Chapter 3 Pharyngeal, laryngeal and tracheal disorders in South African Thoroughbred racehorses: prevalence and relationship with performance

3.1 ABSTRACT

We sought to determine the prevalence of pharyngeal, laryngeal and tracheal disorders in Thoroughbred racehorses in South Africa and to determine their relationship with racing performance. A prospective cross-sectional study was conducted over 3.5 months on 1,005 Thoroughbred racehorses. After racing, videoendoscopic examination of the pharynx, larynx and trachea was performed on unsedated, pre-enrolled racehorses. The presence and characteristics of respiratory tract disorders were evaluated and recorded onto digital video disc. Thereafter, the prevalence of the observed disorders was compared to race career performance records.

Over a 3.5 month period, a single videoendoscopic examination was performed mean ± standard deviation (SD) 24 ± 12.3 min after racing on 1,005 racehorses. The prevalence of arytenoid cartilage asymmetry (grade 2 and 3) in racehorses examined was 2.22% (95% confidence interval [CI]: 1.29 to 3.14%). The median number of lifetime starts was higher in affected horses, with this being significantly so in racehorses with grade 2

arytenoid cartilage asymmetry and idiopathic laryngeal hemiplegia (ILH) ($P < 0.05$). No difference could be shown in median lifetime places between racehorses with grade 1 vs. 2 and 3 arytenoid cartilage asymmetry ($P > 0.05$), but racehorses with grade 2 arytenoid cartilage asymmetry had more lifetime wins ($P = 0.02$) and racehorses with ILH had a greater number of lifetime wins ($P = 0.03$) and lifetime places ($P = 0.01$). Both racehorses with grade 2 arytenoid cartilage asymmetry and ILH had higher lifetime stake earnings ($P < 0.05$). The apparent superior racing performance seen in racehorses affected by various grades of arytenoid cartilage asymmetry was most likely caused by more lifetime starts, which was acting as a confounder in these groups.

Compared to racehorses with grade 1 pharyngeal lymphoid hyperplasia (PLH), racehorses with grade 3 PLH were younger ($P < 0.01$), while racehorses with grade 2 and 3 PLH had fewer lifetime starts, lifetime wins, lifetime places and less lifetime stake earnings ($P < 0.01$). Racehorses with grade 2 PLH had more lifetime places ($P < 0.01$) and lifetime stake earnings ($P = 0.03$) than horses with grade 3 PLH. The apparent impaired racing performance was most likely caused by less lifetime starts, which was acting as a confounder in these groups.

Epiglottic deformity was detected in 0.6% of racehorses (95% CI: 0.12 to 1.08%). Affected racehorses were older ($P = 0.01$) and had more median lifetime starts, lifetime places, lifetime wins and higher lifetime stake earnings than unaffected racehorses ($P = 0.01$). The apparent superior performance was most likely caused by more lifetime starts was again found to be a confounder that probably accounted for the increased life time

wins and places. Epiglottic entrapment was detected in 1.29 % racehorses (95% CI: 0.59 to 2%). There was no difference between affected and unaffected racehorses for median age, sex, lifetime starts, lifetime wins and lifetime places ($P > 0.05$).

Racehorses with grade 3 tracheal mucous had more median lifetime wins, lifetime places and higher lifetime stake earnings compared to horses with grade 2 tracheal mucous ($P = 0.01$). The sixty eight racehorses (6.77%, 95% CI: 5.16 to 8.37%) affected with tracheal cartilage ring spikes were 3.6 times more likely to be male (relative risk = 3.63, 95% CI: 2.07 to 6.35; $P < 0.01$) and had more lifetime wins and lifetime places ($P = 0.03$). The presence of debris was detected in the larynx of 437 racehorses (43.53%, 95% CI: 39.44 to 47.61) and trachea of 220 racehorses (21.89%, 95% CI: 19.02 to 24.81) and did not affect median lifetime wins and lifetime places ($P > 0.05$).

In Thoroughbred racehorses competing in South Africa, the prevalence of pharyngeal, laryngeal and tracheal disorders is varied. Racing performance which was apparently superior in horses affected by arytenoid cartilage asymmetry (grade 2 and 4) and epiglottic deformity; and apparently impaired in horses affected by PLH (grade 2 and 3), was most likely as a result of more lifetime starts which acted as a confounder in these groups of racehorses. Racing performance was not impaired by the presence of epiglottic entrapment, and debris in the larynx and trachea. Superior racing performance was seen in horses with tracheal cartilage ring spikes and grade 3 tracheal mucous. This study also highlights the multifactorial nature of these conditions and the caution that needs to be taken before inferring causality to a particular factor.

3.2 INTRODUCTION

Three North American[21,23,27] and one Australian study[7] have reported on the prevalence of upper respiratory tract abnormalities in Thoroughbred racehorses. Excluding pharyngeal lymphoid hyperplasia, the reported prevalence of upper respiratory abnormalities were 3.4 to 8.1%[21,23,27] and 6.3%.[7]

Using flexible endoscopy, abnormalities identified by these studies included asymmetry of the left arytenoid cartilage,[7,21] left laryngeal hemiplegia,[21,23,27] prosthetic laryngoplasty,[21,23] ventriculectomy,[7] right arytenoid paralysis,[21,23] arytenoid chondropathy,[7,21] ulceration of the arytenoid mucosa,[7] aryepiglottic entrapment,[7,21,23,27] pharyngeal polyps,[21] dorsal displacement of the soft palate,[7,23,27] guttural pouch discharge,[23] nasal stenosis,[23] subepiglottic cyst,[7,23] epiglottic hypoplasia,[7] epiglottic deformity[7] and tracheal mucous.[21,23]

In Thoroughbred racehorses, although certain disorders of the upper respiratory tract such as left laryngeal hemiplegia[8,16] have been associated with reduced exercise capability while others not,[7] there still exists a paucity of objective data to fully evaluate the importance of these and other conditions which are less severe or occur infrequently. Historically, studies presenting data on horses with upper respiratory tract abnormalities have suffered from a selection bias, as horses have been examined only when respiratory strider or poor performance was noted. Although providing useful information, these studies were not an accurate representation of overall health status in a Thoroughbred

racehorse population, and functional consequences of upper respiratory tract abnormalities could not be assessed since racing performance was not evaluated.

A paucity of reports exists on the association between performance and respiratory tract disorders in Thoroughbred racehorses while the prevalence of these abnormalities is largely unknown outside North America[21,23,27] and Australia.[7] The purpose of this study was to firstly document the prevalence of pharyngeal, laryngeal and tracheal disorders in Thoroughbred racehorses competing in South Africa and secondly, to determine their relationship with racing performance.

3.3 MATERIALS AND METHODS

3.3.1 Thoroughbred racehorses

The study was a prospective cross-sectional study of the prevalence of pharyngeal, laryngeal and tracheal disorders and their association with racing performance in Thoroughbred racehorses. Racehorses of either sex, running on turf or sand, competing in flat races between 800 and 3,200 meters at five racetracks in South Africa (Turffontein Race Course [Gauteng Province], Vaal Race Course [Free State Province], Clairwood and Greyville Turf Club [Kwazulu-Natal Province] and Kenilworth Race Course [Cape Town Province]) were enrolled into this study between 4th August and 19th November 2005. These five racecourses are considered representative of the best racing in South Africa. Race day administration of medications such as furosemide is not allowed in

South Africa and drug testing is strictly enforced by the National Horse Racing Authority (NHRA) through screening of urine and blood for prohibited and therapeutic substances. Lists of available horses that were accepted to race were obtained from the NHRA. Eligible racehorses were then identified, trainers contacted individually and permission obtained to examine the horse. Not all trainers allowed their racehorses to participate in this study. Only pre-enrolled horses (24 to 72 hours prior to race day) were entered into the study to avoid a potential enrollment bias. Any racehorse that was refractory to restraint that could comprise the safety of personnel or equipment, and any horse examined after racing that was not pre-enrolled prior to the race, was excluded from this study.

