ETIOLOGY OF RECURRENT LARYNGEAL NEUROPATHY (RLN)/ LARYNGEAL HEMIPLEGIA

Although horses can develop unilateral or bilateral paralysis of the CAD muscle leading to dysfunction of the associated arytenoid cartilage,6-9 unilateral left-sided laryngeal paralysis is most commonly encountered. Because the muscle paralysis results from progressive loss of large myelinated axons in the left recurrent laryngeal nerve,6 recurrent laryngeal neuropathy(RLN) is now considered the most appropriate descriptive term for this condition. Although a genetic basis to the condition is most likely, in the majority of affected horses, a precise etiology is rarely evident, and the term idiopathic laryngeal hemiplegia has commonly been applied as a synonym. Neurogenic atrophy of the intrinsic laryngeal musculature results in progressive loss of both abductor and adductor arytenoid function. Because of this progressive rather than immediate loss of muscle function, varying degrees of abnormal movements of the arytenoid cartilage(s), depending on presence of CAD muscle paresis or paralysis, is frequently observed endoscopically, which strengthens the use of RLN as the most appropriate diagnostic term. RLN occurs in horses from a few months to 10 years of age and older, with large-breed horses (such as Thoroughbreds and draught breeds) more commonly affected than small-breed horses or ponies. The incidence is highest in young horses, often diagnosed before they have started any type of training, or in 2- and 3-year-olds that are racing or are in race training. The prevalence of RLN varies between breeds, with the largest population studied being the Thoroughbred, where between 2.6%15 and 8%14 of horses are reported to be affected.

However, in the heavy draught breeds, an incidence of up to 35% has been reported.16 Following the introduction of pre and post-sale endoscopy at Thoroughbred yearling sales, the incidence of RLN has been widely documented. Although the incidence of complete laryngeal hemiplegia was 2.75% in a study of yearling Thoroughbreds, the overall incidence in the entire yearling Thoroughbred population is thought to be higher. In a study of 427 Thoroughbred sale yearlings, 64% had asynchronous arytenoid cartilage

movements and 25% had laryngeal asymmetry that was significantly associated with decreased racing performance.

In normal horses during sustained high-intensity exercise, the rima glottidis of the larynx dilates fully to maximize airflow. Full arytenoid cartilage abduction is sustained, despite increasing negative inspiratory pressure that occurs as exercise intensity increases. Horses with RLN cannot achieve maximal abduction of the affected arytenoid cartilage, and as negative inspiratory pressure increases, the rima glottidis progressively reduces in size. Subsequently, hypoxemia, hypercarbia, and metabolic acidosis develop more rapidly than in normal horses with the same workload, causing early musculoskeletal fatigue and poor performance.

A specific etiology can be identified in some horses with acquired unilateral or bilateral laryngeal paralysis. The recurrent laryngeal nerve can be damaged as a result of perivascular jugular vein injection, guttural pouch mycosis, trauma from injuries or surgical procedures of the neck, strangles abscessation of the head and neck, and impingement by neoplasms of the neck or chest. Organophosphate toxicity, plant poisoning, hepatic encephalopathy, lead toxicity, and central nervous system diseases have also been shown to cause laryngeal paralysis.

1.Equine Surgery 4th edition by Auer & Stick