

Clinical and endoscopic evidence of progression in 52 cases of equine recurrent laryngeal neuropathy (RLN)

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Keywords: horse; equine laryngeal paralysis; recurrent laryngeal neuropathy; progressive laryngeal paralysis

Summary

Examination of the long-term histories and clinical findings was performed in 351 horses suffering from recurrent laryngeal neuropathy (RLN) to examine for possible evidence of progression of this disorder. Fifty-two out of 351 cases (15%) had 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 7 years. In 30 cases, there was both endoscopic (median deterioration of 3 endoscopic grades; range 1–5 grades) and clinical evidence, with 29 (97%) of these horses concurrently developing sudden-onset, abnormal exercise-related respiratory ‘noises’ and 13 (43%) concurrently reporting reduced exercise performance. In the remaining 22 horses there was solely clinical evidence of RLN progression, including the sudden onset of abnormal exercise-related respiratory sounds in 16 (73%) and the worsening of such sounds in 6 (23%), associated with reduced exercise performance in 13 (59%) of these 22 cases. Endoscopically, 13 (59%) of the latter 22 cases had marked (total or almost total) RLN that did not appear compatible with their previous exercise-performance histories. This evidence of progression of RLN may be of particular significance in disputes concerning horses that are apparently normal at pre-purchase examination but are later shown to have RLN and also in the surgical treatment of less severe cases of RLN.

Introduction

Despite the large body of literature regarding many aspects of recurrent laryngeal neuropathy (RLN) as reviewed by Robertson (1991), Spiers *et al.* (1992), Lane (1993) and Anderson *et al.* (1997), few long-term studies of horses suffering from this disorder have been reported, and it is unclear whether RLN is a congenital or an acquired disease (Lane 1993). A commonly cited long-term study by Baker (1982) found no evidence of clinical or endoscopic progression of this disease. In contrast, the earlier literature has many references to possible cases of progressive RLN (Fleming 1889; Hobday 1935). Recent endoscopic studies of Thoroughbred (TB) yearlings (Embertson 1997) and young racehorses (Anderson *et al.* 1997) showed variation in laryngeal function at repeat examinations. Additionally, Strand *et al.* (2000) presented clinical and race-performance evidence suggestive of

progression of RLN in racehorses, especially over the 6 months prior to referral. The aim of this study is to present clinical and endoscopic evidence that RLN can be a progressive disorder.

Materials and methods

Details of 375 horses suffering from laryngeal paralysis, including 351 cases of RLN and 24 cases of non-RLN laryngeal paralysis and of their clinical and endoscopic examinations, have been presented by Dixon *et al.* (2001). Most of the 24 non-RLN cases of laryngeal paralysis (e.g. those secondary to guttural pouch mycosis or liver failure) had acute onset or rapidly progressive laryngeal paralysis, but such cases are excluded from this report. In this study, all laryngeal movement dysfunction (unilateral or bilateral) of unknown aetiology was classified as RLN. All endoscopic evaluations were performed without sedation. Endoscopic grading of laryngeal paralysis was assessed at rest, prior to and following nasal occlusion and transendoscopic nasopharyngeal flushing with water. A previously described (Dixon *et al.* 2000) 6-grade, endoscopic system for assessing laryngeal function was utilised (Table 1).

Full clinical histories, in particular details of possible abnormal exercise-related respiratory sounds (‘noises’) or of reduced exercise performance, were documented. A complete physical examination, including a specific examination for intercurrent respiratory disease, was performed in all cases and included laryngeal muscle palpation, and resting upper and lower respiratory tract endoscopy. Where possible, horses were clinically evaluated during ridden or lungeing exercise, in particular for the presence of ‘noises’.

Results

Of the 351 RLN cases, 52 horses (15%) had evidence of progression of the degree of RLN. This included clinical evidence, especially of horses with a history of working without making abnormal exercise-related respiratory ‘noises’, later developing such ‘noises’ or of horses making slight ‘noises’ later developing much louder ‘noises’ while performing the same level of work. Some of these cases also had a concurrent history of decreasing exercise tolerance. Such findings indicated the clinical development of, or deterioration of an upper respiratory tract obstruction. The median age at the onset of this progression (in all 52 cases) was 7 years (range 2–15 years).