3.3.2 Endoscopic examination

Following racing, each pre-enrolled racehorse was identified and tagged during parading. Thereafter, tagged racehorses were brought into a dedicated stable for videoendoscopic examinations. Unsedated racehorses were restrained by a handler with a halter and nose twitch in a dedicated examination stable. Videoendoscopic evaluation of one nostril, ipsilateral nasal turbinate, nasopharynx, larynx and trachea was performed and all examinations were recorded onto digital video disc. At least one veterinarian always evaluated the image on the screen at all times. Following insertion of the videoendoscope (Pentax Corporation, Tokyo, Japan: endoscope model number EC3830FK, 1.5 m in length, 38 French in diameter, processor number EPK700) into either nostril, it was passed along the ventral meatus caudally to the nasopharynx. The nasopharynx and

larynx were carefully examined. Following confirmation of the position of the epiglottis and soft palate, the larynx was assessed for symmetry, synchronous movement and debris. Thereafter, the trachea was assessed for tracheal ring symmetry and the presence of mucous or debris up to and including the tracheal bifurcation at the level of the carina. For the purpose of this study, exercise-induced pulmonary hemorrhage was not evaluated.

3.3.3 Grading of pharyngeal, laryngeal and tracheal disorders

Arytenoid cartilage asymmetry was graded 1 to 4.[26] Grade 1 indicated symmetrical synchronous abduction and adduction of the left and right arytenoid cartilages (Figure 3.1); grade 2 indicated some asynchronous movement (hesitation, flutter or abductor weakness) of the left arytenoid cartilage during any phase of respiration and full abduction of the left arytenoid cartilage which could be maintained by swallowing; grade 3 indicated asynchronous movement (hesitation, flutter or abductor weakness) of the left arytenoid cartilage during any phase of respiration and full abduction of the left arytenoid cartilage could not be induced or maintained by swallowing; and grade 4 indicated no substantial movement of the left arytenoid cartilage during any phase of respiration and were subsequently classified as having idiopathic laryngeal hemiplegia (ILH) (Figure 3.2). Those horses with a fixed and immobile, abducted left arytenoid with or without a ventriculectomy were identified as having a prosthetic laryngoplasty and this was confirmed with the trainer or owner.

The severity of pharyngeal lymphoid hyperplasia (PLH) was graded on a scale from 1 to 4.[5] Grade 1 indicated lymphoid hyperplasia limited to < 180° of the dorsal pharyngeal recess (Figure 3.3); grade 2 indicated lymphoid hyperplasia extending to circumference of the dorsal pharyngeal recess (Figure 3.4); grade 3 indicated lymphoid hyperplasia made midline contact of the dorsal pharyngeal recess (Figure 3.5); and grade 4 indicated small masses (which may be abscesses) arising from either the dorsal pharyngeal recess or the pharyngeal walls (Figure 3.6).

Dorsal displacement of the soft palate (DDSP) was defined conservatively by our study and no nasal occlusion was performed as part of the examination (Figure 3.7). DDSP was clinically relevant only if it was present before and after rhinolaryngoscopy was performed, and if repositioning of the soft palate did not occur after 2 swallowing attempts. The length, width and shape of the epiglottis were subjectively evaluated. Epiglottic entrapment was diagnosed if the epiglottis was enveloped by the aryepiglottic fold. Endoscopic evaluations of the guttural pouches were not performed.

Mucous within the trachea was graded 0 to 5.[11] Grade 0 indicated the absence of mucous; grade 1 indicated singular droplets of mucous (Figure 3.8); grade 2 indicated multiple droplets of mucous that is partly confluent (Figure 3.9); grade 3 indicated mucous that is ventrally confluent (Figure 3.10); grade 4 indicated a large ventral pool of mucous (Figure 3.11); and grade 5 indicated profuse amounts of mucous covering > 25% of the tracheal lumen (Figure 3.12).

The racehorses' age and sex as well as racing career performance record immediately preceding the endoscopic examination on race day was extracted from race cards. The data included lifetime starts, lifetime wins, lifetime places (2^{nd}, 3^{rd}, or 4^{th}) and lifetime stake earnings (South African Rand, ZAR).

3.3.4 Data analysis

Data for all racehorses was collated in Microsoft Excel (version 2003) and variables for racehorses that were diagnosed with epiglottic deformity, epiglottic entrapment, arytenoid cartilage asymmetry, prosthetic laryngoplasty, pharyngeal lymphoid hyperplasia, tracheal mucous, tracheal cartilage ring spike, laryngeal dirt and tracheal dirt were compared with variables in unaffected racehorses using the statistical software package NCSS 2006 (Hintze J. NCSS and PASS number cruncher statistical systems, Kaysville, Utah, 2006). Statistical analysis was not performed for other conditions due to the small number of affected racehorses. A Mann-Whitney *U*-Test was used to compare median age, lifetime runs, lifetime wins, lifetime places and lifetime earnings. A chi-squared analysis with Yates correction was used to analyze the sex distribution (male, gelding and female). For all comparisons, a value of $P < 0.05$ was considered significant. Where confounding was suspected, the relationship of the confounding was studied using multiple least squares regression.

3.4 RESULTS

Of the 2,684 eligible racehorses that competed in 230 flat races (194 turf races, 36 sand races) at 28 race meetings, 1,005 horses (37.4%) were endoscopically examined. Endoscopic examinations took place mean ± standard deviation (SD) 24 ± 12.3 minutes after racing. There were 509 females, 491 geldings, and 5 intact males with a mean age of 4 years (95% confidence interval [CI]: 2 to 9 years). Ninety seven trainers participated in this study. This study evaluated and identified PLH, arytenoid cartilage asymmetry, prosthetic laryngoplasty, laryngeal dirt, epiglottic deformity epiglottic entrapment, subepiglottic cyst, DDSP, tracheal stenosis, tracheal cartilage ring spike, tracheal mucous and tracheal dirt.

Following assessment of arytenoid cartilage asymmetry, racehorses were graded 1 (n = 970), 2 (n = 18), 3 (n = 4) and 4 (n = 6), while no grade was allocated to horses with laryngoplasty (n = 7). Results of the Mann-Whitney U-test showed that when compared to grade 1 arytenoid cartilage asymmetry, median values for racehorses with grade 2 arytenoid cartilage asymmetry were older (4 *vs*. 5 years; $P = 0.01$), had more lifetime starts (13 *vs*. 8; $P = 0.047$), more lifetime wins (2 *vs*. 1; $P = 0.02$), and had higher lifetime stake earnings (ZAR 79,567.50 *vs*. 35,287.50; $P = 0.04$) while those with grade 4 arytenoid cartilage asymmetry (ILH) were older (5 *vs*. 4 years; $P = 0.01$), had more lifetime starts (19.5 *vs*. 8; $P = 0.01$), more lifetime wins (3 *vs*. 1; $P = 0.03$), more lifetime places (6.5 *vs*. 2; $P = 0.01$), and higher lifetime stake earnings (ZAR 112,297,50 *vs*. 35,287,50; $P = 0.01$). ILH was detected in a total of 6 racehorses (0.6%, 95% CI: 0.12 to

1.07) consisting of 3 geldings; and 1 gelding, 1 male and 1 female due to a failed prosthetic laryngoplasty. One racehorse each finished in 1st, 4th, 8th and 10th place and two horses each in 3rd place in the race preceding endoscopic examination. Because of the significant difference in lifetime starts it was suspected that this may be confounding the lifetime wins and lifetime places. Multiple least squares regression was therefore performed using lifetime wins or lifetime places as a Y-dependent variable and lifetime starts and arytenoid asymmetry grades as independent variables. The relationship between lifetime wins or lifetime places and the various grades of arytenoid asymmetry was then shown to be insignificant ($P > 0.05$), while there was still a significant relationship ($P < 0.01$) between lifetime starts and lifetime wins or lifetime places. The increased lifetime wins and lifetime places in horses with arytenoid asymmetry are probably therefore due to more lifetime starts and not the arytenoid asymmetry itself.

