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Short Communications

A preliminary report on the possible genetic basis of laryngeal hemiplegia

P. A. PONCET and S. MONTAVON
Swiss Federal Stud, CH-1580 Avenches, Switzerland

C. GAILLARD
Institute of Animal Breeding, University of Berne, CH-3012 Berne, Switzerland

F. BARRELET, R. STRAUB and H. GERBER
Large Animal Clinic, University of Berne, Länggass-Strasse 124, CH-3012 Berne, Switzerland

Introduction

LARYNGEAL hemiplegia has been suspected of having a genetic basis for a long time (Quinlan and Morton 1957; Cook 1970; Mason 1973; Cook 1988) but definite proof of its hereditary nature is lacking. However, Galizzi Vecchiotti and Galanti (1985) have shown that members of eight affected Thoroughbred families suffered in significantly larger numbers from hemiplegia (29 per cent) than did comparable offspring of unaffected sires (3.4 per cent).

A review of the literature on the pathogenesis of equine laryngeal hemiplegia was presented by Cahill and Goulden (1987).

In this report, we present the results of a study involving 47 offspring of an affected stallion compared with a similar number of controls.

Materials and methods

The sire concerned was a French-Riding-Horse stallion used for show jumping, born in 1977 and measuring 165 cm at the withers. he was a son of the Thoroughbred 'Solvedo' (Royal Palace - Persian Gulf).

The horse had a history of progressively worsening inspiratory difficulties related to laryngeal hemiplegia. It was destroyed in 1987 and the left recurrent nerve was examined histologically. Two male offspring developed an inspiratory noise caused by laryngeal hemiplegia which instigated the examination of a larger number of offspring.

Of a total of 59 progeny older than two years, 47 were examined, together with 50 control horses. These offspring descended from 20 different dam's sires and 24 granddam's sires. The control horses represented 25 different sires, excluding 'Solvedo', 24 dam's sires and 29 granddam's sires. Only three stallions are represented on the maternal side of one horse each in both groups.

The age of the offspring of the affected stallion ranged from two to five years and that of the controls from three to 20 years.

Table 1 shows that the proportions of entire horses and geldings are different in the two groups.

All 97 animals were examined endoscopically; 42 offspring of the stallion and all 50 control horses were ridden or lunged for several minutes, five were galloped on pasture and the noise phenomena produced were recorded by two veterinarians.

TABLE 1: Number of progeny of the affected sire and number of controls according to gender

Sex	Number		
	Offspring of affected stallion	Controls	Total
Male (entire)	5	20	25
Gelding	16	3	19
Female	26	27	53
Total	47	50	97

The observations were grouped as follows:

Group 1) normal endoscopic findings, symmetry and symmetric motility of larynx; absence of inspiratory noise at work.

Group 2) left sided asymmetry of the larynx but preserved motility; no inspiratory noise.

Group 3) left sided asymmetry and reduced motility of the larynx. Inspiratory noise.

Group 3 was classified as suffering from laryngeal hemiplegia, **Group 2** was considered suspect. Photographs were taken of all suspect larynges.

Potential genetic markers for laryngeal hemiplegia were sought in the equine major histocompatibility complex and all horses were typed for their Equine Leucocyte Antigen haplotypes.

The height at withers was measured in all horses.

Chi square and t-statistics were used for testing significances.

Results

At post mortem examination, the isolated larynx of the stallion showed marked left sided asymmetry and abductor muscle atrophy. The left recurrent nerve was damaged extensively, especially in its most distal region (close to larynx). Histologically a diffuse loss of fibres with demyelination and signs of remyelination in the remaining fibres as well as profuse Renaut bodies were observed. Some fibres showed Wallerian degeneration.

TABLE 2: Results of clinical investigations

Diagnostic group	Number	
	Offspring of affected stallion	Controls
Group 1 (sound)	25	45
2 (suspect)	11	4
3 (affected)	11	1
males + geldings		
affected and suspect	12	2
sound	9	21
females		
affected and suspect	10	3
sound	16	24

The difference between the offspring of the affected sire and the controls are highly significant ($P<0.001$) if **Groups 2** and **3** are added. When only confirmed cases are considered they are significant (**Group 3**; $P<0.01$). The sexes do not differ significantly from each other ($P>0.4$)

The results of the endoscopic and clinical examinations are given in Table 2. The proportion of affected and suspected horses (47 per cent) was significantly larger ($P<0.001$) in the affected stallion's group than in the controls (10 per cent). The difference is still significant when the suspects are discarded from statistical analysis ($P<0.01$). No significant differences were found between sexes.

The average height at the withers of the affected stallion's offspring (163.9 ± 4.0 cm) was not statistically different from the mean height of the controls (166.6 ± 3.0 cm). The comparison of affected (166.36 ± 4.08 cm) with sound offspring (162.6 ± 4.0 cm) of the stallion concerned, yields a significant difference ($P<0.01$); ie the affected horses were taller than the sound ones. The age structure of both groups is similar.

Ten horses showed signs of follicular pharyngitis. This was felt to be insignificant because five cases were offspring, and five controls. Two horses with follicular pharyngitis also showed typical signs of laryngeal hemiplegia. No association between the occurrence of laryngeal hemiplegia and the ELA-haplotypes was observed.

Discussion

The stallion concerned was suffering from laryngeal hemiplegia. Neuropathological examination of the left recurrent nerve showed changes similar to those described in the literature (Cole 1946). It is thought that the changes represent a primary axonopathy.

The significant difference between this stallion's offspring and the controls seems to confirm the suspicion of a genetic basis to laryngeal hemiplegia (see Quinlan and Morton 1957) and supports the results of Galizzi *et al* (1985). It is unusual, however, to find such a high percentage of affected offspring by a stallion with laryngeal hemiplegia. Kuhn (cited from Manninger and Mócsy 1959) found no increased frequency of laryngeal hemiplegia in 200 offspring by one affected stallion. Quinlan and Morton (1957) investigated 196 progeny of suspected stallions and found a percentage of 10.7 per cent compared with 4.8 per cent in 292 controls ($P<0.05$). Galizzi *et al* (1985) revealed a percentage of 29 per cent in eight affected families and suggested the hypothesis of a dominant gene, which our results support.

These different findings mean that laryngeal hemiplegia may be a phaenocopy as well as being related to a genetic defect. This may be why contradictory results have been reported.

The genetic factor responsible for the predisposition to laryngeal hemiplegia is neither marked nor represented by an equine leucocyte antigen.

Although the heights of the affected stallion's offspring and the control horses did not differ statistically, the mean size of the sound offspring was significantly lower than that of the affected progeny. An association of laryngeal hemiplegia and size does therefore seem likely (Cook 1970). Feeding regimes were identical in both groups.

It seems improbable that the difference in the age distribution of the two populations seriously impairs the validity of our results. The average age of the affected sire's group was lower than that of the controls. It might be argued, therefore, that the controls represent a positive selection. As far as we could ascertain this does not seem to be the case.

The question of whether horses with hemiplegia laryngitis should be admitted to horse-improvement schemes was discussed in the 19th century (Quinlan and Morton 1957; Mason 1973). We feel that the evidence presented here justifies at least the exclusion of affected stallions from such schemes. Although our stallion seems extreme compared to those investigated by Quinlan and Morton (1957) it seems inadmissible that a subsidised stallion should be allowed to produce over 40 per cent of unsound horses.

The results of this study have led to the compulsory endoscopic examination of entire horses presented for approval as subsidised stallions for improvement schemes in Switzerland, even though laryngeal hemiplegia may be caused by non-genetic factors in some cases.

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