HOW THE LIMB WORKS - GAIT ABNORMALITIES IN THE HORSE
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Some disorders cause a marked gait abnormality at a walk. The change in gait is most often due to a loss of mechanical or neurologic function, but it can also be a method of minimizing pain. This presentation discusses various gait abnormalities that can be observed at a walk and reviews the anatomy and limb movement associated with them. Increasing our understanding of these disorders helps increase our understanding of how the limb works. For a more detailed discussion of the treatment options the reader is referred to the references and current literature.

Fibrotic myopathy
Fibrotic myopathy is most often a result of trauma to the semitendinosus muscle with subsequent inflammation, hematoma formation and fibrosis or ossification. Involvement of the semimembranosus, biceps femoris and gracilis muscles is also possible. In cases where an inciting injury was observed, the hind limb has been caught cranially and underneath the horse or slipped forward excessively during sliding stops. It has also been reported after intramuscular injections.1-3 Two congenital cases and three cases associated with peripheral neuropathy have also been reported.1,4

Gait alterations
The gait associated with fibrotic myopathy is likely due to an effective shortening of the semitendinosus muscle and adhesions between the semitendinosus muscle and biceps femoris or semimembranosus muscles. This functional shortening limits protraction of the hind limb, and results in the limb being retracted just before ground contact and contacting the ground in a pronounced vertical slapping motion.

Anatomy
The semitendinosus muscle originates from the transverse processes of the first and second caudal vertebrae, the sacrosciatic ligament, and the ventral surface of the ischiatic tuberosity. It inserts on the tibial crest, crural fascia proper and tuber calcanei. The semitendinosus muscle is innervated by the caudal gluteal and sciatic nerves. Its action is to extend the hip and hock, and flex the stifle.

Treatment
Treatment options include tenotomy of the tibial tendon of insertion of the semitendinosus muscle, or a semitendinosus myotomy procedure involving transection of the muscle fibers at the distal extent of the fibrotic region, typically performed just proximal to the musculotendinous junction.5 The prognosis is favorable, but recurrence of restrictive scar tissue is possible. More extensive myotomy procedures and removal of calcified areas within the muscle have also been reported but have been associated with a higher complication rate.

Stringhalt
Several forms of stringhalt have been described. The Australian or outbreak form of stringhalt is bilateral, occurs in groups of horses on pasture, and is thought to be due to a plant toxin. It has been identified in Australia, New Zealand and California. The Australian form and possibly other forms of stringhalt have been shown to have an underlying neuropathy.6,7

Conventional or classic stringhalt occurs in individual horses and is typically unilateral. The majority of stringhalt cases have no known initiating factors. Some cases are associated with trauma to the dorsal proximal metatarsal region, with the suspected etiologies being adhesion formation involving the lateral digital extensor tendon and altered myotactic response due to injury.8 Other causes of stringhalt may be
peripheral neuropathy associated with neurologic disease such as equine protozoal myelitis. In one report a stringhalt-like gait improved after local anesthesia of the tarsometatarsal and distal intertarsal joints, and resolved after intra-articular corticosteroid treatment. A stringhalt-like gait can also be observed in horses with thin soles after trimming, and horses with hind limb laminitis. These horses have a normal or significantly improved gait on soft footing or after abaxial anesthesia.

Gait alterations
Stringhalt is a gait abnormality characterized by exaggerated hyperflexion of one or both hind limbs.

Anatomy
The lateral digital extensor muscle of the hind limb originates from the lateral collateral ligament of the stifle and the adjacent region of the tibia and fibula. It proceeds lateral to the long digital extensor muscle and enters its tendon sheath in the groove of the lateral malleolus of the tibia. In this region, the tendon and sheath are covered by extensive crural fasciae and the digital extensor retinaculum of the tarsus. Just distal to the tarsus the lateral digital extensor tendon joins the long digital extensor tendon. The lateral digital extensor muscle is innervated by the peroneal nerve. Its action is to extend the digit and flex the hock.

Treatment
When a horse is presented with a stringhalt-like gait, a dietary history, neurologic exam, and search for identifiable sources of pain should be undertaken. Treatment is based on these results if indicated. Lateral digital extensor myectomy/tenectomy is typically indicated for cases of stringhalt associated with dorsal metatarsal trauma or horses with stringhalt of unknown etiology. Improvement after surgery can be dramatic but results are variable and difficult to predict. Preliminary investigations on the effect of Botox treatment have been performed and this may lead to an alternative treatment option in the future.

