CHAPTER 85

Biology and Management of Muscle Disorders and Diseases

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STRUCTURE AND FUNCTION OF MUSCLES

Of the three types of muscle in the body—skeletal, smooth, and cardiac—skeletal muscle makes up about 42% of the body weight in nonathletic horse breeds and about 55% of the body weight in mature Thoroughbred racehorses.[1,2] All movement is the result of contraction of skeletal muscles, and most patients with neuromuscular disease exhibit abnormal movement. Skeletal muscle consists of a central fleshy muscle belly and a tendon at each end. The muscle and its tendon are arranged in the body so that they originate on one bone and insert on a different bone while spanning a joint. As the muscle contracts, shortening the distance between the origin and insertion tendons, the bones move in relation to each other, bending at the joint.[3] Extensor muscles increase the joint angle and flexor muscles decrease the joint angle.

Anatomy

Ninety percent of skeletal muscle consists of muscle cells (also called *myofibers* or muscle fibers), and the remaining 10% is made up of nerves, blood vessels, fat, and connective tissue.[2] The latter surrounds the muscle fibers (endomysium), fascicles (perimysium) and muscle belly (epimysium). A muscle belly has several levels of organization (Figure 85-1). Myofibers are multinucleated elongated cells that span the distance between the tendon ends and have a diameter between 5 and 100 µm. The outer limiting membrane of the fiber is called *sarcolemma*.[3]

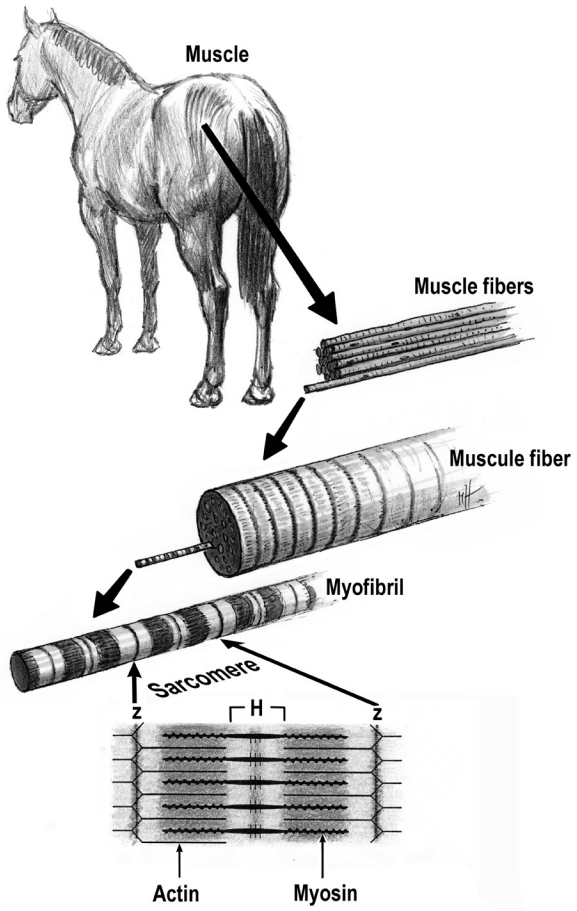

Figure 85-1. Levels of organization within a typical skeletal muscle.

Each myofiber contains thousands of myofibrils that are arranged parallel along its length. On longitudinal histological sections, myofibers show a typical striated pattern of lighter I-bands alternating with darker A-bands, perpendicular to their long axis. Within the I-bands a dense Z-line is visible. The repeating unit between the Z-lines is the *sarcomere*, the basic contractile unit of the muscle fiber.[2,3] At the level of the lighter I-bands, myofibrils only contain thin filaments, composed of the proteins *actin*, *tropomyosin*, and *troponin*. At the level of the darker A-bands, the myofibrils contain overlapping thin and thick filaments, the latter ones composed of the protein *myosin*. Within the A-band, the H-band is defined as the central area where the thick filaments do not overlap with the thin filaments.[2] Myofibrils are surrounded by membranes in the form of tubules that are composed of a transverse tubular system and the sarcoplasmic reticulum. Collectively they are called the *sarcotubular system*. Each muscle fiber is innervated by a terminal branch of an α-motor nerve fiber. The junction between the nerve and the muscle fiber is the neuromuscular junction. All skeletal muscle fibers that are innervated by a single α-motor neuron are called a *motor unit* because all of the muscle fibers within a unit are excited simultaneously and contract in unison.

Physiology of Contraction

It is at the neuromuscular junction that action potentials are generated. The resting membrane potential of the sarcolemma

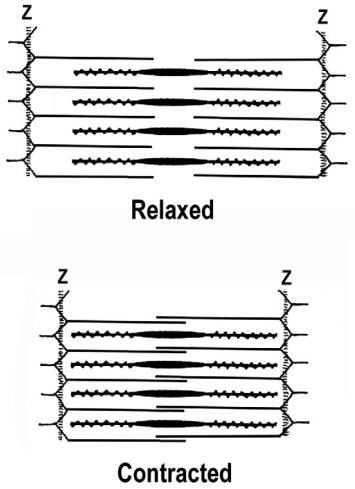

Figure 85-2. The sliding of actin along the myosin molecule results in the physical shortening (contraction) of the sarcomere.

is excited by a synaptic transmission at the neuromuscular junction. Via transverse tubules, the action potential reaches the sarcoplasmic reticulum throughout the muscle fiber.

Skeletal muscle cells convert chemical energy into mechanical energy. At rest, calcium ions are maintained within the sarcoplasmic reticulum of the muscle cell. The arrival of an action potential to the sarcoplasmic reticulum causes the release of calcium ions. These calcium ions perfuse the sarcomere and bind with troponin C causing a conformational change of the troponin-tropomyosin complex, consequently exposing the myosin-binding sites. The globular heads of myosin interact with actin and form a crossbridge that results in sliding of the actin relative to the myosin molecules and a shortening of the total length of the sarcomere (Figure 85-2). This physical shortening of the sarcomeres and ultimately of the myofibril results in contraction and shortening of the muscle belly. As the action potential passes, calcium ions are returned to the sarcoplasmic reticulum, allowing the sarcomere to relax, resulting in lengthening of the myofibrils and muscle.

ATP is needed for this contraction, and creatinine phosphate can be used to replenish ATP stores from adenosine diphosphate (ADP). The enzyme creatine phosphokinase (CPK) catalyzes this conversion and is found in high concentrations within muscle cells. During muscle cell damage, the CPK that is normally contained within intact muscle cells can leak into the bloodstream. Therefore identification of an abnormally high concentration of serum CPK is used routinely to assess skeletal muscle damage. Muscle contraction is only about 40% efficient in converting chemical energy to mechanical energy, and most of the energy is lost as heat. This phenomenon is exploited during shivering to raise core body temperatures. During shivering, antagonistic muscle groups are activated so that they produce no useful work. The chemical energy lost as heat is transferred to the body core to increase temperature.

Muscle Fiber Types

Important differences in muscle fiber types exist, both within and between muscles. They can best be differentiated by analyzing the expression of the specific myosin heavy-chain (MyHC)

isoforms.[4] The MyHC isoforms that have been characterized in adult equine skeletal muscles are types I, IIA and IIX. Equine muscle fibers present either as single isoforms (I, IIA or IIX) or the hybrid form IIA + IIX (IIAX). Type I fibers are slow fibers and are highly efficient and economical for slow repetitive movements and to sustain isometric force. They are not power generators. In contrast, type II fibers create fast crossbridge cycling and develop force rapidly. Type IIX fibers have a 3 times higher velocity of contraction compared to type IIA fibers. Depending on the type and degree of exercise horses perform, the muscle fiber pattern changes. For the maintenance of posture, only type I motor units are required. As the intensity and duration of exercise increase, additional motor units are recruited in the rank order I, IIA, IIAX and finally IIX.[5,6]

Muscle Repair

Muscle has a very limited capacity for regeneration. Depending on the degree and type of injury, satellite cells—a subpopulation of adult skeletal muscle stem cells—are activated and migrate towards the damaged area where they fuse to form multinucleated myotubes that further differentiate over several months to myofibers.[7] For practical purposes, however, new myofibers are not formed after birth. Enlargement and growth of muscle occur via enlargement of myofibers caused by formation of additional myofibrils. Repair of damaged muscle occurs by replacement with connective tissue. Muscle has a good blood supply and is a ready source of fibroblasts. Healing of damaged muscle with scar tissue is usually rapid, but replacement of damaged muscle with large amounts of scar tissue can restrict muscle length and function.

