Havemeyer Foundation
Monograph Series No. 11

Proceedings of a Workshop on

EQUINE RECURRENT LARYNGEAL NEUROPATHY

7th – 10th September 2003
Stratford-upon-Avon, UK

Editors: P. Dixon, E. Robinson and J. F. Wade

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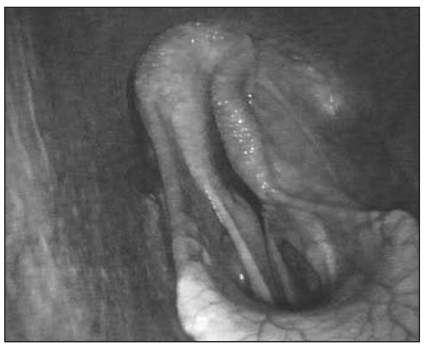

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EDITORS' FOREWORD

Equine laryngeal paralysis, most commonly attributed to the syndrome of recurrent laryngeal neuropathy (RLN), is long recognised as the most important equine upper airway disease of horses. It can cause exercise intolerance and stridor during fast work and in the rare bilateral cases, and severe respiratory distress, even in the resting horse. Despite is, there are still large gaps in our knowledge of its aetiology, pathogenesis, methods of assessment and the critical evaluation of its treatment. Between 20 and 30 years ago, there was significant research into the disease, but there have been few substantive studies since then.

To promote further basic and applied research into this disease it seemed worthwhile to review current knowledge about this disease. This was the main purpose of the present workshop and we invited eminent neurologists to review knowledge of the aetiology of RLN and to compare it with other equine peripheral neuropathies. It was also valuable to gain an overview of some comparative aspects of RLN with laryngeal paralysis in other species. By obtaining consensus on our present knowledge, we went on to suggest areas for productive future research. For example, are sub-clinical neuropathies of other long peripheral nerves present in horses with RLN?

As well as established research scientists, our delegates included practitioners who undertake upper airway examination of young horses at sales. Outlining the difficulties and practical problems encountered by clinicians in Europe, America and Australia, they provided insight into the incidence and possible progression of this disease. Currently, endoscopy is the gold standard for assessing laryngeal dysfunction in the horse. For clinicians to communicate effectively with each other on RLN cases, a variety of grading systems are in use. One aim of the workshop was to reach a consensus as to which endoscopic grading system should be advocated universally. After lengthy discussion, we reached a consensus and time will tell if the international community accepts the suggested system. Some studies have shown that, even with well-defined endoscopic criteria and grading systems, inter-observer variation can occur in the endoscopic grading of laryngeal function. The upcoming results of an interactive video presentation conducted at the workshop may support or refute this. Even allowing for individual variation in interpretation, it is well established that the endoscopic findings in some horses (and foals) may differ from time to time. Presentations addressed the progressive nature of the disease, especially in some older horses.

The advent of endoscopic examination during exercise on a high-speed treadmill has revealed that assessment of laryngeal dysfunction in the resting horse does not always reflect what occurs during high speed exercise. In addition, upper airway obstructions in horses are not always due to laryngeal paralysis. The results of large surveys of both of these topics were presented. Recently, it was found that horses can suffer bilateral laryngeal paralysis in the presence of liver disease and following general anaesthesia and comparison of these cases with RLN may cast further light on the aetiopathogenesis of both disorders.

In the last 2 decades there have been many clinical and physiological studies of the efficacy of treatments for equine laryngeal paralysis. These have addressed both the improvement of airway function and elimination of abnormal sounds. The various treatments currently in use were reviewed including the promising neuromuscular pedicle graft. Comparisons were made with the treatment of laryngeal paralysis in the dog.

It was agreed that future areas for research should include investigations into the aetio-

pathogenesis of this disorder; possible investigations of the genetics of RLN that may eventually eliminate the disease from the breeding population; and multi-centre assessment of the efficacy of RLN treatment, involving large numbers of horses.

A primary aim of the workshop was to reach a consensus on the current state of knowledge. This was particularly challenging with regard to recommendations on grading systems. The consensus statements are presented as the final section of this monograph. We hope that they will 1) provide a useful review for those starting to work on recurrent laryngeal neuropathy and 2) stimulate much discussion that will lead to new investigations into this important disease.

Finally, we thank the participants in the workshop, the organisers and the Havemeyer Foundation. The participants travelled long distances and many gave up valuable time from their practices in order to debate with academics. We hope that both groups gained from the experience. Rachel Pepper and Jan Wade did a superb job of selecting the venue and organising the meeting. The Havemeyer Foundation continues its excellent work in fostering research and education about important equine diseases. We are very grateful for their support.

Paddy Dixon
and Ed Robinson

HAVEMEYER SCIENTIFIC WORKSHOPS

1981 **First International Workshop on Lymphocyte Alloantigens of the Horse**
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October - Cornell University, Ithaca, New York, USA
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1983 **Third International Workshop on Lymphocyte Alloantigens of the Horse**
April - New Bolton Center, University of Pennsylvania, USA
Organiser: Dr D. F. Antczak

1984 **First International Symposium on Equine Embryo Transfer**
October - Cornell University, Ithaca, New York, USA
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October - Mount Joy, Pennsylvania, USA
Organiser: Dr S. M. McDonnell

Bone Remodelling Workshop
October - Corcord, Massachusetts, USA
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1997 **Second International Workshop on Equine Gene Mapping**
October - San Diego, California, USA
Organisers: Drs D. F. Antczak and E. Bailey

Maternal Recognition of Pregnancy in the Mare
January - Dominican Republic
Organisers: Drs W. R. Allen and T. A. E. Stout

Uterine Clearance
March - Gainesville, Florida, USA
Organiser: Dr M. M. LeBlanc

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September - Edinburgh, Scotland
Organisers: Drs D. F. Antczak and F. Stewart

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January - San Diego, California, USA
Organisers: Drs D. F. Antczak and E. Bailey

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March - Banbury Center, Cold Spring Harbor, New York, USA
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April - Lipica, Slovenia
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Equine Placentitis Workshop
October - Lexington, Kentucky, USA
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Septicemia II Workshop
November - Boston, Massachusetts, USA
Organiser: Dr M. R. Paradis

1999

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2000

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July - Saari, Finland
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January - Santa Fe, New Mexico
Organiser: Dr D. P. Lunn

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April - Hungary
Organisers: S. Lazary and E. Marti

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June - Port Douglas, Australia
Organiser: Professor W. R. Allen

International Equine Gene Mapping
July - Brisbane, Australia
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September - Loosdrecht, The Netherlands
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October - Tufts University European Center, Talloires, France
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Infectious Disease Programme for the Equine Industry and Veterinary Practitioners
October - Marilyn duPont Scott Medical Center, Morvan Park, Virginia, USA
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Stallion Behavior IV
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Organisers: S. McDonell and D. Miller

Rhodococcus Equi II
July - Pullman, Washington
Organiser: J. Prescott

Equine Orthopaedic Infection
August - Dublin, Ireland
Organiser: E. Santschi

Inflammatory Airway Disease: Defining the Syndrome
September - Boston, USA
Organiser: Dr E. Robinson

2003 **USDA International Plant and Animal Genome Conference**
January - San Diego, California

Embryonic and Fetal Nutrition
May - Ravello, Italy
Organiser: S. Wilsher

Genomics and the Equine Immunity System
June - Ithaca, New York
Organiser: D. F. Antczak

Fifth International Gene Mapping Workshop
August - Kreuger Park, South Africa
Organiser: E. Baily and E. Vandyke

Equine Recurrent Laryngeal Neuropathy
September - Stratford-upon-Avon, UK
Organisers: P. Dixon and E. Robinson

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SESSION I:

Pathogenesis and pathology

Chairman: Joe Mayhew

DEVELOPMENT AND INNERVATION OF THE LARYNX

C. Hahn

Neuromuscular Diagnostic Laboratory, Royal (Dick) School of Veterinary Studies, The University of Edinburgh, Easter Bush, Roslin, Midlothian EH25 9RG, UK

A brief look at the evolution and development of the larynx goes some way to explain the curious anatomy of this organ. About 400 million years ago, the lungfish evolved the ability to breathe air directly from the external environment, perhaps because its watery home was periodically subject to drought (Ewings 1949). It developed a simple larynx-like slit behind the gills that allowed air into the swim-bladder when the creature was exposed to the atmosphere and that kept water out when it was submerged (Fig 1). As the descendants of the lungfish moved onto land, the swim-bladder evolved into a multi-compartment organ with a large surface area the sole function of which was gas exchange. The larynx in the meantime developed adductor and abductor muscles and lateral cartilages (such as found in the axolotl), then separate arytenoid and cricoid cartilages (newt), primitive thyroid cartilages (alligators and their feathered relatives, the birds) and finally the complex mammalian larynx. As the survival of equids once depended on running long distances to escape predators, horses evolved a larynx that when fully abducted has an aperture that is larger than the trachea itself (this in sharp contrast to the human larynx, where the abducted larynx allows for speech but is only half the diameter of the trachea).

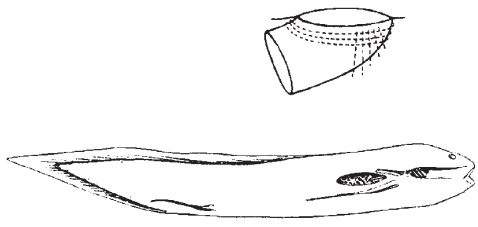

Fig 1: Lungfish with modified swim bladder and dilator and sphincter muscles (modified from Ewings, V. (1949). The Comparative Anatomy and Physiology of the Larynx. William Heinemann, Medical Books, London).

An appreciation of the neuroanatomy of laryngeal innervation is a pre-requisite to understanding the pathology of recurrent laryngeal neuropathy. The main source of laryngeal innervation of the equine larynx is the ipsilateral recurrent laryngeal nerve (rln). Motor neurons of the rln are based in the nucleus ambiguus in the caudal brainstem. This nucleus was recently localised in the horse (Hackett 2000) and was found to be a loosely organised column of cells in the ventrolateral medulla oblongata (Fig 2). A somatotopic distribution of adductor and abductor motor neurons was not apparent but neurons innervating the *cricoarytenoideus lateralis* muscle were observed throughout the nucleus, whereas neurons innervating the *cricoarytenoideus dorsalis* tended to be situated more rostrally.

Nucleus ambiguous axons loop around the parasympathetic nucleus of the vagus to emerge from the brainstem as axons of the internal branch of cranial nerve (CN) XI. They only join the vagus nerve (CN X) on leaving the skull through the jugular foramen and tympano-occipital fissure.

Cranial movement of the head during embryogenesis, and differential degeneration of the 6th aortic arch, resulted in extremely long nerves with the left and right nerves having different pathways. The left nerve loops around the aorta while the right takes a shorter route around the right subclavian artery. Including its vagal course, the total length from neuronal cell body to larynx of the left rln can be up to 250 cm in length, making it twice as long as other motor nerves in the horse and 31 cm longer than the right rln (Cole 1946).

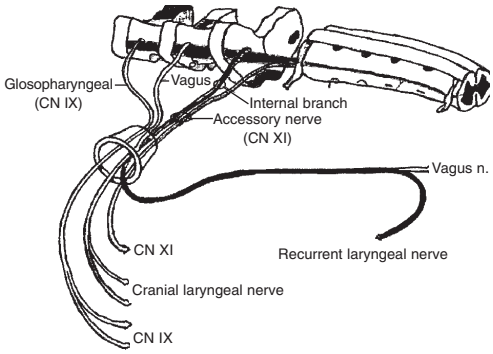

Fig 2: The recurrent laryngeal nerve is supplied by axons originating in the caudal nucleus ambiguus. (Modified from de Lahunta, A. (1983) Veterinary Neuroanatomy and Clinical Neurology, 2nd edn. Saunders, Philadelphia, pp 105).

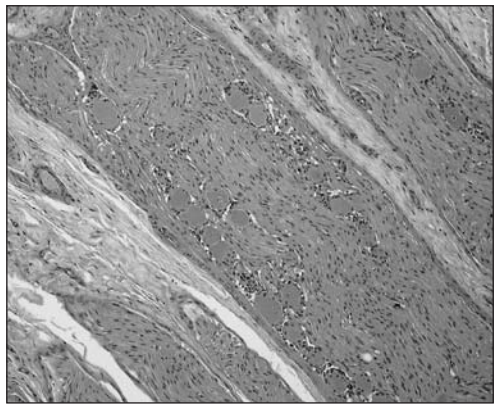

Fig 3: Scattered neuronal cell bodies of the distal vagal ganglion in the proximal vagus nerve.

The normal rln nerve consists of medium sized myelinated fibres with only scattered, smaller diameter fibres present. Myelinated axons in the rln segregate as fascicles within the vagus nerve. However, after these fascicles separate from the vagus as the rln, the axons that are targeted to innervate a particular intrinsic laryngeal muscle are not discreetly clustered within the rln at its origin in the thorax, but instead are mixed among the fascicles throughout its length.

Although the rln is thought of classically as a motor nerve, primary afferent ('dorsal root ganglia') rln neurons have been demonstrated in the proximal and distal vagal ganglia. The distal vagal ganglion is poorly described in the horse but has recently been identified to consist histologically of scattered neurons in the vagus nerve at its bifurcation with the cranial laryngeal nerve (I.G. Mayhew, personal communication, Fig 3). Involvement of sensory axons in horses with recurrent laryngeal neuropathy has not been established. Each nerve then courses cranially to provide motor innervation to the paired intrinsic laryngeal muscles, with the exception of the cricothyroideus muscles. These muscles have a different embryologic origin and are innervated by nucleus ambiguus neurons whose axons join an external branch of the paired cranial laryngeal or vagus nerves (de Lahunta 1983).

It is likely that the complexity and length of this pathway underlies the pathology of recurrent laryngeal neuropathy

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NEUROLOGY OF RECURRENT LARYNGEAL NEUROPATHY AND THE THORACO-LARYNGEAL REFLEX

I. G. Mayhew

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The neurological evaluation of a horse suspected of having abnormal laryngeal function should include a complete neurological examination as left sided recurrent laryngeal neuropathy (RLN) may occur in conjunction with other neurological signs. These rarely relate to brain or spinal cord lesions; more often to peripheral nerve involvement especially within and around the guttural pouch, neck and cranial thorax. Other signs of vagosympathetic trunk involvement, especially swallowing difficulties, Horner's syndrome and sweating over variable areas of the head and neck, can accompany such signs of RLN.

The major neurological diagnostic aids that may be utilised in evaluating cases of RLN include the electromyogram (EMG), nerve conduction velocity (NCV) testing and evaluating the endoscopic and palpable thoraco-laryngeal reflex (TLR) and the electrolaryngeogram (ELG).

ELECTROMYOGRAPHY

In one study (Moore *et al.* 1988), EMG of the dorsal *cricoarytenoidius dorsalis* muscle appeared to have very high sensitivity for the diagnosis of RLN but almost certainly would have a low specificity. EMG would be extremely useful in evaluation of sudden onset of signs consistent with RLN. This is where there is total paralysis of the left (or right) side but no detectable atrophy. Immediate EMG evaluation may reveal very little abnormality, if indeed such signs were due to an acquired lesion, such as a perivascular injection in the cervical region. Monitoring the time course of subsequent atrophy and progression of electromyographic denervation abnormalities could help pinpoint more accurately the exact time of onset of such an acquired disorder.

NERVE CONDUCTION VELOCITY

Steiss *et al.* (1989) determined the characteristics of evoked compound muscle action potentials in the intrinsic laryngeal muscles of control ponies and small horses. They stimulated the proximal vagus and the distal recurrent laryngeal nerve in the cranial cervical region. The mean latencies (ms), from the proximal stimulation site on the vagus nerve and the distal stimulation site on the recurrent laryngeal nerve, to the larynx for ponies and horses are shown in Table 1.

The latencies on the left side were 22% and 26% longer than the right side in ponies and horses, respectively. There was a positive correlation between latency and body length in ponies, but not in horses. True nerve conduction velocities are difficult to determine in such studies because of the difficulty and error in measurement of the length of neural pathways being stimulated. NCV measurement is invasive and does require heavy sedation. However, determining the segmental latency component values could lend further evidence for the neuropathy present in idiopathic RLN being a distal neuropathy.

TABLE 1: Control values for vagal nerve (proximal) and recurrent laryngeal nerve (distal) conduction latencies in ponies and horses (Steiss *et al.* 1989)

	Latency ms; mean (sd)	
	Proximal	Distal
Ponies		
Left	21.7 (3.1)	3.7 (0.9)
Right	17.8 (1.9)	4.0 (0.8)
Horses		
Left	29.4 (3.1)	6.8 (1.8)
Right	23.4 (2.4)	5.7 (1.3)

THORACO-LARYNGEAL REFLEX

Dr Bob Cook in the 1970s serendipitously came up with observations that led to the publication (Greet *et al.* 1980) of the so called 'slap test'. This perhaps more correctly is termed the thoraco-laryngeal reflex (TLR).

The pathway for this reflex is through sensory receptors on the skin or deeper tissues of the dorsal cranial thorax, through dorsal nerve roots to the spinal cord with an ascending pathway in the thoracic and cervical spinal cord. This pathway appears to cross to the contralateral side at least by the time it reaches the brachial intumescence and possibly immediately upon entry into the spinal cord through dorsal nerve roots. The pathway is probably in the lateral funiculus, but may be in the dorsal funiculus of the spinal cord, ascending to reach the dorsal nucleus of the vagus, hence subsequently the contralateral vagal nerve. The efferent pathway around major arteries in the cranial thorax is longer on the left side than the right side, as the recurrent laryngeal nerve leaves the vagus and ascends the neck to innervate intrinsic muscles of the larynx. In the first report (Greet *et al.* 1980), 30 ataxic and 64 non-ataxic horses were evaluated endoscopically for the presence or absence of a TLR. Because 10 of the 41 non-ataxic horses that had RLN demonstrated a normal TLR it was concluded that this test was very unreliable for diagnosis of RLN. However, the data for the horses with neurological disease indicated a very good sensitivity and an excellent specificity for diagnosis of cervical spinal cord disease.

Subsequently, Newton-Clarke *et al*. (1994a) investigated the clinical value of the endoscopically-determined TLR in the diagnosis of spinal cord and brainstem disease. This group looked at 15 ataxic horses (12 of whom were determined to have lesions to explain the signs at post-mortem examination) and 13 control horses. They determined sensitivities in the range of 0.50–0.60 and specificities in the range of 0.70–0.75. On the basis of a 50% potential prevalence of disease this would have raised the positive predictive value from 0.50 to 0.70 and the negative predictive value from 0.50 to 0.64. Their interpretation was that this was a very poor test for diagnosing particularly cervical spinal cord disease.

The same group studied the value of the endoscopically-determined TLR in RLN diagnosis. They evaluated 15 horses with no clinical signs of RLN and subsequently took samples of the *crycoarytenoideus lateralis* muscle for histopathologic examination. Moderate to severe neurogenic muscle atrophy and re-innervation was found in 5 horses with normal TLRs. The remaining horses also had degrees of adductor myopathy. The group determined, as did Greet (1994), that the test was useless for RLN diagnosis. It is possible that 2 of the reasons for such false positive results are that adductor muscle pathology precedes abductor muscle atrophy (Duncan and Baker 1987; Archer *et al.* 1989) and that good evidence of re-innervation was found in most of the horses by Newton-Clarke *et al.* (1994b).

ELECTROLARYNGEOGRAPHY

The original report by Cook and Talhammer (1991) on the electrolaryngeogram (ELR) suggested that most horses had evidence of RLN, at least on the left side. This was based on the assumption that a single pony with the same latencies on the left and right side should be regarded as normal; this has been refuted by other studies (Steiss *et al.* 1989; Hawe *et al.* 2001; R.A. Curtis, unpublished data). Cook and Thalhammer (1991) also determined approximate nerve conduction velocities based on estimates of the reflex pathway length in horses of various sizes. The pony that had identical latencies on each side had a calculated nerve conduction velocity of 63.5 m/s and this figure was used to calculate the

TABLE 2: ELG latency values for control ponies and Clydesdale horses unaffected (Grades 0–1) and affected (Grades 2–4) with RLN (Hawe *et al.* 2001)

Animals	Grade of RLN*	n	ELG latency, ms. Median (range)	
			Left	Right
Ponies	0–1	1050	(34–54)	42 (32–47)
Clydesdales	0–1	7	69 (65–73)	57 (54–58)
"	2	12	69 (60–76)	57 (52–64)
"	3–4	6	70 (65–78)	56 (53–66)

*Based on a 0–5 grading system

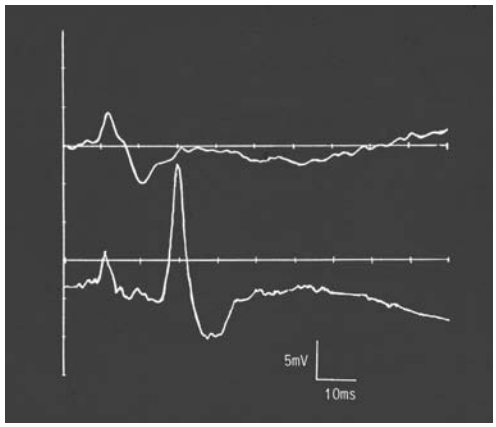

Fig 1: An early brainstem resonse is recorded to a slap test in the top trace. Also an early ELG waveform, possibly indicating electrical activity in the cricothyroid muscle via the cranial laryngeal nerve is recorded in the lower trace (Note: sacle on x- axis is 20 ms).

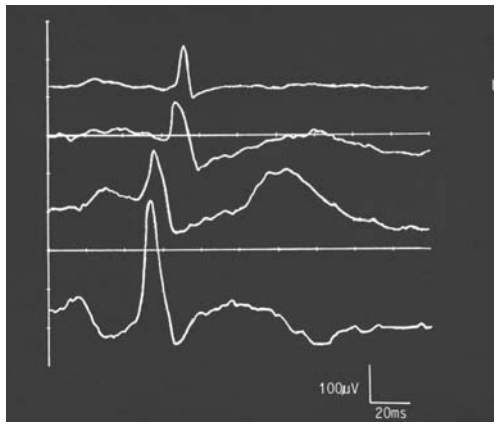

Fig 2: ELG recorded in a pony heavily sedated with detomidine with the head held in a normal position (lowest trace), partially lowered positions (middle 2 traces) and nose-near-the-ground position (top trace).

normal ranges for reflex arks varying between 250 and 400 cm in length.

Hawe *et al.* (2001) undertook a study of 35 Clydesdales and 10 ponies in which they graded the degree of RLN by the classical grading system using endoscopy at rest and post exercise, and compared endoscopic findings with results of laryngeal palpation for muscle mass and performance of the TLR recorded by palpation, endoscopy and the ELG. They found that palpation for muscle mass correlated less well with degree of RLN than the endoscopic TLR. However, the palpable response to the TLR did correlate with the endoscopic grade of RLN, as found by Lane (1993). It is possible that the endoscopic procedure itself or the application of a twitch may interfere with the endoscopic observation of the TLR.

Hawe *et al*. (2001) also determined the latency for the ELG in ponies to be a median of 50 ms on the left and 42 ms on the right. In Clydesdale horses these figures varied depending on the grade of RLN determined and are shown in Table 2. In each category, the left and right latencies were significantly different. These workers corrected the latency values for size of horse using various parameters. Although it was determined that the absolute latency values did correlate with the severity of RLN the latency values corrected for body size measurements did not correlate with the severity of RLN. They concluded that the ELG is not a simple and accurate method for the objective diagnosis and grading of RLN as claimed by Cook and Talhammer (1991) and Cook (1995).

R.A. Curtis (unpublished data) used post processing of digitised ELG latency and velocity estimates to determine that there was a significant association of the grade of RLN (Grades 1 and 2) with right peak latency. However, they found no association of RLN (Grades 1 and 2) with velocity estimates. This is still being prepared for publication and it may well show statistical data to endorse the use of digitised and post processed ELG recordings to categorise groups of horses as having different grades of RLN. However, in an individual animal it may be very difficult to determine statistically whether it is affected or not, let alone what grade it may have.

THE FUTURE

Regarding ELGs, it has been determined (I.G. Mayhew unpublished data) that a brain stem response can be detected as a short latency response to the 'slap test' and that there also can be an early ELG wave form of approximately the same latency (Fig 1). It is suggested that this is activity in laryngeal muscles via a shortened ELG response involving the cranial laryngeal nerve. The utility of this in determining the presence of lesions at various sites is still to be determined.

Additionally, it has also been observed (I.G. Mayhew unpublished data) that in ponies that are sedated with their head held in a normal posture the ELG can be easily recorded. If the head is then slowly lowered to an intermediate position and then to a 'nose-near-the-ground' position the latency of

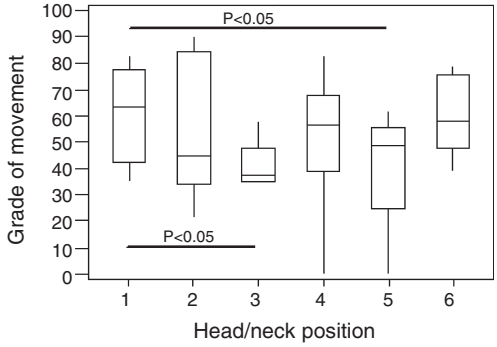

Fig 3: Influence of head and neck position on the degree of contralateral arytenoid adduction occurring during a TLR (Pirie et al. *2003, personal communication). Head and neck positions (time between TLR testing) were: 1) normal resting position – unsedated (5 min); 2) normal resting position, sedated (5 min); 3) head and neck extended, horizontal (8 min); 4) head and neck extended towards ground level (5 min); 5) head and neck extended, horizontal, as 3 (5 min); and 6) normal resting position, sedated.*

the recorded ELG is prolonged (Fig 2). This may reflect an effect on the neural pathway of the TLR, but it is extremely difficult to make sure that the needle electrodes are indeed in exactly the same place as the head and neck are moved in relation to laryngeal muscle action potential generators. Does this potentially indicate a mechanical component to the pathogeneses of RLN?

Pirie *et al.* 2003 (personal communication) attempted to determine the influence of head and neck position on the degree of contralateral arytenoid adduction occurring during a TLR. The 4 point grading system given by 5 observers was transformed to a 0–90 'grade of movement' for each head and neck position as shown in Figure 3.

There was a significant difference for the head and neck extended position (3 and 5) when compared with the neutral position, however there was considerable noise in the data such that definitive conclusions could not be made.

Nollet *et al.* (2002) have data to indicate that the magnetic motor evoked potential in limb muscles is useful in determining the presence and severity of cervical spinal cord disease. It is likely that there is a laryngeal response to cortical magnetic stimulation and this methodology may be applicable to service further studies of the TLR and RLN in general.

