts in the spinal cord to specifically inhibit polysynaptic reflexes, suppressed signs in a horse with chronic bilateral stringhalt but was ineffective in an outbreak of bilateral stringhalt. Baclofen, an analogue of the inhibitory neurotransmitter GABA had no effect in a horse with severe stringhalt.

Although the results of the procedure are unpredictable, surgical removal of the distal muscle belly and tendon of insertion of the lateral digital extensor effects a clinical cure in a proportion of horses with either classical stringhalt or bilateral stringhalt. Of 4 horses with classical stringhalt treated with lateral digital extensor myotenectomy, 2 resolved, and the other 2 improved partially. The surgery was precluded in 2 other horses because of adhesions involving the tendon. Responses to a survey of veterinarians in Australia indicated that more than 50% of over a 100 lateral digital extensor surgeries had been successful in eliminating signs of stringhalt. Even more impressively, 11 of 13 horses with bilateral stringhalt were normal within 12 days of surgery.

The surgery can be performed standing or in lateral or dorsal recumbency. An initial skin incision is made over the lateral digital extensor tendon immediately proximal to its junction...
with the long digital extensor and the tendon is isolated and exposed. A second incision is made over the muscle belly proximal to the hock and the muscle belly is dissected free of overlying fascia. The tendon is severed through the distal incision then the tendon is pulled through its sheath into the proximal incision by exerting traction on the muscle. The muscle belly is then incised 2 cm proximal to the musculotendinous junction. A modification of the technique, which may improve success rate, removes 7 to 10 cm of muscle.\(^\text{39,42}\) After the fascial and skin layers have been closed, a bandage is applied over the surgical site and maintained, with box-stall rest, for 2 to 3 weeks.

**Prognosis**

There is almost no information on outcomes for horses with classical stringhalt; however, it is commonly supposed that most cases persist although they may improve with time.\(^\text{39}\) Of one series of 4 cases that developed after proximal dorsal metatarsal trauma, one recovered, two improved, and one was unchanged for at least 6 months after onset of signs.\(^\text{22}\) Almost all cases of bilateral stringhalt recover normal gait and muscle mass if removed from toxic pasture. The few reported deaths are in horses that either are unable to stand or unable to move to sources of water and feed and are euthanized for humane reasons.\(^\text{17}\) Recovery times of 3 days to 3 years have been recorded with most horses taking 6 to 12 months {Huntington et al. 1989; Dixon and Stewart 1969}.\(^\text{23,31}\) It is unclear to what extent athletic performance returns after clinical recovery from severe bilateral stringhalt. There is evidence that laryngeal dysfunction may persist beyond the time of recovery from gait abnormalities.\(^\text{40}\)

**Prevention**

Exposure to pasture dominated by *H. radicata* or *T. officinale* should be avoided, especially during adverse climatic conditions and in locations previously shown to be associated with stringhalt.

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