SPECIFIC MUSCLE DISORDERS

Fibrotic Myopathy

The classic description of fibrotic myopathy is that of a unilateral hind limb gait abnormality, characterized by an abrupt cessation of the cranial phase of the stride of the affected limb, with the foot suddenly jerked caudad just before it hits the ground (see Figure 85-3 and Video 85-1). This condition is most evident when the horse is observed at the walk from a lateral perspective.[8–14] The overall prevalence of the disease is low. In a study on 39 horses with fibrotic myopathy of the semitendinosus muscle, Quarter Horses (67%) and mares (39%) were overrepresented.[11]

Pathophysiology

In most horses, traumatic injuries that cause adhesions and fibrosis of the semitendinosus muscle are responsible for the condition. Injury to the semimembranosus, biceps femoris, and gracilis muscles has also been identified, though less commonly.[13,15,16] Trauma may result from activities that create extreme tension on the caudal aspect of the hind limb (barrel racing, the full slide of a reining horse), slipping while exercising, falling with the affected hind limb extended underneath the torso and struggling to extract an entrapped hind limb in halters, lead shanks, and fences. In one study, 5 of 18 horses with fibrotic myopathy developed the condition secondary to intramuscular injections.[8] The characteristic gait of horses with fibrotic myopathy can occur temporarily after transportation in a trailer. In this case, it is caused by a localized myositis of the semitendinosus muscle because of prolonged pressure by the rear rope or bar on the muscle. Fibrotic myopathy in the neonate is speculated to be caused by trauma at or soon after birth, although the exact etiology is unknown in congenital cases.[17] Horses born with the gait characteristic of fibrotic myopathy have a taut semitendinosus muscle and tendon, but they do not have palpable fibrosis of the muscle. In a small series of horses, traumatic or degenerative neuropathy causing denervation of the distal semitendinosus muscle resulted in fibrotic myopathy.[9] The underlying neuropathy is the most likely cause in horses when the condition progresses to involve both limbs. If bone forms in the affected tissue, the condition is termed *ossifying myopathy*.

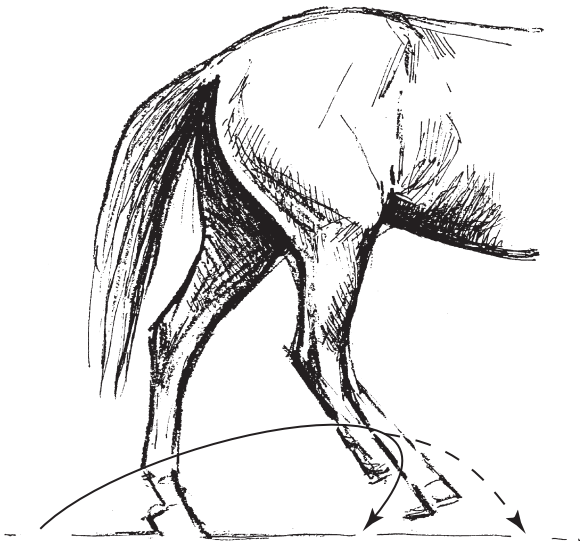

Figure 85-3. Typical motion of the hind limb of a horse affected with fibrotic myopathy of the semitendinosus muscle: the limb is jerked backward just before hitting the ground (see also Video 85-1).

Diagnosis

The diagnosis of fibrotic myopathy is made by observing the abnormal gait and by palpating the fibrotic or ossified muscle areas. This gait abnormality is not painful and is caused by mechanical restriction (either fibrous or osseous) of the affected thigh muscles. The gait abnormality is evident on every stride, is nonresponsive to analgesics, and is not altered by manipulative tests.

The fibrotic or ossified areas of the affected muscles may be palpated on the caudal aspect of the thigh at the level of the stifle joint or immediately above it. Ultrasonography may be helpful in defining areas of fibrosis, whereas radiography is more useful to demonstrate areas of ossification.[8,12,15]

Treatment

Several surgical procedures for the correction of this disorder have been described. These techniques include complete resection of the abnormal tissue,[8] transection of the fibrotic tissue with a scalpel[11,15] or a laser,[16] and a semitendinosus tenotomy/tenectomy near its medial tibial insertion.[12,17,18]

COMPLETE RESECTION OF THE FIBROTIC TISSUE

Complete resection of the fibrotic tissue is no longer recommended because of the relatively high rate of postoperative

complications, including protracted hemorrhage, wound dehiscence, high recurrence rate, and noncosmetic outcomes.[12,13]

TRANSECTION OF THE FIBROTIC TISSUE

Transection of the fibrotic tissue can be performed in the standing horse under local infiltration anesthesia over the affected site. A 5- to 10-cm vertical incision is made over the fibrotic area, just proximal to the musculotendinous union. After blunt dissection and isolation of the fibrotic mass with a forceps, sharp transection is performed.[11] Laser transection reduces bleeding and postsurgical inflammation.[16] A distal Penrose drain is used for 2 to 4 days after surgery.

SEMITENDINOSUS TENOTOMY

Semitendinosus tenotomy is probably the most commonly applied surgical technique. It is performed under general anesthesia in lateral recumbency with the affected limb down, providing access to the medial aspect of the tibia. Landmarks for surgery include the insertion of the muscle on the caudomedial aspect of the tibia just distal to the medial femorotibial joint and caudal to the saphenous vein overlying the gastrocnemius muscle. An 8-cm vertical skin incision is made directly over the palpable tendon and through the subcutaneous and crural fascia until the tendon is exposed. A large, curved Kelly or Crile forceps is passed under the tendon to isolate it from the muscle, and the tendon is transected (Figure 85-4). Resection of a 3-cm segment of the tendon obviates or delays recurrence of the gait abnormality. Fascial layers are closed with interrupted or continuous synthetic absorbable sutures, and the skin is closed with interrupted or continuous nonabsorbable suture material. When the affected limb is pulled forward during surgery, the tendon of insertion of the semitendinosus muscle onto the calcaneal tuber may become very taut in some horses. In that case, an additional 3- to 4-cm-long incision is made directly over the taut tendon, which is then isolated and transected. This incision is caudal and slightly distal to the first incision (see Figure 85-4).

Regardless of the technique, early and sufficient postoperative mobilization are important to decrease the formation of restrictive fibrosis at the transection site.[11,12,16]

Prognosis

Kinematic analysis before and after transection of the insertion tendon or the fibrotic area showed a significant reduction of the abrupt downward and backward movement of the affected limb immediately after surgery.[16] Long-term improvement is less pronounced, although still significantly better compared to before surgery.

Standing scalpel transection of the fibrotic area resulted in partial recurrence of the gait abnormality after surgery in approximately one-third of 39 horses treated, but only 10% regressed to the severity seen before surgery.[11] The tenotomy procedure alone results in few postoperative complications, including less predisposition for reformation of the fibrous band, but it may also not result in complete resolution of the gait abnormality.[17] If a degenerative neuropathy is the cause of the condition, recurrence of clinical signs is likely, regardless of the surgical technique employed.

Stringhalt (Equine Reflex Hypertonia)

Stringhalt, also renamed *equine reflex hypertonia*,[19] is a gait abnormality characterized by involuntary and exaggerated flexion of one or both hind limbs.[20–24] During the cranial phase of the stride, the limb is jerked toward the abdomen (Figure 85-5, Video 85-2). There may be a wide variation in the severity of the signs that occur during every stride at the walk. All breeds can be affected.