Additionally, in 30 of these 52 cases (Table 2; in chronological order of presentation), such clinical evidence was supported by repeated endoscopic findings over the course of this clinical

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TABLE 1: Endoscopic criteria for grading equine laryngeal paralysis

<i>Grade 0</i>	Perfect synchrony and symmetry of the larynx with full bilateral abduction achieved and maintained
<i>Grade 1</i>	Shivering and/or asynchrony of the arytenoid (usually left-sided) present, but full bilateral abduction achieved and maintained
<i>Grade 2</i>	Slight lack of abduction, especially during attempted full abduction (slight asymmetry of larynx present if lesion unilateral)
<i>Grade 3</i>	Moderate lack of abduction, especially during attempted full abduction (moderate asymmetry of larynx present if lesion unilateral)
<i>Grade 4</i>	Marked (but not total) lack of abduction at rest and attempted full abduction (marked asymmetry of larynx present if lesion unilateral)
<i>Grade 5</i>	Total lack of abduction at all times (marked asymmetry of larynx always present if lesion unilateral)

deterioration, with 23 (77%) of the initial and all of the later 30 endoscopic examinations performed by the senior author. The median age at the initial clinical and endoscopic examination of these 30 cases was 6 years (range 2–15 years) and they included 16 Thoroughbreds (TBs), 9 TB crosses, 2 Warmbloods, 2 draught horses and one pony (Table 2).

The initial endoscopic examinations were performed mainly for reasons other than suspected laryngeal dysfunction. Pulmonary disorders are common in this age group and these types of horses, respiratory tract endoscopy was, therefore, performed during the investigation of all such cases at this clinic (Dixon *et al.* 1995) with intercurrent laryngeal findings being contemporaneously documented. Normal laryngeal endoscopic findings i.e. *Grade 0* or *1* RLN, were documented in 17 (56%) of these 30 cases at their initial examination. In 4 of these cases, 2 examiners did not agree on the endoscopic grading (always differing by a single grade) and the mean of both grades are presented for these 6 cases (Table 2).

The median interval between the first and last endoscopic examination was 12 months (range 1.5–48, mean 14.6 months). The median worsening in endoscopic laryngeal grades between the first and last endoscopic examination was 3 grades (range 1–5 grades). Twenty-nine of these 30 horses (97%) were reported to have concurrently developed ‘noises’ and 13 (43%) were reported to have shown concurrent loss of exercise performance.

Particularly clear-cut examples included a 6 year-old, 16.2 hands high (hh/1.67 m), National Hunt TB racehorse (*Case 26*) that was initially examined because of intermittent ‘noises’ heard during galloping which were suspected to be caused by dorsal displacement of the soft palate. Although a mild degree of palpable left laryngeal muscle atrophy was then present, endoscopy at rest and during treadmill exercise (latterly 12.8 m/s at 6° until the horse was fatigued) revealed no detectable laryngeal, nor any other upper respiratory tract abnormality. A diagnosis of intermittent, dynamic nasopharyngeal obstruction was made, and conservative treatment (increased levels of training and use of tongue-tie) was advised. Nine months later this horse began to make continuous and louder ‘noises’ even during trotting. Resting endoscopy now showed almost total (*Grade 4*) left sided RLN. More marked left laryngeal muscle atrophy was now palpable.

A similar example was a 16.2 hh, National Hunt TB racehorse (*Case 30*) which had won races each of the 3 previous years. At age 8 years, the presence of ‘noises’ and associated poor racing

performance developed. Endoscopic examination at this stage showed the larynx to be normal (*Grade 0* RLN) and dorsal displacement of the soft palate was suspected. Following further progression of the above signs over the following year, repeat endoscopy showed total (*Grade 5* RLN) laryngeal paralysis.