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REVIEW OF THE PATHOLOGICAL CHANGES IN EQUINE RECURRENT LARYNGEAL NEUROPATHY

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PATHOLOGICAL CHANGES

The lesions associated with recurrent laryngeal neuropathy (RLN) have been well characterised using light and electron microscopy (Cole 1946; Duncan and Griffiths 1974; Duncan *et al.* 1978; Cahill and Goulden 1986a, b, c, d, e; Duncan *et al.* 1991).

The primary lesions have been demonstrated in nerves, and have been found to be greatest in the distal portions of the left and right recurrent laryngeal nerves. Abnormalities have also been noted proximal and distal to the aorta and in the vagus nerve. Changes noted in the right RLN are less severe than those found in the left. The pathology is characterised by a proximal to distal decrease in large myelinated fibres. However, the same trend, including the presence of Renaut bodies commonly reported in RLN cases, has been shown in 'normal' horses (Lopez-Plana *et al.* 1993). It is unknown if sensory fibres in the recurrent laryngeal nerves are also affected and vagal sensory ganglia should be examined for neuronal chromatolysis.

The primary lesion may be axonal in nature, as indicated by collapsed myelin sheaths without an axis cylinder, increased myelin sheath thickness (potentially due to axonal atrophy), regenerating Schwann cell membrane clusters and paranodal and internodal accumulations of axonal debris and organelles. The latter may be an indication that a defect in the axonal transport systems results in the eventual distal axonal degeneration. In addition there is evidence of extensive myelin damage. Büngner's bands, representing Schwann cell membranes, and onion bulbs made up of proliferating Schwann cells, are commonly found, as are myelin digestion chambers containing central axon fragments. Teased fibre preparations show a marked variation in internodal length and diameter indicating chronic demyelination and attempted remyelination.

Evidence of central changes have been sought, however neither Cahill and Goulden (1986) nor Hackett and Cummings (personal communication) were able to identify lesions in the lower motor neuron cell bodies of the recurrent laryngeal nerves in the nucleus ambiguus of affected horses. Chromatolysis of the lower motor neuron may be expected secondary to the axonal damage, this however is influenced by the proximity of the lesion (Dyck and Thomas 1993). Likewise chromatolysis or neuronal loss in the nucleus ambiguus would be anticipated if the axonal changes are due to somal (cell body) pathology as has been described in Bouvier des Flandres (van Haagen 1980) and the Siberian husky dogs (O'Brien and Hendriks 1986). Unfortunately, there has been no systematic work in the horse evaluating the peripheral or central pathological changes which accompany damage to long axons. Ultrastructural examination of nucleus ambiguus neurons has been attempted but is complicated greatly by the difficulty of identifying the boundaries of the nucleus in the medulla oblongata. It was believed that there was a difference in the number of neurons in horses with RLN compared to normal horses, but small numbers of animals examined did not allow a statistical comparison (Hackett personal communication). There have been no histochemical techniques applied to identify somal changes secondary to the hypothesised transport disorder.

Lesions in the laryngeal muscles innervated by the recurrent laryngeal nerves are characteristic of neurogenic disease. Denervation of the adductor muscles precede abductor involvement and typical changes include scattered angular fibres and

groups of atrophied fibres adjacent to hypertrophied fibres with central nuclei (Duncan and Griffiths 1974; Duncan *et al.* 1991). The first muscle groups affected appear to be the adductor muscles and within the adductor group the *cricoarytenoideus lateralis* is among the earliest and most severely affected muscles (Lopez Plana *et al.* 1993). The chronic, repetitive nature of the disease is further exemplified by the presence of muscle fibre type grouping, as muscle fibre type is controlled by the innervating neuron.

Together, these pathological changes have been classified as a distal axonopathy, with the greater pathology in the left recurrent laryngeal nerve being explained by its greater length. One hypothetical cause of distal axonopathy is a defect in the neuronal soma, as the axon depends on the cell body for metabolic support and sustaining or trophic influences. Indeed, many of the peripheral nerve lesions that are typical in equine motor neuron disease, a disease primarily affecting the cell body, are also observed in RLN including axonal atrophy, proliferated Schwann cell cords (Büngner's bands), loss of myelinated fibres and an increase in endoneurial collagen.

Hypothetical aetiologies

Despite years of work we appear to be no closer to clarifying the aetiology of this common equine disease. Hypotheses range from mechanical causes such as tension and stretch to the recurrent laryngeal nerve and its blood supply during neck movement, growth, or the caudal shift of the heart during embryonic development, to environmental factors, including toxins (reviewed by Cahill and Goulden 1987). The latter have been viewed as unlikely causes of RLN as the neuropathological changes are limited to the recurrent laryngeal nerves.

Myelinopathies and the inherited and metabolic primary axonopathies affect multiple nerves and in other species would be expressed as part of a polyneuropathy and be progressive. Comparable pathology has indeed been noted in foals (Duncan 1992; Harrison *et al.* 1992) and clinical signs of left-sided hemiplegia have been demonstrated to be clinically progressive (Dixon *et al.* 2002). Interestingly, however, there have been no reports of left hemiplegic horses progressing to develop right-sided clinical signs (Dixon, personal communication). On the other hand, it is clear that horses affected with RLN do not show classical clinical signs of polyneuropathy such as megaoesophagus, tetraparesis and muscle atrophy. Notwithstanding, involvement of other long peripheral nerves (common, deep and superficial peroneal and tibial nerves) has been reported by some workers (Cahill and Goulden 1986a; Kannegieter 1989), but was not found by Duncan *et al.* 1978). Similarly, neurogenic muscle changes have been reported to exist in the *extensor digitorum longus* (Cahill and Goulden 1986d) in 3 out of 4 horses suffering from RLN. The above observations however have been isolated, uncontrolled and have not taken into account that age-related pathological changes can be demonstrated in distal limb nerves of horses (Wheeler and Plummer 1989). A detailed study of the peripheral nerves in RLN affected and control animals has not been undertaken.

It should be remembered that axonal degeneration, characterised by distal degeneration that spreads proximally ('dying back'), is the most common pathology seen in peripheral nerve diseases caused by a wide variety of toxic, metabolic, and infectious insults. Some of these processes affect the cell body, and it may be that the axonal dying back process may be initiated in order to conserve energy. How a cell can eliminate part of itself while leaving the rest intact is unknown. Localised axonal degeneration that resembles dying back can also occur in cell culture if the distal portion of the axon is deprived of nerve growth factor, and a similar process may be involved in disease states. Other forms of axonal degeneration that seem distinct from typical dying back occur in various human neurodegenerative diseases such as Alzheimer's, Parkinson's and Huntington's diseases.

Pathological changes of the recurrent laryngeal nerve in RLN has been described in great detail using light and electron microscopy but the tools of the burgeoning science of molecular pathology have not been utilised. A detailed examination of changes in gene regulation and cytokine expression will have to be applied if further details of the pathogenesis are to be uncovered.

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COMPARISON OF RECURRENT LARYNGEAL NEUROPATHY WITH OTHER NEUROPATHIES IN HORSES

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INTRODUCTION

This paper gives an overview of the known and suspected neuropathies in horses, apart from recurrent laryngeal neuropathy (RLN). It will not discuss the primary traumatic neuropathies, polyneuritis equi or the neuronopathy known as equine motor neuron disease.

STRINGHALT

Stringhalt, also known as springhalt and Hahnentritt, is an disease recorded from ancient times that is characterised by a sudden, apparently involuntary, exaggerated flexion of one or both hind limbs during attempted movement. The hind limb motion may be as mild as a slightly excessive flexion to violent movements during which the fetlock or toe will contact the abdomen, thorax and occasionally the elbow with attempted steps leading to a peculiar 'bunny hopping' and plunging type gait. Ultimately marked atrophy of muscles of the hind limb(s), particularly distally, will occur.

Three forms of stringhalt are recognised (Huntington *et al.* 1989). Firstly, the sporadic form usually affects one limb and occurs worldwide. The onset can be preceded by a history of, or evidence of, trauma to the dorsal tarsal region or the dorsoproximal metatarsus. The second form that usually occurs as outbreaks is referred to as Australian stringhalt, but is also seen in New Zealand (Cahill *et al.* 1985), United States (Gay *et al.* 1993), Chile (Araya *et al.* 1998) and Japan (Takahashi *et al.* 2002). Usually there is symmetrical or often asymmetrical involvement of the pelvic limbs, with prominent distal muscle atrophy in severe cases. This form has been associated with exposure to several plants notably *Hypochoeris radicata*, *Taraxicom officinal* and *Malva parviflora* (Huntington *et al.* 1889, 1991; Seddon and Belschner 1926; Cahill and Goulden 1985; Gay *et al.* 1993). These are related species of flat weed, *Taraxicom officinal* being the common dandelion. Finally, there is an atypical form of stringhalt where the thoracic limbs are also affected. There is knuckling of the forelimb fetlocks with prominent extension of more proximal joints and atrophy of the distal musculature, in association with prominent stringhalt in both hind limbs. It is possible that atypical stringhalt is simply a more severe form of Australian stringhalt. Experimental sweet-pea poisoning (lathrysim) causes such a syndrome. Huntington *et al.* (1989) have graded the clinical severity of stringhalt from 1 to 5.

Several authors have treated sporadic stringhalt by lateral digital extensor myotenectomy, with allegedly good results. However, Crabbel *et al.* (1994) retrospectively studied 10 sporadic stringhalt cases where trauma to the dorsoproximal metatarsal region had been confirmed. Four were conservatively treated with controlled exercise and turnout and 6 were treated with extensive myotenectomy. Although the numbers were very small, there appeared to be no real difference in the follow up outcomes of the 2 groups.

Mephensein (Dixon and Stewart 1969), baclofen (Cahill and Goulden 1992) and particularly phenytoin (Huntington *et al.* 1991) appear to be quite useful in the symptomatic treatment of Australian stringhalt cases. Whether phenytoin is useful in the sporadic form has still to be determined. However, at doses of 7–15 mg/kg, sid to tid, several authors (Huntington *et al.* 1991; Wijnberg *et al.* 2000; Takahashi *et al.* 2002) have

indicated substantial clinical improvements with the drug. Notably, when the drug has been discontinued for a few days, signs have returned to then abate with further treatments. In one study (Wijnberg *et al.* 2000), determination of electromyography (EMG) characteristics of 'irritability' also indicated an improvement in the muscle function within the lateral digital extensor muscles following phenytoin treatment. Abnormal EMG patterns were also seen in stringhalt horses (Takahashi *et al.* 2002) but were not detected following clinical improvement with phenytoin treatment.

Huntington *et al.* (1989) determined nerve conduction velocities (NCVs) in the peroneal nerve of 4 horses affected with Australian stringhalt. The mean NCV was 19.5 ± 4.3 m/s. This compared with a NCV of 61 m/s in one control horse. In one of the affected horses, the NCV was repeated after it had recovered clinically and was then determined to be 48.6 m/s.

It is interesting that size and age may be predisposing factors in stringhalt, in so far as older and taller horses tend to be affected (Slocombe *et al.* 1992) in preference to smaller horses such as ponies and native Chilean breeds (Araya *et al.* 1998).

The pathological lesions present in Australian stringhalt have been well studied and undoubtedly represent a distal axonopathy preferentially affecting large diameter axons in long nerves (Cahill *et al.* 1986; Slocombe *et al.* 1992). This explains the muscle atrophy but there must also be selective involvement of γ-efferent fibres to account for the movement disorder with abnormal input via the 1α-afferent fibres to the γ-efferent neurons resulting in inappropriate firing of lateral digital extensor (and other) muscles.

The possibility that the presumed toxic factor that produces Australian stringhalt may play a role in RLN has fascinated several workers over the years. One 15.3 h Thoroughbred horse from a stringhalt outbreak in New Zealand was studied intensively (Cahill *et al.* 1986). This horse had severe stringhalt and grade 5 RLN and had very similar, though perhaps not identical, pathological lesions in the recurrent laryngeal as in pelvic and thoracic limb nerves. However, a more extensive study (Slocombe *et al.* 1992) showed that the changes in long limb nerves and recurrent laryngeal nerves in horses with stringhalt were the same. Another study (Cahill *et al.* 1985) showed that there was endoscopic evidence of abnormal laryngeal function in 10 of 11 horses with stringhalt. Such evidence of RLN was still present while these horses were recovering, or had recovered, over a period of a few days to 18 months. Finally, in horses affected with RLN there is a drop in the proportion of large to small diameter axons in the deep peroneal nerve of affected horses versus control horses, but no limb muscle atrophy or signs of stringhalt (Kannegieter 1989).

It would seem reasonable to conclude that the presumed toxic principle in Australian stringhalt is not the singular cause of idiopathic RLN. However, because the distal portion of long axons in tall horses are predisposed to distal axonopathy then a further insult with the toxin associated with Australian stringhalt could be the precipitating factor in inducing endoscopic and/or clinical evidence of RLN in horses suffering from stringhalt.

Idiopathic neuropathy with 'knuckling'

Japanese workers (Furuoka *et al.* 1994, 1998) have studied 3 cases of so called 'kuckling'. Clinically this is, in fact, the syndrome of prominent upright posture with flexion of the carpi and fetlocks that occurs as an acquired syndrome in weanlings and yearlings. These patients sometimes dragged the toes of their forelimbs. The workers described widespread peripheral nerve wallerian degeneration, which was most prominent distally. The formation of bands of Büngner and regenerative axonal sprouts do make this pathological process consistent with a diffuse distal axonopathy. Neurogenic muscle atrophy with some regeneration was seen with fibre type grouping, particularly in distal muscles. The comment was made that 'laryngeal paralysis was not observed in the present cases'; unfortunately, the basis for this statement was not given.

Scandinavian 'knuckling' horses

A detailed synopsis of 5 outbreaks of a hind limb 'knuckling' syndrome in horses was discussed at a neurology meeting in Sweden in 2001 (K. Gustafsson *et al.*, personal communication). There were a total of 24 cases occurring in an at-risk population of 75 animals. Detailed clinical, paraclinical and pathological investigations were undertaken on numerous affected cases, with only

3 surviving, one of which had recovered. In all cases silage was fed along with poor quality hay.

The clinical syndrome was one of varying degrees of sciatic nerve involvement, some horses showing signs of peroneal neuropathy more prominently and others showing signs of tibial neuropathy more prominently. A total of 9, detailed postmortem examinations were undertaken and in 2 cases there was evidence of mild peripheral wallerian degeneration.

During discussion, 3 other outbreaks were introduced. In Norway, 2 horses showed similar signs on the same property. One was examined at post-mortem, where modest changes in myelin and axon were evident in a peroneal nerve and a thoracic limb nerve. From the 1940s onwards, one practitioner indicated that he had been aware of numerous cases and had seen 10 cases in the last 3 years. One case at least was said to have evidence of wallerian degeneration in sciatic and femoral nerves. Some cases with mild signs improved and in one case the forelimbs were affected and there were lesions in forelimb nerves. Again, the diet consisted of small bale silage, although in one year, in which 3 cases occurred, they were fed only on hay. Finally, 5 out of 6 Norwegian Fjord horses were affected in another outbreak and 2 affected mares gave birth to normal foals.

Further discussion extended to 2 outbreaks in cattle showing evidence of sciatic/peroneal neuropathy, where they would stand with the hindlimbs more caudal than normal and showed either a tendency to stand on the dorsum of the pastern or to have considerable wearing of the hooves. In one case acrylamide was incriminated because it was found in plastic feeding troughs and in the other, vanadium because of an association with a local steel industry producing alloy vanadium steel.

Other toxic neuropathies

Clinically, some cases of lead poisoning can present with laryngeal and pharyngeal paralysis that is most probably associated with a peripheral neuropathy, but these signs are not present consistently (Dollahite *et al.* 1978; Aguilera-Tejero *et al.* 1996; Casteel 2001).

An outbreak of laryngeal paralysis in Arabian foals associated with administration of an organophosphorous anthelmintic haloxon has been described in detail (Rose *et al.* 1981). Because of a problem of large strongyle anthelmintic resistance, an intensive programme of haloxon administration was given to a group of Arabian and part-Arabian foals every 2 weeks from 2 days of age. There was a sudden onset of dyspnoea noted in 6 foals from 23 to 35 days of age. Detailed pathological investigations were carried out and revealed a distal axonopathy of the recurrent laryngeal nerves, worse on the left than the right, and associated bilateral neurogenic laryngeal muscle atrophy. Unfortunately, no limb nerves or muscles were examined. Of the 6 foals, only one totally recovered, the others either were euthanased for other reasons, or died or were euthanased because of the persistent upper respiratory embarrassment.

Additionally, 5 foals had received one or 2 doses of the anthelmintic, showed no signs of dyspnoea but when endoscoped showed Grade 2 RLN. All of these 5 foals recovered normal laryngeal function.

Suspected neuropathies

One investigating group has published some evidence that a primary neural lesion involving the sciatic nerve and its branches may contribute to the acquired sporadic syndrome of equine fibrotic myopathy (Valentine *et al.* 1994). However, with the weight of evidence of many cases having primary muscle pathology it is unlikely that sciatic nerve disease alone is responsible for many cases of this syndrome.

Numerous movement disorders, particularly involving the pelvic limbs in horses, may well involve a primary neuropathy. However, they have not been investigated in detail. One of these would be intermittent abduction of one pelvic limb during the protraction phase of stride. This syndrome could well be explained by an interruption to the 1α-afferent, γ-efferent pathway that almost certainly explains the abnormal movements occurring in stringhalt for example.

Finally, Dr Knottenbelt from Liverpool has presented numerous arguments that head shakers may result from trigeminal neuritis. There does not appear to be a lot of evidence for an inflammatory basis to this disease. However, the possibility that some or all of the head shakers that are seen commonly are related to a trigeminal neuropathy is still very possible. In the authors' experience, horses with overt clinical neuritis (extensive supurative sinusitis and associated surgeries) or histologic evidence of neuritis of the

trigeminal nerve have demonstrated allodynia (painful responses to non-noxious stimuli) as well as hypersensitivity and have not shown the classical syndrome of head shaking but have rubbed and excoriated the side of their face.

Conclusion

It seems reasonable that there are many insults, as discussed above, including vitamin deficiencies and other toxicities, that may target distal, large diameter axons and their myelin sheaths. Therefore these could result in clinical, pathological and/or paraclinical evidence of RLN. It is unlikely that any one of these other causes of RLN is responsible for the majority of idiopathic RLN cases.

Also, if indeed idiopathic RLN is a dying back polyneuropathy, why is some clinical evidence of these other syndromes not seen in severely affected horses?

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LARYNGEAL PARALYSIS IN DOGS

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NON-IDIOPATHIC LARYNGEAL PARALYSIS

Hereditary forms of laryngeal paralysis, that are part of a general neuropathy, are described in the Bouvier des Flandres, Dalmation, Rottweiler, Siberian husky (and crosses) and white coated German shepherd (van Haagen *et al.* 1978; Braund *et al.* 1994; Mahony *et al.* 1998; Ridyard *et al.* 2000). Inheritance patterns have been established in Bouvier des Flandres (van Haagen *et al.* 1981) and Dalmation (Braund *et al.* 1994). Dogs affected with congenital laryngeal paralysis are typically less than 7 months old at presentation and as they are part of a more generalised polyneuropathy or myopathy, they carry a poor prognosis. Congenital laryngeal paralysis has been less commonly described in other breeds, including toy breeds.

In older dogs, acquired laryngeal paralysis may also manifest as part of a peripheral diffuse neuropathy (Braund *et al.* 1989), in association with myasthenia gravis, hypothyroidism (Gaber *et al.* 1985), hypoadrenocorticism and lead or organophosphate poisoning. Laryngeal paralysis can also follow direct trauma to the recurrent laryngeal nerves, eg bite wounds, aggressive use of choke chains; or during thyroid, oesophageal, tracheal (prosthetic ring implants) or cervical spine surgery. It can also occur following damage to the recurrent laryngeal nerves by aggressive neck tumours (eg thyroid neoplasms) or mediastinum (eg lymphosarcoma). Ankylosis of the cricoarytenoid joint has also been described as a cause of laryngeal dysfunction in the dog, (but not the horse).

IDIOPATHIC LARYNGEAL PARALYSIS

Despite the extensive lists of aetiologies of canine laryngeal paralysis, most are due to unknown causes, as is the case with horses and are termed canine idiopathic laryngeal paralysis. This typically affects large breeds, such as Labradors, retrievers, Afghans, and Irish setters and has been recorded less commonly in smaller breeds and cats. One study showed that subclinical, unilateral or bilateral laryngeal paresis or paralysis was present in 25% of dogs undergoing general anaesthesia for non-related reasons (Broome *et al.* 2000).

In equine recurrent laryngeal neuropathy (RLN), pathological changes in laryngeal adductor muscle are more severe than abductor muscle changes. As dysphonia is one of the earliest signs of canine laryngeal paralysis, it may be that preferential adductor compromise also occurs early in the equivalent canine disease in dogs (Braund *et al.* 1988b). Dogs with idiopathic laryngeal paralysis however are generally not presented until the advanced stages of the disease process, not being subject to equivalent athletic demands of horses.

Idiopathic canine laryngeal paralysis is often bilateral and usually affects middle aged to older dogs (median age of 9.5 years in some clinical case studies). This is in contrast to equine RLN, which is primarily unilateral and usually diagnosed in younger horses. As is the case with horses, it has been suggested that male dogs are affected more frequently with idiopathic laryngeal paralysis. Clinical signs in acquired laryngeal paralysis are similar to those of the congenital form, but usually have a more gradual onset than the former. In addition to altered or absent barking (dysphonia), a soft non-productive cough is also common in affected dogs, which may be due to aspiration. Other commonly recorded signs in severely affected dogs include inspiratory stridor and dyspnoea during exercise, excitement or hot weather; and even life-threatening respiratory obstruction (syncope) in more severe cases. These signs are due to the severity and frequent bilateral nature of the

laryngeal paralysis. Older pet dogs with unilateral laryngeal paralysis are seldom presented with clinical signs but working or racing dogs with unilateral paralysis may present with clinical signs that interfere with their work (Greenfield 1987).

Greenfield *et al.* (1997) showed that clinical signs and measurable airflow changes associated with bilateral laryngeal paralysis did not develop in dogs until a median of 38 days following bilateral denervation of the recurrent laryngeal nerves. The reason for such a delay is unclear. It has been suggested that slow development of clinical signs in many naturally occurring cases of canine idiopathic laryngeal paralysis is due to progressive deterioration of the recurrent laryngeal nerve. However, progressive anatomical changes in the laryngeal structures may also be necessary for development of severe clinical signs.

Canine idiopathic laryngeal paralysis can be confirmed by ultrasonography in the conscious dog or laryngoscopy in the lightly anaesthetised subject (deeper anaesthesia will cause immobility of even the normal larynx), taking care to distinguish passive laryngeal abduction associated with deep expiration from normal active laryngeal abduction that occurs during inspiration.

Most publications on idiopathic canine laryngeal paralysis report the diagnosis and treatment of this disorder, or the use of dogs as models to treat human laryngeal paralysis. Few comment on the pathogenesis or epidemiology. Early work suggested involvement of a neurogenic or denervation atrophy of the laryngeal muscles (such as occurs in horses) and this has become widely accepted as the most likely aetiopathogenesis (O'Brien *et al.* 1973). Most studies have either used retrospective case analysis (O'Brien *et al.* 1973); have been restricted to congenital laryngeal paralysis (van Haagen *et al.* 1978, 1981) or have analysed laryngeal innervation in normal dogs (Braund *et al.* 1988 a,b), and so their data and conclusions can be difficult to extrapolate to idiopathic canine laryngeal paralysis. For example, van Haagen *et al.* (1978, 1981) have described histological evidence of neurogenic axonal degeneration of the right and left recurrent laryngeal nerves in cases of congenital laryngeal paralysis

Biopsies of intrinsic laryngeal muscles in cases of idiopathic canine laryngeal paralysis have shown evidence of neurogenic atrophy (O'Brien *et al.* 1973; Love *et al.* 1987). A comparative study of the neuropathology of canine and equine 'RLN' could provide much useful information for both species.

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SESSION 2:

Endoscopic grading systems for laryngeal paralysis

Chairman: Paddy Dixon

4-GRADE SYSTEM FOR EQUINE LARYNGEAL FUNCTION

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The impetus for this grading system was the controversy regarding the clinical significance of various forms of asynchrony and/or asymmetry of the arytenoid cartilage during examination at rest or after exercise. The 4-grade system for assessment of resting laryngeal function was published in 1991 (Hackett *et al.* 1991; Ducharme *et al.* 1991) to meet 3 objectives: 1) a simple practical system; 2) correlation with evaluation at exercise; and 3) a system that was consistent between observers and during re-examination. In this aspect of laryngeal evaluation, the focus is on the respiratory function and more specifically the abductor function of the arytenoid cartilages. It is well known that laryngeal adductors are generally more severely affected when compared to the abductors (Duncan *et al.* 1991). However, adductor deficit is not of clinical significance either for the respiratory or digestive role of the larynx except perhaps from its recognition in the slap test. Although the grade is used in reference to the left arytenoid cartilage, it has also been used to describe the more rare form of failure of right arytenoid cartilage abduction.

The resting 4–Grade laryngeal examination was developed using a population of 108 horses (61 SB, 38 TB, and 9 other breeds that consisted of 66 females, 34 geldings and 8 entire males (6 years ± 3.6 SD). In the early development of the 4–Grade system, 3 main criteria were used: degree of symmetry, degree of synchrony, and degree of abduction. Degree of laryngeal asymmetry was a criterion used in the grading, but was abandoned because of the low intra-observer agreement rate (37%). It was found that in the initial population, all 108 horses could be classified in one of 4 grades (Table 1) summarised in the grading system. A second conclusion was that most of the discrepancies in classification between observers and within observers were due to lack of agreement on the degree of asynchronous movement of the left arytenoid cartilage. In contrast, there was substantial agreement on the ability to recognise full arytenoid cartilage abduction. This criterion (ability to identify full arytenoid cartilage abduction) is therefore, the foundation of the 4-Grade system and, as time has proved, the criterion with the most clinical significance.