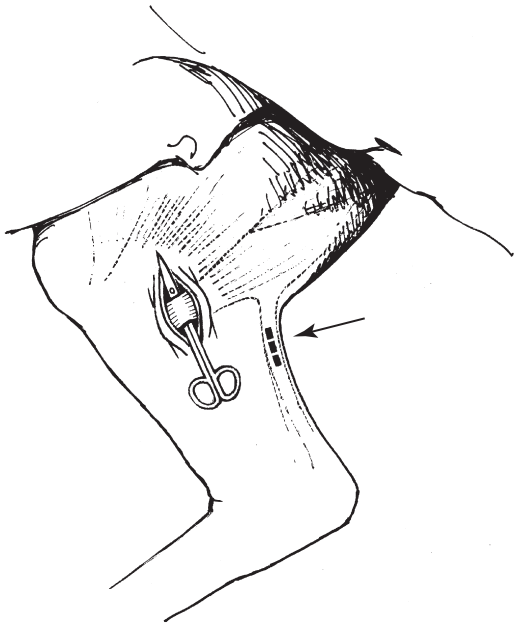

Figure 85-4. Semitendinosus tenotomy. The tendon of the tibial insertion has been isolated on the medial side of the proximal tibia and is ready to be severed. The dashed line *(arrow)* indicates the location for the second incision, which is performed if the insertion of the tendon on the *tuber calcaneus* is taut.

Figure 85-5. Typical motion of the hind limb in a horse with stringhalt: involuntary and exaggerated flexion of one or both hind limbs. (See also Video 85-2.)

Pathophysiology

Two clinical presentations of stringhalt have been described:

CLASSICAL STRINGHALT

Classical stringhalt affects isolated cases and is usually unilateral. The exact etiology of the condition is unknown and it is therefore also referred to as *idiopathic stringhalt*. In some cases however, the condition has been linked to trauma to the dorsoproximal aspect of the metatarsus (MTIII).[22] It is unclear whether the underlying cause is adhesion of the extensor tendons or damage to the sensory nerve endings in the muscle or tendons.[25] Classical stringhalt does not resolve spontaneously.

AUSTRALIAN STRINGHALT

Australian stringhalt is a plant-associated form of stringhalt resulting from the ingestion of several related weeds such as *Hypochaeris radicata* (Australian dandelion), *Taraxacum officinale* (European dandelion), or *Malva* (mallow).[20,21,23,26–30] As the name suggests, Australian stringhalt is most common in Australia, but it has also been reported in New Zealand, North America, Brazil, Chile, Japan, and Europe.[23,26–31] Outbreaks typically occur in horses on pasture during the late summer or fall, particularly after a dry season. There is also a report of a horse developing the condition after ingestion of Hypochaeris radicata in hay.[30] Although the first signs may start on one hind limb, Australian stringhalt is typically bilateral and is also called *acquired bilateral stringhalt*.[23] The onset can be progressive or sudden and depending on the severity of clinical signs, the condition has been graded from I (clinical signs only apparent when the horse is backed or stressed) to V (horse is reluctant to move and hind limbs are kept hyperflexed for minutes).[20] For recumbent horses with hyperextended and tonic hind limbs an additional Grade VI was identified.[30] Australian stringhalt is a diffuse peripheral neuropathy that mainly affects long, large myelinated nerve fibers and results in denervation atrophy of the corresponding muscles. Not only the peripheral nerves of the hind limbs, but also the recurrent laryngeal nerve is commonly affected and horses may also present with clinical signs of laryngeal dysfunction.[20,21,30] Many horses recover without treatment.

Diagnosis

The diagnosis is based on observation of the characteristic gait caused by exaggerated flexion of one or both hind limbs. Some horses show a very mild flexion of the hock during walking, and others show a marked jerking of the foot toward the abdomen, with the foot actually hitting the abdomen in severe cases (see Figure 85-5). The abnormal gait is usually evident with each step (see Video 85-2). Rest and cold weather tend to accentuate the gait, and in some horses, the gait may return to normal with exercise.

Although the gait abnormality has a classic appearance, it should be differentiated from mild forms of intermittent upward fixation of the patella. The patella catches on the medial trochlea of the distal femur in intermittent upward fixation of the patella, and when it releases it gives the appearance of a horse with stringhalt. Acquired bilateral stringhalt should also be differentiated from shivering and especially when walking backwards both disorders can have a very similar clinical appearance.[32] When walking forward, hyperflexion in acquired bilateral stringhalt cases is more abrupt, rapid, earlier in the swing phase and lacks the abduction of the limb that is typically seen in shivering cases.[32]

Treatment

Prevention of ingestion of the plant in cases of *Australian stringhalt* resulted in spontaneous recovery in 50% to 78% of the horses over a period ranging from a few days to 2 years.[20,26,30] Severely affected horses may not recover completely and recurrence has been described.[30] Medical treatment with Phenytoin, a centrally acting anticonvulsant, can be used to reduce peripheral nerve activity and excessive muscle contractions.[33] Although surgical treatment is not recommended for horses with Australian stringhalt, good results were reported in 13 horses.[23] Five horses were able to walk normally immediately after surgery (see later) in both hind limbs, whereas only partial improvement was noted immediately postoperatively in the remaining six horses. Five of them resumed a normal gait after 4 to 12 weeks. The last horse suffered a relapse several weeks postoperatively and eventually recovered spontaneously 9 months after surgery.[23]

Because *idiopathic stringhalt* is rarely associated with spontaneous recovery, lateral digital extensor tenectomy and partial myectomy is the treatment of choice for these horses.[22,34]

The objective of *lateral digital extensor (LDE) tenectomy and partial myectomy* is to remove the LDE tendon, from the last 2 to 10 cm of the muscle to its attachment to the long digital extensor tendon. The procedure may be performed using local anesthesia, or under general anesthesia in dorsal or lateral recumbency. For the standing procedure, a 2-cm line of local anesthetic is injected over the LDE tendon just proximal to its junction with the long digital extensor tendon on the lateral aspect of the MTIII. This site is easily palpable just below the tarsometatarsal joint. Local anesthetic is also infiltrated directly into the muscle belly of the LDE, about 2 cm above the lateral malleolus of the tibia.

The distal incision is made directly over the tendon just proximal to its junction with the long digital extensor tendon. The tendon is subsequently exposed by bluntly dissecting beneath the tendon with a curved Kelly or Ochsner forceps (Figure 85-6, *A*). The proximal incision is located on the lateral aspect of the limb approximately 6 cm above the lateral malleolus of the tibia. It extends through the skin, subcutaneous tissue, and fascia directly over the LDE muscle in a vertical direction, parallel with the muscle fibers. The muscle belly is exposed using blunt dissection, and a heavy curved instrument is placed underneath it (see Figure 85-6, *A*). Before severing the tendon, the surgeon should ensure that the tendon in the distal incision corresponds to the muscle belly in the proximal incision when retracted with the curved instrument. The tendon is severed in the distal incision and is pulled through the tendon sheath by traction on the proximal portion with the heavy curved instrument (see Figure 85-6, *B*). Removing the tendon from its sheath is a maneuver that requires substantial force, particularly if adhesions are present. After the entire length of the tendon has been exteriorized, the muscle is severed at the proximal aspect of the incision, thereby ensuring that at least 2 cm of the muscle is removed (see Figure 85-6, *C*).

The heavy fascia of the proximal incision is closed with a simple-interrupted or continuous pattern of USP size 0 or 1 synthetic absorbable suture material. The subcutaneous tissue is closed with USP size 2-0 synthetic absorbable suture material using a simple-continuous pattern, and the skin is closed with monofilament suture material in a simple-continuous pattern.