Upward patellar fixation
Gait alterations
In a horse with upward patellar fixation, the limb is positioned with the stifle and hock held in extension with the distal limb held in partial flexion. Release of the limb often occurs with a quick and exaggerated flexion. Three situations have been described. Persistent fixation occurs when the patella stays fixed for a prolonged period of time, often requiring assistance or multiple attempts by the horse to release the patella. In cases of persistent fixation, the horse often walks with the limb dragged behind. Intermittent fixation occurs when the patella stays fixed with the limb held in extension behind the horse for several seconds and then is released during a normal step. Momentary fixation occurs when the patella temporarily fixes causing a slight delay in the start of protraction and a slightly exaggerated flexion when the limb is released.

Anatomy
The medial patellar ligament inserts distally in a groove on the proximal medial aspect of the tibial tuberosity and proximally on the medial aspect of the patella through the parapatellar fibrocartilage. Upward fixation of the patella occurs if during maximal stifle extension, the fibrocartilage is elevated and rotated over the medial trochlear ridge of the femur and appropriate release does not occur. Normally, the patella is rotated laterally and elevated slightly by the quadriceps to clear the medial trochlear ridge.

The quadriceps muscle has four components; the rectus femoris, vastus lateralis, vastus medialis and vastus intermedius which originate on the body of the ilium, lateral femur, medial femur and cranial femur respectively. The quadriceps insert on the patella and transmit their action through the patellar ligaments. Innervation of the quadriceps is by the femoral nerve. The primary action of the quadriceps muscle is to extend the stifle joint.
Treatment
Most cases of intermittent upward patellar fixation are young horses with poor quadriceps condition or horses that have had a period extended stall rest. These horses often respond to an exercise program that increases the strength of the quadriceps and surrounding musculature. Cases that are candidates for surgery have intermittent upward patellar fixation despite adequate conditioning programs or have persistent locking that cannot be manually released or recurs after release. Surgical options include splitting of the medial patellar ligament or transection of the medial patellar ligament.11,12

Partial origin of the gastrocnemius tear
Gait alterations
Partial injury to the origin of the gastrocnemius muscle can result in a gait abnormality characterized by slight hyperextension of the hock, lateral rotation of the point of the calcaneus and medial rotation of the toe during the stance phase. This gait is thought to be a method of relieving pain rather than a manifestation of dysfunction.13 Bilaterally, this gait is also observed in horses with generalized weakness and spinal ataxia.14

More extensive injury to the gastrocnemius muscle results in hyperflexion of the hock and partial loss of the reciprocal apparatus.15 Complete transection of the Achilles tendon or complete disruption of both the superficial digital flexor tendon and the gastrocnemius results in a completely dropped hock and minimal ability to bear weight on the limb.

Anatomy
The gastrocnemius muscle originates on the supracondyloid fossa of the femur and inserts on the tuber calcanei. The origin has both a lateral and medial head. The tendon of insertion moves laterally before it attaches deep to the superficial digital flexor tendon on the tuber calcanei. The gastrocnemius muscle is innervated by the tibial nerve. Its action is to flex the stifle joint and extend the hock.

Treatment
The primary treatment option for partial tearing of the gastrocnemius origin is extended rest. The prognosis appears to be good if disruption of the reciprocal apparatus is not evident. Disruption of the caudal component of the reciprocal apparatus carries a guarded prognosis and depends on the degree of disruption.15,16

Sweeney
Sweeney is a condition characterized by shoulder instability and atrophy of the supraspinatus and infraspinatus muscles. Local anesthesia of the suprascapular nerve produces the characteristic sweeney gait, confirming that lack of suprascapular nerve function is a primary cause of the gait abnormality.17 Damage to the suprascapular nerve may be due to direct impact trauma or may be due to a stretching trauma as the nerve courses around the neck of the scapula.18

Gait alterations
During the weight bearing phase of the stride the proximal portion of the humerus moves laterally, resulting in lateral subluxation of the shoulder joint.

Anatomy
The suprascapular nerve originates from the cervical segments C6 and C7 which then converge at the cranial aspect of the brachial plexus. After coursing through the plexus, the nerve courses laterally and over the cranial aspect of the scapular neck prior to innervating the supraspinatus and infraspinatus muscles.
The supraspinatus originates on the supraspinatus fossa and spine of scapula. It inserts on the greater and lesser tubercle of humerus. Its action is to extend the shoulder. The infraspinatus originates on the infraspinatus fossa, and inserts on the greater tubercle of humerus. Its action is to abduct the forearm.

Importantly the supraspinatus and infraspinatus muscle play an important role in lateral stabilization of the shoulder joint. As described by Dyce, the restriction on transverse movements, imposed by the collateral ligaments at most hinge joints, is provided by the tendons of the muscles that closely surround the shoulder, notably the infraspinatus (and to a lesser extent, the supraspinatus) laterally and the subscapularis medially. 