Successful prosthetic laryngoplasty was detected in 7 racehorses (0.7%, 95% CI 0.18-1.21%) consisting of 1 male, 5 geldings and 1 female. No significant differences were observed between horses with laryngoplasty and those without for age ($P = 0.18$), sex ($P = 0.42$), lifetime starts ($P = 0.42$), lifetime wins ($P = 0.69$), lifetime places ($P = 0.14$) and lifetime stake earnings ($P = 0.75$). One racehorse each finished in 1st, 5th, 8th, 10th and 17th place, and two in 4th place each.

Racehorses with PLH were graded 1 (n = 372), 2 (n = 534), 3 (n = 83) and 4 (n = 4) while no grade could be allocated to 12 horses due to poor endoscopic imaging. Compared to grade 1 PLH, racehorses with grade 2 PLH, had less lifetime starts (6 *vs*. 12; $P < 0.01$),

less lifetime wins (0 vs. 1; $P < 0.01$), less lifetime places (2 *vs*. 4; $P < 0.01$), and had lower lifetime stake earnings (ZAR 29,720 *vs*. 53,050; $P < 0.01$); while racehorses with grade 3 PLH were also younger (3 *vs*. 4 years; $P < 0.01$), had less lifetime starts (4 *vs*. 12; $P < 0.01$), less lifetime wins (0 *vs*. 1; $P < 0.01$), less lifetime places (1 *vs*. 4; $P < 0.01$) and had lower lifetime stake earnings (ZAR 5,000 *vs*. 53,030; $P < 0.01$). Those racehorses with grade 2 PLH had more lifetime places (2 *vs*. 1; $P < 0.01$) and higher lifetime stake earnings (ZAR 29,720 *vs*. 5,000; $P = 0.03$) compared to racehorses with grade 3 PLH. Because of the significant difference in lifetime starts it was suspected that this may be confounding lifetime wins and lifetime places. Multiple least squares regression was therefore performed using lifetime wins or lifetime places as a Y-dependent variable and lifetime starts and PLH grades as independent variables. The relationship between lifetime wins or lifetime places and the various grades of PLH was then shown to be insignificant ($P > 0.05$), while there was still a significant relationship ($P < 0.01$) between lifetime starts and lifetime wins or lifetime places. The decreased lifetime wins and places in racehorses with PLH are probably therefore due to less lifetime starts.

Epiglottic entrapment was detected in 13 racehorses (1.29%, 95% CI: 0.59 to 2%) consisting of 3 geldings, 4 males and 6 females (Figure 3.13). No significant differences were observed between racehorses with epiglottic entrapment and horses without for age ($P = 0.62\%$), sex ($P = 0.1$), lifetime starts ($P = 0.54$), lifetime wins ($P = 0.6$) and lifetime places ($P = 0.38$). However, horses with epiglottic entrapment had higher lifetime stake earnings compared to those unaffected (ZAR 78,650 *vs*. 35,885; $P = 0.046$). Two of the thirteen racehorses (males) had epiglottic entrapment (of which 1 was intermittent) and a

subepiglottic cyst concurrently (Figure 3.14), while one racehorse (gelding) had an epiglottis that was entrapped intermittently and malformed. One racehorse each finished in 5th, 7th, 8th, 9th and 11th place, while two horses finished in 2nd, 3rd, 6th and 10th place each in the race preceding endoscopic examination.

Epiglottic deformity was detected in 6 racehorses (0.6%, 95% CI: 0.12 to 1.08%) consisting of 4 geldings, 1 male and 1 female and had a sex distribution that was not significantly different to the other racehorses examined (P = 0.66) (Figure 3.15). Racehorses with epiglottic deformity were older (5 *vs*. 4 years; P = 0.01), had higher lifetime stake earnings (ZAR 87,650 *vs*. 36,000; $P < 0.01$), more lifetime starts (20 *vs*. 8; $P < 0.01$), more lifetime wins (2.5 *vs*. 1; $P = 0.009$) and more lifetime places (6 *vs*. 2; $P = 0.03$). One racehorse finished in 2nd and 8th, and two in 4th and 6th place each in the race prior to endoscopic examination. Because of the significant difference in lifetime starts it was suspected that this may be confounding lifetime wins and lifetime places. Multiple least squares regression was therefore performed using lifetime wins or lifetime places as a Y-dependent variable and starts and epiglottic deformity as independent variables. The relationship between lifetime wins or lifetime places and epiglottic deformity was then shown to be insignificant ($P > 0.05$), while there was still a significant relationship ($P < 0.01$) between starts and wins or places. The increased lifetime wins and places in horses with epiglottic deformity are probably therefore due to more lifetime starts and not the epiglottic deformity itself.

Racehorses with tracheal mucous were graded 0 (n = 5), 1 (n = 412), 2 (n = 291), 3 (n = 164), 4 (n = 71) and 5 (n = 58), while no grade could be allocated in 4 racehorses due to the large volume of blood present in the trachea. Compared to grade 2 tracheal mucous, racehorses with grade 3 tracheal mucous had more lifetime wins (1 *vs*. 1; $P = 0.02$), more lifetime places (3 *vs*. 2; $P = 0.01$) and higher lifetime stake earnings (ZAR 47,880 *vs*. 33,225; $P = 0.01$) while horses with grade 4 tracheal mucous had more lifetime starts (10 *vs*. 7; $P = 0.046$).

Tracheal debris was detected in 220 racehorses (21.91%, 95%CI: 19.02 to 24.81%) consisting of 117 geldings, 38 males and 65 females with a sex distribution that was not significantly different to unaffected racehorses ($P = 0.11$). There were no significant differences between affected and unaffected racehorses for median age ($P = 0.91$), lifetime stake earnings ($P = 0.84$), lifetime starts ($P = 0.73$), lifetime wins ($P = 0.76$) and lifetime places ($P = 0.74$).

Laryngeal debris was detected in 437 racehorses (43.53%, 95% CI: 39.44 to 47.61%) consisting of 223 geldings, 59 males and 155 females with a sex distribution that was not significantly different to unaffected racehorses ($P = 0.14$). There were no significant differences for racehorses with laryngeal debris and those without for median age ($P = 0.28$), lifetime stake earnings ($P = 0.43$), lifetime starts ($P = 0.57$), lifetime wins ($P = 0.87$) and lifetime places ($P = 0.35$).

Tracheal cartilage spikes were detected in 68 racehorses (6.77%, 95% CI: 5.16 to 8.37%) consisting of 10 geldings, 53 males and 5 females with a sex distribution that was significantly different to unaffected racehorses ($P < 0.01$) (Figure 3.16). Affected racehorses were nearly 4 times more likely to be males (relative risk = 3.63, 95% CI: 2.07 to 6.35); and had more lifetime wins (1 *vs*. 1; $P = 0.03$) and more lifetime places (4 *vs*. 2; $P = 0.03$). There was no significant differences for racehorses with tracheal cartilage spikes and those without for median age ($P = 0.82$), lifetime stake earnings ($P = 0.06$) and lifetime starts ($P = 0.13$).

One racehorse (gelding) had tracheal stenosis and finished in 4th place, two racehorses had subepiglottic cysts consisting of 2 males that finished in 2nd and 8th place; while DDSP was identified in two racehorses consisting of 1 male and 1 female that finished in 5th and 7th place. Statistical analysis for performance could not be performed for these conditions as the number of affected racehorses was too low.