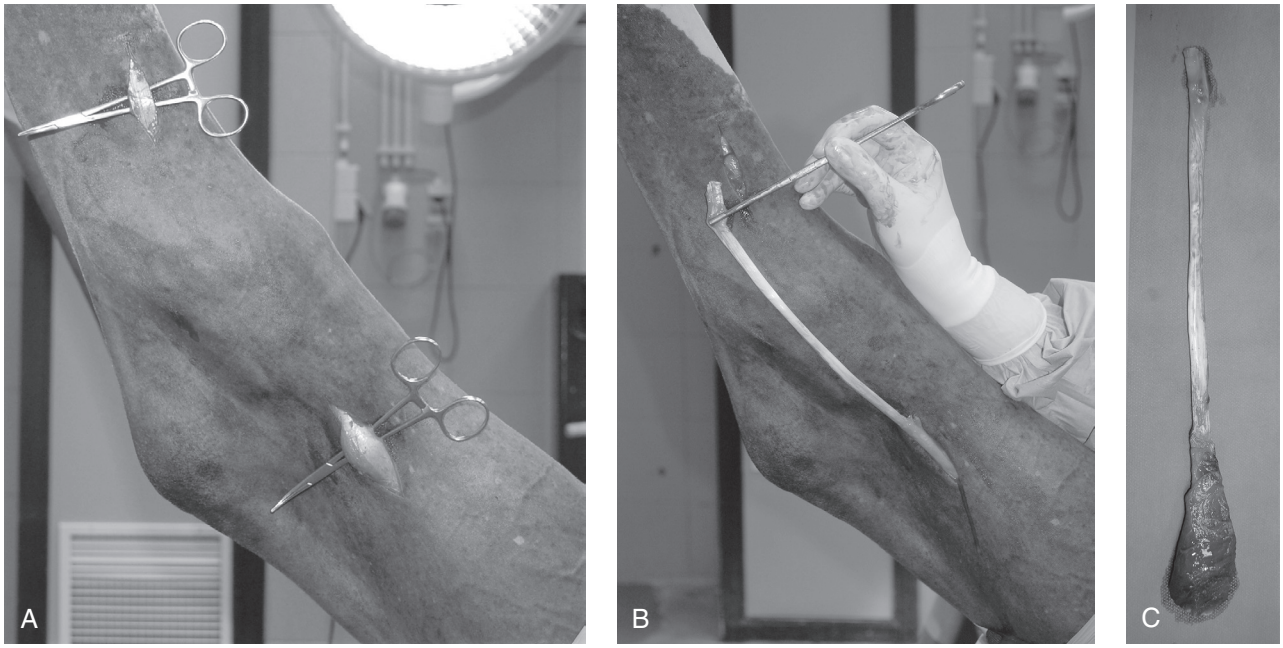

Figure 85-6. LDE tenectomy and partial myectomy. (A) The muscle belly and tendon of the LDE muscle have been isolated through two separate incisions and are elevated with a forceps. (B) The LDE tendon has been transected at the distal incision and has been pulled through the proximal incision. (C) The removed tendon including at least 2 cm of the muscle.

The distal incision is closed with skin sutures only. The wounds are covered with a sterile dressing, and the entire limb is bandaged for 14 days to control seroma formation. The horse is confined to a stall for this time, after which the sutures can be removed. The horse is then given 1 week of controlled hand walking, and normal exercise can resume 3 to 4 weeks after surgery.

Complications of surgery include seroma formation, dehiscence, infection, and failure of the abnormal gate to improve.

Prognosis

The prognosis for *Australian stringhalt* is fair. Many horses recover spontaneously, although a few suffer from stringhalt of such severity that they are euthanized.[30] The prognosis for *classical stringhalt* is guarded to favorable, because surgery is unpredictable and can result in partial to complete recovery. Many horses show improvement after surgery, but the degree of improvement varies from slight to a complete remission.[22] Improvement may occur immediately or take several months.

Peroneus Tertius Disorders

The peroneus tertius (PT) (also known as fibularis tertius) is an entirely tendinous muscle in the horse that lies in between the long digital extensor tendon and the tibialis cranialis muscle on the dorsal aspect of the crus. It originates together with the long digital extensor muscle in the extensor fossa on the cranial lateral aspect of the distal femur. At the level of the proximal lateral aspect of the trochlear ridge of the talus, the PT forms a sleeve through which the tibialis cranialis muscle passes.[35,36] Distal to this sleeve, the tibialis cranialis tendon divides into medial and dorsal branches. The PT tendon divides into four distinct tendons attaching to portions of the central, 3rd and 4th tarsal bones, MTIII and the calcaneus.[37]

The PT tendon is an important part of the reciprocal apparatus that coordinates flexion and extension of the stifle and tarsus (Figure 85-7). The tarsocrural joint is flexed by the active contraction of the tibialis cranialis muscle and the passive pull of the tendinous peroneus tertius.

The PT can rupture or be injured anywhere along its course. In a retrospective study of 27 cases, the location of the PT rupture was identified in 24 cases: 2 cases involved the origin (within 5 cm of its attachment to the distal femur), 11 cases the midbody, and 11 cases the distal aspect of the PT, just proximal to the sleeve formed by the PT.[35] Rupture at the origin of the PT tendon is typically associated with an avulsion fracture of the distal femur. Given their anatomic relationship, the long digital extensor muscle must also be involved. Ruptures at the origin of the PT are usually seen in foals[38–41] but they have also been reported in adult horses.[35] It has been speculated that foals may be more likely to suffer avulsion injuries as a result of inherent weakness in their bones.[40]

Pathophysiology

Trauma is the most common cause for rupture to the PT. By virtue of the anatomy described earlier, flexion and extension of the tarsus and stifle must occur simultaneously if the PT is intact. Therefore rupture of the PT has been surmised to be caused by an overextension of the tarsus while the stifle is held in flexion. This may occur when the horse slips with a hind limb extended backward, during the exertion of a rapid start, or when the limb is trapped and the horse struggles to free it. A full-limb cast extending to the proximal tibia may also cause rupture if the horse slips with the leg extended behind or struggles against the cast.[42] In a retrospective study of 27 cases, blunt trauma resulting in a closed injury was caused by falling down (10 cases), slipping (4 cases), limb caught in stall bars (3 cases),

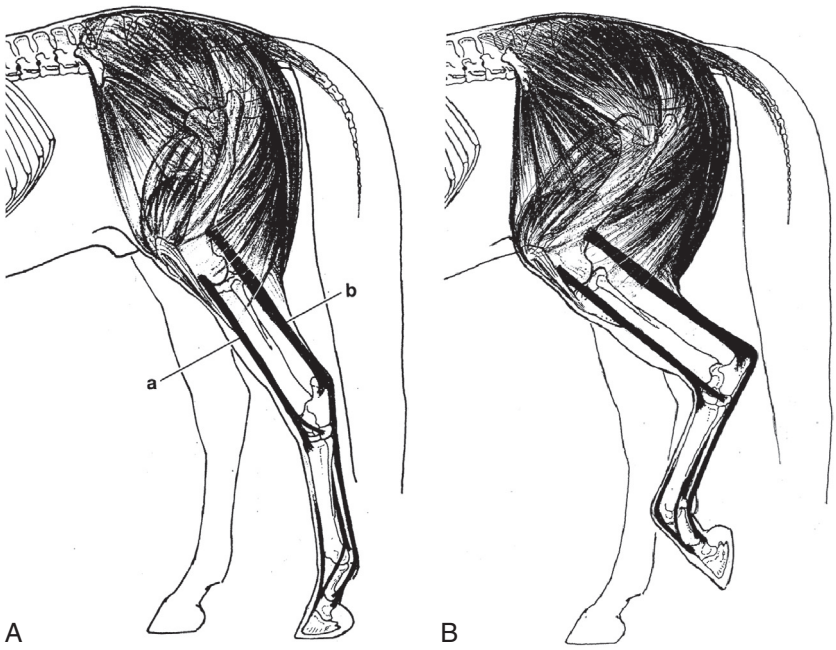

Figure 85-7. (A) Correlation of the anatomic structures composing the reciprocal apparatus of the horse. *a,* The peroneus tertius tendon originates on the muscular fossa of the distal femur and inserts at the small tarsal bones as well as MTIII, whereas the lateral part courses towards the fourth tarsal bone. *b,* The gastrocnemius muscle originates from the plantar fossa of the distal aspect of the femur and with the Achilles tendon, unites with the soleus and accessory tendons before it inserts on the tuber calcanei. (B) Illustration of the hind limb reciprocal apparatus demonstating that flexion of the tarsus or stifle results in flexion of all remaining joints in the hind limb.

and running into a solid object (2 cases). The PT was disrupted in 6 of 27 cases because of a laceration resulting in an open injury.[35]

Diagnosis

The classic sign of a complete PT rupture is the ability to extend the tarsus while the stifle is flexed.[35] A characteristic dimple forms in the common calcaneal tendon on the caudal lateral aspect of the crus, just proximal to the calcaneal tuber (Figure 85-8). When standing, the horse may bear weight normally, but when the horse walks the tarsus does not flex along with the stifle (Video 85-3). A lameness at a trot was reported as a Grade 1 (out of 5) in 3 horses, a Grade 2 in 6 horses, a Grade 3 in 10 horses, and a Grade 4 in 2 horses.[35] With avulsion injuries, horses can be severely lame initially, and there may be accompanying distention of the femorotibial and femoropatellar joints.[35,38–41] In the acute phases of a rupture, digital palpation may reveal some swelling and pain in the craniolateral aspect of the tibia, but typically there is little evidence of pain on physical examination.