A second major issue to resolve regarding any laryngeal grading system is identifying the correct laryngeal grade. Therefore, we and others (Archer *et al.* 1991; Ducharme *et al.* 1991) evaluated various conditions during examination. It was discovered that full abduction could be induced consistently by nasal occlusion or by inducing a swallow by touching the tip of the epiglottis with a video-endoscope. In addition, in many cases, the use of a respiratory stimulant was found not only to increase the respiratory frequency, but also the degree of abduction of the arytenoid cartilages. It was also determined that the use of sedation such as xylazine hydrochloride decreases the degree of abduction and affects the degree of laryngeal asynchrony (Valdez-Valquez *et al.* 199). The nasal side that the endoscope is passed influences the viewer's perspective of the larynx and decreases the repeatability of the grading system. Therefore, it was recommended that laryngeal grading should be performed in the resting animal without sedation, always using the same nostril and if needed, with the use of the twitch (Ducharme *et al.* 1991).

The real value of any resting grading system is its correlation with laryngeal function at exercise and performance. Four studies have correlated the resting laryngeal grade to the degree of abduction of the arytenoid cartilage during strenuous

TABLE 1: 4-Grade classification system for assessment of equine laryngeal respiratory function in unsedated horses examined at rest*

Laryngeal grade	Definition
I	Synchronous and full abduction of the arytenoid cartilages.
II	Asynchronous movement (hesitation, flutter, abduction weakness, etc.) of the left arytenoid cartilage during any phase of respiration. Full abduction of the left arytenoid cartilages (when referenced to the right) is observed either by swallowing, nasal occlusion or the use of respiratory stimulants.
III	Asynchronous movement (hesitation, flutter, abduction weakness, etc.) of the left arytenoid cartilage during any phase of respiration. Full abduction of the left arytenoid cartilages (when referenced to the right) cannot be induced either by swallowing, nasal occlusion or the use of respiratory stimulants.
IV	Midline or paramedian position of the left arytenoid cartilage and no substantial movement of the left arytenoid cartilage can be induced by swallowing, nasal occlusion or the use of respiratory stimulants.

*Update from Hackett *et al.* (1991)

TABLE 2: 3-Grade classification system for assessment of equine laryngeal respiratory function in horses examined during exercise*

Laryngeal grade	Definition
A	Full abduction of the arytenoid cartilages during inspiration.
B	Partial abduction of the left arytenoid cartilages between full abduction and the resting position.
C	Abduction less than resting position including collapse into the right half of the rima glottidis.

*Update from Rakestraw *et al.* (1991)

exercise on high-speed treadmill. In the initial study (Rakestraw *et al.* 1991), 49 horses were evaluated while exercising on a 5% inclined high-speed treadmill for 8 min at a maximum speed of 8.5 m/s. Hammer *et al.* (1998) compared the outcome of 26 Grade III horses to exercising grade in horses travelling up to 14 m/s. Martin *et al.* (2000) described the correlation of 73 horses with Grade II, III and IV to exercising grade in horses travelling up to 14 m/s. Finally, we reviewed the outcome of 313 horses exercising on the treadmill at our hospital at speeds up to 16 m/s (Rakestraw *et al.* 1991). These 4 studies were combined for an analysis to establish the results of the comparison of the resting laryngeal Grade in 461 horses: 204 horses had a laryngeal Grade I, 114 had a laryngeal Grade II, 126 had a laryngeal Grade III, and 17 had a laryngeal Grade IV. The results are summarised in Figure 1. It was found that 100% of horses with a laryngeal Grade I, and 96% of horses with a laryngeal Grade II had full arytenoid cartilage abduction (Grade A) throughout exercise. All horses without significant movement of the arytenoid cartilage at rest (Grade IV), experienced collapse of the arytenoid cartilage (Grade C). Most horses (85%) with laryngeal Grade III had some degree of arytenoid cartilage collapse. Furthermore they were a range of outcome in horses with laryngeal Grade III, indicating that horses with this type of laryngeal grade need further evaluation, such as examination during exercise. The results of this study indicate that failure to obtain and maintain arytenoid abduction at rest is the major criterion to triage horses that probably have an abnormal arytenoid cartilage function at exercise. This finding was collaborated further by another group of investigators (Christley *et al.* 1997) who looked at indices of ventilation during exercise. They found that the only criterion in the resting laryngeal grade at rest that differentiates horses with abnormal indices of ventilation at exercise was the presence or absence of full arytenoid cartilage abduction. Indeed, horses that did not have full

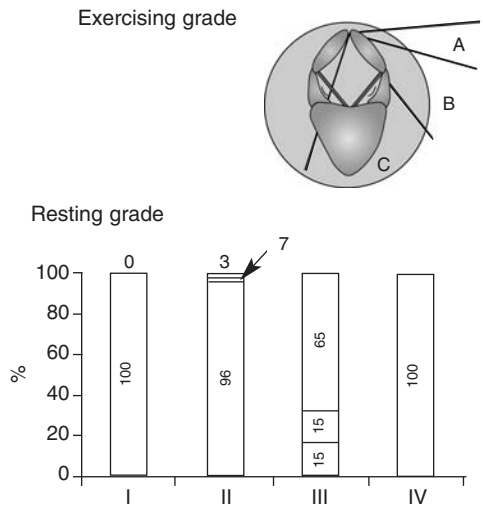

Fig 1: Correlation of 4-grade resting laryngeal grade to exercising grade in 443 horses.

arytenoid cartilage abduction at rest experienced statistically more severe exercise-induced hypoxemia and hypercarbia. And finally, when Stick *et al. (*2001), studied the correlation between resting laryngeal grade in yearlings and future performance in Thoroughbred racehorses, they found that yearlings with laryngeal Grade-I and -II had significantly better racing performance as adults, compared with yearlings with Grade-III arytenoid cartilage movements.

In summary, since its introduction 12 years ago, the 4-grade system has been validated using correlation between resting examination and treadmill examination, as well as between resting examination and future performance. This allows the equine practitioner an accurate, repeatable mean to identify horses that are likely to experience clinical disease during exercise (Grade III and IV) and those whose laryngeal function is normal (Grade I and II).

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5-POINT GRADING SYSTEM OF LARYNGEAL FUNCTION IN HORSES DURING QUIET BREATHING

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Clinicians are generally obsessed with scoring or grading disorders which they encounter, whether it be the severity of feather pecking in chickens, hip dysplasia in dogs or function of the equine larynx. The purpose is invariably to provide an objective and repeatable method to document observations. This 5-point scheme to grade the endoscopic perception of equine laryngeal motility at rest in the stable, including on return from exercise – quiet breathing has been used at Bristol since the early 1980s and in Australasia since 1993 (Lane 1993; Kannegeiter and Dore 1995). It is not very different from the 4-point scale used in North America (Rakestraw *et al.* 1991) and has been continued simply because it has been found to be workable in everyday equine laryngological practice.

Grade 1: All movements, both adductory and abductory are synchronised and symmetrical regardless of whether sedated or examined before or after exercise. A 'mirror' effect is achieved through a perspective artifact whereby the right arytenoid appears less abducted when the endoscopy is performed through the right nostril, and the left is similarly less abducted when the larynx is viewed via the left nasal chamber.

Grade 2: All major movements are symmetrical and a full range is achieved. Transient asynchrony, flutter or delayed opening may be seen (Baker 1983).

Grade 3: Asymmetry of the rima glottidis at rest due to reduced motility by the left arytenoid cartilage and vocal fold. On occasions, typically after swallowing or during the nostril closure manoeuvre, full symmetrical abduction is achieved.

Grade 4: There is consistent asymmetry of the rima glottidis but with some residual active motility by the left arytenoid cartilage and vocal fold. Full abduction is not achieved at any stage.

Grade 5: True hemiplegia. There is obvious and consistent asymmetry of the rima glottidis with no residual active motility by the left arytenoid cartilage and vocal fold. No responses to the 'slap' test are provoked.

At the request of a major owner/breeder video-endoscopic recordings were made of the upper respiratory tracts of 3,497 yearlings during a 15 year study and the findings have been reviewed. The study has provided a unique opportunity to estimate the prevalence of laryngeal disorders as and to assess the distribution of functional anomalies in the Thoroughbred. Although the subjects represent an elite group of horses on the basis of genetic selection, unlike previous investigations (Pascoe *et al.* 1981; Raphael 1982; Baker 1983; Lane *et al.* 1987; Sweeney *et al.* 1991), these were unbroken and, therefore untried animals. Initially, approximately half of the yearlings had been purchased, some privately and some at auction, and half were homebred. In recent years the majority of the yearlings examined have been homebred and yet the distribution of the findings has remained constant. The findings in regard to the distribution of functional grading are shown in Table 1.

The 12 yearlings for which no grade was assigned included 7 afflicted with the fourth branchial arch defect syndrome and an additional 5 with malfunction on the right side and where no physical explanation was discovered.

TABLE 1: Laryngeal function grading of 3,497 yearlings

RLN grading	1	2	3	4	5	Not graded
	784	2006	617	69	9	12
%	22.4	57.4	17.6	2.0	0.026	0.34

All horses showing Grade 5 were later confirmed to show severe obstructive dyspnoea and that most of the Grade 4 horses were also clinically diseased. This is in line with previous findings (Morris and Seeherman 1991) which showed that dynamic collapse of the left arytenoid cartilage (ACC) and vocal fold (VCC) developed in the overwhelming majority – 20 out of 27 horses with Grade 4 (sic) motility at rest. The prevalence of clinically significant recurrent laryngeal neuropathy (RLN), ie Grades 4 and 5, was found to be 2.26% and this can be regarded as a base level for an unselected population of Thoroughbred horses. It compares with previous reports of selected groups (Table 1).

The greatest diversity of opinion hinges on the significance of Grade 3 motility and whether this represents a performance-limiting malfunction, or whether it should be viewed as a variant of normality. This grading does not discriminate between the abilities to achieve and to maintain full abduction as these are subjective judgements. A large number of group, listed and stakes race winners have come from the horses with this grading. The numbers of Grade 3 horses which required, or were subjected to corrective surgery later in life, is not known but from studies of animals examined by highspeed treadmill endoscopy (see page 47) it is safe to conclude that the majority are 'normal', showing sustained symmetrical abduction of the arytenoid cartilages and vocal folds throughout exercise. However, horses with Grade 3 RLN are at a significantly greater risk of sustaining ACC and/or VCC at exercise than horses with Grades 1 and 2 RLN.

Although the designation of a Grade 1 or 2 score during quiet breathing does not convey absolute protection against ACC or VCC during exercise, susceptible horses invariably show other features such as palpable atrophy of the *crico-arytenoideus dorsalis* muscle and a progressive inspiratory noise at exercise.

In conclusion, the 5-point grading scheme for RLN has proved to be a practicable means to assess laryngeal function in Thoroughbreds that has a useful predictive value with regard to the likelihood of clinical disease, provided it is used in conjunction with such techniques as palpation and an exercise test. Assessments of upper respiratory tract function that rely on endoscopy during quiet breathing alone should be regarded as inadequate.

TABLE 2: Previous endoscopic surveys to assess laryngeal function in horses

Authors	Group size	Population	RLN incidence (sic. grades 4 and 5)
Pascoe *et al.* (1981)	235	Horses in training	2.6%
Raphael (1982)	479	"	3.3%
Baker (1983)	537	"	4.7%
Lane *et al.* (1987)	6860	Yearlings at sale	0.96%
Sweeney *et al.* (1991)	678	Horses in training	4.0%

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ENDOSCOPIC GRADING SYSTEMS FOR LARYNGEAL PARALYSIS - 6 GRADE SYSTEM

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There is general agreement that laryngeal endoscopy is currently the gold standard for diagnosing equine laryngeal paralysis. Endoscopic evaluation during high-speed treadmill exercise is even more accurate in the rare cases that show normal laryngeal function (including laryngeal asynchrony and arytenoid shivering) at rest, but have a significant deterioration during fast exercise. Treadmill endoscopy is also the optimal technique for assessing *degree* of laryngeal abductor dysfunction, which is determined most accurately at fast work (Morris and Seeherman 1990). However, facilities for treadmill endoscopy are limited and such procedures involve training of horses, delay in obtaining results, technical expertise and capital outlay. Therefore, for the foreseeable future, resting endoscopy remains the standard method for diagnosis and assessing the degree of equine laryngeal paralysis.

Resting laryngeal endoscopy using flexible endoscopes has been practiced for 30 years and there is widespread consensus that asynchrony and shivering (usually left sided) of a larynx that can obtain and maintain full abduction are generally insignificant. Such findings are now rarely viewed with the same degree of suspicion as they were prior to work such as that of Morris and Seeherman (1990) who showed conclusively that horses with laryngeal asynchrony and flutter can maintain normal laryngeal function during high speed treadmill endoscopy.

In the author's opinion, even permanent, low degrees of laryngeal asymmetry can be present with no or minimal functional effects. High-speed treadmill endoscopy of such cases has shown normal laryngeal function during strenuous exercise. The author also disagrees with any classification system, which rates normal function as 'Grade 1' of that dysfunction. It must be more rational to have normal as zero, with increasing degrees of dysfunction classified numerically.

The widely used laryngeal paralysis grading system of Ducharme *et al.* (1989) and Hackett *et al.* (1991) separated laryngeal function into 4 grades, ie totally synchronous; asynchrony/flutter; asymmetry; and total hemiplegia. However, the major disadvantage of that system is that it classifies all asymmetry (incomplete abduction) of the larynx into a single category. Consequently, horses with very mild asymmetry (which may be functionally normal) are classified in the same group as horses with almost total laryngeal paralysis which, during treadmill exercise, will have a grossly abnormal larynx. The sub-division of this category into 3 subgroups provides a 6-grade system, which allows a better assessment of laryngeal function, as illustrated in Table 1 (Dixon *et al.* 2004). The 5-grade system of Lane (1993) partially addressed this issue but it is believed that the proposed 6-grade system is more advantageous. Embertson (1997) also sub-divided the resting endoscopic grades of recurrent laryngeal neuropathy (RLN) that describe asymmetric movements into 2 grades, and Hammer *et al.* (1998) sub-divided this same grade, as assessed during high speed treadmill exercise, into 3 grades.

For a resting endoscopic examination, the endoscope is inserted via the right ventral meatus and positioned midline in the nasopharynx. The symmetry and synchrony of arytenoids is observed during quiet breathing, following swallowing (induced by trans-endoscopically flushing water through the nasopharynx) and during temporary nostril occlusion (to assess the degree of maximal arytenoid abduction).

In a study where 2 independent observers endoscopically examined and graded 45 horses using the above 6-grade laryngeal endoscopic

TABLE 1: Grading of RLN by resting endoscopic examination; Dixon *et al.* (2000)

Grade	Endoscopic findings
0	(Normal) perfect synchrony of arytenoid movement, and symmetry of appearance, full bilateral arytenoid abduction achieved and maintained (eg during nasal occlusion on excitement).
1	(Normal) asynchronous arytenoid movements, ± presence of arytenoid or vocal shiver but full symmetrical arytenoid abduction achieved and maintained.
2	(Mild paresis) slight arytenoid asymmetry, incomplete arytenoid abduction or complete but transient abduction ie unable to maintain full abduction.
3	(Moderate paresis) obvious arytenoid abductory deficit and arytenoids asymmetry.
4	(Severe paresis) marked but not total arytenoid abductory deficit and asymmetry, very little arytenoid movement.
5	(Total paresis [hemiplegia]) no arytenoid movements detectable.

technique, a highly significant ($P<0.001$) positive correlation (Spearman rank correlation coefficients; right = 0.817; left = 0.913) was obtained between observers (Hawe *et al.* 2001). The paired left endoscopic scores did not differ significantly ($P<0.05$) between observers. Additionally right and left RLN grades allocated post exercise endoscopy (n = 16) by both assessors showed a highly significant ($P<0.001$) positive correlation (Hawe *et al.* 2001). As previously noted with endoscopic grading of laryngeal function by Archer *et al.* (1989); Ducharme *et al.* (1989); Hackett *et al.* (1991); Christley *et al.* (1997), having pre-determined, clearly defined criteria for endoscopic grading helps minimise subjectivity and inter-assessor variation in the above study.

These laryngeal endoscopic grading systems are semi-quantitative and by increasing the number of grades, it becomes theoretically more difficult for different workers to use them. Notwithstanding, it is in reality easy for clinicians to recognise resting laryngeal asymmetry and then to differentiate between horses that have, mild, moderate or severe (but not total) laryngeal paresis. We believe by using such a 6-grade endoscopy system, clinicians can document more accurately the degree of equine laryngeal paralysis present and would advocate its use as the endoscopic standard for laryngeal function evaluation.

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SESSION 3:

Progression and endoscopic variation

Chairman: Paddy Dixon

LONG-TERM LONGITUDINAL STUDY OF LARYNGEAL FUNCTION IN 187 FOALS

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The sale of foals to be retained to go into training or to be resold as yearlings – 'pin-hooking' – forms a major component in the marketplace for Thoroughbred horses. Prospective purchasers seek to take precautions to ensure that the animals concerned will be suitable for resale, or can be trained effectively for racing. It has become fashionable for foals to be subjected to endoscopic examinations on behalf of prospective purchasers prior to sale. Some breeders also seek endoscopic examinations of foals ahead of sales if only for peace of mind. However, is endoscopy of foals a worthwhile procedure and are the results dependable?

A number of congenital structural abnormalities of the upper respiratory tract (URT) can be diagnosed accurately by a combination of palpation and endoscopy (Embertson 1997). These conditions include midline clefts and other defects of the palate, pharyngeal and sub-epiglottal cysts and fourth branchial arch defects. These abnormalities are collectively relatively unusual and arise in no more than 0.5% of Thoroughbreds born (see page 49). A major interest for foal purchasers often centres on laryngeal function and the identification of recurrent laryngeal neuropathy (RLN). To assess the reliability of the interpretation of laryngeal function, a group of 197 foals were examined by endoscopy and 187 were available for re-examination one year later. The video-endoscopic records were reviewed 'blindly' – the findings are shown in the Table 1 and the obvious conclusion is that there are inconsistencies in the 2 series of results. The laryngeal function of some foals appeared to be within normal limits, but then to

TABLE 1: Comparison of laryngeal function of 197 foals with the same horses one year later

		Yearling grades (187)					
		1 (32)	2 (112)	3 (37)	4 (6)	5 (0)	NA
Foal grades (197)	1 (36)	**12**	14	4	0	0	6
	2 (123)	17	**86**	16	1	0	3
	3 (29)	3	11	**12**	2	0	1
	4 (9)	0	1	5	**3**	0	0
	5 (0)	0	0	0	0	**0**	0

10 horses (NA) were not available to be examined as yearlings

TABLE 2: Comparison of the laryngeal function grading of 187 yearlings with their median winnings between 2 and 4 years of age

Laryngeal grade	1 (32)	2 (112)	3 (37)	4 (6)	5 (0)
Median Winnings (£)	1933	5358	5367	724	0

have 'deteriorated' over the following 12 months, while others, which would have been considered unsuitable had they been examined prior to sale as foals turned out to be totally normal. There was no foal or yearling in the group studied which was afflicted with any of the congenital abnormalities mentioned above. In summary, endoscopy of foals is not an absolutely dependable technique and decisions on whether to buy or to reject should not be based upon this technique.

A review of subsequent racing performances at 2, 3 and 4 years of age showed that the median stakes winnings of the yearlings in this study were greatest in the Grade 3 group (Table 2).

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ENDOSCOPIC OBSERVATIONS ON LARYNGEAL SYMMETRY AND MOVEMENTS IN YOUNG RACING HORSES

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Respiratory endoscopists have found arytenoid cartilage movement during quiet respiration in horses to be remarkably variable. This variability, according to the findings of Baker (1983), does not progress in the individual animal to hemiplegia. About 87% of the horses Baker studied, however, were 3 years of age or over. It has been shown that the pathological changes characteristic of denervation and re-innervation in the intrinsic laryngeal muscles dramatically increase in prevalence and severity in Thoroughbred horses during the yearling to 2-year-old period (B.H. Anderson, unpublished data). Presumably, if asynchronous arytenoid movements are related to these pathological changes, then progression of asynchrony to hemiplegia is most likely to be found in animals of this age.

An endoscopic survey of young racehorses was performed (Anderson *et al.* 1997) to examine the prevalence and character of laryngeal movements during quiet respiration. The main aim was to determine whether those arytenoid movements that could possibly reflect the efficiency of left dorsal cricoarytenoid muscle function changed over a period of time.

Of the 462 horses examined, 439 were Thoroughbreds and 23 were Standardbreds, 250 were less than 2 years of age (6–21 months), and 202 were 2 years old. One hundred and nine of these horses were examined again 16 months later. Arytenoid movements were given one of 4 grades. Grades 1 and 2 were considered normal and unlikely to be the result of abnormal left dorsal cricoarytenoid muscle function, whilst Grades 3 and 4 were considered likely, or almost certainly the result of abnormal left dorsal cricoarytenoid muscle function (a grading system similar to that used by many endoscopists for laryngeal examinations). The percutaneous prominence of the muscular process of left and right arytenoid cartilages, endoscopic arytenoid movement on left and right sides, age, sex and breed were recorded. Chi-squared analysis was used to determine the association between age, breed, sex and the other recorded variables, and the presence or absence of abnormal laryngeal movements.

At the first examination, 48% of the horses had Grade 1, 37% grade 2, 15% Grade 3 and 0.2% Grade 4 left laryngeal movements. Of the horses examined 16 months later 52% had Grade 1, 33% Grade 2, 14% Grade 3 and 1% Grade 4 left laryngeal movements. Fifteen percent of horses with Grade 1 and 8% with Grade 2 initially were found to be Grade 3 at the subsequent examination. Conversely, 53% of horses with Grade 3 initially were found to be Grade 1 and 21% Grade 2 at the subsequent examination. One horse that was Grade 3 at the initial examination was Grade 4 at the subsequent examination. Overall, 43% of horses were graded the same, 29% were given a 'better' grade and 28% were given a 'worse' grade.

Age and sex were not associated with abnormal left laryngeal movements. The presence of abnormal arytenoid movements was significantly less in Standardbreds, but significantly higher, in those horses that had a more prominent muscular process of the left arytenoid cartilage. The number of Grade 2 and 3 laryngeal movements recorded on the left side was significantly higher than the right.

Why such a variation in grades occurred is not known but one or some combination of the following possibilities could have been involved: the repeatability of the endoscopic examination technique used was unsatisfactory; the cut-off point between normal (Grade 1 and 2) and

abnormal (Grade 3 and 4) laryngeal movement grades was inaccurate; the inter-relationship between pathological changes in the left arytenoid muscles and the arytenoid movements considered to be indicative of these changes is unreliable or erroneous; in young Thoroughbred horses, changes in grade of arytenoid movements frequently occur. In the present study, 8 of 11 of the horses changing from what was considered normal laryngeal movements to abnormal, changed from Grade 1 to Grade 3. Similarly, of those horses changing laryngeal grade from what was considered abnormal to normal, 10 of 14 changed from Grade 3 to Grade 1. Although the results of this study will be affected by the repeatability of the endoscopic procedure used it is unlikely that errors were made in differentiating Grade 3 laryngeal movements from Grade 1. Not withstanding the other factors mentioned, in this age group it appears that: a) asymmetrical laryngeal movements are common; b) laryngeal movements may interchange between what is considered normal and abnormal; c) development of more obvious degrees of asynchrony is low (12%); and d) development of laryngeal hemiplegia in horses that have endoscopic evidence of deficient left abductor muscle function is also low (5%).

Recent investigations

More recently, Dixon *et al.* (2002) reported on endoscopic and/or clinical progression of recurrent laryngeal neuropathy (RLN) in older national hunt and sport horses (predominantly Thoroughbred). Fifty-two of the 351 horses examined (15%) showed evidence of progression of the degree of laryngeal dysfunction over a median period of 12 months (range 1.5–48 months) with the onset of progression occurring at median age of 7 years.

The results of this study and the one reported above (involving predominantly Thoroughbreds) indicate that the progression of RLN or alternatively, clinically significant arytenoid abductor dysfunction, could vary between 5 and 15%. The age of onset of the deterioration in arytenoid function is, however, markedly different. The reason for this is unknown. In addition, Dixon *et al.* (2002) have reported that the time or rate at which progression can develop may be as short as 6 weeks. In other cases deterioration can take months to years. This has important implications for examination of horses for sale and supports the clinical impressions of veterinarians involved in sales endoscopy.

In contrast to the above study, Dixon *et al.* (2002) found no evidence of improvement in laryngeal function in the clinical cases examined. In another endoscopic study of the laryngeal movements of Thoroughbreds (Lane 2000) reported on the results of 2 examinations conducted 12 months apart on 197 foals. At the time of the second examination 187 yearlings were available. Video-endoscopic records were reviewed 'blindly' on 3 occasions. The results showed marked inconsistencies in the 2 series. The laryngeal function of some foals appeared to be within normal limits, but appeared to have deteriorated over the following 12 months. Conversely, 9 foals examined initially were observed with marked abductor deficiency (Grade 4 of 5) but when examined 12 months later one was considered normal (Grade 2 of 5), 5 were considered equivocal (Grade 3 of 5) and 3 remained Grade 4. While it was concluded that endoscopy of foals is not reliable and decisions on whether to buy or to reject horses should not be based upon this technique, it is possible that some of the variation in laryngeal function could be the result of successful re-innervation of de-innervated intrinsic laryngeal musculature.

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VARIATION IN LARYNGEAL FUNCTION IN MATURE HORSES AND PROGRESSIVE CASES OF RECURRENT LARYNGEAL NEUROPATHY (RLN)

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The literature on 'roaring' in the 17th and 18th centuries cites many reports of cases of recurrent laryngeal neuropathy (RLN) that appear to be progressive. However, due to lack of endoscopic verification of the precise cause of the upper airway stridor, it is possible that some of these cases were non-RLN upper airway abnormalities. In contrast to this early literature, repeated endoscopic examinations of National Hunt racehorses over a number of years by Baker (1982) found no evidence of progression of RLN. However, more recent observations by Embertson (1997) in foals in the USA and Anderson *et al.* (1997) in 2–3-year-olds in New Zealand showed variation (improvement and deterioration) in laryngeal function between examinations. There may be physiological reasons why 'normal' equine larynges (including those displaying arytenoid shivering or asynchrony, but can remain fully abducted) show such variation but endoscopic interpretation of laryngeal function is also subject to both inter- and intra-observer variation at repeat examinations (Ducharme *et al.* 1991; Hackett *et al.* 1991).

The histology (fibre-type grouping on ATP-ase histochemistry) of RLN affected muscles shows it to be a dynamic disease; affected muscles show evidence of cyclical de-innervation and re-innervation (Gunn 1973; Duncan *et al.* 1974; Cahill and Goulden 1986). Based on the above muscle histochemistry, RLN affected laryngeal function may improve or worsen, depending on the balance between laryngeal muscle de-innervation or re-innervation at the examinations.