3.5 DISCUSSION

This study is the first to report the prevalence of pharyngeal, laryngeal and tracheal disorders in a natural population of high quality Thoroughbred racehorses in South Africa. Overall, there was a low prevalence of grade 2 and 3 arytenoid cartilage asymmetry, ILH, epiglottic entrapment, subepiglottic cyst and epiglottic deformity; while more severe grades of PLH, laryngeal debris, tracheal debris, tracheal mucous and tracheal cartilage ring spikes had a higher prevalence. Arytenoid cartilage asymmetry (grade 2 and 3), ILH and epiglottic deformity did not result in impaired racing performance but these horses had significantly more lifetime starts. Racehorses with grade 3 PLH were more likely to be younger and have impaired lifetime racing performance. The presence of tracheal and laryngeal dirt and epiglottic entrapment did not modify racing performance. Furthermore, racehorses with tracheal cartilage ring spikes (which were more prevalent in males) and tracheal mucous (grade 3) had better lifetime racing performance.

3.5.1 The prevalence of pharyngeal, laryngeal and tracheal disorders

Thoroughbred racehorses may have an endoscopic examination performed of the upper respiratory tract at different stages of life. Disorders may be identified during pre- or post sale endoscopic examination as a yearling,[26] or later in life when presenting for respiratory disease. Although studies have identified upper respiratory tract disorders in foals[17] and yearlings,[1,2,18] extrapolation of such results to more mature horses may be

inaccurate as was reported with laryngeal asymmetry detected in foals[17] and yearlings[1] versus older horses.[1,17] While these reports[1,2,17,18] are useful, data may not be applicable to racehorses. Poor performance, stridor or a combination of the aforementioned may be reasons for endoscopic examination in racehorses.[14,19,20] Such surveys of horses suffer from a selection bias and may not accurately reflect the true prevalence of upper respiratory tract disorders in a natural population of racehorses. Also, no association was made between upper respiratory tract disorders and athletic performance. Reports do exist on the prevalence of respiratory tract disorders in horses,[7,21,23,27] however only one study has made an association between athletic performance and upper respiratory tract disorders[7] but excluded PLH and tracheal disorders.

Grade 2 and 3 arytenoid asymmetry was detected in 2.2% of racehorses in this study as compared with 3.8% of Thoroughbred racehorses competing in North America,[21] 1.4% of Thoroughbred racehorses in Australia,[7] and 47% Thoroughbred racehorses New Zealand.[1] This study compares favorably with the Australian report that used a similar grading scale for arytenoid asymmetry, also evaluated higher quality racehorses soon after racing (on average 32 minutes post-race).[7] Other studies have reported much higher prevalence of arytenoid asymmetry using similar grading criteria[1] while another did not define their criteria for arytenoid asymmetry.[4] Plausible reasons may exist for the wide variation in reported prevalence of arytenoid asymmetry and include variation in criteria for grading, experience of the grader, selection criteria for endoscopic examination, quality of the racehorses examined, timing of endoscopic examination and affect of locality.

ILH was detected in 0.6% of racehorses in this study as compared to 1.3% to 3.3% of Thoroughbred racehorses competing in North America,[21,23,27] 0.3% of Thoroughbred racehorses competing in Australia,[7] and 1% of Thoroughbred racehorses competing in New Zealand.[1] The low prevalence reported by this study and another,[7] may be due to the higher quality of racehorses examined and the fact that all the horses were pre-enrolled and not selected for endoscopic evaluation based on abnormal stridor or poor racing performance.

Epiglottic deformity was detected in 0.6% of racehorses in this study compared to 0.1% of Australian Thoroughbred racehorses.[7] A subepiglottic cyst was detected in 0.2% of racehorses in this study compared to 0.2% of North American[23] and 0.1% of Australian Thoroughbred racehorses.[7] Similarly, there was a low prevalence of epiglottic entrapment (1.3%) in this study compared to 0.74 to 2.1% of racehorses competing in North America[21,23,27] and 0.9% of racehorses competing in Australia.[7]

DDSP was detected in 0.2% of racehorses in this study compared to 0.74 to 5.2% of North American racehorses[15,23,27] and 0.5% of Australian racehorses.[7] The low prevalence of DDSP may have resulted form the conservative definition employed by this study and the fact that nasal occlusion was not performed as part of the endoscopic evaluation.

Together, grade 2 to 4 PLH was detected in 63% of racehorses competing in this study compared to an overall prevalence of 29.5% and 34.2 % of Thoroughbred racehorses

competing in North America.[23,27] The prevalence of PLH reported in this study is difficult to compare with previous studies due to the use of different grading criteria.[15,23,27] However, similar to previous studies that found an association between age and PLH,[15,23,27] this study also confirmed that younger racehorses were affected by more severe grades of PLH (grade 3).

Laryngeal debris was detected in 43.5% of racehorses in this study compared to 1.3% of racehorses competing in North America,[21] while tracheal debris was present in 21.9% of South African racehorses compared to 0.4 to 0.9% of North American racehorses.[21,23] Debris may be caused either by inhaled sand or dirt during racing and their presence within the respiratory tract may be dependant on degree of mucociliary clearance. The higher prevalence of debris as reported in this study may be due to endoscopic examinations that were performed soon after racing before effective mucociliary clearance occurred and by the use of more advanced videoendoscopy equipment that aided detection of debris.

Although tracheal cartilage ring spikes have been noted to occur,[24] their prevalence in a natural population of Thoroughbred racehorses has not been previously reported to the author's best knowledge. These spikes are epithelium-covered cartilages which protrude into the tracheal lumen from the tracheal ventrum.[24] Tracheal cartilage spikes were detected in 6.8% of racehorses in this study and occurred more often in male racehorses. The clinical significance of this condition and why it occurs more commonly in males is still unclear.

Tracheal mucous was detected in 99.5% of racehorses in this study compared to 6 to 6.8% of Thoroughbred racehorses competing in North America.[21,23] Another study performed 1,900 endoscopic examinations on racehorses ≥ 24 hours post-race and detected tracheal mucous in 41.3% of racehorses.[15] This study examined horses on average 24 minutes after racing compared to evaluations performed much later in racehorses at rest.[15] The observed differences in prevalence of tracheal mucous may be due to a close temporal relationship between endoscopic examination and racing.

3.5.2 Association with athletic performance

Upper respiratory tract disorders may affect the athletic performance of horses. Surveys of horses with upper airway disorders have demonstrated that DDSP[12] and ILH[8,10,16] is associated with impaired performance, mucosal erosions had no effect on performance,[7] while epiglottic entrapment and grade 2 arytenoid cartilage asymmetry was associated with enhanced performance.[7] Plausible reasons exist why DDSP and ILH may impair athletic performance such as reduced oxygen consumption,[16] while it is unclear what the reason may be for enhanced performance seen in epiglottic entrapment.

In this study, the apparent superior performance with grade 2 arytenoid cartilage asymmetry and ILH could be partially explained by its association with the number of lifetime starts, which was acting as a confounder. So while it appeared that horses with grade 2 arytenoid cartilage asymmetry and ILH had better racing performance due to more lifetime wins and/or lifetime places, and higher lifetime stake earnings, this was

probably a function of the lifetime starts. This raises the question of whether presence of arytenoid asymmetry and ILH is related to the frequency of racing, with horses with more lifetime starts being at higher risk. Our findings agree with a previous report that found grade 2 arytenoid cartilage asymmetry did not impair performance[7] as this degree of asymmetry still allows maximal arytenoid abduction[22] and is not associated with hypoxaemia.[8] Numerous studies have reported a negative association between ILH and racing performance based on impaired gaseous exchange in horses exercised on a treadmill[8,16] and track.[6] However, these studies suggest that race performance may not be adversely affected since no effect of ILH on run time was reported.[6,16] This study is in agreement with previous studies that ILH was not associated with impaired race performance[6,16] but the reason for this in light of the confounding effect of lifetime starts is now debatable.