The evidence in the remaining 22 cases, which included 12 TBs, 7 TB crosses, 2 Warmbloods and a Clydesdale, median age 7 years (range 3–11 years) (Table 3) is solely clinical evidence. In 16 (73%) horses it included the sudden development of ‘noises’ and, in 6 (27%) cases the worsening of existing ‘noises’, over a median period of 12 months (range 0.5–48, mean 17.1 months). The onset or worsening of these ‘noises’ was reported to be associated with reduced exercise performance in 13 (59%) of these cases. Endoscopic examinations following referral showed a marked degree of RLN (i.e. *Grade 4* or *5*) in 13 of these 22 cases (59%) that appeared to be incompatible with their previous exercise performance history (Table 3). Endoscopically, the other 9 cases (41%) had lesser degrees of RLN (*Grade 2–3.5* RLN) (Table 3). A marked example was a 6-year-old National Hunt racehorse (*Case 5*) that showed progressively increasing ‘noises’ and marked loss of exercise tolerance over 5 months. Endoscopy then showed *Grade 5* (total RLN), which was incompatible with its previous excellent racing history and absence of ‘noises’.

There was no clear-cut evidence of progression of the degree of RLN in the remaining 299 cases of RLN (Dixon *et al.* 2000). However, these included 19 cases where RLN was detected at prepurchase examination, a situation where a reliable history was unlikely to be available. In 35 other cases referred for poor performance examination, RLN was not suspected and, therefore, evidence of progression is also unavailable. In 112 cases, abnormal ‘noises’ were detected soon after the horses first began fast work and these horses were then immediately referred for diagnosis and treatment. In the remaining cases, there was absence of a reliable, long-term clinical history, or of a history of animals suffering from the same level of clinical signs, with both of these situations precluding detection of possible progression of RLN. However, it remains possible that RLN was progressive in some of these 299 cases.

Discussion

This study presents historical, clinical and endoscopic evidence that laryngeal abductory function can deteriorate, sometimes slowly and progressively, other times more acutely, in about 15% of RLN affected horses. Thirty of these 52 cases had endoscopic and clinical evidence of deterioration in laryngeal abductory function. It is accepted that the endoscopic interpretation of laryngeal function is subject to inter- and intra-observer variation at repeat examinations (Ducharme *et al.* 1991; Hackett *et al.* 1991). However, it is difficult to attribute all of the deterioration in endoscopic findings observed in the current cases (median worsening of 3 endoscopic RLN grades) to such factors, as clearly defined endoscopic criteria had been utilised in this study. Additionally, as a single observer performed 53 of the 60 endoscopic examinations, minimal inter-observer variation should have occurred.

In the remaining 22 cases this evidence was solely clinical evidence, especially of the development or worsening of abnormal respiratory noise production during exercise and of decreased exercise tolerance. The interpretation of such clinical findings can be subjective, especially that of exercise performance, where the

TABLE 2: Details of 30 horses with endoscopic and clinical evidence of progression of recurrent laryngeal neuropathy