The main pathological features of the recurrent laryngeal nerve in horses suffering from RLN suggest a chronic progressive disorder. Griffiths (1991) suggested that some horses with sub-clinical RLN lesions would later develop clinical disease. In view of the above noted pathological lesions of the laryngeal muscle and the recurrent laryngeal nerve, it is perhaps not surprising that some cases of RLN are in fact progressive.

Dixon *et al.* (2002) found that 52 out of 351 horses (15%) with RLN showed progression in the degree of the disease over periods ranging from 6 weeks to over 4 years (mean 12 months). The median age at the onset of progression was 7 years. In 30 cases there was both endoscopic evidence, with a median deterioration of 3 endoscopic grades (range 1–5 grades), and clinical evidence, with 97% of these horses concurrently developing sudden-onset of abnormal exercise-related respiratory noises and 43% concurrently reporting reduced exercise performance. In some of these cases the degree of dysfunction changed from normal function to total hemiparesis. Some of these cases were examined on treadmills on a number of occasions, absolutely confirming that there are progressive and often major changes in laryngeal function.

Of the remaining 22 horses, there was just clinical evidence of RLN progression, including the sudden onset of abnormal exercise respiratory sounds in 73% and the worsening of such sound in 23%. These abnormal 'noises' were associated with reduced exercise in 59% of these cases. Although examined endoscopically on just a single occasion, 59% of the 22 cases had marked (total or almost total hemiparesis) that did not appear compatible with the previous exercise performance history. Although 52 cases in the study had apparent deterioration of laryngeal function, no improvement in laryngeal function was detected in any cases in the current study, in

contrast to the findings of Flemming (1889) and Anderson *et al. (*1997).

More recently, even more acute cases of laryngeal function variation were recorded, including a case where 2 competent equine veterinarians clinically examined (including during exercise) a racehorse pre-purchase, and during a sale, and neither found any detectable abnormality. Within 4 days of the sale the horse started to make a 'noise' and endoscopic examination a week later showed marked laryngeal dysfunction. The author's referral population includes very few young racehorses, but such deterioration in laryngeal function may also occur in this age group.

The main significance of the above findings concerns interpretation of differences in clinical or endoscopic findings between veterinarians, especially at sale times. Provided that the initial clinical and/or endoscopic examinations are performed by competent veterinarians, and that a standard and identifiable endoscopic grading system is used, any differences in clinical and endoscopic findings detected at a later stage must be considered as possibly being due to further deterioration of laryngeal function during this period. Some delegates at this Havemeyer Workshop (Embertson 2004; Anderson 2004) presented further evidence of cases of short-term deterioration in laryngeal function, further clarifying to the sales authorities and the litigation lawyers that not all such differences are due to negligence.

Another practical aspect of recognition of progressive deterioration in laryngeal function concerns the treatment of such cases. Cases that initially have normal laryngeal function, and are later found to have a mild to moderate degree of laryngeal paralysis, may be treated by ventriculectomy and/or vocalcordectomy (varying on their work discipline). However, with further progression of the laryngeal hemiparesis, affected horses may require laryngoplasty at a later date. Clinicians should consider whether cases of RLN are progressive by historical, endoscopic and clinical findings, and if progression is strongly suspected, perhaps laryngoplasty should be performed on the first occasion on such horses, even if severe degrees of laryngeal dysfunction are not present at that time.

It is concluded there is now irrefutable historical, clinical and endoscopic evidence that equine laryngeal function can deteriorate often slowly but on occasions very rapidly (over weeks) in a proportion of RLN cases.

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SESSION 4:

Diagnosis of RLN and non-RLN URT disorders

Chairman: Eric Parente

SALES: PROBLEMS IN DIAGNOSIS OF RLN – UK PERSPECTIVE

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Tattersalls, Ltd Conditions of Sale re Wind Conditions in Thoroughbred Yearlings or Horses in Training:

1978–1983	A horse is returnable if it can be heard to make a characteristic abnormal inspiratory sound when exercised actively.
1984	As above but add and/or has laryngeal hemiplegia when examined with the endoscope.
1985	Makes a characteristic abnormal inspiratory sound AND has laryngeal hemiplegia (RLN).

After the horse is sold the purchaser appoints a veterinary surgeon to listen when the horse is lunged and if it makes any abnormal inspiratory noise it is endoscoped. If it is found to have recurrent laryngeal neuropathy (RLN) the purchaser returns the horse to Tattersalls for adjudication by their panel. The panel has included the authors since 1984 with occasional alternates. Yearlings are returnable within 24 h of sale, and horses in training within 7 days. The panel examines the horse soon after its return, often later on the same day as the original test by the purchaser's veterinary surgeon. Each member of the panel palpates the larynx for atrophy, cartilage symmetry and surgical scars. The larynx is also palpated immediately after exercise for fremitus and the right arytenoid depression test. These observations provide supportive evidence and with the exception of surgical scars would not be decisive. The yearling is then lunged on both reins at the canter which may be repeated on a particular rein but it is never lunged to exhaustion and the exercise lasts only a few minutes. The lunging ring is well designed with good acoustics and a fibresand surface. Horses in training are ridden at a fast canter uphill on a polytrack surface with members of the panel standing close to the end of the canter to hear the animal pass by. This exercise can be repeated if necessary. The horse is then allowed to settle down after the exercise before it is endoscoped. Originally the panel endoscoped horses before exercise but this policy was changed in order to mimic the examination by the purchaser's veterinary surgeon and eliminate bias in the exercise test. The horse is scoped without a twitch, if possible, and only sedated if essential and with the agreement of the vendor and purchaser. The larynx and pharynx are examined via the right nostril in order to reduce the parallax effect for left sided RLN. During the examination the nostrils are blocked and deglutition is stimulated. The panel does not employ a slap test. Until recently each member examined the larynx via the same endoscope in sequence. A video-endoscope is now used but the examination is not recorded on tape

Endoscopic criteria for RLN which mean the horse will be returnable include gross resting asymmetry of the rima glottidis, failure to achieve symmetric or full abduction and failure to maintain symmetric abduction during a complete inspiration, ie premature weakness. Each panelist forms his own view but sometimes members have discussed certain aspects such as the character of the inspiratory noise or the endoscopic findings. In most cases the decision was unanimous but in 11.5 % or 41 cases out of 356 between 1989 and 2002 majority decisions were reached.

Problems with the system include incomplete examination by a purchaser's veterinary surgeon and some veterinarians have a higher percentage of passes among those they return to the panel. Judgement of noise can be difficult in yearlings

TABLE 1: Summary of race records of yearlings returned for their wind under Tattersalls' Condition of Sale and Control 1978–1984

1978-1983
27 returned for whistling/roaring only
(52% did not run; 22% won; 2.8 average starts per horse)

1984
28 returned for whistling/roaring and/or RLN.
(39.3% did not run; 39% won; 5.9 average starts per horse)

1978-1984
controls – the 2 sold before and the 2 after the returned lot (19.5% did not run; 37.9% won; 6.67 average starts per horse)

Lane *et al.* (1987)

TABLE 3: Tattersalls Yearling Sales 1987–1993: Summary of matched control study of yearlings returned for their wind

7,463 yearlings sold
78.8 examined by purchasers' vets
1.65% referred to panel
0.67% failed
4: 1 colts: fillies
(Sex ratio of all catalogued yearlings 1.27:1)

1987–1993 Racing Performance
Aged 2–4 years
Failures
Ran 8.48 mean races **Won** mean 1.32 races
Controls – (the 2 sold before and the 2 sold after the referred lot and of the same sex)
Ran 13.71 mean races **Won** mean 2.18 races

J. Griffiths Project (in preparation) 1987–1993

TABLE 2: Tattersalls Yearling Sales 1987–1995: Summary of matched control study of yearlings returned for their wind

1987–1995 (9,524 yearlings sold)
78.8% examined by purchasers' vets
1.56% referred to panel
0.65% failed
0.91% passed
At least 3:1 colts: fillies

1987-1995 Racing Performance
Aged 2–4 years

Passes	10.85 mean races 1.87 wins
Controls	9.09 mean races 1.41 wins+
Failures	7.32 mean races 0.97 wins*
Controls	10.69 mean races 1.42 wins+

S. White Thesis, University of London
* Significant difference; + Controls – one sold before and one after the lot referred to the panel and of the same sex

which are unfit or untrained and very often inspiratory noises will disappear with one or two lunging sessions. Panel members occasionally differ in their interpretation of the character of the inspiratory noise or on the grading of the endoscopic findings. For some years vendors were able to insure against their yearling being returned by the wind panel, either early in the year without a preliminary test or one month before selling following a lunging test by their own veterinary surgeon. The authors suspect that there were some dubious claims.

In the Horses in Training Sales the veterinary surgeons of purchasers often lunge the horse before returning it to the panel. The horse with RLN may make a whistle or roar on the lunge but be silent when galloped in a straight line.

Other upper airway conditions have included pharyngeal lymphoid hyperplasia of varying degrees. The more severe cases often make abnormal inspiratory sound, sometimes indistinguishable from the characteristic roar or whistle required by the Conditions of Sale. Dorsal displacement of the soft palate was seen but none was permanent and no palatal deformities were found. One horse with epiglottic entrapment did not have RLN and was not returnable. Four cases of fourth branchial arch anomalies were identified. One of these showed no evidence of laryngeal asymmetry or RLN and was passed. Two were failed and returned to the purchaser and one was taken back by the vendor before the panel completed its examination. Two cases of right sided RLN, which did not have fourth branchial arch anomalies, were identified and returnable. Cases of thin, flimsy epiglottis were noted but were not among those which were failed.

Three studies have been prepared for publication on the findings of Tattersalls' panel.

An interesting feature of the Table 1 findings is that those which were returned on the basis of sound only performed significantly less well than those which were returned with RLN. Figures from Tables 2 and 3 confirmed that the horses which were adjudicated as returnable by the panel, raced and won significantly less often than their sex matched controls sold in the same sale.

The panel periodically review the modus operandi and diagnostic criteria. They have considered including other congenital and incurable

disorders such as branchial arch anomaly or palatal defects. However, their low incidence and, in a few cases of fourth branchial arch anomaly the difficulty of diagnosis, have persuaded them to leave the Conditions of Sale unchanged. Also, if other diseases were included, endoscopic examination of foals would increase significantly. As the trade in buying foals to sell on as yearlings is so important, widespread pre-sale scoping of foals would lead to misleading advice which would not be in the interests of the horse, purchasers or vendors (Lane *et al.* 1987). The panel also discussed the use of video-endoscopy but were dissuaded as it was believed that a recording would have to be made, which would then be used for intense discussion by lawyers. More recently this view has been relaxed and the panel are now able to see the endoscopic findings simultaneously and a better assessment is made in a shorter period of time.

This task of adjudicating such a Condition of Sale is pragmatic at best and it is not in the interests of the auctioneers to have too many yearlings returned as was the case in 1984 (2.75%). The percentage of returns has thus rested at less than 1% in the 20 years that the authors have conducted this task. It has also reduced the number of unsound yearlings being submitted to the sales as vendors have been more careful in checking them beforehand. No major litigation has ensued and analysis of our results reassure us that we are diagnosing a performance-limiting disease.

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SALES: PROBLEMS IN DIAGNOSIS OF RLN-USA PERSPECTIVE

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INTRODUCTION

Examination of the upper airway (UA) has become an important part of the purchase examination performed on horses intended for athletic endeavours. The examination is obviously done to avoid purchasing a horse with an abnormality that will adversely affect the ability of the horse to train or race. The focus of this paper is the problems encountered evaluating the UA of the immature racehorse (weanling, yearling, 2-year-old), particularly in reference to arytenoid function.

The Thoroughbred (TB) sales are where most veterinarians have gained experience in evaluation of the UA of the immature horse. The UA examination of the yearling at the sales ground in the USA essentially consists of a quick visual examination of the head, an endoscopic examination of the UA (usually through one nasal passage), and infrequently external palpation of the larynx. The TB sales companies have placed conditions of sale in the sales catalogue that allow for the return of a horse to the seller if specific abnormalities of the UA are found immediately post sale. This provides the buyer with reasonable assurance that the UA is normal and theoretically lessens the need for pre-sale UA endoscopy. However, even though the UA of an individual may pass the conditions of sale, it may not pass the criteria the examining veterinarian considers acceptable. This has resulted in pre-sale UA examination of most horses at the yearling sales.

The UA conditions of sale found in the sales catalogue, in addition to the pre-sale UA examination has led most vendors to have the UA of their horses evaluated prior to the sale. This avoids unwelcome surprises at the sale. The process has evolved to the point that relatively few horses arrive at the sale with one of the abnormalities listed in the conditions of sale. With the addition of pre sale scrutiny, it is now rare to encounter a post-sale dispute regarding the UA. The UA abnormalities listed in the conditions of sale are laryngeal hemiplegia, rostral displacement of the palatopharyngeal arch, epiglottic entrapment, permanent dorsal displacement of the soft palate, severe arytenoid chondritis or chondroma, sub-epiglottic cyst, and cleft palate.

The definition of laryngeal hemiplegia in the Keeneland catalogue is ‘consistent immobility or inability to fully abduct the arytenoid cartilage’. In the Fasig-Tipton catalog the definition is the same except the word ‘consistent’ is replaced by ‘complete’. Arytenoid movement is probably the area of most concern during the UA purchase examination.

PERFORMING THE UPPER AIRWAY EXAMINATION

Problems encountered in performing an examination of the UA can make diagnosis of recurrent laryngeal neuropathy (RLN) a difficult task. The vendor can be uncooperative and allow few or no UA examinations pre-sale. There has been some concern from vendors regarding the endoscopic procedure physically harming the horse, especially with popular yearlings that may be subjected to numerous UA exams. The possibility of physical damage to the UA is very low, but not zero. There has also been some concern about harm to the sales value of the yearling if a negative opinion of the UA is not kept confidential.

A few yearlings are intolerant of the endoscopic procedure, making the examination

itself difficult. This creates a small risk of injury to the horse or the people involved with the procedure. Sedation is usually not an option. Thus, experienced handlers become very important.

To address the above concerns, some vendors will allow only a few experienced veterinarians to examine endoscopically certain yearlings. They may ask other veterinarians wanting to examine the UA to contact the few veterinarians who did examine the horse, for their opinion. However, in general, most vendors allow as many UA examinations as requested. There is a direct relationship between the number of veterinary examinations of the UA and the number of potential buyers.

Interpreting upper airway findings

Accurate interpretation of the findings from UA examination is not difficult for most horses as they are well within normal limits. However, for some horses this can be challenging. A veterinarian is expected to determine suitability of the UA for racing during a brief single examination. The pharynx/larynx is observed at rest, after swallowing, and usually during nasal occlusion.

In most immature horses the arytenoids are well abducted throughout much of the UA examination, or easily maximally abduct following swallowing, or when the UA is stressed during nasal occlusion. Rarely is complete paralysis of an arytenoid found in a yearling. In the experience of the author's practice this occurs in less than 0.2% of the general population of thoroughbred yearlings examined during the first three-quarters of their yearling year. The arytenoid function of yearlings that fall between complete paralysis and relatively easily achieved maximal abduction can be difficult to interpret and requires good judgment to determine suitability for purchase.

It is recognised that many normally functioning arytenoids that are neither perfectly symmetrical nor synchronous, do not become dysfunctional. It is also generally believed that complete arytenoid paralysis is usually preceded by progressive, deterioration of arytenoid function of variable duration. Thus, when evaluating the UA of a sales yearling the arytenoids that do not function in an ideal fashion do raise the level of concern.

It has been noted that arytenoid movement in some yearlings can change mildly over a few to several months, over a few days, or even from morning to afternoon. This may change the opinion of the examining veterinarian of whether the UA is acceptable. Some yearlings are uncooperative and cannot be induced to fully abduct the left and right arytenoids even with nasal occlusion. The author considers this within normal limits for this age if they are relatively symmetrical and synchronous. In some yearlings one arytenoid (usually the left) does not fully abduct, even with nasal occlusion. The author considers this normal if the arytenoid abducts to at least 95% of what would be considered maximal abduction.

Is a yearling within normal limits if an arytenoid can fully abduct briefly after swallowing and fully abduct briefly with nasal occlusion, but not maintain abduction? This becomes a judgment call on whether the abduction was maintained long enough to be considered acceptable by the examining veterinarian.

Recording findings (grading systems)

Being able to record endoscopic findings in a consistent manner is important. This allows subsequent review of notes to determine accurately the status of the UA during that examination. The veterinarian can then explain to a client or another veterinarian what was seen at that time. This also provides a comparative reference for a subsequent examination of the same horse and for UA findings of other horses.

Different grading systems for arytenoid movement have been used. This can make communication between veterinarians difficult unless the actual description of the arytenoid movement is used. Some of the grading systems proposed and used have had from 4 to 10 different categories for arytenoid movement. Some veterinarians have abandoned these grading systems and just describe what was seen. Some veterinarians combine all the findings of the UA endoscopic examination and give the entire pharynx/larynx a letter grade.

A relatively simple grading scale that is easy to use and widely accepted is needed. The problem with having too few categories is the broad range of arytenoid movements that fit in a single category. A system with too many categories becomes too complicated to use easily.

Regardless of the grading system used, some arytenoids will not fit precisely into a specific category. However, a widely accepted grading

system used by veterinarians all over the world, will provide consistent and more accurate communication between veterinarians.

PREDICTING FUTURE FUNCTION OF THE ARYTENOID

It is now well recognised that arytenoid movement within an individual can change over variable periods of time, both for the better and for the worse. Thus, making accurate predictions in immature horses for future function can be difficult. In general the younger the horse, the more difficult this becomes.

It is important for buyers and sellers to understand that regarding arytenoid movement, opinions on suitability for athletic function are based on the examination performed that day. This may result (has resulted) in horses that appear normal having subsequent problems, and in horses that do not quite appear normal, having no subsequent problems.

There is less risk for the veterinarian and the buyer to purchase a horse with what is considered a normal UA and avoid purchasing a horse with a questionable UA, than to take a chance on a horse with a questionable UA.

In summary, although the knowledge regarding arytenoid movements in young horses has improved, there will continue to be very good racehorses that had less than ideal arytenoid function as a young horse.

SALES: PROBLEMS IN THE DIAGNOSIS OF RLN – AUSTRALASIAN PERSPECTIVE

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In an ideal world treadmill video-endoscopy would be used to determine the clinical significance of varying degrees of recurrent laryngeal neuropathy (RLN) identified during post sale endoscopic examinations. This is not practical and so the less perfect procedure of resting endoscopy must be relied on. Unfortunately, this technique will not predict with 100% accuracy which horses will make a 'whistling' or 'roaring' noise at fast exercise.

In New Zealand and Australia a 5-point grading system is used to help endoscopists determine the clinical significance of RLN. Grade 1 and 2 laryngeal movements are considered within normal limits. Grade 3 laryngeal movements are equivocal. Grade 4 and 5 laryngeal movements are due to clinically significant RLN (supported by a number of treadmill studies showing respiratory impairment during fast exercise in these animals). Grade 4 and 5 horses fail the post sale endoscopic examination.

Horses with Grade 3 laryngeal function pass the post sale endoscopic examination. Endoscopy in these horses reveals slight laryngeal asymmetry at rest but full abduction can be achieved by the arytenoid cartilage. However, activity is generally reduced on one side (usually the left). Full bilateral abduction can be stimulated either by partial asphyxiation (nasal occlusion manoeuvre) or by inducing swallowing, but is frequently not sustained. Although some of these horses may have impaired athletic performance, recent treadmill studies (Lane 2000) indicate that in at least 75% full bilateral arytenoid abduction is maintained during fast exercise. Fortunately for endoscopists at sales within the United Kingdom a dynamic component is added to the testing procedure by using 'wind testing' and this aid helps improve accuracy in determining clinically significant RLN. Under this system horses with Grade 3 laryngeal movements that also make a characteristic 'whistle or roaring noise' when lunged at the canter, fail post sales examinations. Without the benefit of an exercise test and because the majority of Grade 3 horses have normal upper respiratory tract function at fast exercise, failing such horses in New Zealand and Australia would be very problematic. This is an area of *caveat emptor* and would seem to be fair to both vendors and purchasers.

Using a static method to describe a dynamic process has limitations. A wide range of defective laryngeal movements exist and cut off points can never be as precise as one would wish. Problem cases are likely. In addition, even apparently normal horses (Grade 1 or 2 laryngeal movements at rest) have been found to have abnormal laryngeal movements (Grade 3–5 or dynamic arytenoid collapse) (Kannegieter and Dore 1995; Lane 2000) and respiratory noise when galloped on the treadmill. Furthermore, it is sobering to appreciate that the progression of RLN in Thoroughbred horses to degrees which may be clinically important is estimated as 5–15%, (Anderson *et al.* 1997; Dixon *et al.* 2002). More alarming for endoscopists however is that this progression may occur quickly, over some few weeks or months.

The challenge in the sales environment is to be confident that a horse can fully abduct the arytenoid cartilages. As a matter of quality control, all horses with Grade 3 laryngeal movements are examined by a second veterinarian. In horses in which laryngeal abductor function is questionable, video-endoscopic examination is made and reviewed carefully to determine that full symmetrical abduction of both cartilages is present.

Recently, it has come to the attention of examining veterinarians that some of the horses they are examining appear sedated. On entrance to the stall, such horses are quiet, often have a lower head carriage, droopy lips and may have slight protrusion of the third eyelid. The use of such drugs as acetylpromazine and valium by vendors may be the cause. It is clear from experience and documented literature that some sedative agents, notably xylazine, can reduce the range and frequency of laryngeal movements. Often horses which had normal movements (Grade 1 and 2) will develop Grade 3 laryngeal movements following sedation. While it is unlikely that Grade 4 laryngeal movements will be induced following sedation it is possible that horses with Grade 3 movements which may be borderline (ie have difficulty in achieving full arytenoid abduction) could be altered to such a degree that determining accurately if full arytenoid abduction is attained could be difficult. Veterinarians are advised that if their clients are using such agents to 'calm' horses in the sales environment, this may jeopardise the interpretation of laryngeal function by endoscopists. Certainly the horses most at risk are those with some degree of neuromuscular dysfunction. Presumably sedative agents work on depression of neuromuscular function.

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DIFFERENCES BETWEEN RESTING AND TREADMILL ENDOSCOPIC FINDINGS IN REGARD TO RLN

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Earlier in this workshop presentations were made regarding a series of schemes by which to grade equine laryngeal motility during quiet breathing, ie when the patient is standing in the stable or has recently returned from exercise. In fact, for the majority of equine clinicians such endoscopic examinations form the lynch-pin for diagnosis in horses showing signs suggestive of dynamic upper respiratory tract (URT) obstruction, but how dependable are the findings.

The acid test for any grading scheme for recurrent laryngeal neuropathy (RLN) must be how well the findings at rest translate into the laryngeal function during maximal exercise. The most common cause of disquiet or dispute in regard to the interpretation of endoscopic findings in horses arises with those horses showing Grade 3 RLN motility, ie the 18% of the total population which show asymmetry during quiet breathing but which are capable of full symmetrical abduction after swallowing or during the nostril occlusion manoeuvre. Prospective purchasers at sales are advised frequently not to bid for such animals, either because it is perceived that Grade 3 represents a performance-limiting malfunction as it stands, or that horses showing such laryngeal motility are likely to deteriorate to become Grade 4 or 5 later. What evidence is there that either of these perceptions are true?

Dixon *et al.* (2002) have shown that isolated individual horses from all RLN grading groups may show progression of malfunction and that horses with Grades 1 and 2 (sic) scores are no less likely to sustain deterioration of laryngeal function than those showing Grades 3 and 4 (sic) motility. Earlier Baker (1983) had suggested that for the overwhelming majority of horses laryngeal function, as perceived endoscopically, remains unchanged throughout life.

In a study of 459 Thoroughbred horses referred to the University of Bristol's Equine Sports Medicine Centre the RLN gradings of horses examined at rest on arrival were compared with the diagnosis achieved by endoscopy during highspeed treadmill endoscopy (HSTME) see Table 1 (Franklin 2002).

Thus, in a selected population of horses that were referred for the investigation of poor performance, often with a history of abnormal respiratory noise, only 29/82 (35%) animals with Grade 3 RLN at rest were confirmed to show dynamic collapse of the left arytenoid cartilage and/or vocal fold during treadmill exercise. These results also show that there were isolated cases (19/338–5.6%) where horses showing 'normal' laryngeal motility – Grades 1 and 2 - during quiet breathing showed dynamic arytenoid or cord collapse under exercise conditions. Also, 4/23 (17%) of horses with Grade 4 RLN failed to show dynamic collapse of the vocal fold or arytenoid

TABLE 1: Comparison between laryngeal function at rest and findings during highspeed treadmill endoscopy in 459 Thoroughbred horses

82 Grade 1 at rest yielded	1	ACC on treadmill
	3	VCC
256 Grade 2 at rest yielded	11	ACC
	4	VCC
82 Grade 3 at rest yielded	20	ACC
	9	VCC
23 Grade 4 at rest yielded	19	ACC
	0	VCC
6 Grade 5 at rest yielded	6	ACC
Others	10	

ACC = arytenoid cartilage collapse
VCC = vocal cord collapse

cartilage although the rima glottidis was slightly asymmetric at all stages of the exercise test. These observations are at odds with a previous report which came to the more simplified conclusion that all horses with Grade 1 and 2 RLN showed full sustained abduction during treadmill exercise (Morris and Seeherman 1990), and the same authors reported that 20 out of 27 horses with Grade 4 RLN (sic) showed ACC or VCC during treadmill exercise (Morris and Seeherman, 1991). Kannegeiter and Dore (1995) also used the 1–5 grading scheme and showed that while resting scores provide a useful overall guide, exceptional horses appear 'normal' at rest but sustain dynamic collapse during treadmill exercise and, vice-versa, that a small number of 'abnormal' horses become 'normal' under exercise conditions. In all instances where dynamic collapse of the vocal cord and/or arytenoid cartilage occurred in the Bristol cases there was an obvious inspiratory noise, and in most instances, palpable atrophy of the intrinsic musculature on the left side of the larynx was detected.

To the unaided human ear the inspiratory noises produced by horses afflicted with axial deviation of the ary-epiglottal folds (ADAEF) is not distinguishable from the 'roaring' or 'whistling' associated with RLN. In this series of 459 horses there were 10 horses with ADAEF and no other concurrent form of dynamic collapse in the URT, 40 where it was associated with palatal malfunction, and 7 where there was concurrent dynamic collapse of the arytenoid or vocal fold. These findings are comparable with those reported elsewhere (Parente *et al.* 1994; Kannegeiter and Dore 1995; King *et al.* 2001). The overall conclusion is that URT obstructions are commonly complex with the dynamic collapse of multiple structures which can only be established by HSTME.