Racehorses with PLH (grade 2 and 3) had apparent impaired racing performance. Affected racehorses had fewer lifetime starts, fewer lifetime wins, fewer lifetime places, lower lifetime stake earnings and were younger. Within the different grades of PLH, superior racing performance was seen in racehorses with grade 2 compared to grade 3 PLH, due to more lifetime places and higher lifetime stake earnings. However, similar to arytenoid asymmetry, lifetime starts was having a confounding effect and the apparent effect on performance was probably as a result in differences in the number of lifetime starts rather than the presence of the disorder. This would be more consistent with previous studies that have reported no association between PLH and racing performance.[3,13,15]

Epiglottic deformity also appeared to be associated with superior racing performance as these horses were older, had higher lifetime stake earnings, more lifetime starts, more lifetime wins, and more lifetime places. However, once again lifetime starts was acting as a confounder making the result difficult to interpret. Causes of epiglottic deformity are varied and may have congenital, inflammatory or traumatic origins, or develop secondary to chronic entrapment by the aryepiglottic fold or soft palate. Previous studies have reported epiglottic deformity but were unable to make an association with performance due to inadequate statistical power[7] and selection criteria.[28]

Superior lifetime racing performance was seen in racehorses with tracheal cartilage spikes as affected racehorses had more lifetime wins and more lifetime places. In this case, lifetime starts did not appear to be a confounder. It is uncertain as to how enhanced racing performance is related to the presence of tracheal cartilage spikes.

Epiglottic entrapment was not associated with impaired racing performance. Although lifetime starts, lifetime wins, and lifetime places were similar between affected and unaffected racehorses, horses with epiglottic entrapment had higher lifetime earnings. This study is in agreement with another study that suggested epiglottic entrapment does not impair performance.[7] This is perhaps not surprising since previous reports could not document impaired gaseous exchange[16] nor changes in upper airway pressures (despite medical or surgical intervention) in horses with epiglottic entrapment.[29]

Tracheal mucous (grade 3) was associated with superior performance as affected racehorses had more lifetime wins, more lifetime places and higher lifetime earnings. Moreover, horses with grade 4 tracheal mucous had more lifetime starts. This is surprising since increased tracheal mucous is associated with exercise-induced arterial hypoxemia following a standardized treadmill test[9,25] indicating that tracheal mucous may be associated with reduced performance. However, the presence of tracheal mucous was not found to affect treadmill performance in racehorses after assessment of total run time, peak running speed, speed at a heart rate of 200 beats/minute and distance to fatigue.[9] Another study has reported that more severe accumulations of mucous in racehorses was associated with a higher race finish position and thus poor racing performance, however did not include detailed racing performance results and evaluated racehorses ≥ 24 hours post race.[15] The reason for the superior performance reported by this study may again be related to lifetime starts although this is less clear in this case. Our results may also have been affected by examining higher quality racehorses with improved fitness levels and the examination of racehorses soon after racing.

Due to the low prevalence of DDSP, tracheal stenosis and subepiglottic cysts, the relationship between the disorder and racing performance could not be made. It should be noted that although resting endoscopic examination as performed by this study may have only detected structural abnormalities within the respiratory tract, while dynamic or functional obstructive abnormalities can only be observed using high-speed treadmill examinations. Therefore, although the following abnormalities may have been present in the study population, this study could not identify disorders such as progressive laryngeal

hemiplegia, intermittent DDSP, pharyngeal collapse, tracheal collapse, epiglottic entrapment, epiglottic retroversion, axial deviation of the aryepiglottic folds and axial deviation of the vocal folds.

In Thoroughbred racehorses competing in South Africa, the prevalence of pharyngeal, laryngeal and tracheal disorders is varied. Racing performance was not impaired by the presence of epiglottic entrapment, and debris in the larynx and trachea. Superior racing performance was seen in horses with tracheal cartilage ring spikes and grade 3 tracheal mucous. This study also highlights the multifactorial nature of these conditions and that clinicians should be cautious to infer causality to a particular disorder.

3.6 CONCLUSIONS

This study identified left arytenoid asymmetry, ILH, epiglottic deformity, epiglottic entrapment, DDSP, PLH, laryngeal and tracheal dirt, tracheal mucous, and tracheal cartilage ring spikes in Thoroughbred racehorses post-race. An association with sex was identified as tracheal cartilage ring spikes occurred more often in male racehorses. Superior racing performance was identified in racehorses with grade 3 tracheal mucous and tracheal cartilage ring spikes.

3.7 FIGURES AND TABLES

Figure 3.1 Symmetrical abduction of the arytenoid cartilages in a Thoroughbred racehorse.

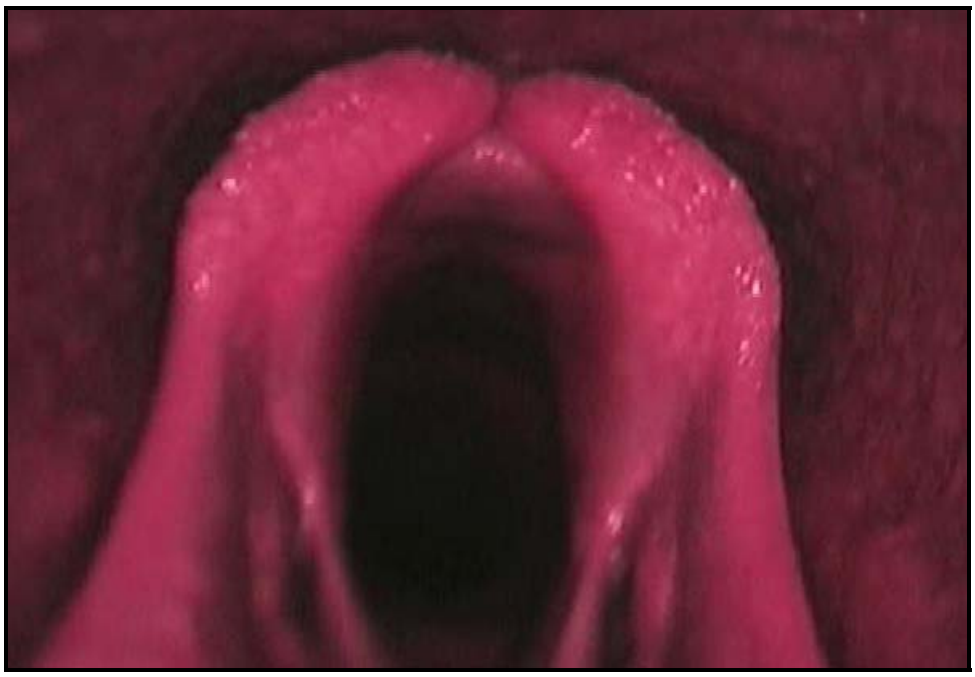


Figure 3.2 Idiopathic laryngeal hemiplegia in a Thoroughbred racehorse.