The clinical diagnosis is confirmed using ultrasonography of the craniolateral aspect of the crus. The PT tendon can normally easily be recognized as the most echogenic structure. Rupture results in an increased size and a heterogeneous appearance of the tendon (Figure 85-9).[43] Radiography of the stifle is necessary for diagnosing avulsion fractures.

Treatment

Horses with avulsion injuries of the origin of the PT tendon are best evaluated arthroscopically.[38–40] Small bone fragments can be removed, but large bone fragments that are incorporated into the joint capsule are better left in place. Six weeks of stall rest followed by controlled exercise for 3 months is recommended. Ruptures of the midbody and insertion are treated conservatively using stall rest and a controlled return to exercise. Lacerations are treated with routine wound management.

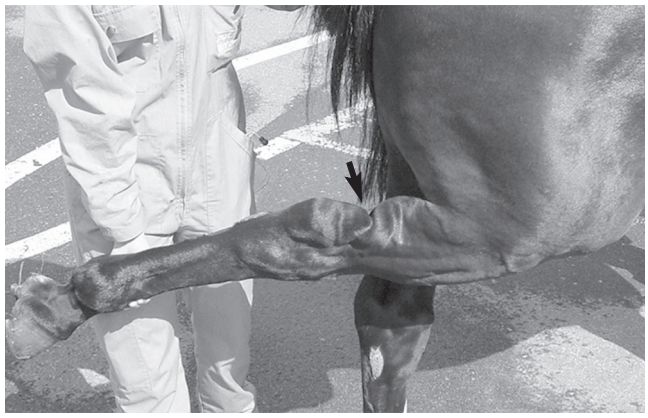

Figure 85-8. A horse with rupture of the peroneus tertius. The tarsocrural joint is extended while the stifle is flexed. Note the characteristic dimple in the contour of the caudal distal aspect of the crus *(arrow).* (See also Video 85-3.)

In a retrospective study of 27 cases, rupture of the PT was treated with stall rest for a median time of 16 weeks (range, 0–52 weeks), with or without hand walking. After stall rest, horses were turned out to pasture for a median of 12 weeks

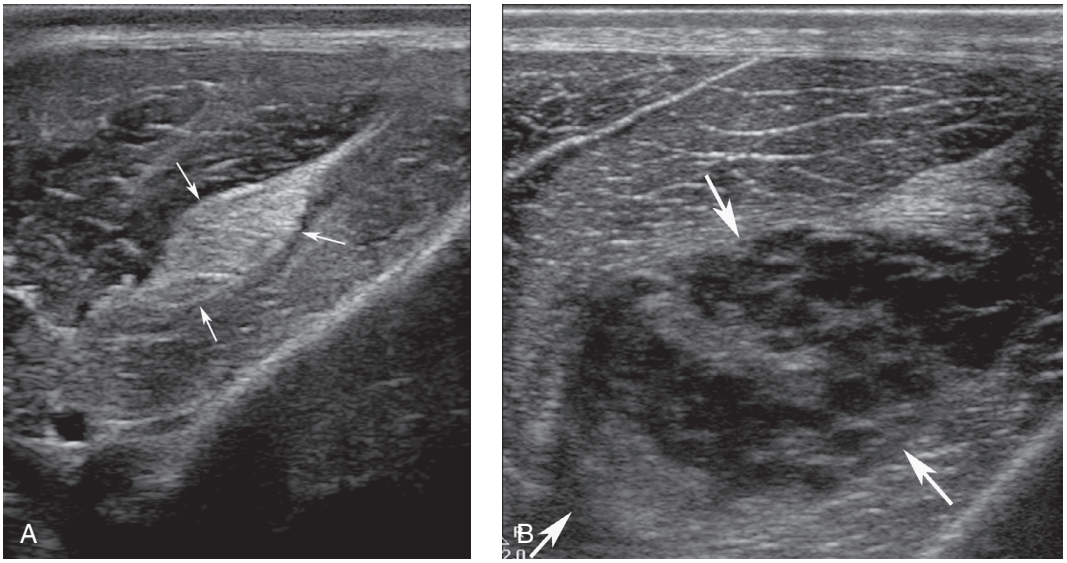

Figure 85-9. (A) Normal transverse ultrasonographic image of the PT in the proximal aspect of the crus. The PT is visible as the most echogenic structure *(arrows)*. (B) Transverse ultrasonographic image of a midbody rupture of the PT with complete loss of the normal tendon architecture *(arrows)*. (Courtesy K. Vanderperren, Ghent University.)

(range, 4–48 weeks) and were then gradually returned to their previous level of exercise over a mean period of 8 weeks (range, 4–12 weeks).[35]

Prognosis

Of the 23 horses available for long-term follow-up, 18 (78%) returned to their previous level of exercise, and 5 (21.7%) were euthanized because of persistent lameness.[35] Premature return to exercise was incriminated as the cause for reinjury, which occurred in 3 horses. Horses were significantly less likely to return to their intended use if they were racing at the time of injury (13 times less likely) or if a structure in addition to the PT tendon was damaged at the time of injury (8 times less likely). Neither age, an open or closed injury, lameness at presentation, ultrasonographic size of the lesion, location of the rupture, nor duration of rehabilitation had a significant influence on the outcome.[35] The value of serial ultrasonographic examinations to monitor the healing of the PT tendon before returning a horse to exercise was emphasized.

Adult horses with proximal avulsion injuries have a guarded prognosis for full soundness, but in foals several successful cases have been reported.[38–41]

Rupture of the Gastrocnemius Muscle

The gastrocnemius muscle has a medial and lateral muscle belly, both of which arise from the caudodistal aspect of the femur. They combine to form the gastrocnemius tendon, which inserts on the calcaneus and is a major part of the calcanean tendon. The gastrocnemius muscle is a component of the caudal aspect of the reciprocal apparatus, together with the superficial digital flexor muscle and tendon.