Case	Breed	Height (hh)	Gender	Work	Age at first visit (years)	RLN grade changes (left/right)	Time between endoscopies	Clinical features
1	TB	16.3 (169 cm)	G	NH Racing	10	2–3.5	4 months	No ARN previous season, ARN and fading progressively developed this season
2	TB	16.1 (165 cm)	F	NH Racing	5	0–2	9 months	No ARN last season, progressive ARN this season, full sister had RLN
3	TB	16.2 (167 cm)	G	NH Racing	6	(2/0)–(5/2) Bilateral	17 months	ARN and poor work -(Gr 2)-HD. Later poorer work, now bilateral RLN, (Gr 5/2)
4	TBx	18 (181 cm)	G	Hunting	5	3–4	7 months	Progressive ARN, half brother also had RLN
5	Pony	15 (152 cm)	F	Hacking/RC	15	0–2	1 year	Developed loud ARN when aged, occasionally stridor at rest – bilateral RLN?
6	TBx	17 (171 cm)	G	Eventing	7	1–2	2 years	Progressive worsening ARN over 2 seasons
7	TBx	16.3 (169 cm)	G	Eventing	7	1–4	20 months	Marked clinical and endoscopic progression between 2 visits to the hospital
8	TBx	16.3 (169 cm)	G	Eventing	6	0–3	5 months	As 4 y.o. -no ARN, at 6 yrs –ARN (Gr 2) HD and VC, 5 m later ARN worsened (Gr 3 RLN)
9	TB	16.1 (165 cm)	G	NH Racing /Hunting	8	2–3	7 months	Won races previous year, ARN developed May 89 (Gr 2), worse ARN Dec 89 (Gr 3)
10	TB	17.3 (179 cm)	F	Eventing	8	2–4	4 months	ARN only (Gr 2) July 89, ARN and poor exercise performance (Gr 4) Nov 89
11	TB	16.1 (165 cm)	F	NH Racing	6	0–2	17 months	DDSP Feb 90, (Gr 0) , July 91 (Gr 2) RLN
12	TB	16.2 (167 cm)	G	NH Racing	7	2–5	9 months	From mild to total RLN over single season
13	TBx	16 (162 cm)	G	Eventing	7	1–4	4 years	92 ARN (Gr 1), 93 progressed to (Gr 3), 96 progressed to (Gr 4)
14	TBx	16.3 (169 cm)	G	Hunting	6	3–5	4 months	Sudden onset of ARN, rapid RLN progression
15	TB	16.2 (167 cm)	G	Hunting	5	2–5	2 years	(Gr 2) RLN had HD and VC; 2 yrs later total RLN
16	WB	17.2 (176 cm)	F	Showing	5	2–4	1 year	Progressive RLN suspected by referring vet
17	TB	16.2 (167 cm)	G	NH Racing	6	0–5	2 year	Poor performance - endoscopy by referring vet. 92, worsening of signs and referral 94
18	TBx	17.2 (176 cm)	G	Hacking/RC	4	0–4	21 months	First visit (Gr 0) for pulmonary disease
19	TBx	16.3 (169 cm)	G	Hacking/RC	3	0–3	6 weeks	(Gr 0) at pre-purchase exam. 6 wks later ‘whistling’ developed and RLN diagnosed
20	TBx	16.3 (169 cm)	G	Hunting	8	1.5–5	15 months	HD in 1996, Initial endoscopy by referring surgeon - increasing ARN since
21	TB	16.3 (169 cm)	G	Hacking/ RC	2	0–5	7 months	Multiple endoscopies for choke showed progression of RLN. Full brother had RLN
22	TB	16.1 (165 cm)	G	NH Racing	5	2–4.5	10 months	ARN and mild paralysis Sept. 97, worsening signs and (Gr 4) July 98
23	WB	16.3 (169 cm)	G	Eventing	7	0–4.5	21 months	Development of ARN and epistaxis in fast work
24	Clyd	17.3 (179 cm)	G	Show Driving	9	3–5	3 years	Deteriorating performance, increase in ARN and in severity of paralysis found
25	TB	18 (181 cm)	G	NH Racing	11	1.5–4	10 months	Worsening of ARN last season. Initial endoscopy by referring vet
26	TB	16.2 (167 cm)	G	NH Racing	6	0–4	9 months	Intermittent ARN (Gr 0) on treadmill, worsening ARN
27	TB	16.2 (167 cm)	G	Flat racing	3	0–4.5	1 year	Raced as 2 yr old, next season - developed ARN and poor performance
28	TB	16.3 (169 cm)	G	NH Racing	7	0–3	13 months	Won races, passed pre-purchase exam. Later worsening ARN and poor work
29	Ir Dr	17.1 (174 cm)	G	Show Jumping	6	0–5	2 years	Slight whistling for 2 yrs has progressed to severe respiratory distress
30	TB	16.2 (167 cm)	G	NH Racing	8	0–5	1 year	Won for 3y, developed ARN and poor work, DDSP initially? (Gr 0) later (Gr 5)

TB: Thoroughbred; TBx: Thoroughbred cross; WB: Warmblood; Clyd: Clydesdale; Ir Dr: Irish Draught; RLN: recurrent laryngeal neuropathy; ARN: abnormal (exercise-related) respiratory ‘noises’; HD: Hobday operation (ventriculectomy); VC: vocalcordectomy; tb: tieback operation (laryngoplasty); DDSP: dorsal displacement of the soft palate; RC: Riding Club; NH: National Hunt; G: Gelding; F: Female; m: Month.