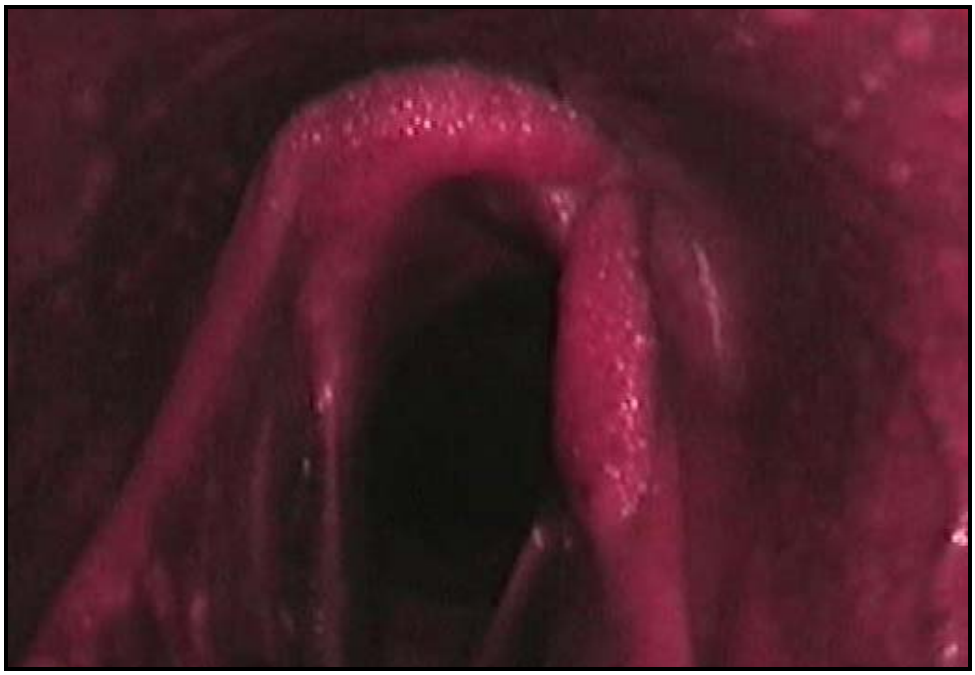


Figure 3.3 A Thoroughbred racehorse with grade 1 pharyngeal lymphoid hyperplasia.

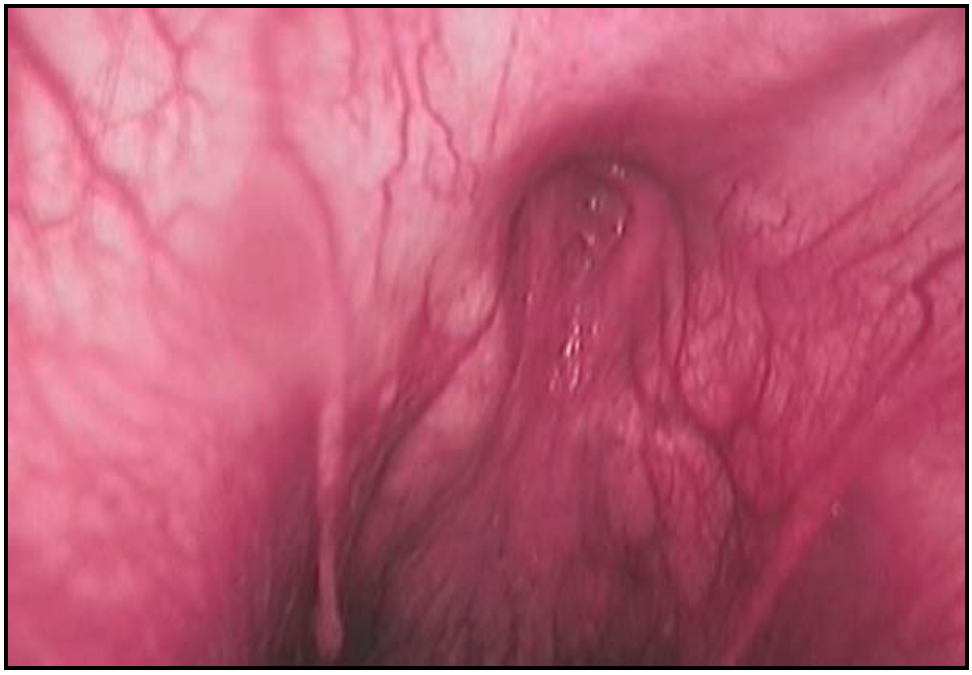


Figure 3.4 A Thoroughbred racehorse with grade 2 pharyngeal lymphoid hyperplasia.

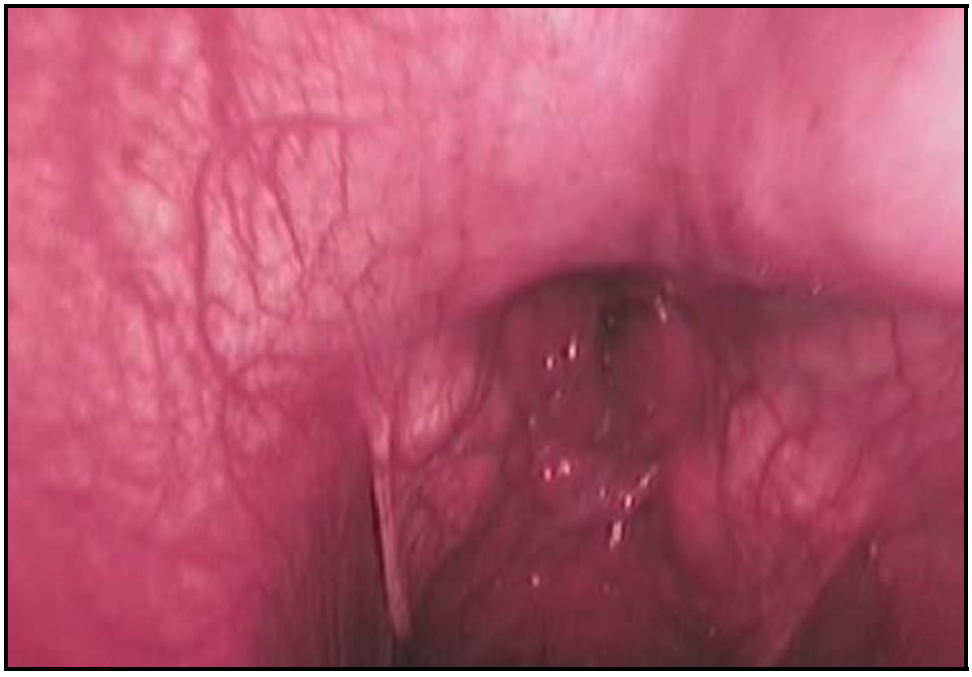


Figure 3.5 A Thoroughbred racehorse with grade 3 pharyngeal lymphoid hyperplasia.

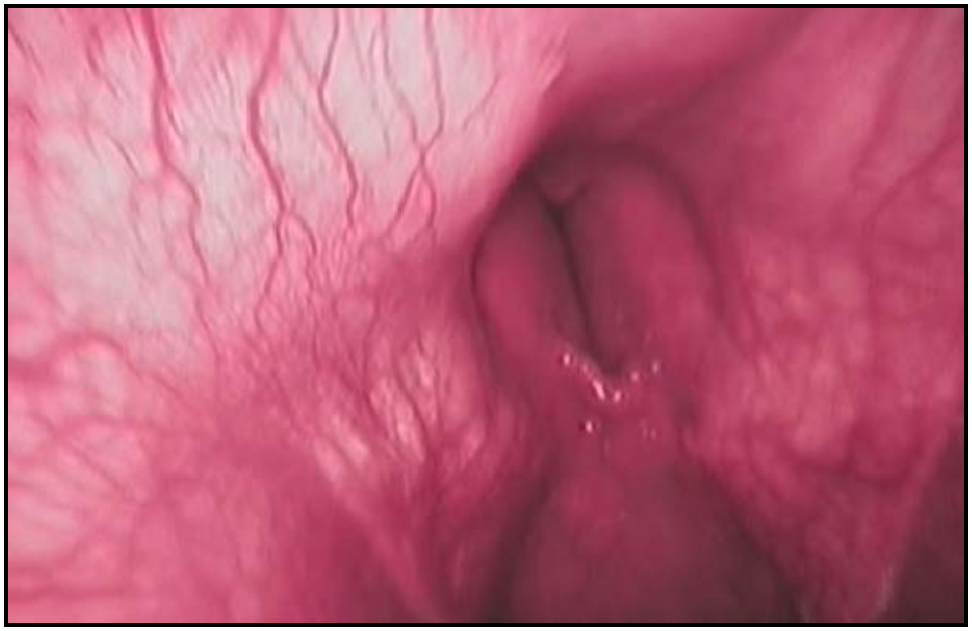


Figure 3.6 A Thoroughbred racehorse with grade 4 pharyngeal lymphoid hyperplasia.