Only few cases of rupture of the gastrocnemius have been documented in mature horses [44,45] and the condition is more common in foals, where it has been associated with dystocia, assisted delivery and uncoordinated attempts to stand postpartum.[46,47] Rupture can also be caused by external trauma or by falling with the hind limb extended under the body.[45]

Diagnosis

The gastrocnemius muscle typically tears from its origin at the caudal and distal aspect of the femur, and the avulsion may be partial or complete. The clinical signs are determined by the degree of dysfunction of the reciprocal apparatus. In partial ruptures, the animal is acutely lame and shows a partially dropped tarsus or a gait abnormality characterized by lateral rotation of the calcaneus and medial rotation of the toe.[44] In total ruptures involving gastrocnemius and superficial digital flexor muscles, the reciprocal apparatus is dysfunctional and the animal cannot bear weight on the affected limb. When an attempt is made to place weight on the limb, the tarsus collapses and the stifle joint becomes hyperextended (Figure 85-10, *A*).[46,47] The diagnosis is confirmed with ultrasonography showing disruption of the gastrocnemius muscle, usually from its origin on the caudodistal femur. Radiographic examination of the stifle should be performed to rule out additional pathology and avulsion fractures from the supracondylar region of the femur.[46,47]

In a retrospective study of 28 foals with gastrocnemius rupture, concurrent disease processes such as enterocolitis, flexural limb deformities, encephalopathy, pneumonia, rib fractures, sepsis, spinal cord trauma, and superficial digital flexor tendon rupture occurred in 61% of affected animals.[47]

Treatment

Management involves stall rest alone or in combination with stabilization of the affected limb in the presence of significant disruption of the reciprocal apparatus.[47] Stabilization can be achieved using custom-made splints or sleeve casts applied to the dorsal or plantar aspect of the tarsus, keeping it in a normal weight-bearing position.[45,47] A tube cast over the tarsal region can also be used (see Figure 85-10, *B*). The metatarsophalangeal

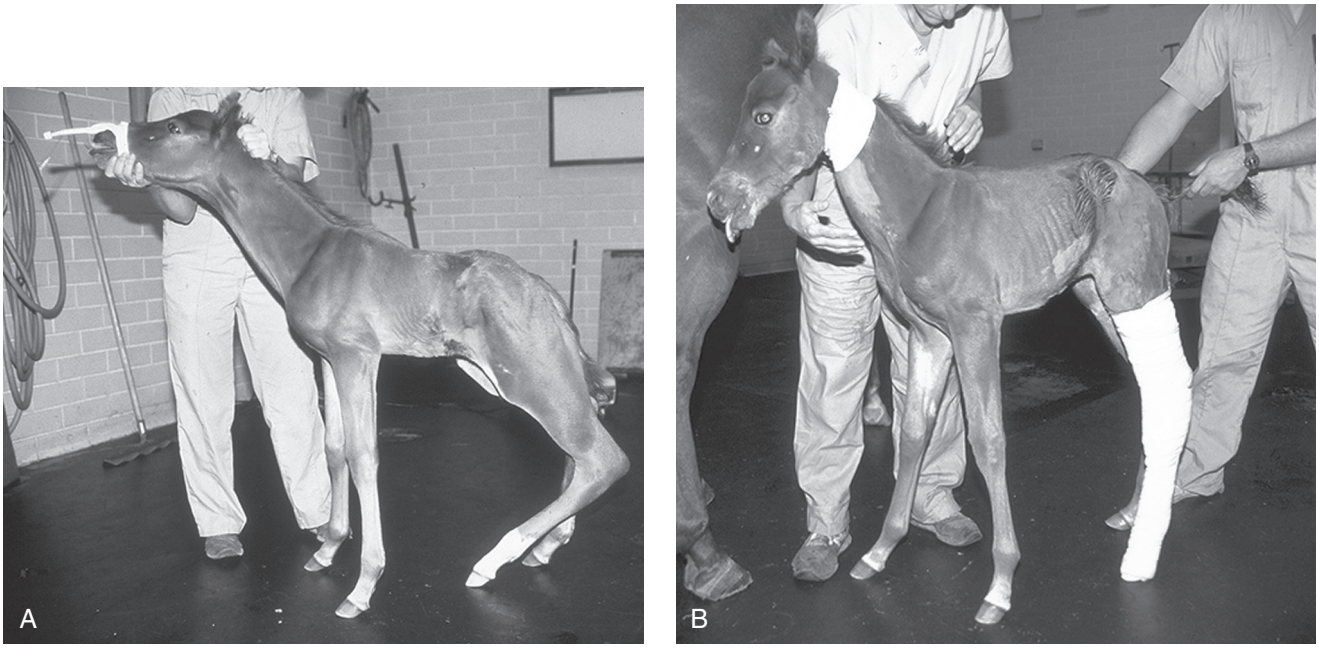

Figure 85-10. Three-week-old Quarter Horse foal with an avulsion of the gastrocnemius muscle. (A) Weight-bearing results in marked flexion of the tarsus and hyperextension of the stifle. (B) Application of a tube cast over the tarsus allows the foal to bear weight.

region should not be incorporated into the splint or cast to avoid tendon laxity. The splint or cast should be changed at regular intervals and be left in place for 4 to 5 weeks, followed by a Robert Jones bandage for an additional 2 to 4 weeks.[45–47]

The main complication is the development of pressure sores from prolonged casting or splinting.[46,47] Other complicating issues include acute hemorrhage warranting blood transfusion, and abscess formation in the gastrocnemius muscle.[47]

In adult horses, the prognosis is poor for complete ruptures, but favorable with conservative management of partial ruptures.[44,45] In foals, the prognosis is favorable for partial and complete ruptures. In a retrospective study of 28 thoroughbred foals with rupture of the gastrocnemius muscle, 82% of those aged 2 or more years at the time of the study were able to race.[47] Foals without a concomitant illness were significantly more likely to race.[47]

Rupture of the Extensor Carpi Radialis Tendon

Rupture of the extensor carpi radialis tendon is a rare condition in the horse and is usually the result of a traumatic insult, such as a kick or laceration at the distal cranial aspect of the radius, stumbling and falling on a road (coronation), or contact between a forelimb and a sharp object.

Diagnosis

Horses suffering from a rupture of the extensor carpi radialis tendon have a peculiar gait. Protraction of the involved forelimb results in hypermetric elevation, because the flexor muscles are not opposed by their main antagonist, the extensor carpi radialis muscle (Figure 85-11, Video 85-4).[48,49] Palpation of the cranial aspect of the carpal and distal radial region reveals a defect in the area of the missing tendon. In cases with a laceration, the distal tendon stump is often seen at the injury site.

Treatment

Lacerations should receive standard treatment (see Chapter 27): lavage of the tendon sheath, careful wound débridement, and skin closure with or without a drain. As the tendon typically ruptures at its insertion, suturing the tendon ends is usually not possible. The limb should be placed in a cast or splint bandage that prohibits carpal flexion for 6 weeks. If a drain has been used, the splint bandage should be changed after 3 days and the drain removed. The horse is restricted to a box stall and the splint bandage or cast is changed at regular intervals. Hand walking is initially the only exercise, and it is gradually increased.[48,49]

Prognosis

Generally, a good prognosis for return to a successful athletic career is given for rupture of the extensor carpi radialis tendon.[49] Complications include infection of the tendon sheath and dehiscence of the sutured tendon or laceration. In such cases, conservative management involving daily wound care and bandage changes is required. The prognosis in complicated cases is guarded for return to an athletic career.

Cribbing/Wind-Sucking

Cribbing or crib-biting is a stereotypical vice: a repetitive, relatively invariant pattern of behavior with no apparent goal.[50] A cribbing horse places his upper incisors on a fixed object, pulls backward, contracts the neck muscles and draws air into the cranial part of the esophagus emitting an audible grunt.[50–53] This behavior is also called "wind-sucking" or "aerophagia" because it seems that the horse is swallowing air. It has however been shown that cribbing is not related to deglutition and swallowing air into the stomach. There is a transient dilation of the upper esophagus and the characteristic noise of wind-sucking coincides with the