Clearly, not all clinicians have access to the facilities for endoscopy during highspeed treadmill exercise. The observations above confirm that endoscopy at rest should never be used in isolation from other diagnostic techniques, and that the possibility of erroneous diagnosis is reduced considerably when endoscopy is used in conjunction with palpation findings and attendance at an exercise test to listen for untoward respiratory noises.

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NON-RLN UPPER RESPIRATORY TRACT DISORDERS FOUND IN A SURVEY OF 3,497 THOROUGHBRED YEARLINGS

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Surveys of structural and functional abnormalities of the upper respiratory tract of the horse have previously been based upon selected populations, for example, those submitted to public sales (Lane *et al.* 1987) or in training (Pascoe *et al.* 1981; Raphael 1982; Baker 1983; Sweeney *et al.* 1991), where unsaleable or untrainable individuals will have been excluded earlier. In addition, no survey of dynamic dysfunctional conditions, occurring only under exercise, has been attempted to date. Thus, it is generally held that dorsal displacement of the soft palate (DDSP) and recurrent laryngeal neuropathy (RLN) are the 2 most frequently encountered disorders of the region. The prevalence of non-RLN upper respiratory tract (URT) disorders found during the video-endoscopic study of 3,497 yearlings was as follows:

Fourth branchial arch defects (4-BAD)	7 cases	0.20%
Sub-epiglottal cyst (SEC)	5 cases	0.14%
Epiglottal entrapment (EE)	2 cases	0.06%
Right laryngeal malfunction	5 cases	0.14%

Hast (1972) has described the development of the larynx in the early human embryo and showed that the extrinsic structures are derived from the fourth branchial arch and the intrinsic structures from the sixth arch. 4-BAD is a syndrome of irreparable congenital defects resulting from a failure of development of some or all of the derivatives of the fourth branchial arch (Lane 1993). The structures involved are the wings of the thyroid cartilage, the cricothyroid articulation, the cricothyroideus muscles and the cricopharyngeal sphincter muscles. Any permutation of aplasia or hypoplasia of these structures may arise uni- or bilaterally.

The condition has been identified in other breeds such as the Hanovarian, warmbloods, Welsh Section A ponies and the Haflinger (Cook 1974; Goulden *et al.* 1976; Wilson *et al.* 1986; Deegan and Klein *et al.* 1987; Klein *et al.* 1989; Dixon *et al.* 1993). Generally, has been reported under titles such as rostral displacement of the palato-pharyngeal arch (RDPA) or cricopharyngeal-laryngeal dysplasia.

The author has reviewed the findings in 60 4-BAD-afflicted Thoroughbred horses and the results are summarised here. The presenting signs of horses with 4-BAD are variable and reflect the severity of the absence of the structures involved. However, in order of frequency, the signs are abnormal respiratory sounds at exercise (50 out of the 60 horses), belching (13), nasal discharge (10), coughing (10) and recurrent colic (5). The involuntary aerophagia and eructation sometimes may be confused with the noises produced by 'wind-suckers'.

There is no current evidence that the syndrome is genetically transmitted. A complete evaluation of the extent of 4-BAD can only be made at exploratory surgery or autopsy but the combined findings of palpation, endoscopy and radiography are generally sufficient to justify a diagnosis. When the cartilage components are defective, an unusually wide gap can be palpated between the caudal margin of the thyroid and the rostral edge of the cricoid, whereas in the normal larynx the 2 structures overlap. The 2 endoscopic features to alert the clinician to the possibility of 4-BAD are RDPA (33 cases) where the caudal pillars of the soft palate form a cowl, which partly obscures the corniculate processes dorsally and defective arytenoid motility (45 cases). 4-BAD is the most common explanation for apparent right sided RLN (Tulleners *et al.* 1996) and there is a marked over-

representation of right sided cases. In the 60 cases 15 were bilateral, 39 were right unilateral and in 6 the defects were confined to the left side. In one instance 4-BAD was only detected as dynamic RDPA during treadmill exercise. RDPA itself is simply an endoscopic symptom of a major underlying laryngeal disorder and should not be regarded as a disease in its own right. It arises when the upper oesophageal sphincter muscles are not present to provide an anchor for the palatal arch caudal to the apices of the corniculate processes of the arytenoid cartilages. When the crico- and thyro-pharyngeus muscles are absent, there is a failure to close the upper oesophagus so that lateral radiographs will reveal a continuous column of air extending from the pharynx into the oesophagus. The RDPA is seen as a 'dew drop' intruding into this air column from the dorsal wall. Repeated aerophagia leaves those animals without an upper oesophageal sphincter susceptible to episodes of colic which may be life threatening. Three horses have died or been destroyed through tympanitic and one other has required surgical decompression.

The performance records of 51 of the horses have been traced: 22 were unnamed; 29 were named but unraced; 11 raced but were unplaced; 4 were placed and 7 won, albeit all in minor competitions. Thus, horses afflicted with 4-BAD are generally ineffective athletes.

Five cases of right laryngeal malfunction, other than the 7 cases of 4-BAD, were identified in the stud survey of yearlings. Three of these were later subjected to surgery with a view to prosthetic laryngoplasty, but were found to be inoperable by virtue of hypoplasia of the muscular process of the right arytenoid cartilage. Given that the arytenoid cartilages develop from the sixth branchial arch should a 6-BAD syndrome be added to the clinical vocabulary?

It is generally believed that sub-epiglottal cysts are congenital and if this is so the incidence of 0.14% demonstrates that the disorder is indeed rare. In contrast the aetiopathogenesis of epiglottal entrapment is not known and the results of repeated endoscopic examinations confirm that at least some cases are acquired. Thus, the identification of only 2 cases in 3,497 yearlings does not reflect the overall incidence. Similarly, arytenoid chondropathy is known to be an acquired disorder and a failure to record a single case even in quite a large population of young horses is not significant.

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NON-RLN URT DISORDERS IDENTIFIED DURING POST SALE ENDOSCOPIC EXAMINATION OF 5,559 TB YEARLINGS (1997-2002) IN NEW ZEALAND

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At the 1995 National Thoroughbred Yearling sales in New Zealand, post sale endoscopic examinations of the larynx and pharynx were introduced. Following the fall of the hammer, the purchaser is given the opportunity to have his/her horse endoscoped by a veterinarian – approved by the auctioneer – who then examines the larynx and pharynx of the presented horse for the presence of, and only of, one or more of the following 6 conditions: a) Laryngeal hemiplegia; b) Subepiglottic cyst(s); c) Persistent dorsal displacement of the soft palate; d) Epiglottic entrapment; e) Rostral displacement of the palatopharyngeal arch; and f) Arytenoid chondritis or chondroma. The examination is of a pass or fail nature and the endoscopist is under no obligation to reveal the findings of the examination to the purchaser or vendor and merely records the result.

Of the 5,559 horses examined between 1997–2002 (Tables 1 and 2), 41 failed the conditions of sales, a failure rate of 0.74%. Eight of these horses had left sided recurrent laryngeal neuropathy (RLN) and will not be discussed further. A further 3 horses had markedly deficient right sided arytenoid cartilage abduction, presumably due to RLN.

Of the other 30 horses 19 failed due to inflammatory conditions, including 12 due to arytenoid chondritis, 6 because of mechanical interference with arytenoid abductor function (principally pharyngeal/guttural pouch masses or severe epiglottic inflammation) and one with epiglottic chondritis. A further 11 horses failed because of non-inflammatory lesions including 2 horses with persistent dorsal displacement of the soft palate, 5 horses with epiglottic entrapment, 3 horses with rostral displacement of the palatopharyngeal arch and one horse with hypoplasia of the soft palate.

These results indicate that less than 1% of horses presented for post sale endoscopy fail the examination and that inflammatory conditions accounted for 46% (19/41) of all failures with arytenoid chondritis the most common (30% or 12/41). Follow up showed that in a significant number of horses (42% or 17/41), which failed the conditions of sale on the day of examination treatment, either surgical or medical, resulted in a saleable horse.

It is interesting to note the high number of horses affected with arytenoid chondritis. Compared to results from other endoscopic

TABLE 1: Number of endoscopic examinations at the National Yearling Sales (1997–2002) and failure rate

Year	No. endoscopic examinations	No. horses failing conditions of sale	Percent of horses failing conditions of sale
1997	691	4	0.58
1998	928	8	0.86
1999	698	3	0.42
2000	1,028	12	1.2
2001	1,161	5	0.43
2002	1,053	9	0.86
Total	**5,559**	**41**	**0.7**

TABLE 2: Reasons for and number of horses failing conditions of sale at the National Yearling Sales (1997– 2002)

Condition	Number	Comments
Arytenoid chondritis	12	Some horses with focal lesions have been treated by debridement and have been sold or are racing
Epiglottic entrapment	5	All treated and sold
Persistent dorsal displacement of soft palate	2	Both resolved with antibiotic/anti-inflammatory treatment
Right side Grade 4 or 5 laryngeal movements	3	Aetiology unknown 1 horse had grade 4 laryngeal movements and rostral displacement of the palatopharyngeal arch
Right side Grade 4 or 5 movements due to mechanical obstruction	4	3 dorsopharyngeal masses 1 severe epiglottic inflammation All resolved with treatment
Left side Grade 4 or 5 laryngeal movements (ILH)	8	1 horse had what appeared to be a unilateral rostral displacement of the pharyngeal arch as well as ILH 7 horses ILH
Left side Grade 4 or 5 laryngeal movements due to mechanical obstruction	2	1 dorso pharyngeal mass 1 severe epiglottic inflammation All resolved with treatment
Rostral displacement palatopharyngeal arch	3	
Chondritis left side of epiglottis	1	Failed because the chondritis resulted in an obstruction of the rima glottidis
Hypoplasia of soft palate	1	Broken in, trialled but retired

examinations at sales around the world, the problem is unusually common in New Zealand. The exact reasons for this are unknown.

In addition, a number of horses have been recognised that have arytenoid mucosal injury (Anderson 2000). These mucosal injuries manifest as small erosions/ulcers, or alternatively, raised areas of epithelial injury. They are found on the medial surface of each arytenoid just above where the vocal cords join onto the arytenoid cartilages (the vocal processes) and may also be found on the vocal cords themselves. They may be hyperaemic or even have small, slightly purulent centres and can vary in colour from red to white. Although occasionally unilateral most often there are 2 lesions, one on either cartilage and they are referred to as 'kissing lesions'. Even if there appears to be only one lesion, close scrutiny of the opposite arytenoid cartilage usually reveals a small area of accompanying injury. The exact cause of these lesions is not known. Reports in the literature regarding such lesions in horses are sparse but they have been reported in yearling Thoroughbreds at horse sales in Australia (Kelly *et al.* 2003). Review of laryngeal diseases and injuries in man, cattle and other species indicates that the cause(s) of these injuries is likely multifactorial with infectious causes having a major role (Fig 1). Mucosal inflammation or mucositis occurs and is followed by mucosal ulceration when the superficial epthelium is denuded. Because there is no submucosa, the mucosal epithelial layer is tightly adhered to the underlying perichondrium of the arytenoid cartilage. Pressure, the result of both intrinsic and extrinsic trauma over the thin, relatively immobile, mucosal layer results in erosion and ulceration. Once the mucosal barrier is breeched bacterial infection of the underlying structures is possible. If the infection remains localised a relatively quick healing response occurs with or without the use of antibitotic/anti-inflammatory treatment. Healing

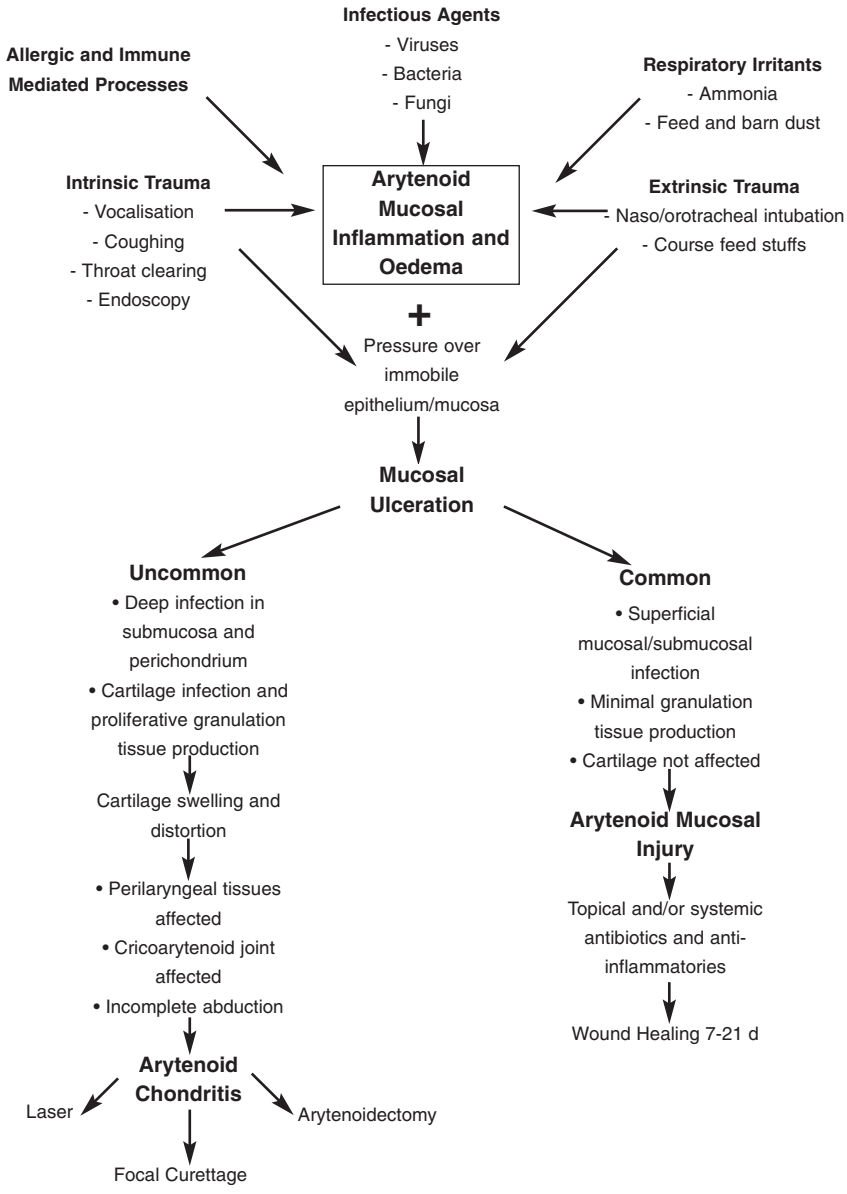

Fig 1: Suggested pathogenesis and outcome of arytenoid mucosal injury.

occurs by epithelialisation, mild fibroplasia (granulation tissue production) and some contraction. Typically, the underlying arytenoid cartilage is not affected. The potential for such injuries to progress to the more serious arytenoid chondritis (discussed below) is thought to be low (Smith 2000; Kelly *et al.* 2003; B.H. Anderson, unpublished data) but happens occasionally. Kelly *et al.* (2003) reported idiopathic mucosal lesions of the arytenoids cartilages in 21 Thoroughbred yearlings (0.63% of 3,312 horses having post sale upper respiratory tract examinations over a 5 year period). In 2 horses (10%) granulomas developed at the site of ulceration and in one horse (5%) chondropathy developed.

Strictly defined, arytenoid chondritis means inflammation of the arytenoid cartilage. However, the condition usually referred to as arytenoid chondritis is an inflammatory swelling involving the arytenoid cartilage, peri-arytenoid tissue,

laryngeal mucosal surface and dorsal muscular structures. Endoscopically this condition is recognised as a medially displaced, swollen, misshapen, arytenoid cartilage. In most cases the cartilage is reddened or hyperaemic. There may be reduced or absent cartilage movement in more severe cases. Discharging pus-filled lesions on the medial border of the arytenoid cartilage may be present as well as ulcers or epithelial swellings ('kissing lesions') on either cartilage. Intraluminal projections of granulation tissue may also be present.

It is not clear what factors or conditions are important in determining if infection becomes established within the arytenoid cartilage rather than remaining in superficial tissues. What has become difficult in the sales environment is determining if infection or inflammation is confined only to the mucosa or if cartilage is involved. Differentiating generalised mucosal oedema from cartilage enlargement, and determining if luminal projections of granulation tissue involve the underlying cartilage or not is not always easy. Experience gained from surgical treatment has revealed that if luminal projections of granulation tissue are marked, invariably the underlying cartilage is affected. However, each case is considered 'on the day' and a decision on the absence or presence of chondritis is aided by careful examination of video-endoscopic pictures.

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NON-RECURRENT LARYNGEAL NEUROPATHY (RLN) CAUSES OF EQUINE LARYNGEAL PARALYSIS

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Non-recurrent laryngeal neuropathy (non-RLN) accounts for the small proportion (6% Goulden and Anderson 1981; 11% Dixon et al. 2001) of horses with laryngeal paralysis in which a probable cause can be identified. Conversely, RLN cases have no such detectable underlying cause.

Non-RLN laryngeal paralysis may be a sequel to localised injury to the vagus or recurrent laryngeal nerves at any site along their circuitous courses. Such injury may occur in disorders of the guttural pouch (mycosis, rupture of the *rectus capitis ventralis* muscles, temporohyoid fracture, trauma), pharynx (trauma, abscessation, neoplasia), neck (perivascular/perineural irritant injection reactions, trauma, iatrogenic nerve damage during oesophageal and thyroid surgery) or mediastinum (neoplasia, abscessation). Non-RLN paralysis may also be a manifestation of a generalised disorder, eg a polyneuropathy or myopathy. Bilateral laryngeal paralysis, which is considerably less common (2–6%) than unilateral paralysis, almost invariably results from generalised neuromuscular disorders.

Liver disease is a common cause of bilateral laryngeal paralysis (Mayhew 1989; Pearson 1991; McGorum *et al.* 1999). McGorum *et al.* (1999) recorded bilateral laryngeal paralysis in 7 of 50 horses with primary hepatic disease, all of which had hepatic encephalopathy and hyper-ammonaemia. All cases presented with loud inspiratory stridor and many were referred for investigation of suspected primary upper respiratory tract obstruction. In all cases, endoscopy revealed total bilateral paralysis, with both arytenoids passively adducted to the midline during inspiration. Ponies were affected more often than horses, but this probably reflects an increased frequency of liver failure in ponies rather than increased susceptibility. The laryngeal paralysis was often temporary, worsening during exacerbations of encephalopathy and resolving with restoration of hepatic function. No gross or histopathological abnormalities were identified in the laryngeal muscles, the recurrent laryngeal nerve or other peripheral nerves of affected horses.

The pathogenesis of this complication remains unclear. Most reported cases are in horses with liver failure and hepatic encephalopathy, but it is unknown whether it occurs with compensated liver disease. Similarly, while it is reported in horses with pyrrolizidine alkaloid induced liver disease (Pearson 1991; McGorum *et al.* 1999), the role of this substance, which may be neurotoxic (Cooper and Huxtable 1999), is unclear. As the laryngeal paralysis may be temporary, and no histo-pathological lesions have been identified, it may reflect neuromuscular dysfunction rather than pathology. Such dysfunction could occur by mechanisms akin to those that cause hepatic encephalopathy. Alternatively, it may represent a form of peripheral neuropathy, a common sequel to human hepatic disease. The pathogenesis of peripheral neuropathy in human liver disease patients is unknown, but may involve metabolic inhibition of axonal membrane function, metabolic damage to Schwann cells and/or disordered insulin metabolism akin to diabetic neuropathy.

Interestingly, all 3 ponies with liver disease induced bilateral laryngeal paralysis that had post-mortem examinations, also had pituitary adenomas, but no overt clinical hyperadreno-corticism (McGorum *et al.* 1999). While pituitary adenomas are relatively common in older ponies, and may not relate to the laryngeal dysfunction, increased production of pituitary derived peptides may contribute to the pathogenesis of this complication. However, Pearson (1991) made no

reference to pituitary adenomas in 2 ponies with liver failure and inspiratory dyspnoea, which were subjected to detailed post mortem examination.

Rarely, horses may develop post operative laryngeal paralysis, sometimes complicated by secondary severe pulmonary oedema and/or haemorrhage (Abrahamsen *et al.* 1990; Dixon *et al.* 1993, 2001). These cases had variable recovery of laryngeal function, with one horse showing complete resolution within 24 h, while another had residual laryngeal dysfunction after one year. Post-operative laryngeal paralysis was most likely to have resulted from excessive head/neck extension, which could induce neural stretch injury or cause neural hypoxia via occlusion of the vasa nervorum. Alternatively, laryngeal paralysis may have resulted from compression of the recurrent laryngeal nerve against a rigid structure in the neck. Myopathy, persistent hypoxia and pre-existing laryngeal dysfunction are additional factors that may contribute to development of this complication. Temporary post operative laryngeal paralysis has also been reported in a dog.

A variety of toxic peripheral neuropathies may cause equine laryngeal paralysis including delayed organophosphate induced toxicity, Australian stringhalt, lead poisoning and plant poisoning. However in all such cases, laryngeal paralysis is clearly part of generalised disorder that affects multiple nerves, and so presents little diagnostic problem. Delayed organophosphate induced toxicity leads predominantly to degeneration of long axons in peripheral nerves and spinal cord, and results from covalent binding of organophosphates to 'neuropathy target esterase'. Organophosphate induced laryngeal paralysis may be permanent (Rose *et al.* 1981; Duncan and Brook 1985). Ingestion of *Lathyrus spp.* and *Cicer arietinum* (chick pea) may cause equine laryngeal paralysis. The toxic principles include beta-N- oxalylamino-L-alanine, an excitatory amino acid which causes neuropathy with distal axonal degeneration. Feeding experiments with *Lathyrus sativus* (Indian vetch) indicated that, even with prolonged feeding, only a minority of horses were affected. Lead toxicosis primarily targets peripheral nerves, and approximately 13% of horses with lead toxicosis develop laryngeal dysfunction (Sojka *et al.* 1996), which may or may not recover with time. The pathogenesis of lead neurotoxicosis is multi-factorial and includes inactivation of thiol and cysteine-containing enzymes, and Zn-, Cu-, Fe-dependent enzymes, resulting in neural oxidative stress, and toxicity of cell membranes resulting in increased membrane permeability.

Hyperkalaemic periodic paresis, a generalised myasthenic disorder, commonly presents with episodic upper airway obstruction. While airway obstruction in this disorder is multi-factorial, laryngeal spasm or paralysis occurs in approximately half of affected horses. The severity and incidence of upper airway dysfunction may be reduced by medical treatment (Carr *et al.* 1996).

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SESSION 5:

Treatment of equine laryngeal paralysis

Chairman: Geoff Lane

DECISION MAKING IN PRACTICE FOR TREATMENT OF RECURRENT LARYNGEAL NEUROPATHY

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Recurrent laryngeal neuropathy is one of the commonest respiratory conditions encountered in equine practice. Nowadays endoscopy is available and used routinely in the vast majority of practices, and confirmation of the diagnosis in at least the more advanced case is relatively straightforward. It is the interpretation in the less severely affected horse that represents a far greater challenge to the less experienced clinician and one that can lead to difficulty when treatment options are to be considered.

The author has used a 10 grade assessment of the disease based upon the endoscopic appearance of laryngeal function and at least 3 other systems will be discussed at this meeting. The key factor in deciding what, if any, surgical therapy is appropriate for an individual horse, is the degree of its disability, which can be extremely difficult to assess.

Having briefly and unsuccessfully used neuromuscular pedicle grafting, the author's surgical treatments are based upon more traditional approaches. In the UK, surgical ablation of the vocal cord and ventricle are still accepted as valid procedures in the 'noisy' horse with satisfactory performance. In the author's hospital this is performed this using a diode or Nd:YAG laser in the standing patient. This has proved an attractive option for clients, although no more effective than using the traditional approach. The author believes that it is of benefit when combined with laryngoplasty in reducing the incidence of wound problems significantly.

Laryngoplasty is reserved for horses which are 'short of air' and performance is clearly suboptimal. The author uses 2 implants (a braided elastic and a coated braided polyester suture) as this technique seems to produce the most reliable results. Owners are counselled carefully regarding post operative management in particular. Time spent at this stage in communication is well worthwhile in reducing misunderstandings and client dissatisfaction in the post operative period. The prognosis for the combined operation is hard to assess. In a survey undertaken by the author involving over 100 horses, a detailed response was obtained in 66 cases. Of these 49 (ie nearly 75%) were deemed to be performing with a major improvement after surgery (ie with little or no evidence of laryngeal obstruction). Ten additional cases had significant performance improvement despite evidence of persistent laryngeal obstruction. Chronic sepsis necessitated the removal of implants in 3 horses but only one was removed because of dysphagia.

The implications of the survey were that careful patient selection is vital. Three of the unimproved group had undergone previous laryngeal surgery, which might be considered a bad prognostic sign. Clearly good results are easier to achieve in horses which have lower respiratory demands at exercise and in patients with a proven athletic record, as these animals tend to respond better to surgical procedures.

LARYNGEAL RE-INNERVATION IN THE HORSE

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Laryngeal re-innervation has been well documented in the human literature as a successful treatment for a range of laryngeal dysfunctions (Tucker and Rusnov 1981; Tucker 1978). Investigation into the potential use of laryngeal re-innervation in horses with laryngeal hemiplegia has occurred due to the complications that can occur with prosthetic laryngoplasty.

EQUINE LARYNGEAL RE-INNERVATION

The first studies into laryngeal re-innervation in horses were reported in 1989 (Ducharme *et al.* 1989a,b,c). The nerve muscle pedicle graft, nerve implantation and nerve anastomosis techniques were all investigated in ponies. In these experimental ponies, the recurrent laryngeal nerve was transected at the time of re-innervation surgery. While the first 2 techniques demonstrated histological evidence of re-innervation, the authors concluded that laryngeal function was insufficient to allow for maximal exercise. Importantly this study identified the *omohyoideus* muscle as an accessory muscle of respiration and, therefore, suitable for use as a donor muscle along with its nerve supply – the first or second cervical nerves.

In 1990, the nerve muscle pedicle graft technique, using the first cervical nerve and *omohyoideus* muscle, was evaluated on experimentally induced cases of left laryngeal hemiplegia in Standardbred horses (Fulton *et al.* 1991). In that study histologic evidence of re-innervation was demonstrated (Fulton *et al.* 1992) as was a return of laryngeal function, verified by upper airway flow mechanics studies (Fulton *et al.* 1991). This study demonstrated that the paralysed *cricoarytenoideus dorsalis* muscle could be re-innervated and that in vigorously exercising horses, upper airway function could return to baseline levels between 6 and 12 months after surgery.

