Is it weak, lame or neurological?

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INTRODUCTION
Gait abnormalities are commonly encountered in equine veterinary work and reaching a diagnosis to enable provision of appropriate prognosis and treatment is important for both patient and client. It can be difficult to distinguish between the relative contribution of weakness, lameness and central or peripheral nervous system involvement. Mixed deficits also occur that can complicate the clinical picture.

LAMENESS
The term 'lameness' is generally reserved for a gait abnormality associated with pain; in most cases the deficit is consistent between strides (either at the walk or trot). Localising the site of the painful focus is a key component of the lameness examination, and involves flexion tests, palpation and application of hoof testers. Confirmation is usually made by a variety of local analgesic techniques. Furthermore, most musculoskeletal lameness will improve with systemic nonsteroidal anti-inflammatory medication.

In some horses however, extensive and repeated lameness investigations fail to identify any cause for the gait deficit. In these cases, and particularly in those that have not shown any response to a 'phenylbutazone trial', other potential causes of the abnormality should be considered. Mechanical deficits for example, may be a non-painful manifestation of an old musculoskeletal injury. Some mechanical deficits are readily recognised by their characteristic presentations (for example fibrotic myopathy and upward fixation of the patella).

A commonly overlooked, but important sub-category of lameness cases have compression of caudal cervical spinal nerve roots (Ricardi and Dyson 1993). Similarly, radiculopathy is a common feature of cervical disc prolapse in humans with localised cervical and shoulder pain and parasthesia of the arm and hand. In horses, cervical radiculopathy may be static or dynamic in nature. In the latter cases gait abnormalities may only be seen with the head and neck flexed (or collected) or may be exacerbated by turning. Horses with radiculopathy usually have articular process degenerative joint disease, or synovial cysts and occasionally cervical fractures. In these cases there is compression of nerve roots within the intervertebral foramina(e) (Moore et al. 1992). Some equine cervical radiculopathy cases that present as lameness are associated with localised shoulder or thoracic limb muscle atrophy. EMG and/or muscle biopsy is useful in determining whether the atrophy is associated with a neurogenic cause (damage to the lower motor neurone) or whether it is associated with disuse. Some, though not all, horses with cervical radiculopathy have concomitant cervical pain and/or signs of spinal cord compression, such as ataxia and upper motor neurone weakness.

In man, radiculopathy of lumbar spinal nerves may manifest as sciatica and lumbar back pain. Similarly, thoracolumbar spinal nerve root compression may be a source of chronic back pain in horses, although definitive diagnosis is difficult. Vertebral spondylosis and discospondylitis affecting the lumbar vertebrae, diagnosed by radiography, ultrasonography and scintigraphy has been described in horses (Jeffcott 1975, 1980; Sweers and Carstens 2006). As diagnostic options are limited, it remains unclear to what extent horses are affected by nerve root compression in the back. Lumbar vertebral trauma may affect spinal nerve roots that contribute to the lumbar-sacral plexus, causing pelvic limb lameness, and if severe, weakness (see below) and muscle atrophy.

WEAKNESS
Weakness in horses may be associated with dysfunction of upper motor neurones, lower motor neurones, neuromuscular transmission or the muscles themselves. Weakness is characterised by knuckling, stumbling, toe dragging and inability to bear weight. In horses, most neuromuscular and primary muscle disorders that cause weakness are generalised conditions such as botulism and hyperkalaemic periodic paralysis. Disorders of lower motor neurones may be generalised (e.g. equine motor neurone disease) or localised (e.g. radial nerve paralysis). Chronic, lower motor neurone dysfunction and disease results in muscle atrophy of the supplied muscle groups. Muscle fasciculations are a common feature of generalised muscle, neuromuscular and lower motor neurone disease, whereas localised peripheral nerve involvement tends to be associated with flaccidity (and focal muscle atrophy). Upper motor neurone dysfunction can be difficult to distinguish from the proprioceptive deficits that characterise spinal ataxia, particularly given that both abnormalities frequently occur together.

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ATAXIA
The commonest cause of ataxia in horses is cervical vertebral malformation/stenosis. Most cases are readily recognised by varying degrees of erratic (abducted and adducted) foot placement, truncal sway, hypermetria, hypometria and dysmetria. In mild cervical compressive disease, the abnormalities may only be evident within the pelvic limbs; these cases can be confused with pelvic limb lameness, especially when the signs are asymmetric. Gait abnormalities associated with ataxia are usually variable and do not respond to analgesic therapy. Often these cases require assessment for musculoskeletal and neurological disease.

DYSFUNCTION OF MYOTACTIC REFLEX PATHWAYS
 Syndromes such as Stringhalt are thought to be due to dysfunction of the afferent (sensory) component of normal myotactic reflexes that normally maintain motor tone in posture and gait. Although Stringhalt is probably the most widely recognised of these disorders, other related disorders probably exist. These gait abnormalities may be asymmetric, and when subtle they can be suggestive of a musculoskeletal lameness. Often, however, with true neurological disease, signs may be influenced (presumably via upper motor neurone control) and vary (often becoming exaggerated) when the horse is led over obstacles such as curbs or from one surface to another.

CONCLUSION
Differentiating neurological disease from musculoskeletal disease is usually possible on the basis of comprehensive lameness and neurological examinations. Subtle cases are the biggest challenge. In these, a methodical approach, combined with ancillary testing can help in differentiating the underlying cause although in some cases, progression of signs may be necessary before a diagnosis is reached. The clinician should also be aware of the possibility of concurrent conditions contributing to the gait abnormality.

REFERENCES

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