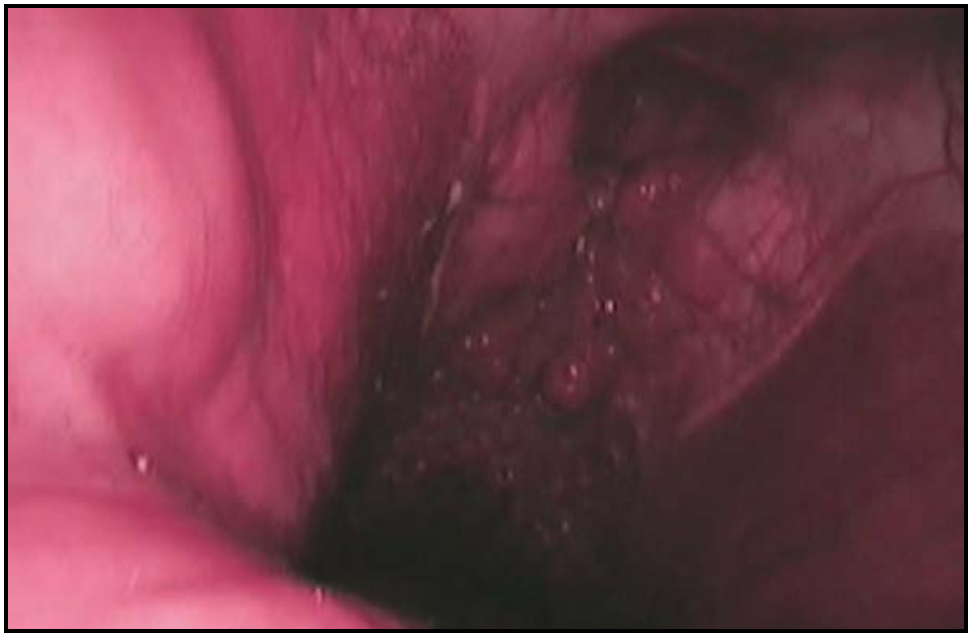


Figure 3.7 A Thoroughbred racehorse with dorsal displacement of the soft palate.

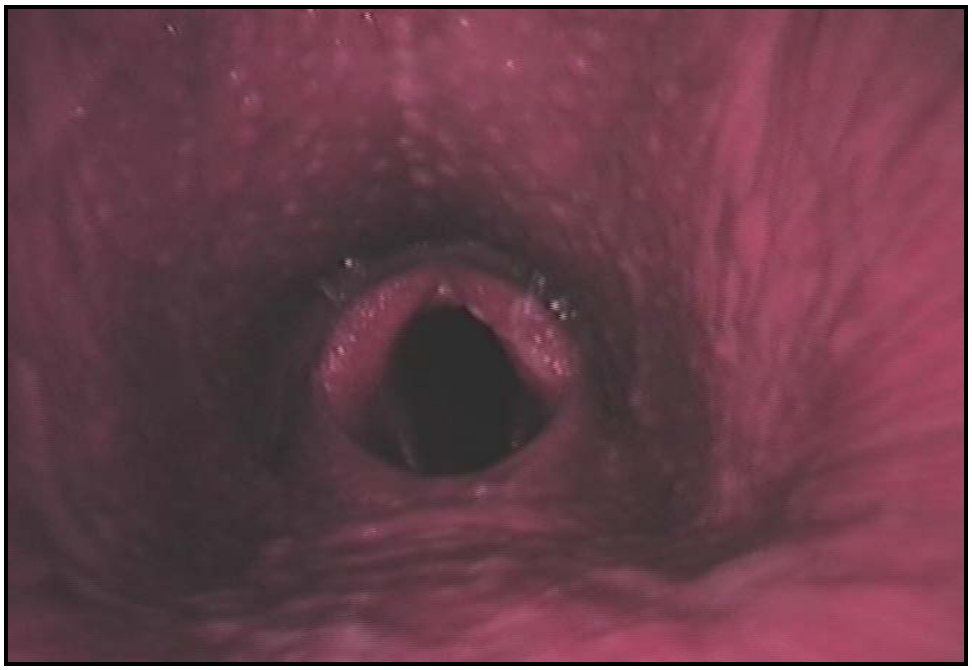


Figure 3.8 A Thoroughbred racehorse with grade 1 tracheal mucous detected by tracheobronchoscopy.

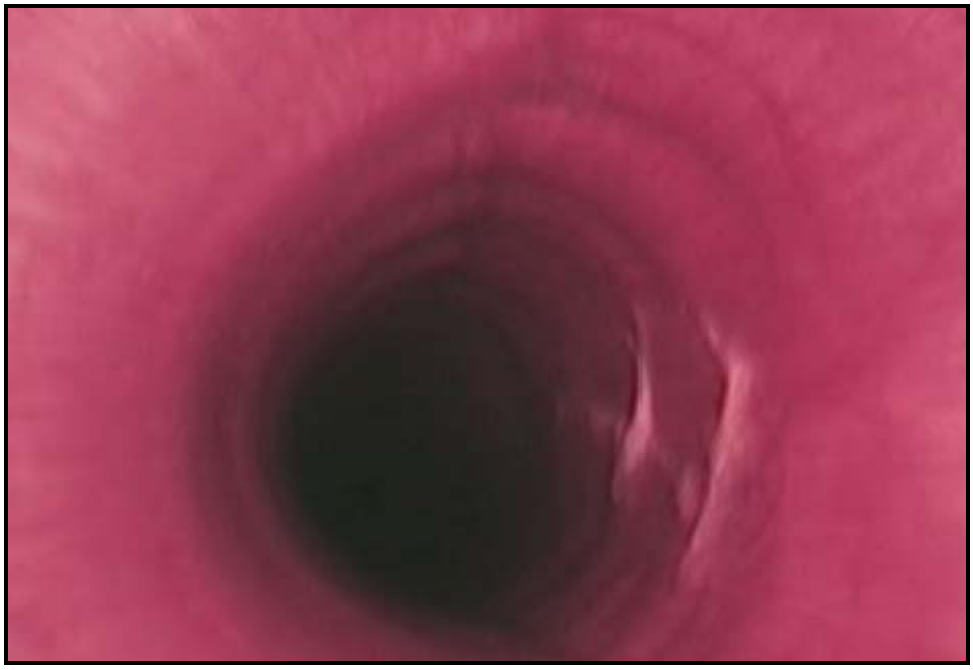


Figure 3.9 A Thoroughbred racehorse with grade 2 tracheal mucous as detected by tracheobronchoscopy.