Re-innervation has also been attempted using a muscle pedicle graft created from the right CAD muscle. It was hoped that muscle-to-muscle neurotisation would result in return of function to the paralysed left CAD muscle; however this attempt was unsuccessful (Harrison *et al.* 1992).

Since 1991 the nerve muscle pedicle graft technique has been used in selected clinical cases of laryngeal hemiplegia and hemi paresis in 129 Thoroughbred, 10 Standardbred and 7 Warmblood horses. The following is a brief description of the surgical technique, post operative care, complications, and follow-up results in these cases.

SURGICAL TECHNIQUE

The nerve muscle pedicle graft is performed with the horse under general anaesthesia. An incision is made along the ventral border of the linguo-facial vein followed by accurate dissection of the left first cervical nerve as it passes over the lateral aspect of the larynx to where it meets the *omohyoideus* muscle, an accessory muscle of respiration (Ducharme *et al.* 1989a) The first cervical nerve branches are followed to their point of insertion into the *omohyoideus* muscle. A small block of muscle is removed from the *omohyoideus* muscle with the fine branch of the first cervical nerve attached – up to 5 branches can be isolated.

Exposure of the recipient muscle, the *cricoarytenoideus dorsalis* (CAD) muscle is achieved by rotating the larynx laterally. The pedicle grafts are inserted into individual pockets in the CAD muscle fibres of the CAD muscle. A

single 4-0 polydioxanone suture is used to hold the pedicle graft into the CAD muscle. A stent bandage is usually sutured over the skin incision and an elastic bandage is used to apply pressure over the incision area, minimising the opportunity for seroma formation.

Since September 2000 left cordectomy using a diode laser in combination with the nerve muscle pedicle graft has been used. The cordectomy is routinely performed the day following the nerve muscle pedicle graft procedure in the standing sedated horse.

Horses are routinely kept confined to a stall for 2 weeks following surgery. After stall confinement, a further 2 weeks in a day yard followed by paddock turnout for 12 weeks is normally recommended. At this stage it is advised that the horse should go into training – 16 weeks post operatively. When the horses are returned to exercise it is advised that episodes of fast exercise are introduced as early and as frequently as possible. As the *omohyoideus* muscle is an accessory muscle of respiration, considerable respiratory effort must be undertaken to activate the first cervical nerve.

After 6 weeks of training, trainers/owners are requested to present the horse for endoscopic assessment of the larynx. At rest, the left arytenoid cartilage most commonly looks exactly as it did prior to surgery. Two diagnostic reflexes have been developed to stimulate contraction of the *omohyoideus* muscle and therefore the newly innervated CAD. The first involves stretching the head and neck upward as high as possible while observing the larynx closely through the endoscope. If re-innervation has occurred, there is often a spontaneous flicker or single abduction of the left arytenoid cartilage. The second reflex involves pulling back rapidly with a finger or thumb on the commissure of the lips. Again a sudden abduction of the left arytenoid cartilage occurs if re-innervation has been successful. This reflex can be stimulated from the left or right side of the head.

Complications

Complications associated with laryngeal re-innervation have been few when compared to prosthetic laryngoplasty. The most frequent complication has been seroma formation 3–5 days following surgery. The use of a compressive neck bandage that encircles the neck rostral and caudal to the poll and maintained for 4–6 days post operatively has reduced this to some extent. Some seromas have become infected, and these have been treated with antibiotics selected from culture and sensitivity results. One horse developed a large hematoma immediately post operatively that required the incision to be re-opened and the vessel ligated.

Results

Thoroughbreds

Raced Thoroughbreds – 63 horses were included in this group, 24 were Grade 4 horses, while 39 horses had Grade 3 laryngeal hemiplegia. Of the 59 horses available for follow up, 95% went on to start in one or more races. The average length of time from surgery to race one was 7.5 months for Grade 3 horses and 8.6 months for Grade 4 horses. The earliest that re-innervation was identified was at 4 months, and the latest at 9 months. Following surgery, the horses raced an average of 12.5 times each. Of the 59 horses, 32 (54%) won one or more races after surgery.

To analyse the effectiveness of the nerve muscle pedicle graft, the following 4 variables, before and after surgery, were calculated for each horse: total performance ranking, total prize money, performance ranking per start, and prize money per start.

1. ***Total performance ranking***
   Thirty-four of the 59 (58%) horses had an improved total performance rank after surgery.
2. ***Total prize money***
   Thirty-one of 59 horses (53%) earned more prize money after surgery.
3. ***Performance ranking per start***
   Thirty-four of the 59 horses (58%) had improved performance ranking per start after surgery.
4. ***Prize money per start***
   Thirty-four of the 59 horses (58%) earned more money per start after surgery than before.

Unraced Thoroughbreds – 66 horses were included in this group, 19 were Grade 3 left laryngeal hemiplegia (LLH) while 47 had Grade 4 laryngeal function. Of the 66 horses, 39 (60%) went on to start in at least one race, 15 horses were considered to be failures, 2 died, 3 were retired for

TABLE 1: Prize money per start for each year of racing of LLH-affected horses treated with a nerve muscle pedicle graft compared with the national average (1996-2001) for Australian horses

Year of racing	NMP graft horses $/start	National average $/start
2-year-olds (n=2)	$1895.00	$2038.00
3-year-olds (n=34)	$2822.00	$1425.00
4-year-olds (n=18)	$1227.00	$1289.00
5-year-olds (n=6)	$1054.00	$1167.00
6-year-olds (n=1)	$1050.00	$1114.00

reasons unrelated to the surgery, 4 were lost to follow up, and 3 are still convalescing. The average age at their first race was 3.1 years. The 39 unraced Thoroughbreds that raced following surgery had an average of 10.6 starts each.

For the unraced horses that raced after surgery, money earned per start after surgery was compared with the Australian average - Table 1 demonstrates that each year, LLH-affected horses treated with a nerve muscle pedicle graft performed about the same as the national average.

Standardbreds

Ten horses underwent nerve muscle pedicle graft. Three horses were retired for reasons unrelated to surgery prior to resuming racing and one horse was considered a failure and did not race. Six horses returned to the race track. Of these 5 won races and 3 earned more money than before surgery.

Warmbloods

Seven horses were operated on. The oldest horse at time of surgery was 8-years-old and had been hemiplegic for 2 years prior to surgery. Five of the 7 horses went on to compete at a higher level than before surgery.

CONCLUSIONS

The biggest disadvantage of the nerve graft surgery is the time from surgery to the first race. Time to first race for Group 1 horses was 7.5 and 8.6 months for Grade 3 and Grade 4 laryngeal function respectively, while following prosthetic laryngoplasty time to first race is 5.8 months (Hawkins *et al.* 1997).

In yearlings or early unraced 2-year-olds, the nerve muscle pedicle graft is a reasonable surgery to offer owners. In the case of a 5-year-old Thoroughbred gelding, prosthetic laryngoplasty could be the logical choice

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EVALUATION OF RLN TREATMENT EFFICIENCY – LARYNGOPLASTY

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The first recorded attempt at laryngoplasty (LP) appears to have been by Moeller, who some 200 years ago, transcutaneously sutured the affected arytenoid to the thyroid cartilage in 'roarers' to prevent it collapsing into the airway (Cadoit 1893). However, this form of laryngoplasty never became popular and ventriculectomy was the standard surgical procedure for laryngeal paralysis in the late 19th and the 20th century until the introduction of the current laryngoplasty treatment by Marks *et al.* (1970). Recent surveys have shown in Britain (Bathe 1993) and in the United States (Hawkins *et al.* 1997; Hammer *et al.* 1998; Strand *et al.* 2000) that laryngoplasty is currently the most widely used treatment for equine laryngeal paralysis.

At least 11 studies have shown laryngoplasty to be of value, as assessed by absence of or reduction in abnormal exercise related respiratory 'noises' post operatively; or reported improvement in exercise performance, as reviewed by Dixon *et al.* (2003a). Some of their surveys also compared race times, or race earnings pre- and post surgery. A number of physiological studies including the early work of Bayly *et al.* (1984) and of Tetens *et al.* (1996) and those of Weishaupt (Weishaupt *et al.* 2003) have shown improvements in airflow mechanics or in arterial blood gases following LP surgery. However some of these studies were performed in experimental ponies under laboratory conditions. Despite the above volume of evidence of its efficacy, laryngoplasty is not always successful in clinical cases and some horses suffer significant post operative problems.

Obtaining and even more importantly, maintaining the required degree of arytenoid abduction is the key to success of laryngoplasty. Maximum levels of abduction are not required to allow maximal exercise performance. The degree of LP abduction obtained can be assessed semi-qualitatively using a number of grading systems. A recent study has shown a very strong statistical correlation between maximal abduction (ie Grade 1 abduction – where the arytenoids are at 90 degrees to the vertical, or even in some occasions beyond that level and maximal Grade 2 ie arytenoids close to 90 degrees to the vertical) with aspiration and coughing (Dixon *et al.* 2003a). The absence of noise at 12 months plus post operatively correlated significantly with the degree of laryngeal abduction present at 6 weeks.

There is progressive loss of arytenoid abduction post operatively in most cases, especially in the first few weeks following surgery (Dixon *et al.* 2003b). The reasons for this abductory loss are not understood fully. Most cases of recurrent laryngeal neuropathy (RLN) have preferential atrophy of their adductor muscles (Duncan *et al.* 1991) and so laryngeal adductor deficits are usually worse than abductor deficits - in contrast to Semon's law. Consequently, arytenoid adductory tension on the prosthesis is unlikely to be the main cause of abductory loss. A more likely explanation is that during swallowing, full adduction of the arytenoids occurs as the pharyngeal muscles (including the caudal constrictors) constrict sequentially in a peristaltic fashion to push the food bolus from the pharynx into the oesophagus. Consequently a surgically abducted arytenoid that is protruding laterally will be subjected to repeated adductory pressures during swallowing. This may decrease the degree of abduction progressively, in some cases totally.

Very many surgeons also perform concurrent ventriculectomy or ventriculo-cordectomy with laryngoplasty - an insurance perhaps, in case of laryngloplasty failure? Some clinicians question if

these procedures should be performed concurrently. A pertinent question posed recently by Jim Schumacher is whether a ventriculectomy is necessary if an ipsilateral vocalcordedctomy is performed? Does ventriculectomy just cause more scarring of the lateral ventricular wall and also prevent effective suturing of the vocalcordectomy wound that could limit intra-laryngeal scarring at the site of vocalcordectomy?

There is little doubt that the fibroelastic tissues of the vocal fold are tensed by laryngoplasty (as digitally assessed at laryngotomy) and will then place permanent tension on the prosthesis. Therefore, performing a concurrent vocal-cordectomy can remove some of that adductory pressure on the prosthesis and so help main laryngoplasty abduction. Recent work has shown that ventriculo-cordectomy can decrease abnormal noises in horses with laryngeal hemiplegia to even a greater degree than laryngoplasty (Brown *et al.* 2003). This is a further reason for performing concurrent ipsilateral ventriculo-cordectomy along with laryngoplasty. It can also be an 'insurance' because if the laryngoplasty loses much of its abduction, the affected arytenoid should at least be fixed in a position where it cannot obstruct the contralateral side of the larynx. At the same time the concurrent vocalcordectomy/ventriculectomy will hopefully increase the ventral laryngeal airway to help with airflow during exercise.

The prevention of sutures cutting into the cartilages at their anchor points would seem important in preventing excessive loss of abduction. A variety of suture materials have been used for laryngoplasty and all have different merits. Thicker braided prostheses may place less focal pressure on the cartilages and therefore less 'pull through' may occur with these. However, if braided non-absorbable sutures become infected, a persistent external sinus tract may well occur. Although monofilament stainless steel wires have the advantage of allowing adjustment of laryngoplasty abduction (either loosening or tightening) at surgery or during further surgery, it is possible that their fine calibre may promote 'pull through' the cartilage. The use of elasticated sutures has the advantage that they may allow continued tension to occur even with 'pull through'. Nemeth (1987) used an absorbable suture and later reported satisfactory results in a high proportion of cases, which is difficult to explain scientifically. Further studies into improving laryngoplasty suture retention are described by Parente (2004).

The success of treating equine laryngeal paralysis varies on how a successful outcome is defined (Ducharme and Hackett 1991). If the presence of an abnormal 'noise' was the primary complaint of the owner, then elimination of this noise constitutes a surgical success. In contrast Hawkins *et al.* (1997) suggested that elimination of exercise intolerance, and not necessarily of noises, should be the main criterion to consider for LP success in horses. The latter may be difficult to quantify for many reasons. A review of published clinical case studies by Dixon *et al.* 2003a shows a reported reduction, with circa 75% of horses reported to have total absence of noises following laryngoplasty. This is similar to the 73% recorded by Dixon *et al.* (2003a) in a study on 200 older mixed-work horses.

A draw back of using noise elimination as the sole criterion to identify success of LP may be that horses may make 'noises' and yet have no significant upper airflow obstruction. Conversely the elimination of noises does not necessarily indicate the presence of optimal laryngeal airflow (Brown *et al.* 2003). In addition, some owners are poor at detecting abnormal noises as noted by Spiers *et al.* (1983) who detected abnormal noises in horses where the owner could not detect them. This is also a common finding by the author when less experienced owners present cases of RLN for examination. The assessment of the effects of LP on exercise performance can be subjective and many studies including those of Ducharme and Hackett (1995), Kidd and Slone (2002) and Dixon *et al.* (2003a) have shown that laryngoplasty is most likely to improve performance in non-racehorses. Such cases are likely to have severe or total laryngeal paralysis prior to presentation.

Despite the amount of evidence on the value of larynglоplasty, this procedure needs to be evaluated further by larger physiological studies in clinical cases, pre- and post surgery. Such studies may be difficult to conduct, bearing in mind the reluctance of owners and trainers to bring horses back for further treadmill evaluations, even more so when they perceive their horse to be now performing satisfactorily.

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IMPROVEMENTS IN LARYNGOPLASTY

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Despite recognition of laryngeal hemiplegia for over 100 years and development of the laryngoplasty procedure over 30 years ago, there have been no major changes to treatment of laryngeal hemiplegia since its description by Marks *et al.* (1970). There is still reluctance by trainers to have the procedure performed as it does not return the horse to 'normal', and has potential complications. Furthermore, failure to maintain abduction of the arytenoid, both short-term and long-term, is a documented problem. This has led to interest in modifications of the standard laryngoplasty and alternative procedures. Unfortunately, alternative procedures such as re-innervation have had limited success and require a longer return to return to training.

A minor but significant improvement in laryngoplasty is removal of the vocal cord. While early research did not detect a significant improvement in airway mechanics with a ventriculectomy, most surgeons now believe that removal of the vocal cord is beneficial based on both clinical and experimental evidence. Treadmill endoscopy clearly demonstrates the deviation of the vocal cord during inspiration if it is not removed as an adjunctive procedure with laryngoplasty. While the sacculectomy may not truly stabilise the cord, there is some question whether a sacculectomy is beneficial by creating fibrous support for the abducted arytenoid.

The majority of efforts to improve laryngoplasty deal with maintenance of arytenoid abduction and thus suture retention (Dixon *et al.* 2003). Causes of failure are thought to be associated with suture pull out, assumed through the muscular process based on experimental models (Dean *et al.* 2001). Yet, more often it appears to be 'loosening' in clinical cases that results in decreased abduction. One technique developed to minimise loosening was concurrent recurrent laryngeal neurectomy while performing laryngoplasty for horses with remaining laryngeal motion (Davenport *et al.* 2001). The hypothesis was that cycling from remaining muscular pull resulted in suture loosening. Performing the neurectomy was ineffective in improving the prognosis relative to standard laryngoplasty.

Other methods to maintain arytenoid abduction include techniques to place sutures and different suture materials. Mechanical testing of *in vitro* models has been promising but these methods do not yet have long-term clinical follow-up and may present greater difficulty in placement *in vivo* than present standard procedures.

We have pursued 2 other methods. We now often approach the muscular process from behind the cricopharyngeus muscle. This can be performed with appropriate positioning of the horse under general anaesthesia. Dissection is performed easily just above a branch of the cranial thyroid vein and just caudal to the cricopharyngeus muscle through dense fascia to access the muscular process of the arytenoid. This approach should minimise any slack of the suture material by fascial interference and prevent crossing of suture that could occur when passing multiple strands under the cricopharyngeus muscle belly.

An approach to improve stability of the larynx with laryngoplasty has also been developed by creating an arthrosis of the cricoarytenoid (CA) joint. The hypotheses for the experimental model were: 1) that surgical destruction of the CA joint could be performed using a motorised burr via a routine laryngoplasty surgical approach; 2) that surgical destruction of the CA joint with a motorised burr in conjunction with laryngoplasty would result in joint fusion and improved stability

of the arytenoid cartilage; and 3) that surgical destruction of the CA joint using a motorised burr would not result in any complications in relation to laryngeal/pharyngeal tissues or laryngeal function.

Eight horses with normal laryngeal function were prepared for aseptic surgery and a standard laryngoplasty. Three horses were controls, receiving a standard laryngoplasty and 5 had debridement of the CA joint as well as a standard laryngoplasty. To access the CA joint, the tendinous insertion of the *cricoarytenoideus dorsalis* muscle was transected from the caudal border of the muscular process. The muscular process was retracted cranially exposing the lateral CA joint capsule. The capsule was incised exposing the articular cartilage which was debrided with a 2 mm motorised burr. Two #5 polyester sutures were placed through the cricoid cartilage and muscular process in typical fashion. The video-endoscope was placed through the nostril to assess any penetration of laryngeal mucosa with the prosthetic sutures and the position of the arytenoid. The loops of suture were tied independently while observing the abduction of the arytenoid with the endoscope. Soft tissues and skin were closed routinely. All horses were given antimicrobials and anti-inflammatories for one week with stall rest, then turned out on pasture for another 83 days.

Endoscopy was performed and recorded the morning after surgery and at the conclusion of the study. Still images were captured and digitised after induced maximal abduction. The degree of arytenoid cartilage abduction was calculated by using an approach similar to one previously described. A line was drawn connecting the most dorsal and ventral points of the glottis and extended dorsally for a distance one third of the dorsoventral height of the larynx. Tangential lines were then drawn from that point to the edge of each corniculate. The angle between the 2 lines was recorded as the angle of abduction, and the right to left quotient was determined by dividing the right angle by the left angle.

All horses were subjected to euthanasia 90 days from the time of surgery. The larynges of the horses were removed intact with approximately 20 cm of trachea immediately after euthanasia for mechanical testing. The right arytenoid was fixed in maximal abduction with one #2 polyester suture between the muscular process and the cricoid cartilage. The specimens were secured to a wooden board by needles placed through the epiglottis. A 16 gallon vacuum cleaner was secured to the trachea and adjusted to produce flows of 10, 20, 30, 40, 50 l/s. Airflow was measured with a 5 cm-diameter 'fleish-type' pneumotachometer placed between the adaptor and the vacuum cleaner. Translaryngeal pressure difference, and translaryngeal impedence, were calculated at the different airflows – with the suture intact and after it was cut free from the cricoid cartilage without disrupting any fibrous tissue around the muscular process. Digital photographs were obtained at the various airflows with and without the suture cut from the cricoid. From the photographs, the right to left angle quotient were measured and calculated as described.

The results indicate fusion of the CA joint provides additional stability to a standard laryngoplasty in this experimental model. No horses experienced any post operative complications.

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VENTRICULECTOMY/CORDECTOMY

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The vocal cords (folds or plica vocalis) form the ventrolateral limits of the rima glottidis. When horses with laryngeal hemiplegia are examined by video-endoscopy while exercising on a high speed treadmill, one can observe the left vocal fold to be collapsing in the airway and therefore, restricting (Derksen *et al.* 1986; Shappel *et al.* 1988; Tetens *et al.* 1996) the ventral diameter of the larynx (Fig 1). In some cases there is also an associated ipsilateral arytenoepiglottic fold collapse. Finally, in a small percentage of horses, there is also bilateral vocal fold collapse. Because it was recognised early in the study of this disease that the vocal fold and ventricle contributes to the upper respiratory noise and poor performance, ventriculectomy with or without cordectomy were introduced. Indeed, ventriculectomy, cordectomy and arytenoidectomy were introduced and then abandoned in the 19th century by Gunther. At the beginning of the 20th century, ventriculectomy was re-introduced by Professor William L. Williams and popularised by Sir Frederick Hobday. The procedure was extended to a ventriculo-cordectomy late in the 20th century. This modification was supported by treadmill observation that indicated that a ventriculectomy did not always prevent collapse of the ipsilateral vocal fold, the source of the upper airway obstruction of the ventral aspect of the rima glottidis. Over the last decade, some surgeons have begun to remove only the vocal cord.

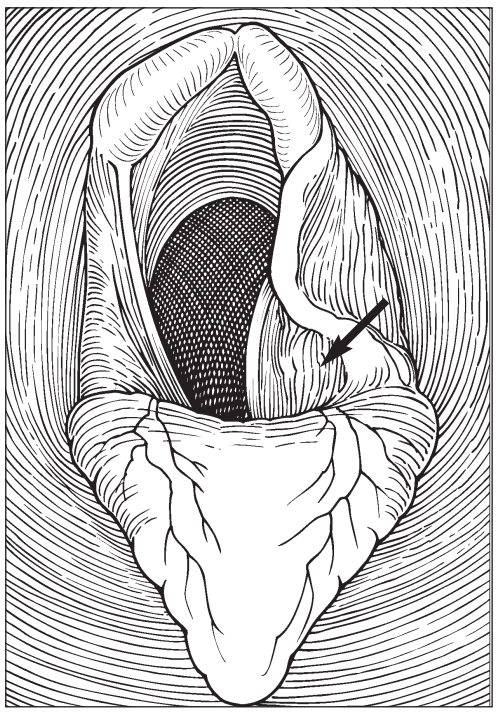

Fig 1: Schematic of Grade IV laryngeal hemiplegia experiencing dynamic collapse of left vocal folds (white arrow) during exercise. Note associated collapse of the left arytenoepiglottic folds (black arrow).

The value of unilateral or bilateral ventriculo-cordectomy/cordectomy (VC/C) in horses with Grade IV recurrent laryngeal neuropathy (RLN) is controversial. This is partially because the ventriculectomy alone (without vocal fold removal) is of little value. The ventriculectomy was initially thought to induce adhesions between the left arytenoid and thyroid cartilages that would limit axial displacement of the arytenoid cartilage during exercise. However, Shappel *et al.* (1988), determined impedance during exercise at up to 7.2 m/s on a 6.38° incline and found no measurable evidence that ventriculectomy alone yields any improvement in the size of the rima glottidis. Although this study has been criticised for assessing performance in submaximal stress, it is hard to conceive that a procedure that shows no

mechanical advantage at low speed would be of some benefit at a higher speed. The value of ventriculectomy as an added procedure to the laryngoplasty was evaluated in 3 studies (Derksen *et al.* 1986, Shappel *et al.* 1988, and Tetens *et al.* 1996) and revealed the same finding ie ventriculectomy is not needed so laryngoplasty alone is the treatment of choice for restoration of upper airway mechanics, both at maximal and submaximal exercise. Despite these findings, most surgeons continue to perform a VC/C with laryngoplasty in horses with RLN.

There are multiple reasons for the belief that ventriculo-cordectomy or cordectomy are useful procedures. Firstly, it is felt that the upper airway mechanic data is not sufficiently sensitive as horses with a laryngoplasty do not have, on visual inspection, a normal airway yet they have normal upper airway mechanics. Perhaps other indices such as arterial blood gases should be used to assess the effect of laryngoplasty on ventilation. In one such study, Edwards (1996), found that a laryngoplasty did not normalise blood gases in horses exercising at 14 m/s on a high speed treadmill indicating that after laryngoplasty, airway size is not restored to normal. This latter finding is consistent with the video-endoscopic evidence of the larynx at exercise that the airway is not restored to normal after laryngoplasty. In the same study, unilateral ventriculo-cordectomy did improve airway mechanics (using impedance flow of 24 l/s) over a laryngoplasty alone, but still did not normalise blood gases or airway mechanics, indicating the airway is not restored to normal in horses exercising at 14 m/s. On the contrary, bilateral ventriculo-cordectomy did not have any advantage over only a laryngoplasty based on airway mechanics data in horses exercising at speeds up to 13 m/s (Tetens *et al.* 1996). The difference in the findings may be due to the sensitivity of the different indices of upper airway mechanics used in both studies and differences in the study design.

Should the use of ventriculo-cordectomy/ cordectomy (VC/C) be considered as the sole treatment of horses affected with Grade IV RLN? Most recently (Derksen 2004a,b), unilateral laser cordectomy or bilateral ventriculo-cordectomy were shown to improve airway mechanics in experimentally induced left laryngeal hemiplegia, although to a much lesser degree than laryngoplasty. This degree of improvement may be sufficient in horses travelling at low speed, and there is some data suggesting that in horses such as draft horses, the airway mechanics are improved with ventriculo-cordectomy. Airway sounds should also be a concern when judging these surgical techniques. Objective data became available when Brown *et al.* (2004) found, based on sound analysis, that bilateral ventriculo-cordectomy can reduce the abnormal upper airway sounds in horses with laryngeal hemiplegia. Further data (Derksen 2004a,b) indicated that unilateral laser cordectomy or bilateral ventriculo-cordectomy alone restores normal upper airway sounds in horses with experimentally created left laryngeal hemiplegia. Laryngoplasty alone was inferior to VC/C in the improvement in upper airway sound that it yielded.

What about the use of ventriculo-cordectomy/ cordectomy (VC/C) as the sole treatment of horses affected with Grade III B RLN? There is no data at this time to answer this question. It is known that horses with partial arytenoid cartilage collapse at exercise have vocal fold collapse. In those horses where the degree of collapse of the arytenoid cartilage is no less than the position of the arytenoid cartilage after a laryngoplasty, the author feels that removing the vocal fold would be of value and would not expose the horse to the possibility of failed laryngoplasty and thus, a worsening of the situation.

SURGICAL PROCEDURE

The horse is placed in a stock for restraint and an intravenous jugular catheter placed under aseptic conditions. A specially padded halter is used to elevate the head during the procedures; sedation results in marked relaxation such that the buckles on the halter must be appropriately padded. The head elevation is facilitated by having a ring on the nose band of the halter and on the ceiling or front of the stock. For sedation of the average 450 kg horse, the authors used a mixture of 5 mg detomidine and 5 mg butorphanol followed with one or 2 repeated detomidine (2 mg) injections as needed. To desensitise the upper airway and the right nasal cavity, a solution containing 50 cc lidocaine hydrochloride and 10 cc 0.15% solution of phenylephrine is applied through the biopsy channel of the video-endoscope.

