Neuropathic and myopathic causes of gait abnormalities and lameness

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Neuropathic and myopathic causes should always be considered in an animal with a gait abnormality or lameness, particularly when more conventional methods of assessment have failed to reveal a diagnosis. Though several disorders cause characteristic signs, others, particularly early during the clinical course can be confused for skeletal problems. This review summarises these diseases.

STRINGHALT
Stringhalt is a gait abnormality characterised by sudden and exaggerated flexion of one or both pelvic limbs, generally during walking. Severity varies from a mild gait deficit to severe movements where the distal limb may contact the abdomen or elbow. Occasionally an atypical form of the disorder is seen where there is additional involvement of the thoracic limbs (Jeffcott, Friend et al. 1986) with fetlock knuckling and extension of proximal joints. In long standing cases, distal muscle atrophy is recognised. Clinical signs sometimes vary within the same animal, perhaps decreasing after the onset of exercise or changing, depending on the ground surface.

This syndrome can be sporadic or can occur in outbreaks. The sporadic form is usually unilateral and may be precipitated by traumatic injury to the dorsolateral tarsus or metatarsus or may have no obvious inciting cause (Crabill, Honnas et al. 1994). Experimentally tibial neurectomy produces similar signs (Mayhew 1989). Outbreaks of the syndrome however are believed to have a toxic cause. Historically referred to as Australian Stringhalt, these cases are reported worldwide, in multiple animals at pasture, often late in dry summers (Huntington, Jeffcott et al. 1989; Gay, Fransen et al. 1993; Araya, Krause et al. 1998; Torre 2005). Bilateral signs are common. Several plants are implicated, including Hypochoeris radicata (flat weed or false dandelion); Taraxacum officinale (common dandelion) and Malva parviflora (cheeseweed or little mallow) (Seddon and Belschner 1926; Torre 2005). Experimentally, lathyrism (sweet pea toxicosis) produces the same signs (Mayhew 1989).

Evidence suggests that toxic stringhalt is a distal axonopathy. Peroneal nerve conduction velocities were markedly reduced in 4 horses in comparison with a control animal (Huntington, Jeffcott et al. 1989). Pathological evaluation reveals involvement of large diameter (motor) axons of long nerves accounting for neurogenic muscle atrophy (Cahill, Goulden et al. 1986; Slocombe, Huntington et al. 1992), although involvement of sensory and muscle spindle regulatory (gamma efferent) fibres probably accounts for the bizarre gait deficits that are recognised early in the clinical course (Mayhew 2005). Spinal cord involvement has not been totally excluded however, and indeed neurolathyrism in humans is associated with anterior horn grey matter and upper motor neuron lumbar spinal cord lesions caused by mitochondrial toxicity (Ravindranath 2002).

Both sporadic and toxic forms respond to lateral digital extensor myotenectomy (Crabill, Honnas et al. 1994; Torre 2005), with allegedly good results, although conservative treatment can also be successful. A variety of medical treatments, including bacofoen, phenytoin and mephensein have all been tried (Dixon and Stewart 1969; Huntington, Seneque et al. 1991; Cahill and Goulden 1992). Phenytoin in particular (for toxic stringhalt) has been administered at 7-15 mg/kg SID-TID with success (Huntington, Seneque et al. 1991; Cahill and Goulden 1992; Wijnberg, Back et al. 2000; Takahashi, Kitamura et al. 2002).

SHIVERS
Shivers or shivering is a syndrome that occurs primarily in Draught and Warmblood breeds and occasionally others (Davies 2000). It is characterised by intermittent positional and postural tonus or tremor of usually the pelvic limb. Often the limb is held hyperflexed at the hock and may quiver in this position. Signs may be exacerbated on backing or lifting the limb and occasionally the tail head is held elevated (Mayhew 1989). A report by Valentine et al. describing two Belgian horses suggested that shivers syndrome may be associated with polysaccharide storage myopathy (EPSM) (Valentine, de Labanta et al. 1999) however the high prevalence of this myopathy and shivers within this breed, probably accounts for the two conditions occurring simultaneously and coincidentally within the same animals (Firshman, Baird et al. 2005). Unfortunately, no other lesions have been described that can account for the clinical signs seen in shivers.

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KNUCKLING SYNDROMES

Both thoracic and pelvic limb knuckling syndromes have been described. With the forelimb syndrome, there is a prominent upright posture at the fetlock or knuckling over with the horse standing on the dorsal aspect of the joint, akin to foals with acquired flexural deformities. Widespread Wallerian nerve degeneration and neurogenic atrophy, is reported consistent with a distal axonopathy (Furuoka, Mizushima et al. 1994; Furuoka, Okamoto et al. 1998) somewhat similar to the thoracic limb signs reported in atypical forms of Stringhalt (Huntington, Jeffcott et al. 1989).

Mayhew describes outbreaks of an often fatal pelvic limb knuckling syndrome occurring in multiple horses in Scandinavia (Mayhew 2005). Horses exhibited signs characterised by sciatic neuropathy or sometimes its peroneal or tibial branches. Wallerian degeneration was seen in at least some cases. A toxic cause was suspected but has not been confirmed.

MYOPATHIES

Polysaccharide storage myopathy is most commonly associated with exertional rhabdomyolysis syndrome, although gluteal muscle atrophy (Valentine, de Lahunta et al. 1999), back pain and stiffness has also been described (Quiroz-Rothe, Novales et al. 2002). The disease is recognised in Quarter horses (PSSM) and a variety of other breeds (EPSM) including Warmbloods and Draught breeds (Valentine, McDonough et al. 2000; Valentine and Cooper 2005). Muscle histology reveals excessive glycogen accumulation and abnormal polysaccharide material that resists amylase digestion (Firshman, Valberg et al. 2006). Clinical improvement is seen following adjustment of the diet, in particular, reducing soluble carbohydrate and replacing it with fat (Ribeiro, Valberg et al. 2004).

Occasionally, idiopathic myopathies are diagnosed in horses that present with signs of back pain and / or lameness. Histopathology of these cases reveals signs consistent with muscle regeneration but absence of abnormal polysaccharide (Piercy – unpublished observation).

Fibrotic myopathy is a mechanical lameness characterised by an abrupt cessation to the swing phase of protraction of the pelvic limb, with the hoof normally slapping the ground. Usually the gait abnormality is seen in horses with previous hamstring muscle tears (or other trauma) (Irwin and Howell 1981), although intra muscular injection and sciatic nerve damage have also been suggested as possible causes (Turner and Trotter 1984; Valentine, Rousselle et al. 1994). Diagnosis can be made by palpating a fibrotic mass in the hamstring musculature, or visualising it with ultrasound in combination with the classic clinical signs. Surgical management, either transection or excision of the fibrotic mass (Irwin and Howell 1981; Turner and Trotter 1984), or tenotomy of the tibial insertion of the semitendinosus muscle are described (Gomez-Villamandos, Santisteban et al. 1995).

REFERENCES