Treatment
Both surgical and medical therapies have been reported for management of sweeney. Surgical management involves decompression of the suprascapular nerve by creation of a small notch in the cranial aspect of the scapula with or without suprascapular ligament transection. Medical therapy consists of anti-inflammatory medication and extended rest. The prognosis for both treatments depends on the extent of the original injury but in general appears to be favorable.

Deep digital flexor tendon rupture

Gait alterations
When the deep digital flexor tendon is ruptured, the coffin joint hyperextends during weight bearing and the toe is elevated from the ground. This gait is observed after traumatic laceration or internal rupture of the deep digital flexor tendon but is also observed in foals with severe laxity of the deep digital flexor tendon muscle unit.

Anatomy
In the forelimb, the heads of the deep digital flexor muscle originate from the medial epicondyle of the humerus, the olecranon, and the caudal radius, and insert as a single tendon on the palmar surface of the third phalanx. In the hind limb, the heads of the deep digital flexor muscle originate from the lateral condyle and lateral border of the tibia.

Innervation of the deep digital flexors in the forelimb is from the median and ulnar nerve. Innervation of the deep digital flexors in the hind limb is from the tibial nerve. Action of the deep digital flexors in the forelimb is to flex the digit, flex the carpus and extend the elbow. Action of the deep digital flexors in the hind limb is to flex the digit and extend the hock.

Treatment
Depending on the cause, location and extent of the injury, corrective shoeing, cast/splint application alone or cast/splint application with direct suture repair have been used in the treatment of deep digital flexor tendon lacerations. The prognosis for traumatic laceration of the deep digital flexor tendon is guarded, especially when associated with sepsis of the digital flexor tendon sheath.

Radial nerve paralysis
The radial nerve or the roots that form it can be damaged during soft tissue or bone trauma. Fractures associated with radial nerve damage include C7/T1 fractures and humeral fractures. Prolonged lateral recumbency may result in compression of the nerve as it crosses the musculospiral groove of the humerus or compression of the brachial plexus between the scapula and the rib cage. It may also lead to ischemia of the triceps musculature.
**Gait alterations**
The elbow is hyperextended or "dropped" and the carpus flexed. 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. If the horse is able to bear weight, the elbow and carpus usually “give” during mid-stance and the horse quickly leans on the opposite limb. Protraction of the limb is limited and comes primarily from extension of the shoulder joint.

**Anatomy**
The radial nerve innervates the extensors of the elbow, carpus, and digit. It arises primarily from the ventral nerve roots of T1 in the brachial plexus. The triceps has three heads: the long, lateral and medial, which originate on the caudal border of the scapula, lateral humerus, and medial humerus respectively. Insertion is on the olecranon. Innervation of the triceps is from the radial nerve. The primary action of the triceps is to extend the elbow.

The extensor carpi radialis extends the carpus and flexes the elbow. It originates on the lateral epicondyle of the humerus and inserts on the proximal metacarpus. Innervation of the extensor carpi radialis is from the radial nerve.

**Treatment**
Causes other than radial nerve paralysis for a dropped elbow appearance should be ruled out. Splinting the carpus in extension often allows the horse limited use of the limb and can be an important component of therapy. In addition, anti-inflammatory therapy, extended rest and support of the contralateral limb are often indicated. No definitive studies have been performed but horses with radial nerve neuropathy are suggested to have a good prognosis when associated with lateral recumbency positioning, a fair prognosis when associated with soft tissue trauma and a poor prognosis when associated with humeral fracture.¹⁸

**Peroneus tertius rupture**
The peroneus tertius is an important component of the reciprocal apparatus. The reciprocal apparatus connects the action of the hock and stifle allows the horse to remain standing with a minimum of muscular effort. In addition, the reciprocal apparatus synchronizes movement of the hock and stifle during the swing phase of the stride.²³

**Anatomy**
The peroneus tertius is entirely tendinous in the horse. It originates with the long digital extensor muscle on the extensor fossa of the lateral femoral condyle. It inserts on the third metatarsal, third tarsal, calcaneus and fourth tarsal bone. Its action is to (passively) flex the hock when the stifle is flexed.

**Gait Alterations**
As the horse protracts the limb the stifle flexes but the hock remains extended and the lower limb appears to be “hanging” during protraction. When the limb is passively flexed the stifle and hock move independently from each other.

**Treatment**
In the majority of horses extended rest with ultrasonographic monitoring of healing is recommended. Horses with avulsion fractures of the insertion or origin may require direct fixation if possible. A favorable prognosis is expected for closed midbody tears.²⁴ Results of studies are mixed, but it is the author’s impression that horses with midbody tears have a much better prognosis than horses with lacerations or avulsion fractures.