SHIVERS AND MOVEMENT DISORDERS

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Locomotion is a mechanically complex task that involves the precise activation and inhibition of many muscles, the pattern of which varies between gaits. The key features of simple forward gaits are rhythm, ipsilateral coordination of flexors and extensors across the same or different joints in a limb, as well as left/right and fore/hind coordination. Pain can disrupt this pattern causing classic signs of lameness, however, neural pathways can also be disrupted resulting in a gait abnormality or movement disorder not associated with pain.

At a spinal level, central pattern generators (CPGs) located in the thoracic and lumbar spinal cord afford the spinal cord nearly autonomous rapid control of basic locomotor rhythms. More complex locomotion requires adaptability from the simple and fast-acting to the complex and long-lasting. Higher neural structures, including the brainstem, cerebellum, and motor cortex are required for more complex movement. Disruption in the function of one of these regions in the nervous system can produce movement disorders. Because movement disorders fall into a grey area between the lameness and neurology they remain poorly understood. The purpose of our research is to develop a clinical characterization of locomotor-induced movement disorders in horses and to identify pathophysiologic bases for these conditions.

Clinical characterization of Movement Disorders

The Neuromuscular Diagnostic Laboratory solicited owners of horses with movement disorders loosely termed Shivers to fill in an on-line questionnaire and submit videos of their horses walking, turning, backing and lifting their limbs. Over 200 owners described their horse’s movement and many provided videos of their horses. Based on examination of this material several distinct categories of movement disorders were apparent.[1]

1. Dystonia of the head and neck
   - Resistance to manual flexion of either forelimb
   - Trembling of the forelimb once flexed
   - Patterned extension and twisting of the neck to one side with movement of the lips and eyelids when forelimb is manually flexed
   - Normal backwards and forwards walking

2. Hyperflexion of a hindlimb
   - Occurs when manually picking up the limb
   - Normal backwards and forwards walking
3. Hitch
   - Hyperflexion of the hindlimb, a pause and then placement at a normal speed back to the ground
   - Occurs intermittently for a few strides when walking forward
   - Backwards walking occurs normally and willingly

4. Stringhalt
   - Hyperflexion of the limb forward and under the belly while walking forward followed by a rapid return of the limb to the ground
   - Abnormal movement occurs for many strides when walking forward and occurs walking backward.
   - Horse walks backward willingly
   - Hyperflexion persists at a trot
   - May be bilateral or unilateral

The above movement disorders were not classified as ‘Shivers” as there could be many different neural or pain pathways that contributed to the abnormal movement. For the purposes of our research, Shivers was defined as a disorder that always affected the ability to walk backwards.

5. Shivers
   - Occurs with backwards walking especially if horse has been standing still, transported or stressed
   - Forward walking and trotting are normal
   - Difficulty with farrier work due to inability to hold limb flexed when standing
   - Backwards walking occurs with resistance and reluctance
   - Hyperflexion: Hyperflexion and abduction of the hindlimb, muscle trembling of the flexed hindlimb, a pause and then placement at a normal or slightly increased speed back to the ground when walking backwards
   - Hyperextension: in this alternative form hyperextension of the hindlimb occurred causing extreme difficulty flexing to move backwards and a shuffling backward gait of only a few strides.

6. Advanced Shivers
   - Hyperflexion and abduction of the hindlimb, muscle trembling of the flexed hindlimb, a pause and then placement at a rapid speed back to the ground when walking backwards
   - Alternatively, hyperextension of hindlimbs and inability to flex the limb or to walk backwards
   - Occurs consistently with backwards walking, when pushed sideways, when turned sharply or for a few strides walking forward
   - Backwards walking occurs with resistance and reluctance
   - Trotting is unaffected
   - Inability to trim hind feet because horse cannot hold the limb flexed and will slam the foot to the ground
   - Occasional tremor of upper lip
Epidemiology of Shivers

In our epidemiologic study of Shivers we found that clinical signs often began at < 7 yrs and progressed in 74% of cases.[2] Owner-reported additional clinical signs included muscle twitching (85%), muscle atrophy (44%), reduced strength (33%) and exercise intolerance (33%). Shivering horses were significantly taller (>16.3 hands high) with more geldings affected than mares. No potential triggering factors or effective treatments were reported. We concluded that Shivers is a chronic, often gradually progressive movement disorder that usually begins before 7 years of age with a greater prevalence in tall male horses.

Pathophysiologic basis for Shivers

Given that neither the neurophysiological nor the pathological mechanisms of Shivers were known and no neuroanatomical locus of the disease had been identified, we undertook a detailed analysis of the central nervous system and skeletal muscles of horses with Shivers and clinically normal control horses. No abnormalities were identified in the examined hindlimb and forelimb skeletal muscles or the associated peripheral nerves. There was no evidence of polysaccharide storage myopathy in any Shivers case. Axonal swellings were a common pathologic abnormality in many regions of the central nervous system of both control and Shivers horses. Using special immunohistochemical stains (calbindin stain) 80-fold more degenerative axonal swellings (spheroids) were found in Purkinje cell axons within the deep cerebellar nuclei of horses with Shivers when compared to controls. The immunohistochemical and ultrastructural characteristics of the lesion combined with their functional neuroanatomical distribution indicate, for the first time, that Shivers is characterized by end terminal neuroaxonal degeneration in the deep cerebellar nuclei, which results in context-specific hypermetria and myoclonus.[3]

Reference