TABLE 3: Clinical and endoscopic details of 22 cases with clinical evidence of progression of recurrent laryngeal neuropathy (RLN)

Case	Period over which progression suspected	Breed	Gender	Height (hh)	Work	Age at first visit	RLN grade	Clinical features
1	Weeks	TB	G	16.1 (165 cm)	Show Jumping	8	2	Sudden onset of ARN
2	Weeks	TBx	G	17.2 (176 cm)	RC	10	4	Sudden onset of ARN
3	6 months	TBx	G	16.1 (165 cm)	Eventing	4	5	ARN development- had HD. 6 m later return of ARN
4	Weeks	TBx	G	17 (171 cm)	Hunting	5	4	Sudden onset of ARN
5	5 months	TB	G	16 (162 cm)	NH Racing	6	5	Progressive worsening of ARN and work
6	2 years	TB	G	17.2 (176 cm)	NH Racing	9	2	1990 won races - gradual onset and progression of ARN and poor work since
7	4 months	TB	G	16.1 (165 cm)	NH Racing	4	4	No noise when worked as 3 yo, onset of ARN at beginning of next season
8	Weeks	TB	G	16 (162 cm)	NH Racing	4	2	Sudden onset of ARN in 1st season
9	2.5 months	TBx	F	15.3 (160 cm)	Hunting/Eventing	8	2	Passed prepurchase exams. 10 w later developed ARN
10	1 year	TB	G	16 (162 cm)	Eventing	9	2	ARN worsened further after our exam - became a showjumper
11	4 years	TBx	F	17 (171 cm)	Hunting/Eventing	7	4	4 yrs off work (3 foals), when came back in work, ARN and poor work present
12	1 year	WB	G	17.1 (174 cm)	Dressage	6	4	1 yr ago passed prepurchase vetting – now ARN even at canter
13	2 years	TB	G	16 (162 cm)	Eventing	11	5	Purchased 2 years earlier (had HD) worsening ARN since
14	5 months	TBx	F	16.3 (169 cm)	Eventing	4	5	No ARN initially, then ARN in gallop, later worsened to ARN at trot
15	22 months	TBx	F	16.1 (165 cm)	Hunting	8	4.5	2 m after purchase ARN developed - has become worse in last 20 m
16	2 years	WB	G	16.3 (169 cm)	Hunting	7	5	Bought 4 y ago, developed ARN 2 y ago worsening ARN since
17	9 months	Clyd	G	18.2 (186 cm)	Driving/showing	4	3.5	Progressive development of ARN
18	1 year+	TB	M	16.2 (167 cm)	Flat Racing	3	4.5	History of ARN, worsened over last year
19	46 months	TB	G	16.3 (169 cm)	NH Racing	10	5	Progressive worsening of ARN and work
20	Few weeks	TB	G	16.2 (167 cm)	NH Racing	7	3	Sudden onset and progressive loss of exercise ability
21	2–3 months	TB	G	16.1 (165 cm)	NH Racing	7	3	Concurrent laryngeal- cricopharyngeal dysplasia
22	3 months	TB	G	16.2 (167 cm)	NH Racing	8	2	Raced successfully previous season, recent onset of ARN

TB: Thoroughbred; TBx: Thoroughbred cross; WB: Warmblood; Clyd: Clydesdale; RLN: recurrent laryngeal neuropathy; ARN: abnormal (exercise-related) respiratory 'noises'; HD: Hobday operation (ventriculectomy); RC: Riding Club; NH: National Hunt; G: Gelding; F: Female; M: Male.