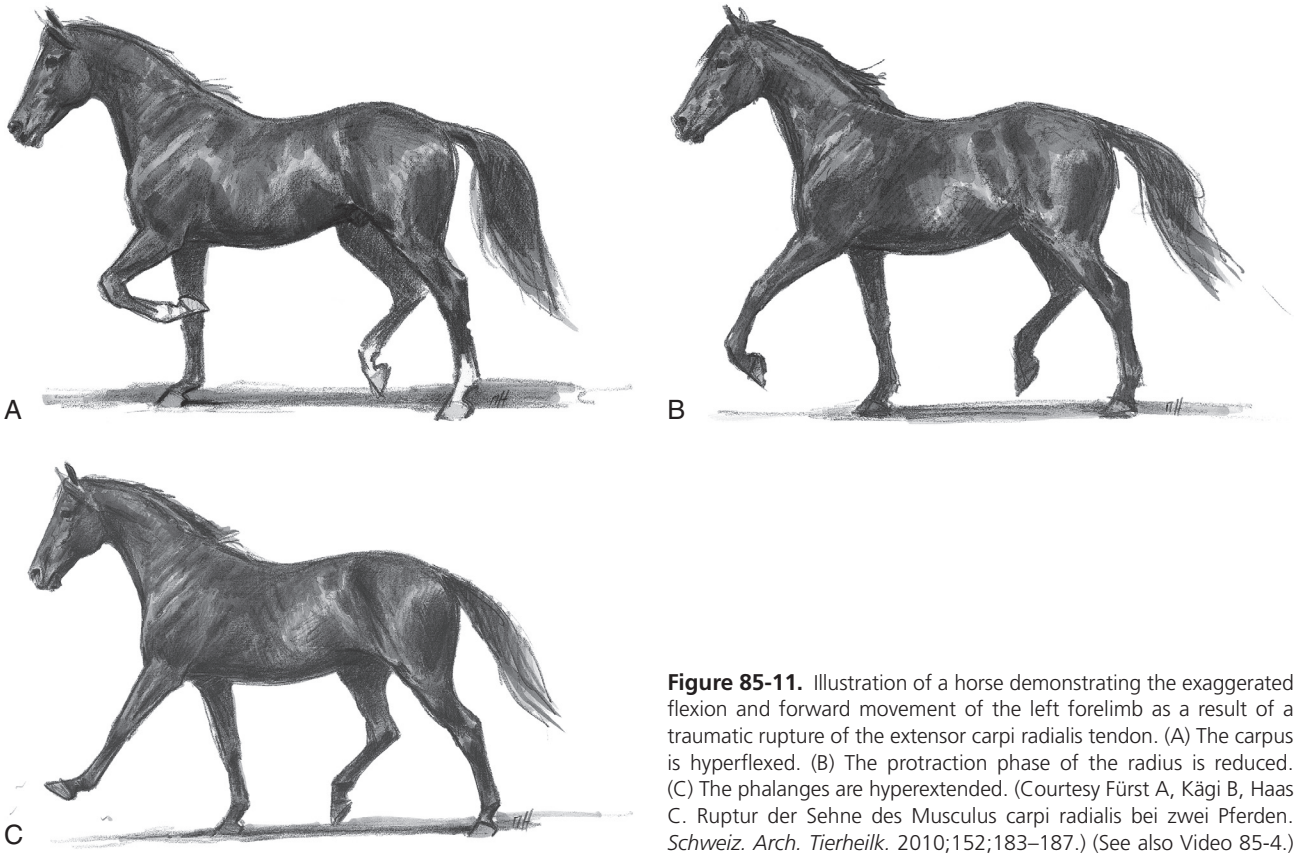

Figure 85-11. Illustration of a horse demonstrating the exaggerated flexion and forward movement of the left forelimb as a result of a traumatic rupture of the extensor carpi radialis tendon. (A) The carpus is hyperflexed. (B) The protraction phase of the radius is reduced. (C) The phalanges are hyperextended. (Courtesy Fürst A, Kägi B, Haas C. Ruptur der Sehne des Musculus carpi radialis bei zwei Pferden. *Schweiz. Arch. Tierheilk.* 2010;152;183–187.) (See also Video 85-4.)

in-rush of air through the cricopharynx. The esophageal dilatation is relieved when the air returns to the pharynx and only small quantities of air pass caudally.[51] The estimated prevalence of cribbing in North American and European horse populations is between 2% and 10%.[51–54] Cribbing has been associated to unthriftiness, tooth wear, temporohyoid osteoarthropathy, and chronic and recurrent colic.[55–57] More specifically, cribbing horses have been shown to have a 67 to 72 times higher risk for developing epiploic foramen entrapment.[58,59] A possible explanation for the latter is the recorded significant increase in intraabdominal pressure during cribbing and several hours afterwards.[60] Cribbing is generally viewed by owners as problematic and undesirable. Moreover, there is a certain degree of stigma associated with cribbing and affected horses may be refused to be stabled together with other horses because of the wide-held belief that horses copy the behavior from other cribbing horses.[53,54,61] When horses are isolated for that reason, a welfare issue can certainly be raised.

Etiology

The precise etiology of cribbing is unknown but is likely multifactorial. There seems to be a breed predisposition, with Warmbloods and Thoroughbreds having a 2 to 5 times higher risk for developing this oral vice compared to other breeds like Quarter and Arabian horses.[52,53,62] Although two studies recorded a higher prevalence of cribbing in stallions and geldings compared to mares,[52,63] more research is needed to confirm this sex predisposition. A genetic basis for cribbing has long been suspected and recently, the heritability of this trait in a population of cold-blooded Finn horses was estimated to be $h^2 = 0.68$. This is high and confirms that genetic factors do play a role in the onset of cribbing in horses.[63]

Cribbing has been associated with altered neuroendocrine physiology with the behavior being an adaptive response to stress.[50] Gastrointestinal irritation can also play a role and foals with gastric ulceration have been shown more at risk to develop cribbing.[64] Prevention of oral vicelike behaviors should be based on optimizing housing and management conditions, including paddock-weaning, allowing tactile contact and social interaction with other horses, daily pasture or paddock free movement, high amounts of roughage and little or no concentrates.[50,62,65]

Therapy

NONSURGICAL MANAGEMENT

Although therapy of cribbing should focus on prevention by providing optimal housing and management conditions as mentioned above, horses that have acquired this vice often do not respond to a change in environmental conditions alone.

There are several nonsurgical treatment options that either aim at preventing the horse from grasping a solid object with the incisors (electrified surfaces, surfaces with an unpleasant taste, muzzle, etc.) or use of a variety of collars that restrict or punish neck flexion. Leather straps with or without a ventrally located piece of articulating metal are placed snugly around the throatlatch of the horse to discourage contraction of the ventral neck muscles and arching of the neck. More severe straps have metal prongs that pierce the skin when the neck is flexed. Cribbing straps are adjusted to allow the horse to eat and breathe normally and are worn at all times except during exercise. A recent study comparing three of these nonsurgical treatment

options found all of them equally effective in reducing cribbing, though none of them could achieve a complete elimination of the behavior.[66]

SURGICAL MANAGEMENT

The original procedure described by Forssell in 1926 intended to inhibit the neck flexion in cribbing horses consists of a combined myectomy of the omohyoideus, sternothyrohyoideus and sternomandibularis. This surgical procedure has since been modified, mainly to reduce the cosmetic blemish that results from the resection of a large muscle mass in the ventral neck. One modifications is resection of the ventral branch of the accessory spinal nerve (cranial nerve XI) that innervates the sternomandibular muscle, the largest and most powerful muscle involved in cribbing. Because of the poor results achieved with this technique alone, the neurectomy is presently combined with a myectomy of the omohyoideus and sternothyrohyoideus, a procedure named the 'modified Forssell procedure'.[67]

Modified Forssell procedure

The horse is placed under general anesthesia in dorsal recumbency with the head positioned at a 30-degree angle to the horizontal.[67] After surgical preparation, a 30-cm incision is made on the ventral midline of the neck through the skin, the edges of which are retracted laterally, exposing the ventral surface of the paired bellies of the omohyoideus and the sternothyrohyoideus and the cranial aspect of the sternomandibularis muscle. Careful attention to hemostasis minimizes staining of the areolar tissue and consequently simplifies identification of the nerve. A plane of dissection is created on the medial aspect of the sternomandibularis muscle about 5 cm caudal to the musculotendinous junction. The ventral branch of the spinal accessory nerve is located on the dorsomedial aspect of this muscle by carefully rolling the muscle belly laterally. Curved hemostatic forceps are placed under the nerve, which is elevated until a sizable portion of it can be exteriorized. Contraction of the sternomandibularis muscle after the nerve is pinched with a hemostat forceps confirms isolation of the correct nerve. Removal of at least 10 cm in length from its insertion in the muscle is advised to ensure complete neurectomy.[67] This is important because the ventral branch of the accessory nerve divides into two to seven terminal branches before entering the sternomandibular muscle. This same procedure is subsequently repeated on the opposite side of the neck.

