THE PATHOLOGY OF LARYNGEAL HEMIPLEGIA IN THE HORSE

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ABSTRACT

A review of the literature on equine laryngeal hemiplegia indicated that further investigations were warranted, in order to define more clearly the underlying pathogenic processes. This investigation was undertaken on one Standardbred and fourteen Thoroughbred horses, of which four were clinically affected with laryngeal hemiplegia, five subclinically affected and six were apparently normal. Their left and right recurrent laryngeal nerves were examined in detail, as were hindlimb nerves from two of the clinically affected animals. A variety of histological techniques were employed, including light and electron microscopy of resin embedded nerve, and single teased fibre preparations. Quantitative data obtained from these investigations was evaluated statistically. Some intrinsic laryngeal muscles, and the *extensor digitorum longus* muscle were investigated. In addition, the motor nucleus of the recurrent laryngeal nerve fibres, the *nucleus ambiguus* , and long central nerve fibre tracts were examined.

The results demonstrated that the neuropathy of equine laryngeal hemiplegia is characteristic of a distal axonopathy. Rather than being a disease of the left recurrent laryngeal nerve, as it has previously been considered, a generalised neuropathological process affecting long and large diameter nerve fibres was found. The primary site of the lesion was shown to be the axon rather than the myelin sheath. Distal axonal atrophy was demonstrated. As well as the involvement of peripheral nerve fibres in the disease process, some evidence for the involvement of long fibres of the central nervous system was found.

In addition, an investigation of the pathology associated with stringhalt, another nervous condition of horses, was performed on one animal. It was demonstrated that this was also a distal axonopathy, although a number of differences from idiopathic laryngeal hemiplegia were observed. It was an acute rather than a chronic process, and lacked any involvement of central fibres.

In conclusion, it was suggested that a number of previously postulated causes for equine laryngeal hemiplegia could be dismissed, on the basis of the finding of a generalised distal axonopathy. This included those which suggested mechanical damage to the left recurrent laryngeal nerve, such as stretch or compression. In light of the findings, the most likely aetiology underlying the pathology of equine laryngeal hemiplegia was considered to be an acquired or inherited metabolic defect affecting energy production in the axon, with the resultant inability to support the distal areas of long, large diameter nerve fibres.

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