expectations of many owners appears to exceed the ability of their horses. Consequently, it could be argued that in these 22 horses the presented evidence simply reflects the subjectivity of these clinical findings. However, the onset of loud 'noises' in 16 of these 22 horses (73%) that were reported to not make such 'noises'

previously, the concurrent reported loss of exercise tolerance in 13 (59%) of these 22 cases and the endoscopic evidence of marked RLN changes (i.e. *Grade 4* or *5* RLN) in 13 (59%) of these 22 cases when they were eventually examined endoscopically, is strong evidence that the RLN was in fact progressive in these 22

horses. In some cases (e.g. *Cases 5 and 14*), the presence latterly of total, left-sided laryngeal paralysis along with the presence of loud 'noises' and poor exercise tolerance was simply incompatible with their previous histories of successfully performing strenuous work without making 'noises' unless there was a deterioration in laryngeal function in the interim.

The current findings disagree with the results of a long-term study by Baker (1982) who endoscopically examined 168 TB National Hunt racehorses, most over 5 years old, at annual intervals on at least 3 occasions. At the initial examinations, 49% of horses had asynchronous laryngeal movements (equivalent to *Grade 1* RLN in this study) with the remaining 51% having synchronous movements (*Grade 0* RLN in this study). At repeat examinations (1 or 2 years later), 93% of horses continued to have a similar endoscopic laryngeal appearance, 6% changed from normal to asynchronous movements, and 1% from asynchronous to normal movements. No progression from laryngeal asynchrony to asymmetry was observed in any horse. However, Baker and colleagues had previously noted that while most horses developed clinical RLN at an early age, cases with late-onset clinical signs had been reported to them (Duncan *et al.* 1977).

Goulden and Anderson (1981) noted that 15 of 55 racehorses with RLN had a gradual onset of clinical signs over periods of weeks to months. The other 40 cases had sudden onset of clinical RLN, with some cases reported first to show signs when they returned to racing as 3-year-olds, having being asymptomatic as 2-year-olds. Hillidge (1986) found postexercise endoscopic evidence of laryngeal asymmetry in 14 of 169 horses on a TB farm (all horses sedated with xylazine for endoscopy). Surprisingly, none of 32 weanlings in this farm were affected, suggesting that the endoscopic appearance of this disease was age-related.

Evidence of long-term changes in laryngeal function was recorded by Anderson *et al.* (1997) who performed (resting) endoscopy on 109 horses, (TBs and Standardbreds, most under 2 years old when initially examined) on 2 occasions, 16 months apart. At the initial endoscopy (using a 4 grade system similar to that of Hackett *et al.* (1991)), they found 55 horses to have *Grade 1* RLN (synchronous larynxes), 35 with *Grade 2* (laryngeal asynchrony but full abduction) and 19 with *Grade 3* RLN (laryngeal asymmetry). At the repeat examinations, the grade of RLN was found to be similar in 43% of horses, improved in 29% and worsened in 28%. When endoscopic changes occurred, they were of a single RLN grade in 71% of cases and of 2 RLN grades in 29%. While some of the above recorded variation could be attributed simply to the poor repeatability of endoscopic grading, it is less likely that the changes of 2 RLN grades observed in some cases could be attributed to this factor. Anderson *et al.* (1997) suggested that the observed improvement in laryngeal function in 29% of horses could be attributed to regeneration of nerve fibres and compensatory hypertrophy of unaffected laryngeal muscle fibres.

Likewise, Embertson (1997) reported variation in the endoscopic appearance of larynxes of TB yearlings when re-examined, some days or even hours later. Embertson also stated that total laryngeal paralysis is often preceded by a progressive deterioration in laryngeal function. Similarly, Cook (1965) noted that about 20% of RLN cases develop in horses over 6 years of age. Robertson (1991) also noted that some RLN cases could be progressive. In Britain, other equine clinicians have also recognised progressive cases of RLN (J.G. Lane, personal communication). However, in view of the apparent substantial (15%) incidence of progressive cases of RLN found in this study,

it is surprising that progressive cases have previously not been more widely and factually documented.