After the bilateral neurectomy, the myectomy is performed. A 30-cm (12-in) section of the combined bellies of the omohyoideus and sternothyrohyoid muscles is removed (Figure 85-12). These muscles are identified at the cranial aspect of the incision, just ventral to the larynx, and they are freed from the larynx and fascial attachments to the linguofacial vein and thyroid gland. Curved scissors or other curved instruments are passed under the muscles to ensure inclusion of all parts of the muscle bellies, which are then transected. These muscles bellies are subsequently grasped and "peeled" caudad. The areolar tissue connecting them to the trachea is sharply dissected. Using scissors, the omohyoideus muscles are sectioned obliquely and the sternohyoid bands transversely at the caudal aspect of the incision. The sternothyroid bands of the sternothyrohyoideus muscles are elevated from the trachea and removed. Special attention is given to hemostasis of the muscle stumps to prevent hematoma formation.

Two Penrose drains are inserted in the wound and exteriorized through separate stab incisions on both ends of the incision. The subcutaneous tissues are closed with a simple continuous suture pattern using USP No. 0 or 1 synthetic absorbable suture material, and the skin is closed with any suitable pattern and material. To help eliminate dead space, a stent bandage is sutured over the incision to provide constant pressure to the wound and to protect it during the initial phase of healing.

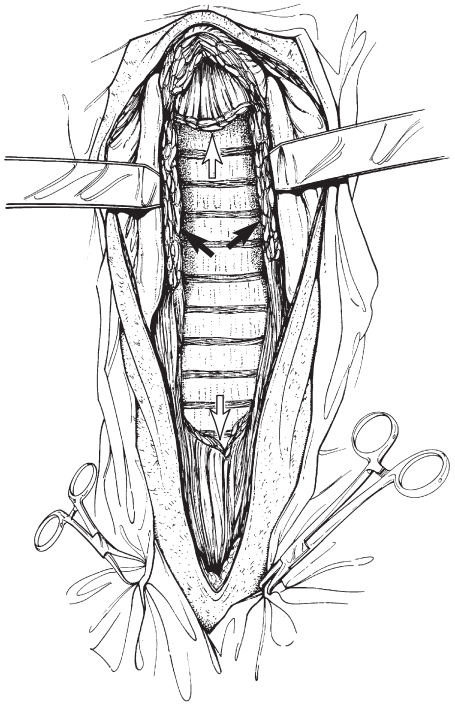

Figure 85-12. The modified Forssell procedure for cribbing is performed by a myectomy of the omohyoideus and sternothyrohyoideus muscles and by a neurectomy of the ventral branch of the spinal accessory nerve. *Black arrows,* Omohyoideus muscle stumps; *white arrows,* stumps of the sternothyrohyoideus muscles.

After surgery, the horse is confined to a stall or small paddock for 2 weeks. The stent bandage is normally removed 2 or 4 days postoperatively. The Penrose drains are removed 4 to 6 days after surgery, followed by the skin sutures 10 to 14 days postoperatively. Exercise can be resumed after 21 days. Hematomas or seromas can be managed conservatively or by drainage. Antibiotic therapy initiated perioperatively can be continued until the drains are removed.

More recently, two additional modifications were described to the above technique, both aiming at reducing the development of tendonlike fibrous tissue between the transected muscle ends that could result in recurrence of the behavior.[69] The first modification is a more cranial transection of the muscles. The head is positioned in full extension and the incision is made from the basihyoid bone extending 34 cm caudally. After the standard neurectomy procedure, the omohyoideus and sternohyoideus are excised at the level of the incision, and the tendinous insertions of the sternothyroideus muscles on the thyroid cartilage laminae are transected. The second modification is the use of a Nd:YAG laser to transect the muscles, to improve hemostasis and decrease the risk of postoperative hematoma and seroma formation.[69]

Prognosis

The modified Forssell procedure results in an excellent cosmetic appearance after surgery with no deformation of the neck and a

barely visible scar in the vast majority of cases.[68] Complete elimination of cribbing behavior is obtained in 30% to 100% of cases[68–73] but few studies have large case numbers. A recent long-term (>2 years) follow-up study on 33 crib-biting horses operated using the modified Forsell procedure showed a complete remission of cribbing behavior in 61% of cases.[73] However, a success rate of 80% was obtained when only horses operated within the first 6 months from the initial signs of crib-biting were included.[73] This association between cribbing duration and success of surgery was confirmed in a large long-term follow-up study of horses treated with the Laser assisted modified Forssell's procedure (90 horses evaluated for >1 year). There was an 86% reduction in odds of obtaining a successful outcome for horses cribbing for more than 3 years compared to horses cribbing less than 3 years.[68]

That same study also recorded wound healing complications (seroma, hematoma, wound infection, prolonged incisional drainage, and dehiscence) in 22% of horses, despite the use of the laser. A (univariate) significant association was found between wound healing complications and unsuccessful outcome, thus underlining the importance of good postoperative wound care. The authors indicated that maintaining the pressure bandage for 2 weeks was helpful to reduce dead space and seroma formation and shorten the overall recovery time.[68]

Calcinosis Circumscripta

Pathophysiology

Periarticular mineralized masses have been reported in several species, including horses.[74–76] In horses, these have been referred to as "calcinosis circumscripta" or "tumoural calcinosis."[74] In a retrospective study on cutaneous nodular and proliferative lesions submitted to a veterinary diagnostic lab on horses, donkeys, and mules over a 3.5-year period, 116 nonneoplastic nodular and proliferative lesions were identified and comprised 18% of all lesions submitted. Calcinosis circumscripta was identified as the most common nonneoplastic nodular lesion in horses up to 10 years of age.[77] They are most commonly located on the lateral aspect of the stifle joint, adjacent to the proximal fibula. There are isolated reports of lesions on the neck, shoulder, pectoral region[74] as well as two cases of partially or fully intraarticular calcinosis circumscripta lesions of the carpus.[76,78] Although the etiology of calcinosis circumscripta is unknown, it is likely to be initiated by a traumatic incident resulting in dystrophic mineralization.[74,76] Though few reports have investigated serum chemistries of affected horses, hypercalcemia and hyperphosphatemia have not been reported. This is in contrast to human tumoral calcinosis where a hereditary metabolic dysfunction of phosphate regulation results in massive periarticular calcinosis.[75] This is an argument not to use the terms 'calcinosis circumscripta' and 'tumoral calcinosis' interchangeably in horses.[76]

Diagnosis

During physical examination, a hard, spherical, well-circumscribed, nonpainful subcutaneous mass, 3 to12 cm in diameter can be palpated, typically at the lateral aspect of the lateral femorotibial joint. The overlying skin is intact and freely moveable, but the lesion itself is almost immoveable relative to the underlying structures.[74] Lesions may be bilateral. Typically, these horses present for cosmetic reasons and do not have any lameness.

On radiographic examination a soft tissue density, irregularly infiltrated with small, highly radiopaque amorphous granules is visible.[74,76] Histology performed after surgical excision confirms the diagnosis: the mass is composed of multiple loculi of various shapes and sizes that contain amorphous basophilic extracellular material and mineral deposition, surrounded by giant cells and macrophages. The surrounding capsule and internal septae are composed of mature fibrous connective tissue.[74,76]

Treatment

Treatment is indicated only if the cosmetic appearance is unacceptable to the owner, or if there is lameness caused by the mass. Surgical excision is the only treatment option. Attention to aseptic technique is imperative, since the mass often attaches to the lateral femorotibial joint capsule, which may be accidentally entered during surgery. Closing dead space, a tension-relieving closure using mattress sutures and a sutured stent bandage are advised because the wound at the lateral aspect of the stifle is prone to dehiscence.[79] If the joint capsule was opened, septic arthritis is a potential serious complication.[74,80]

Prognosis

For horses that are not treated, the mass remains and is unlikely to cause complications. The prognosis after surgical excision is good and regrowth has not been reported. Incisional complications are the most important reason for failure and attention to wound care is therefore very important.

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