The author performs the procedure using a diode laser fibre or ND: YAG laser. The video-endoscope is inserted in the right nostril and the laser fibre passed through into the biopsy channel

such that it is positioned over the axial surface of the left vocal fold. The incision must start at the caudal and ventral aspect of the vocal fold and extend to the rostral aspect of the vocal cord. Care should be taken not to extend the incision to the abaxial surface of the vocal cord at this time because there are significant blood vessels at the junction of the rostral and abaxial edges of the vocal cord. The video-endoscope is then placed through the left nasal cavity to give a better perspective of the next incision. A grasping bronchoesophagoscopic forceps (product no. 8280.62, Richard Wolfe Medical Instruments Corporation, Vernon Hills, Ill.), bent with an arc of approximately 30° to conform to the curve of the nasal passage and pharynx, is inserted onto the right nasal cavity until it becomes visible on the dorsal aspect of the epiglottic cartilage. Elevation of the head at this point facilitates the grasping of the vocal cord immediately dorsal to the rostral incision. The vocal fold is then pulled axially and rostrally such that the laser fibre does not inadvertently touch the right vocal fold. This forms a triangle of vocal fold (apex in the forceps). The base of this triangle of the vocal cord is incised vertically starting 3–4 mm distal to the vocal process of the arytenoid's cartilage. The vertical incision is continued until the ventral incision is reached and vocal cord excised. Care must be taken not to lase the contralateral vocal cord during the latter process. Post operatively, the horses are administered systemic antibiotics for 7 days and phenylbutazone for 5 days. The surgical site is usually healed in 2–3 weeks.

Currently, the author still performs ventriculo-cordectomy or cordectomy to treat exercise intolerance in horses working at low velocity (like draft horses), horses with partial collapse of the left arytenoid cartilage and/or vocal fold at exercise, and in association to a laryngoplasty. It is also used to reduce/normalise abnormal airway sounds in horses with Grade III or IV RLN.

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VENTRICULO-CORDECTOMY FOR TREATMENT OF RECURRENT LARYNGEAL NEUROPATHY: 75 CASES IN A MIXED POPULATION OF HORSES

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Introduction

There are few reports in the literature of the efficacy of ventriculectomy, cordectomy or ventriculo-cordectomy (VC) for the treatment of equine recurrent laryngeal neuropathy (RLN). Laryngoplasty (LP) with or without ventriculectomy or vocal cordectomy are currently the techniques of choice for the treatment of RLN for most equine surgeons. Recent experimental studies have shown that VC significantly reduces abnormal respiratory noises in horses with experimentally induced laryngeal hemiplegia (Brown *et al.* 2003), and this technique has been recommended if reduction of respiratory noise is the primary objective of surgery. VC has also been shown to improve upper airway function in horses with experimentally induced laryngeal hemiplegia, but not to baseline levels (Brown *et al.* 2003). Kidd and Slone (2003) suggested that inclusion of a vocalcordectomy along with the LP procedure was important in terms of eliminating respiratory noise in clinical cases. Vocal cord collapse has been reported to occur without significant arytenoid cartilage collapse in horses with lower grades of RLN undergoing high-speed treadmill endoscopy (Hammer *et al.* 1998; Lane 2003; Dixon and Barakzai, unpublished observations), and it is possible that for such cases, VC would provide a good alternative to LP. To the authors' knowledge, there has been no large study of the clinical use of VC published to date.

Aims

This study proposed that for performance horses (ie racehorses) with low grades of RLN (Grade 2 or 3, using a 6-grade system, Dixon *et al.* 2001), or for non-performance horses with any grade of RLN, VC alone would reduce clinical signs of RLN. The study also aimed to determine the rate of post operative complications and owner's assessment of the value of surgery and compare these to LP + VC surgery (Dixon *et al.* 2003a,b).

Materials and methods

Seventy-five horses which underwent unilateral ventriculectomy and vocalcordectomy at the Royal (Dick) School of Veterinary Studies (R(D)SVS) for treatment of idiopathic RLN were selected for subjective restrospective analysis. Surgery was performed via a ventral laryngotomy incision, and after sharp excision of the laryngeal ventricle and vocal fold, the crico-thyroid membrane was closed with 3.5 metric polyglactin 910 (Vicryl, Ethicon). Cases were endoscoped at one day, 6 days and 6 weeks post operatively. A postal questionnaire was sent to all owners/trainers after the horses had been back in work for at least one year following surgery, which was followed up by a telephone questionnaire if there was no response within 2 months.

Results

The breeds included 50 Thoroughbreds, 21 Thoroughbred crosses, 1 Clydesdale and 3 ponies. The work of horses in this study included 40 National Hunt racehorses, one flat racehorse, 12 hunters, 7 eventers, 3 showjumpers, and 12 miscellaneous work loads. The median age was 6 years (range 3–15 years), and median height was 16.2 hh (range 12.0–18.2 hh). Presenting signs included abnormal exercise related respiratory 'noises' detected by the owner/trainer (53%), poor exercise performance (11%), abnormal noises and

poor performance (33%), and abnormal respiratory noises detected at a pre-purchase veterinary examination (3%). The median pre-operative endoscopic grade of RLN was 2 (range 2–5), with 74 horses afflicted with left sided RLN and one afflicted with right sided RLN. Twenty-nine percent of horses had endoscopic evidence of lower airway disease on resting endoscopy.

Complications of surgery: Sixty-two percent of horses had no discharge from the laryngotomy wound when the horse returned home, one week post operatively. In 20% of horses, the discharge had resolved by 2 weeks post operatively, and in 3% of horses the laryngotomy discharge persisted for more than 4 weeks. Twenty-two percent of horses coughed after surgery (Fig 1), of which 27% coughed whilst eating and the remainder coughed at times unassociated with eating. Sixty-six percent of horses did not make abnormal noises post operatively (Fig 2). Nine percent continued to make abnormal noise at the canter, 21% made noises at the gallop, and 4% of owners were unsure if abnormal noises were present. Of the 23 horses which made abnormal noises at exercise post operatively, 19 returned to full work regardless, and 6 were diagnosed with other disorders in the year post-operatively including 3 with dorsal displacement of the soft palate, one with facial paralysis, one with pulmonary disease and one false nostril atheroma.

Post operative performance: Ninety-three percent of horses returned to full work after

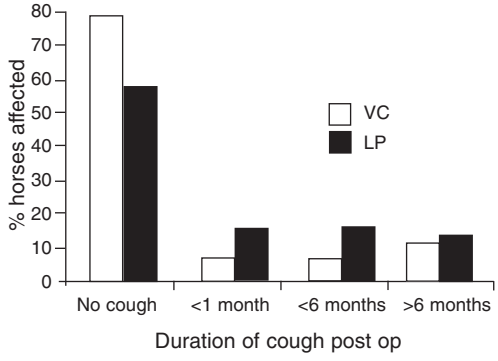

Fig 1: Bar chart showing % of horses affected with coughing post operatively for both ventriculo-cordectomy (VC) and laryngoplasty with ventriculo-cordectomy (LP) procedures (Dixon et al. 2003a).

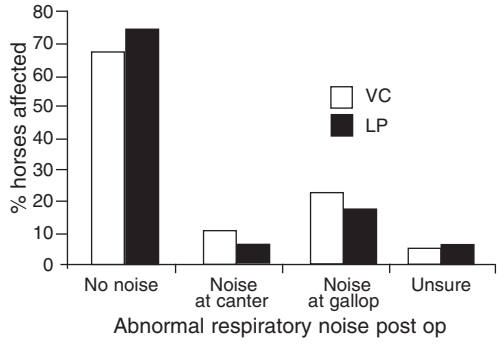

Fig 2: Bar chart showing % horses making abnormal respiratory noises post operatively for both ventriculo-cordectomy (VC) and laryngoplasty with ventriculo-cordectomy (LP) procedures (Dixon et al. 2003a).

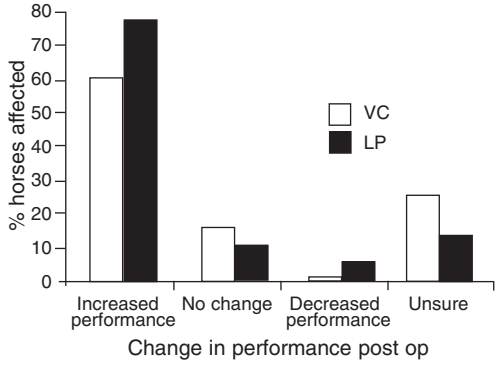

Fig 3: Bar chart showing % horses which the owner/trainers considered had increased, decreased or no change in exercise performance post operatively for both ventriculo-cordectomy (VC) and laryngoplasty with ventriculo-cordectomy (LP) procedures (Dixon et al. 2003b).

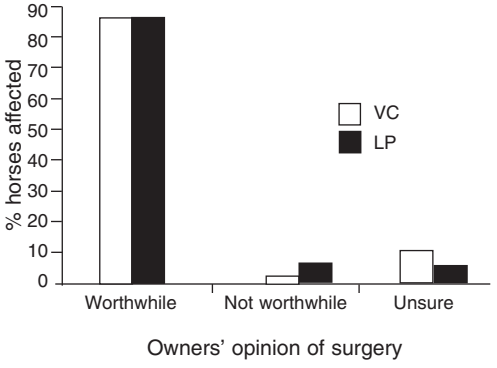

Fig 4: Bar chart showing the owner/trainers' overall opinion of surgery for both ventriculo-cordectomy (VC) and laryngoplasty with ventriculo-cordectomy (LP) procedures (Dixon et al. 2003b).

surgery, 6% were able to perform reduced work, and one horse was retired. Fify-nine percent of owners reported that the horse's performance had increased as compared to pre-operative performance, 16% reported no change, and the remaining owners were unsure of any such change (Fig 3). Overall, 86% of owners considered the surgery to be worthwhile, 3% did not consider it to be worthwhile, and 11% were unsure of its value (Fig 4).

Discussion and conclusions

When compared to LP procedures, VC had a lower rate of post operative complications. Unsurprisingly, healing of the laryngotomy wound was very similar to healing in horses that underwent LP with VC (Dixon *et al.* 2003a), with the vast majority of horses having no discharge from the surgical site at 2 weeks post operatively. However, in horses which underwent LP plus VC, an additional 19% had wound complications (seromas, suture abcesses etc) associated with the LP wound. Although 22% of horses coughed post VC, this is considerably fewer than the 43% of horses which coughed after LP, and this is unsurprising because after VC the arytenoid cartilages are not fixed in an abducted position and should be able to adduct and protect the airway during deglutition. Additionally, in the majority of horses which did cough post VC, coughing was not associated with eating and may therefore be attributable in some cases to pre-existing lower airway disease (present in 29% of horses) rather than dysphagia due to surgical interference.

A larger percentage of horses were reported to make abnormal respiratory noises post VC (34%) as compared to LP combined with VC (27%) (Dixon *et al.* 2003a) even though the group which underwent VC alone had a lower pre-operative median grade of RLN than those undergoing VC combined with LP. This is surprising given that the majority of noise is thought to arise from vibration of the vocal cord in horses afflicted with RLN (Hammer *et al.* 1998; Kidd and Slone 2002). Similarly, slightly fewer horses were considered subjectively to have increased exercise performance post operatively as compared to horses which underwent LP, but the overall rate of owner satisfaction was very similar for the 2 procedures.

This study shows that ventriculo-cordectomy is a useful alternative to laryngoplasty for selected cases of RLN and is associated with a low post operative complication rate and a high rate of owner satisfaction.

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SESSION 6:

Evaluation of RLN treatment efficacy

Chairman: Norm Ducharme

TREATMENT OF RECURRENT LARYNGEAL NEUROPATHY: PHYSIOLOGICAL AND PERFORMANCE EVALUATION

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In exercising horses with recurrent laryngeal neuropathy (RLN) there is dynamic collapse of the affected arytenoid cartilage during inspiration and an inspiratory upper airway obstruction. Severity of the collapse and obstruction depends on degree of laryngeal paresis (Martin *et al*. 2000). The obstruction is characterised by increased inspiratory trans-upper airway pressure, inspiratory impedance and inspiratory time, and decreased inspiratory flows, respiratory frequency and minute ventilation. Hypoxemia and hypercapnia are also observed. (Derksen *et al.* 1986). Treatments for laryngeal hemiplegia include: prosthetic laryngoplasty ventriculectomy, ventriculo-cordectomy, laser cordectomy, partial, total and subtotal arytenoidectomy and laryngeal re-innervation.

PROSTHETIC LARYNGOPLASTY

The prosthetic laryngoplasty technique was first described by Marks *et al.* (1970). The goal of the procedure is to produce mechanical abduction of the arytenoid cartilage midway between normal resting and full abduction. Laryngoplasty returns upper airway flow mechanics to baseline levels by 30 days after surgery (Derksen *et al*. 1986; Shappell *et al.* 1988). Stabilisation of the affected arytenoid is more important than the degree of abduction. Indeed, there is no correlation between degree of arytenoid abduction and residual airway obstruction following surgery (Russell *et al.* 1994). Prosthetic laryngoplasty is the treatment of choice for RLN in horses where airway obstruction and exercise intolerance are the primary concern (Russell and Slone 1994; Hawkins *et al.* 1997). However, post operative complications are common and include prosthetic failure, dysphagia, coughing and infection (Hawkins *et al.* 1997).

VENTRICULO-CORDECTOMY

Ventriculectomy or sacculectomy, refers to the removal of the laryngeal saccule. Ventriculectomy aims to produce abduction of the affected arytenoid cartilage by formation of adhesions between the arytenoid and thyroid cartilages and to reduce filling of the ventricle with air during inspiration. Vocal cordectomy is often performed alone or in conjunction with ventriculectomy (ventriculo-cordectomy). These procedures may be performed unilaterally or bilaterally and remaining tissues are left to heal by second intention or sutured. Variations in surgical techniques are likely to influence efficacy. Unilateral ventriculectomy alone does not improve upper airway function 30 days after surgery (Shappell *et al.* 1988). Both unilateral vocal cordectomy and bilateral ventriculo-cordectomy reduce, but do not eliminate, upper airway obstruction in laryngeal hemiplegia affected horses. These effects are evident 30 days following surgery. The beneficial effects of prosthetic laryngoplasty on upper airway flow mechanics are not enhanced with bilateral ventriculo-cordectomy (Tetens *et al.* 1996).

ARYTENOIDECTOMY

Arytenoidectomy is rarely the first choice of surgery for RLN but it is indicated in cases of failed prosthetic laryngoplasty or arytenoid chondritis. The total arytenoid cartilage may be removed, (total arytenoidectomy), the muscular process may be left in place (partial arytenoidectomy), or the corniculate process may be spared (subtotal arytenoidectomy).

Partial arytenoidectomy combined with bilateral ventriculo-cordectomy improves upper

airway flow mechanics in exercising horses with experimentally induced laryngeal hemiplegia (Lumsden *et al.* 1994) but flow volume loop analysis shows that some airway obstruction remains. Complications following arytenoidectomy are common and include dysphagia and coughing. In search of a technique with fewer complications, subtotal arytenoidectomy was proposed. However, this fails to improve upper airway function (Belknap *et al.* 1990). Endoscopic evaluation during exercise following subtotal arytenoidectomy demonstrates that the unsupported corniculate process collapses into the airway during inhalation.

Laryngeal re-innervation

As RLN treatments are ineffective or associated with serious complications, there has been interest in finding a more physiological solution, eg laryngeal re-innervation. Branches of the first cervical nerve and associated *omohyoideus* muscle are transplanted into the affected *crico-arytenoideus dorsalis* muscle. The nerve muscle pedicle graft technique is effective in restoring upper airway flow mechanics in horses with experimentally induced laryngeal hemiplegia (Fulton *et al.* 1991). As the first cervical nerve is an accessory muscle of respiration, and the nerve is activated only during exercise, surgical success can only be assessed at exercise. It may take up to a year for upper airway flow mechanics to return to normal. Associated complications are mild and rare. Fulton *et al. (*2003) reported that the nerve muscle pedicle graft technique is as effective as prosthetic laryngoplasty in returning Thoroughbreds to competitive racing and therefore it is recommended when complications linked with other techniques are unacceptable, or when time between surgery and return to athletic activity is less important.

Effect on racing performance

How effectively can surgery restore athletic performance in RLN-affected racehorses? Stick *et al. (*2001) showed that elite Thoroughbred yearlings with Grade 3 or 4 RLN had reduced performance as adults. Assuming that they received the best available care, this suggests that current surgical treatments cannot fully restore athletic performance in an elite Thoroughbred with RLN. This is supported by other studies (Strand *et al*. 2000).

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TREATMENT OF RECURRENT LARYNGEAL NEUROPATHY: EVALUATION BY RESPIRATORY SOUND ANALYSIS

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Upper respiratory disease is suspected in an exercising horse when an abnormal respiratory noise is heard and when the horse's performance is reduced. While performance reduction can be caused by dysfunction in many systems, respiratory noise during exercise is specific for an upper airway problem.

For sport horses, respiratory noise caused by conditions such as recurrent laryngeal neuropathy (RLN) can be more important than the obstruction itself. There is information in the literature describing the efficacy of surgical procedures in improving upper airway flow mechanics in exercising horses with RLN, (Derksen *et al.* 1986; Shappell *et al.* 1988; Tetens *et al.* 1996) but there is little information about noise reduction. This information is also important for racehorses, as residual respiratory noise after surgery is often interpreted as failure to improve upper airway flow mechanics (Russell and Slone 1994; Hawkins *et al.* 1997; Kidd and Slone 2002). However, the relationship between noise and upper airway obstruction has not been evaluated critically (Derksen 2003).

Recording and analysing respiratory sounds in exercising horses

The first challenge associated with the quantitative evaluation of respiratory sound in exercising horses is the squelching of extraneous noises. To accomplish this, a dynamic unidirectional microphone is placed in such a way that the recording microphone is directed towards the nostrils and rests approximately 4 cm from the horse's nose. The microphone is connected to a cassette recorder containing an automatic gain control and a compression circuit. The combined features of this system reduce extraneous noises. The recorded sounds are evaluated using computer-based spectrum analysis (Derksen *et al.* 2001).

RLN and respiratory noise

Exercising horses with RLN make a distinctive inspiratory noise. In affected horses, high-intensity sounds are present throughout inhalation (Cable *et al.* 2002; Franklin *et al.* 2003). This sound is characterised by 3 frequency bands called formants which are centred at approximately 400, 1700, and 3700 Hz. Quantitative indices describing the loudness and character of the sound include inspiratory sound level and the sound intensity of the 3 inspiratory formants (Derksen *et al.* 2001). The sound intensity of the formant 2, centred at about 1700 Hz, is most important, because this formant is in a frequency range where human hearing is most acute.

Efficacy of surgical procedures in reducing noise caused by RLN

Surgical techniques recommended for the treatment of RLN include prosthetic laryngoplasty, the nerve muscle pedicle graft technique, ventriculectomy, ventriculo-cordectomy, laser cordectomy, and total, partial, and subtotal arytenoidectomy. Thus far, we have only studied the effect of prosthetic laryngoplasty and bilateral ventriculo-cordectomy on respiratory noise caused by experimentally induced laryngeal hemiplegia. Thirty days after surgery, bilateral ventriculo-cordectomy has no effect on inspiratory noise. However, at 90 and 120 days after surgery most indices of inspiratory noise, including the sound intensity of formant 2, return to baseline levels. Inspiratory sound level remains slightly but

significantly elevated. Thus, bilateral ventriculocordectomy effectively reduces inspiratory noise associated with laryngeal hemiplegia.

The effects of prosthetic laryngoplasty on upper airway noise in laryngeal hemiplegia affected horses are already evident 30 days after surgery. However, key indices of sound intensity, including the sound intensity of formant 2, remain elevated relative to baseline levels. This indicates that prosthetic laryngoplasty is less effective than ventriculo-cordectomy in reducing laryngeal hemiplegia associated noise. The efficacy of prosthetic laryngoplasty in reducing airway obstruction and noise is variable in individual horses. The degree of remaining airway obstruction following surgery and the amount of residual respiratory noise are not correlated. Therefore residual respiratory noise following prosthetic laryngoplasty cannot be used to determine the degree of remaining airway obstruction in individual horses.

Interestingly, there is a significant positive correlation between the degree of arytenoid abduction and inspiratory noise following surgery. That is, the more arytenoid abduction achieved, the greater the residual noise. The degree of arytenoid abduction following surgery does not correlate with residual airway obstruction.

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SPIROMETRIC AND ENDOSCOPIC ASSESSMENT OF SURGICAL TREATMENT IN HORSES WITH LARYNGEAL HEMIPLEGIA

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INTRODUCTION

The outcome of a 'tie back' operation is routinely judged, based on the endoscopic re-evaluation, the changes in the quality and volume of the respiratory noise and the performance history of the equine athlete. Exercise spirometry is a valuable method to quantify airflow limitation of upper airway diseases (Shappell *et al.* 1988; Belknap *et al.* 1990; Lumsden *et al.* 1993, 1994; Tetens *et al.* 1996; Weishaupt *et al.* 1998*)* and is therefore used in the authors' clinic to assess the functional improvement after this surgical intervention.

The relationship between the degree of abduction by laryngoplasty and the extent of functional improvement is poorly investigated. As over-abduction of the paralysed arytenoid may result in coughing and/or dysphagia, an optimal lateralisation aims at correcting each athlete individually in relation to its future athletic career.

The aim of this study was to quantify the functional improvement of a combined laryngoplasty-ventriculectomy procedure through spirometric assessment and to correlate function with the arytenoid abduction angle determined endoscopically before and after surgery.

MATERIAL AND METHODS

Horses were trained to canter on a high-speed treadmill. Time and flow parameters were measured with an ultrasonic flowmeter (Spiroson Scientific®, Isler Bioengeneering AG, Dürnten, Switzerland). The levels of exercise intensity were adjusted to the horse's individual capabilities. The left abduction angle was surveyed on endoscopic pictures using the method described by Reutter (Reutter *et al.* 1994). A combined laryngoplasty-ventriculectomy procedure was performed to correct the laryngeal hemiplegia. After a rehabilitation and retraining period of 5 months, the horses were reassessed using the identical exercise protocol. With the owner's consent, 19 Warmblood horses with left laryngeal hemiplegia Grade 4/4 (Rakestraw *et al.* 1991) were assessed in this way.

RESULTS

Before surgery, beginning airflow limitation could be observed already at the trot; the conclusive inspiratory flow limitation occurred usually only at canter intensities of 6.0-7.5 m/s at 6% incline (heart rates 158-209/min). The inspiratory flow curve was characterised by a plateau phase and peak values were limited at 48 ± 9.9 l/s (mean ± SD; range 26–63 l/s). At expiration no limitation was obvious and peak flow reached 69 ± 9.8 l/s (range 49–94 l/s). Inspiratory time was always prolonged. Horses with severe inspiratory, dynamic collapse of the paralysed arytenoid, showed an initial flow peak with a subsequent characteristic drop of flow. At the canter, 7 of the 19 horses changed their 1:1 locomotion-to-respiration coupling (LRC) intermittently or permanently to 2:1.

Differences in the degree of abduction and of functional parameters after surgery are listed (Table 1).

Independent of the LRC strategy, peak inspiratory flow (PIF) increased over all horses by 46.2% and thus proportional to the abduction angle. Minute ventilation (VE) increased in average by 30.9%.

Comparing the increase of PIF and VE with the changes of the left abduction angle no significant correlation could be found (Pearson,

TABLE 1: Mean difference ± SD (percentage difference)

A_{abd}	7.1 ± 7.7	(+42.3%)*		
	LRC 1:1		**LRC 2:1**	
f_R	0.3 ± 1.3	(+0.3%)	32 ± 14.5	(+50.1%)*
t_{insp}	-3.5 ± 3.2	(-6.1%)*	-10.8 ± 4.5	(-16.9%)*
VT	2.5 ± 1.2	(+18.1%)*	-0.4 ± 4.5	(-1.7%)
VE	269 ± 108	(+18.4%)*	593 ± 265	(+54.9%)*
PIF	16.4 ± 7.0	(+31.5%)*	28.9 ± 9.5	(+73%)*
PEF	3.5 ± 4.7	(+5.6%)	7.5 ± 6.1	(+11.2%)*

A_{abd}, abduction angle [degree]; f_R, respiratory rate [1/min]; t_{insp}, inspiratory time fraction [%]; VT, tidal volume [l]; VE, minute ventilation [l/min]; PIF, peak inspiratory flow [l/s]; PEF, peak expiratory flow [l/s]
* significant difference (paired t-test, P<0.05)

P<0.05). However, looking at the plots, 2 groups could be discerned: In two thirds of the patients, PIF increased linearly with increasing abduction angle so that an increase of one degree improved PIF by 1.2 l/s (R^2 = 0.815). In the other horses respiratory function improved although the surgical correction was inadequate but enough to prevent dynamic collapse of the paralysed arytenoid.

Discussion

Based on geometric considerations, the area of the left half of the rima glottidis, outlined by the arytenoid cartilage, the vocal cord and the midline, increased proportionally up to an abduction angle of 45°. In this range, PIF increased by approximately one litre per second with every further degree of abduction. Inappropriate surgical corrections (0–10° abduction) improved respiratory function disproportionately by preventing dynamic collapse. For abduction angles above 45°, the geometric calculations indicate that the laryngeal aperture does not further increase substantially, as the area is estimated to be a function of the sinus of the angle. The functional benefit of abduction angles >50° – which are usually aimed at in racehorses – has still to be investigated.

In conclusion, sole endoscopic assessment may not reflect in all cases the functional improvement of a laryngoplasty-ventriculectomy procedure.

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MANAGEMENT OF CANINE LARYNGEAL PARALYSIS

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In the horse recurrent laryngeal neuropathy has been recognised as a clinical disorder for centuries, but in the dog laryngeal paralysis was first documented only 30 years ago (O'Brien *et al.* 1973). It is likely that the condition is not new but newly recognised and in former times it may have been confused with cardio-pulmonary failure or non-responsive idiopathic millophylline deficiency (Table 1).

The major differences between the canine and equine presentations of the neurogenic failure of the intrinsic laryngeal musculature are that in dogs the disease is typically bilateral by the time that clinical signs appear, and older animals are likely to be involved. A congenital and inherited version of the disorder is recognised in the Bouvier de Flandres (van Haagen 1978) and the husky (Hendricks and O'Brien 1985), but the majority of canine patients are over 10 years of age at presentation. Two thirds of afflicted dogs are male or neutered male and those weighing 25–35 kg are most susceptible. The best recognised predisposed breeds include the Labrador retriever, Afghan hound, Irish setter, and English and German pointers (Lane 1986; see Table 2). Giant breeds are rarely involved probably because of their limited longevity.