The older literature also has possible examples of progression of RLN. For example, Fleming (1889) cites examples from the detailed British army veterinary records of horses sent to South Africa, including that "*Mr Lambert himself took out a fine charger, and within 3 months after landing was a roarer*". In his treatise on equine laryngeal paralysis, Moeller (1888) of the Berlin Veterinary School also recorded that 'roaring' often progressively worsened in horses between age 3 and 6 years. Similarly, Fleming (1889) noted that in Britain most horses develop 'roaring' between age 2 and 7 years and that "*not a season passes but some promising animal, having accomplished wonders at 2 years of age, is good for nothing, or for nothing but short spins, at 3 because it has begun to make a noise*", a finding also reported by Hobday (1935), Goulden and Anderson (1981) and also noted in this study (*Case 27*, Table 2, *Case 18*, Table 3). Fleming (1889) also cited some less common cases of horses where 'roaring' had improved spontaneously. Because endoscopy was unavailable for these earlier studies, it is possible that upper respiratory tract abnormalities other than RLN (e.g. epiglottic entrapment) were in fact present in some of these cases. However the pathological studies of Fleming (1889) showed consistently that only abnormalities present in clinical cases of 'roaring' (or *laryngismus paralyticus* as then also termed) were left sided recurrent laryngeal nerve and laryngeal muscle changes.

Although the 52 cases in this study had apparent deterioration in laryngeal function, no cases with improvement in laryngeal function were detected in the current study, in contrast to the findings of Fleming (1889) and Anderson *et al.* (1997).

The laryngeal muscles of RLN affected horses are characterised by pathological changes indicative of repeated denervation and re-innervation (Gunn 1973; Duncan *et al.* 1974; Cahill and Goulden 1986), which is suggestive of a progressive lesion. Anderson (1984) showed that pathological changes indicative of denervation and re-innervation in the laryngeal muscles of TBs increased rapidly at between ages 1 and 2 years. The major neural lesion in RLN is also suggestive of a chronic progressive disorder (Griffiths 1991). The latter proposed that some cases with sub-clinical RLN lesions would later develop clinical disease. Bearing in mind the pathological lesions present, it is perhaps not surprising that the clinical and endoscopic degrees of laryngeal dysfunction could also progress in this disorder. Absolute confirmation of progression in RLN would require serial histological evaluations of laryngeal muscles or nerves, but it is difficult to envisage how such pathological studies could be performed.

The findings of this study may have particular implications concerning disputes over the presence of RLN at pre- and post-purchase examinations. In 3 of the present cases (*Case 19*, Table 2: *Cases 9, 15*, Table 3) the recorded deterioration in laryngeal function occurred within 10 weeks of purchase, and in 2 cases, was the source of conflict between vendor and purchaser. In the third case (*Case 9*, Table 3), the same clinician who passed the horse at prepurchase examination diagnosed RLN 10 weeks later, following the development of clinical signs in the interim. In the light of the above evidence, it should be considered that differences in clinical and endoscopic findings between veterinarians who perform pre- and postpurchase examinations, perhaps just weeks apart, may in some cases, simply reflect clinical progression of RLN, rather than inter-assessor variation,

or as has even been alleged in some such cases, to negligence by the veterinarian who performed the prepurchase examination

The current findings may also have implications concerning the treatment of RLN cases. For example, less severe cases of RLN, especially in nonracehorses, are sometimes treated by ventriculectomy and/or vocal cordectomy. However, if such cases appear to suffer from progressive RLN, it may be more prudent to treat them by laryngoplasty initially, rather than waiting for further possible deterioration in laryngeal function, and then having to perform a 2nd general anaesthesia and surgery.

In conclusion this survey presents strong clinical and endoscopic evidence that RLN is a clinically progressive disorder in about 15% of affected horses.

Acknowledgements

Our thanks to our colleagues in practice for referring these cases, to Christina Gkioni and Lucy Smith for help with data retrieval and analyses and to I. G. Mayhew and N. Chandler for reviewing this paper. B. McGorum, D. Railton, C. Hawe and K. Pickles were Horserace Betting Levy Board Scholars during the period of this study.

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