TABLE 1: Major presenting signs reported by owners of 750 dogs with acquired laryngeal paralysis presented at UBVS

Stridor	656
Gagging/retching cough	434
Changed bark	327
Reduced exercise tolerance	317

The presenting signs for canine laryngeal paralysis (Table 1) include stridorous breathing even at rest, reduced exercise tolerance, a moist retching cough and loss of bark. Owner expectations of older dogs often lead to delayed presentation.

Ventriculo-cordectomy has never been proposed as a means to manage canine laryngeal paralysis (Table 3) but initially a radical partial laryngectomy was used (O'Brien *et al.* 1973). This comprised the excision of the true and false vocal folds as well as the ventral projections of the arytenoid cartilages. An unacceptable complication rate was achieved (Ross *et al.* 1991).

Laryngoplasty ('tie-back') surgery represents one of the few instances where an equine technique has been transferred to canine surgery. The technique first proposed a lateralisation of the disarticulated arytenoid on each side of the larynx to the wing of the thyroid cartilage using a ventral approach (Harvey and van Haagen 1975). The technique most widely used today comprises a combined prosthetic abductor implantation with a lateralising suture on the left side of the larynx only (Lane 1982; LaHue 1989). The left lateral approach is similar to that used in the horse but the small muscular process of the dog is insufficiently robust to hold fixation sutures. Thus, the arytenoid is disarticulated to access the thicker body of the cartilage. Although the canine version of the disease is invariably bilateral a unilateral solution is sufficient for the demands of the sedentary life of an elderly dog. Only working dogs such as Huntaways are subjected routinely to bilateral surgery (Burbidge *et al.* 1993).

The results of 'tie-back' surgery in dogs are excellent in experienced hands and the incidence of complication is much lower than for the equivalent procedure in horses (White 1989).

TABLE 2: Breed prevalence in 750 dogs with acquired laryngeal paralysis presented at UBVS

Breed	UBVS Cases		UK control population (Thrusfield, 1989)
	No	**%**	**%**
Labrador*	307	40.9	10.7
Afghan hound*	84	11.2	NR < 3
Irish setter*	61	8.1	NR < 3
Golden retriever*	43	5.7	3.8
English springer spaniel	20	2.7	2.9
Border collie	16	2.1	4.5
Flat coat retriever	14	1.9	NR < 3
English setter	13	1.7	NR < 3
Standard poodle	13	1.7	NR < 3
St Bernard	10	1.3	NR < 3
Finnish spitz	10	1.3	NR < 3
Rhodesian ridgeback	9	1.2	NR < 3
Weimaraner	7	0.9	NR < 3
Airedale	6	0.8	NR < 3
English cocker spaniel ‡	6	0.8	4.3
Greyhound	6	0.8	NR < 3
Other + crosses	125	-	-
German SD ‡	0	-	8.4
Yorkshire terrier ‡	0	-	6.0
Jack Russell terrier ‡	0	-	5.2
West Highland terrier ‡	0	-	3.9

* Significantly over-presented breeds
‡ Significantly under-presented breed
NR Insufficient numbers recorded but less than 3 percent

TABLE 3: The development of surgical treatments for acquired laryngeal paralysis of dogs

Date	Technique	Authors
1973	Partial laryngectomy	O'Brien *et al.*
1975	Unilateral arytenoid lateralisation by ventral approach, using routine tracheotomy intubation	Harvey and Venker-van Haagen
1982	Unilateral arytenoid lateralisation by lateral approach without routine tracheotomy intubation	Lane
1982	Bilateral arytenoid lateralisation by ventral approach	Rosen and Greenwood
1983	Castellated laryngofissure and vocal fold resection	Gourley *et al.*
1986	Modified castellated laryngofissure with arytenoid lateralisation	Smith *et al.*
1986	Abductor prosthesis	Lane (also cited by LaHue 1989)
1986	Composite laryngoplasty combining unilateral abductor prosthesis with lateralisation	Lane
1993	Bilateral arytenoid lateralisation using bilateral lateral approaches	Burbidge *et al.*

Apart from follow-up data from owners attempts have been made to measure the physiological impact of the airway obstruction and its relief using arterial gas tensions (Love *et al.* 1987) and tidal breathing flow-volume analysis (Amis *et al.* 1986). However, elderly dogs are not amenable to controlled exercise regimes on treadmills and techniques to assess respiratory obstruction in dogs are limited.

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SESSION 7:

The future

Chairman: Ed Robinson

IS RLN INHERITED? NEWER TECHNIQUES TO HELP INVESTIGATE THIS QUESTION

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Several papers have been published which suggest that laryngeal hemiplegia has a genetic basis. In many of these studies it has been observed that the offspring of affected stallions are more likely to be affected than the offspring of unaffected control stallions. For example, in one recent study examining 47 offspring of an affected stallion, 11 were affected with laryngeal hemiplegia and another 11 were suspect whereas, in a control group of 50 offspring, only one affected and 4 suspect individuals were seen. The difference is significant at the $P<0.01$ level. A significant difference in the average height at the withers of the affected stallion's affected offspring was noted compared to his unaffected offspring. Some authors have proposed that a dominant gene may be responsible for the disease. One group looked for an association between particular equine leucocyte antigen (ELA) haplotypes and laryngeal hemiplegia with negative results.

The development of a genetic linkage map for the horse provides the molecular tools to attempt to map diseases and traits with a genetic basis in the horse. A map with 359 microsatellite markers, with markers assigned to every chromosome, was published in 2000. Further work has doubled the number of markers mapped on the linkage map. The recent publication of the first radiation hybrid (RH) map of the horse enables the power of comparative genetics to be applied to genetic studies in the horse. The horse RH map identifies conserved evolutionary segments between the horse and human genomes. These resources have been supplemented by the construction of a high quality bacterial artificial chromosome (BAC) library, containing 10-fold coverage of the horse genome, which provides researchers with ready access to cloned genomic copies of nearly all horse genes. Increasing numbers of horse expressed sequence tags (ESTs) from multiple tissues are also now being deposited in the sequence databases.

The availability of these molecular tools to undertake genetic characterisation of diseases and traits in the horse presents many opportunities to improve the health of horses. Combining sophisticated clinical expertise and molecular genetics in the area of recurrent laryngeal neuropathy should prove fruitful.

MULTICENTRE TRIALS FOR EFFICACY OF TREATMENT

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Over the last 30 years, there have been many surveys and reports from multiple centres describing treatment results for a large number of horses with recurrent laryngeal neuropathy (RLN) (Russel and Sloane 1994; Hawkins *et al.* 1997; Strand *et al.* 2000; Kidd *et al.* 2002; Dixon *et al.* 2003a,b). In addition, there is reasonable experimental data available for evaluating the patency of the upper airway after the current surgical options: ventriculectomy, ventriculocordectomy, laryngoplasty, subtotal arytenoidectomy, partial arytenoidectomy, and laryngeal innervation (Derksen *et al.* 1986; Shappel *et al.* 1988; Tetens *et al.* 1996). Recently, more knowledge has been gained about methods to objectively measure upper airway sounds after these various treatments.

Is there a need for multicentre trials, and what benefits would a centrally coordinated multicentre trial provide? Before we answer these questions, consider that the success rate in various surveys for racehorses treated for laryngeal hemiplegia (LH) ranges from 50 to 70%. Also, significant complications such as tracheal aspirations, persistence of upper respiratory noise, incisional seromas, incisional infection, and chondritis are still seen. It is necessary to know the optimal way to manage these complications in addition to reducing their number and severity. It would be useful to know: 1) the odds ratio that a horse will develop chondritis or decreased abduction after an incisional seroma; 2) the desirable degree of abduction to be targeted at surgery related to a horse's specific activity; 3) whether more horses return to work after a partial arytenoidectomy than after a laryngoplasty; whether the Netherlands' elimination of horses with laryngeal hemiplegia and some degree of laryngeal hemiparesis from breeding considerations decreased the incidence of the disease; and 4) whether laryngeal re-innervation is useful for reducing upper respiratory noise in horses performing at submaximal exercise levels. Clearly there are many unanswered questions that remain.

WHAT CORE OBJECTIVES WOULD A MULTICENTRE TRIAL ACHIEVE?

1) Accumulation of significant data for evaluating treatment results stratified by subtypes of horse and activity.

2) A group could be set up to develop recommended guidelines for treating and managing LH complications based upon review of the data evidence. This group could also help set research guidelines or priorities.

HOW WOULD THIS WORK?

'The Equine Recurrent Laryngeal Neuropathy (ERLN) group' could oversee the study design (ie inclusion criteria). The data would be entered at the point of collection only by participating investigators using a web-based program that would allow anyone to view the data being accumulated. For example, the treatment could be stratified by breed, age at the time of treatment, and activity (jumper, hunter, racehorses [point to point, flat race, steeple chase, 3 day event, quarter horse, barrel racing, and endurance] and show horses). The type of treatment (laryngeal re-innervation, laryngoplasty, partial arytenoidectomy) and technical details such as type and number of sutures, pattern of anchorage, and experience of surgeon would also be recorded. Criteria for evaluating results consistently would be agreed upon beforehand so success would be uniformly defined.

Treatment complications are a critical factor to evaluate in the author's opinion. One of the first benefits of a multicentre trial could be the knowledge obtained about complications and their management, which have not been well addressed historically. For example, data collected could show whether immediate re-operation or patience is best for handling acute dysphagia. The ERLN group could make recommendations such as those shown in Figure 1 based on their review of current evidence. Aside from diagnosis and treatment considerations, blood samples could be used for genomic testing, histopathological samples could be sent to different laboratories with different focuses, etc, and the understanding of the heritability of RLN could be increased.

Advantages in establishing research criteria and priorities

Most researchers use different exercise protocols and testing criteria. The group should establish recommended exercise protocols in order to obtain consistent data. For example, they could discuss the pros and cons for: 1) using exercise protocols at maximal heart rate on an incline at lower speeds vs maximal heart rate at maximal speed; 2) different research protocols for sprinters compared to horses that perform at longer distances; 3) the best indices of airway mechanics data (ie arterial blood gases, flow volume loops and their indices, pressure flow curves and their indices, pulse oximetry); 4) standardising sound collection procedures for sound analysis; and 5) setting priorities in the treatment: method of anchoring the prosthesis, degree of abduction targeted, best post operative feeding protocol, post operative diet, etc.

The ERLN group could help identify areas that have been researched enough and those in need of further investigation.

What are the hurdles?

The needs for an agency to fund the establishment of the ELH group, organise a meeting, set up data collection, and maintain a website. The organisational procedures of similar organisations, such as the Cochrane Collaboration (www.cochraneconsumer.com), could be

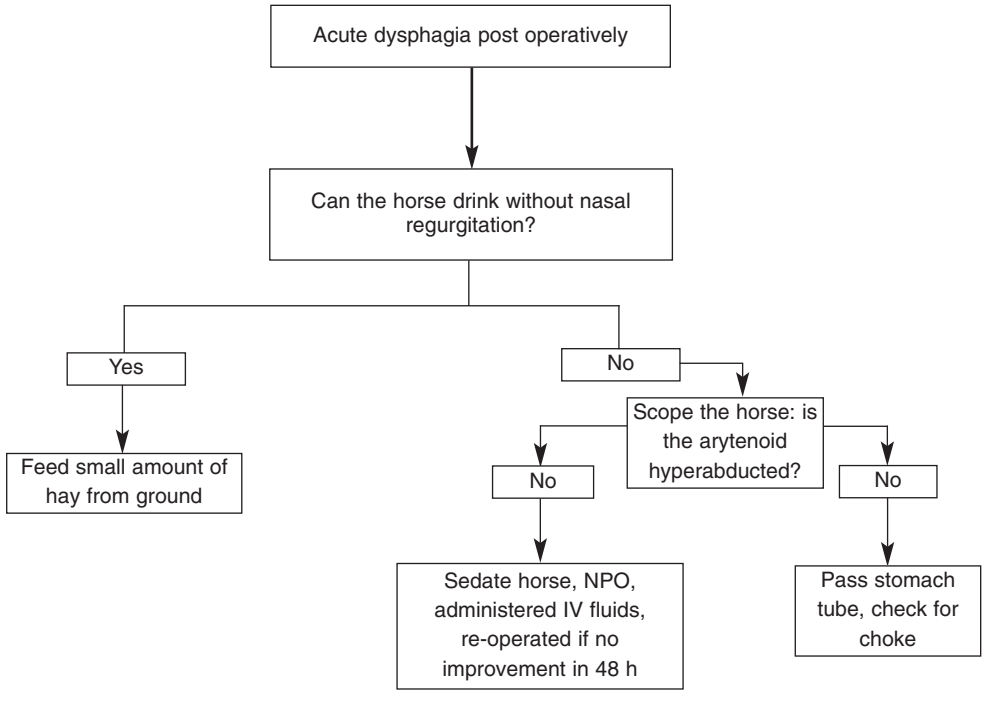

Fig 1: Example of guidelines to be established by the ERLN group.

followed. Distances to be travelled by an international group would be an issue.

It is not a trivial issue to establish criteria that truly assess performance. There have been multiple studies of horses' performance post laryngoplasty, but even objective performance indices are affected by subjective issues, such as track surface, degree of fitness, race availability, etc. It is difficult to evaluate a 2-year-old racehorse that has not raced or has only had one start or to apply objective criteria to non-racehorses. Perhaps only horses that have measurable success parameters (racehorses, grand prix jumping, etc) would be evaluated, but this is a difficult issue that must be resolved. The methods for collecting data must be established. It is perhaps easier with racehorses, but how are complications evaluated, who reports it, and who contacts the owners?

Potential benefits

The establishment of an equine health group that focuses on the equine larynx and could coordinate multicentre trials whose design and sample size would allow accumulation of a large data set so confounding variables are more likely to be circumvented. This group could review and promote the best evidence for effective treatments and management of their complications. The group's study would allow veterinarians and horse owners to make informed decisions in managing their horses based on evidence-based medicine.

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WORKSHOP SUMMARY

CONSENSUS STATEMENTS ON EQUINE RECURRENT LARYNGEAL NEUROPATHY

General

1. The term recurrent laryngeal neuropathy (RLN) is preferable to idiopathic laryngeal hemiplegia (ILH) to describe a disease that can manifest as laryngeal paresis or paralysis.
2. RLN is mainly a disease of the left side of the larynx.
3. Clinically and endoscopically RLN occurs with higher frequency in larger horses than ponies, but the relationship between body size and presence of disease is unresolved.
4. Abnormal inspiratory sounds during exercise, commonly referred to as 'roaring or whistling' are a principal clinical sign of RLN.
5. RLN can cause reduced exercise performance.
6. Other dynamic inspiratory obstructions of the upper airway such as axial deviation of the aryepiglottic folds, arytenoid chondritis, and nasopharyngeal collapse can cause similar clinical signs to RLN.

Anatomy of the recurrent laryngeal nerve

1. The recurrent laryngeal nerve provides the motor innervation of all the equine laryngeal muscles except the cricothyroid, which is innervated by the cranial laryngeal nerve.
2. The cell bodies of the lower motor neurons of the recurrent laryngeal nerves are located in the nucleus ambiguus.
3. The recurrent laryngeal nerve comprises efferent and afferent nerves, principally medium sized, myelinated axons that are not discretely clustered according to their target muscle.
4. The proportion of motor and sensory fibres in the recurrent laryngeal nerves is unknown.
5. The right and left recurrent laryngeal nerves loop around the subclavian artery and aortic arch, respectively.
6. The left recurrent laryngeal nerve is the longest nerve in the horse and is believed to adhere tightly to the fascia of the aorta.

Pathology and pathogenesis

1. Although the disease clinically presents almost exclusively as a consequence of reduced abduction (see Table 1 for definition of terminology) of the left arytenoid cartilage due to dysfunction of the *cricoarytenoideus dorsalis* muscle, the ipsilateral adductor muscles are preferentially denervated in RLN.
2. The lesions of RLN, which include preferential degeneration of large diameter axons, more prominent distally, are indicative of a distal axonopathy.
3. The presence of similar lesions in other long nerves (eg phrenic or peroneal) of affected horses needs more investigation to determine if RLN is a mono – or a polyneuropathy.
4. RLN is not simply a result of nerve length; although the recurrent laryngeal nerve of large horses is about 3 m, other mammals have nerves up to 6 m long (eg giraffe's recurrent laryngeal nerve) without neuronal degeneration.
5. The neuromuscular pathological changes present in RLN indicate ongoing, continual or intermittent injury of the recurrent laryngeal nerve with repeated attempts at regeneration.
6. Horses with other recognised neuropathies, for example stringhalt or Swedish knuckling disease, can undergo complete recovery but it is unclear if this can occur in RLN.

TABLE 1: Definitions of terminology used to describe endoscopic appearance of the larynx

Abduction	Movement of the corniculate process of the arytenoid cartilage away from the midline of the rima glottis
Adduction	Movement of the corniculate process of the arytenoid cartilage toward the midline of the rima glottis
Full abduction	Most of the corniculate process of the arytenoid cartilage lies horizontally (90 degrees to the midline of the rima glottis)
Asymmetry	A difference in position of the right and left corniculate processes relative to the midline of the rima glottis
Asynchrony	Movement of the corniculate processes occurs at different times. This can include twitching, shivering and delayed or biphasic movement of one arytenoid

7. Even though there is histological and histochemical evidence of regeneration of nerves and reinnervation of muscles, clinical and endoscopically evident recovery is uncommon in RLN.
8. It is not known if chromatolyis of the motor neurons of the nucleus ambiguus occurs in RLN. This information is critical to classification of the type of neuropathy present in this disorder.
9. Nerve and muscle lesions potentially consistent with RLN have been observed in fetuses and neonatal foals. This topic needs more investigation because the presence of RLN in early life has major implications in the pathogenesis of the disease.
10. The relationship between endoscopic changes in laryngeal movement and neuromuscular pathology in RLN is incompletely understood.
11. If RLN is a neuropathy that affects only one nerve, it would be a unique mammalian neuropathy.
12. There is a total absence of information on the molecular pathology of RLN.
13. Canine idiopathic recurrent laryngeal neuropathy is similar to equine RLN in that it affects larger breeds but differs in that the canine disease is usually bilateral and affects older dogs clinically.
14. There is evidence to suggest a certain degree of heritability of RLN. However, the mechanisms involved (structural or functional weakness) and mode of inheritance remain unclear.

Other causes of laryngeal paresis or paralysis

1. Dysfunction of the recurrent laryngeal nerve can also be caused by perivascular injections, guttural pouch mycosis, cranial thoracic masses, as a consequence of general anaesthesia (possibly due to hyperextension of the head and neck during surgery), or other iatrogenic causes during cervical surgery.
2. Bilateral laryngeal paresis commonly accompanies lead poisoning and can also occur with liver disease, following general anaesthesia and with organophosphate toxicity and some plant toxicities.
3. The most common cause of right side laryngeal dysfunction is cricopharyngeal laryngeal dysplasia (4th branchial arch defect [4-BAD]).

DIAGNOSIS

General

1. Examination of a horse suspected of having RLN should include the following: a history to determine the animal's exercise performance, nature of possible abnormal exercise related respiratory sounds and when they occur; palpation of the larynx for muscular atrophy, and endoscopic examination.
2. An evaluation of the respiratory tract that depends on endoscopy alone is incomplete.
3. When there is a mismatch between endoscopic and historical and clinical findings, high-speed treadmill endoscopy is indicated to evaluate the function of the larynx during strenuous exercise.

Guidelines for endoscopic examination for the purpose of laryngeal evaluation

1. Evaluation should be performed with as little restraint as needed for safety of horse and personnel.
2. The use of chemical restraint can alter laryngeal function.
3. To achieve consistency in the endoscopic appearance of the larynx, it is recommended that the same nostril be used routinely for introduction of the endoscope.
4. The initial endoscopic examination should be conducted before rather than after exercise. This allows observation of a wider range of laryngeal movements.
5. During the endoscopic examination, laryngeal function should be observed during quiet breathing, swallowing and nasal occlusion.

Clinical grading of RLN

1. Although a number of grading systems are available and validated, they consistently agree on the following:

   a) Inability to achieve full abduction of the affected arytenoid cartilage during examination is likely to be associated with compromised respiratory function during exercise.

   b) Glottic asymmetry at end-exhalation and asynchronous arytenoid movement during inhalation are not cause for concern as long as such horses can attain and maintain full bilateral abduction of their arytenoid cartilages.
2. The grading systems shown in Tables 2 and 3 is recommend by participants in the workshop.
3. There was no consensus on the functional significance of obtaining full arytenoid abduction that is not maintained symmetrically.
4. In the majority of horses, laryngeal function remains constant over time, but in some horses, laryngeal function can deteriorate over a few weeks to years regardless of initial endoscopic appearance.
5. Endoscopic evaluation of the larynx in weanlings for presence of RLN is an unreliable predictor of their laryngeal endoscopic appearance as yearlings.

Other diagnostic procedures

1. Other tests of recurrent laryngeal function include palpatory and endoscopic assessment of the thoraco-laryngeal reflex ('slap test') and measurement of nerve conduction velocity, ie electrolaryngeogram. Currently, none of these procedures is believed to be as effective as endoscopic examination for diagnosing RLN.

TREATMENT OF CLINICAL SIGNS ASSOCIATED WITH RLN

1. Surgical treatments used for relief of the clinical signs of RLN include ventriculectomy with or without vocalcordectomy, prosthetic laryngoplasty, various degrees of arytenoidectomy, and nerve muscle pedicle grafts (reinnervation surgery).
2. These procedures vary in their efficacy in the reduction of abnormal exercise related sounds and the relief of airway obstruction.
3. Most of the following conclusions are based on studies in experimentally induced laryngeal hemiplegia. In the following section 'RLN' signifies the naturally occurring condition.

Abnormal upper respiratory sounds

1. Prosthetic laryngoplasty alone significantly reduces the abnormal upper airway noise in RLN afflicted horses.
2. Following prosthetic laryngoplasty, there is no clear correlation between the degree of residual upper respiratory noise and the magnitude of surgical arytenoid abduction.
3. The degree of noise reduction after laryngoplasty may be more a factor of stability of the arytenoid cartilage rather than the degree of arytenoid abduction.
4. Bilateral ventriculo-cordectomy can reduce abnormal upper airway sounds in horses with experimentally induced left laryngeal hemiplegia.
5. Unilateral ventriculo-cordectomy can reduce abnormal upper airway sounds in RLN afflicted horses.

TABLE 2: Grading system of laryngeal function performed in the standing unsedated horse†

Grade	Description	Sub-grade
I	All arytenoid cartilage movements are synchronous and symmetrical and full arytenoid cartilage abduction can be achieved and maintained	
II	Arytenoid cartilage movements are asynchronous and/or larynx asymmetric at times but full arytenoid cartilage abduction can be achieved and maintained	.1 Transient asynchrony, flutter or delayed movements are seen .2 There is asymmetry of the rima glottidis much of the time due to reduced mobility of the affected arytenoid and vocal fold but there are occasions, typically after swallowing or nasal occlusion when full symmetrical abduction is achieved and maintained
III	Arytenoid cartilage movements are asynchronous and/or asymmetric. Full arytenoid cartilage abduction cannot be achieved and maintained	.1 There is asymmetry of the rima glottidis much of the time due to reduced mobility of the arytenoid and vocal fold but there are occasions, typically after swallowing or nasal occlusion when full symmetrical abduction is achieved but not maintained .2 Obvious arytenoid abductor deficit and arytenoid asymmetry. Full abduction is never achieved .3 Marked but not total arytenoid abductor deficit and asymmetry with little arytenoid movement. Full abduction is never achieved
IV	Complete immobility of the arytenoid cartilage and vocal fold	

† Description generally refers to the left arytenoid cartilage in reference to the right. However this grading system can apply to the right side (ie right Grade III-1)

6. Following ventriculo-cordectomy abnormal upper airway sounds can continue to decrease for up to 90 days after surgery.
7. Bilateral ventriculo-cordectomy is superior to laryngoplasty alone in reduction of abnormal sounds in horses with experimentally induced left laryngeal hemiplegia.

Exercise intolerance

1. Unilateral ventriculectomy is ineffective in restoring normal airflow in horses with experimentally induced left laryngeal hemiplegia.
2. Prosthetic laryngoplasty can restore normal airflow in horses with experimentally induced left laryngeal hemiplegia.
3. Prosthetic laryngoplasty combined with bilateral ventriculectomy can restore normal airflow in sport horses with RLN.
4. In most horses subjected to prosthetic laryngoplasty, the degree of abduction of the arytenoid cartilage will decrease by varying degrees in the post operative period.
5. Evidence from studies of sport horses with RLN and horses with experimentally induced laryngeal hemiplegia indicates that the post operative degrees of residual airway obstruction and the degree of abduction of the arytenoid cartilage are poorly correlated.
6. Maximal abduction of the arytenoid cartilage during prosthetic laryngoplasty may be unnecessary to restore laryngeal airway function and is probably undesirable by causing increased risk of aspiration and coughing.
7. Subtotal arytenoidectomy combined with ipsilateral ventriculectomy is ineffective in restoring airflow in horses with experimentally induced left laryngeal hemiplegia.

TABLE 3: Grading system of laryngeal function‡ as assessed in the horse during exercise†

Laryngeal grade	Definition
A	Full abduction of the arytenoid cartilages during inspiration
B	Partial abduction of the left arytenoid cartilages (between full abduction and the resting position)
C	Abduction less than resting position including collapse into the contralateral half of the rima glottidis during inspiration

‡Description generally refers to the left arytenoid cartilage in reference to the right. However this grading system can apply to the right side (ie right Grade III.1-B)
†Update from Rakestraw, P.C., Hackett, R.P., Ducharme, N.G., Nielan, G.J., Erb, H.N. (1991) A comparison of arytenoid cartilage movement in resting and exercising horses. *Vet. Surg.* **20**, 122-127

8. Bilateral ventriculo-cordectomy and unilateral laser cordectomy modestly improve upper airway function following experimentally induced laryngeal hemiplegia.
9. Partial arytenoidectomy combined with bilateral ventriculectomy improves airflow in horses with experimentally induced left laryngeal hemiplegia.
10. Laryngeal reinnervation surgery can be equally as effective in the restoration of a horse's racing performance as prosthetic laryngoplasty.

EXERCISE INTOLERANCE AND ABNORMAL UPPER RESPIRATORY SOUNDS

1. The implications of the above data are that prosthetic laryngoplasty combined with ventriculo-cordectomy/cordectomy comprise an effective surgical remedy for the treatment of both the abnormal sound and exercise intolerance present in horses with RLN.
2. Reinnervation surgery by use of the nerve muscle pedicle graft is an effective alternative to the above.

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