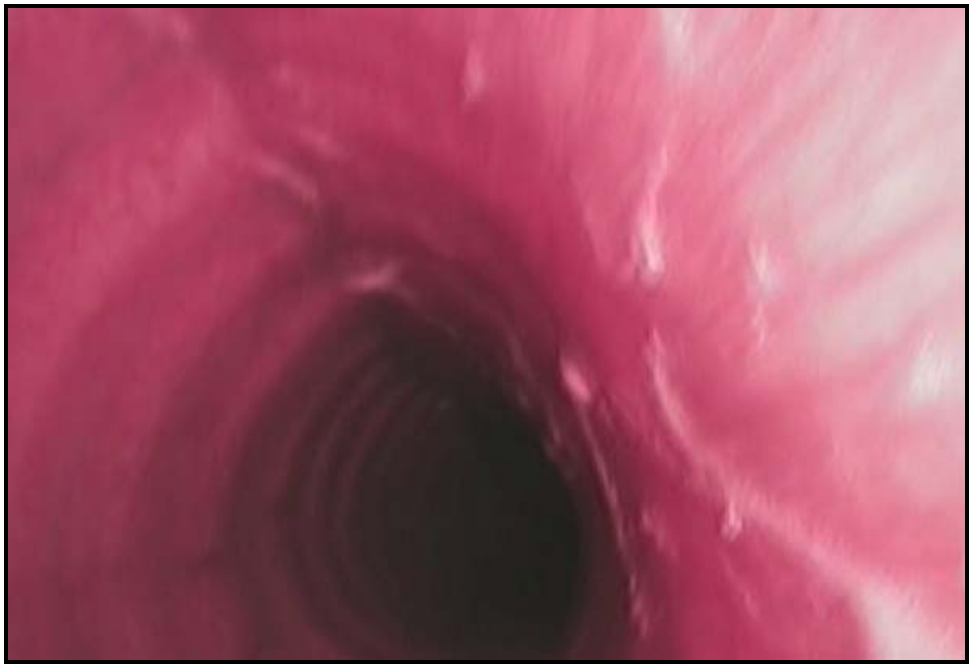


Figure 3.10 A Thoroughbred racehorse with grade 3 tracheal mucous as detected by tracheobronchoscopy.

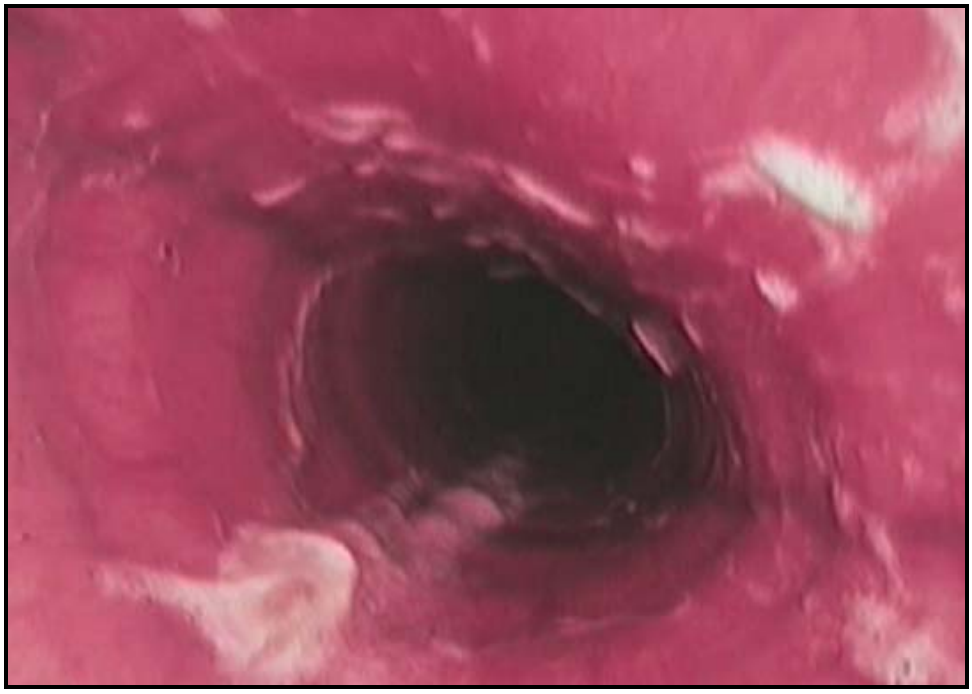


Figure 3.11 A Thoroughbred racehorse with grade 4 tracheal mucous as detected by tracheobronchoscopy.

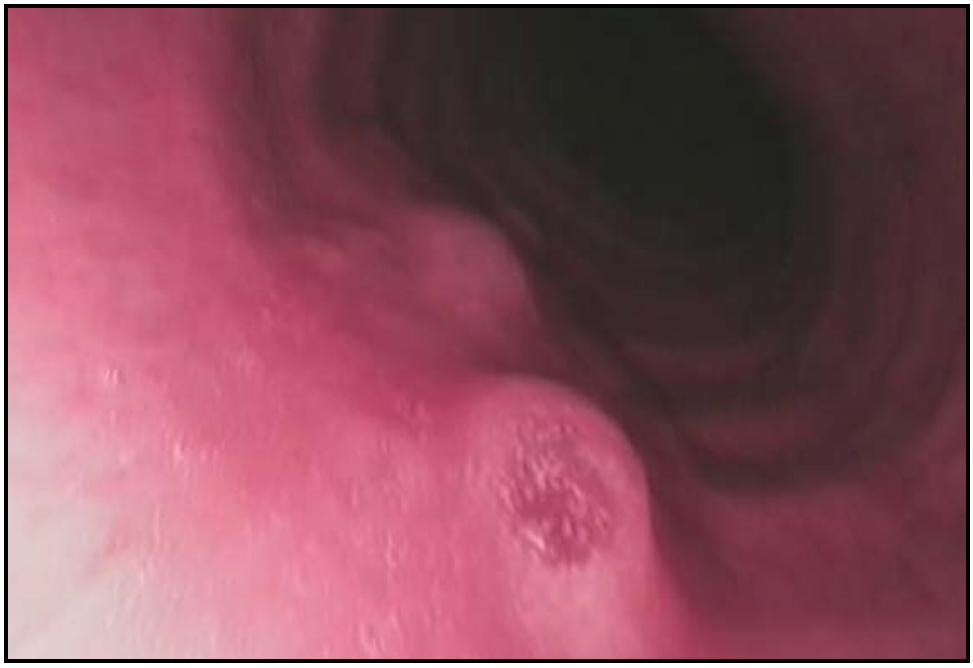


Figure 3.12 A Thoroughbred racehorse with grade 5 tracheal mucous as detected by tracheobronchoscopy.

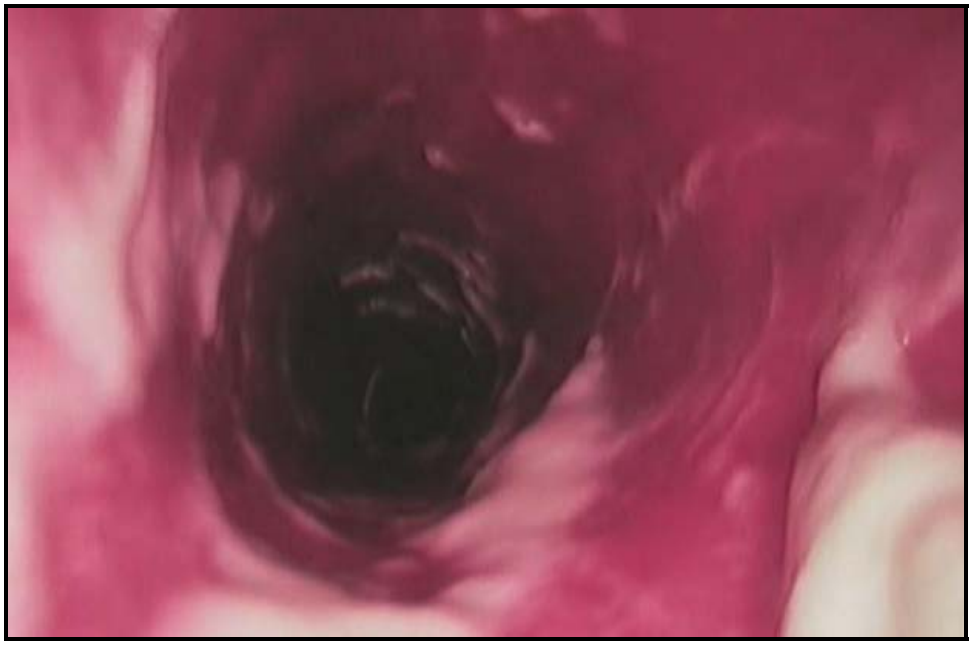


Figure 3.13 A Thoroughbred racehorse with epiglottic entrapment.

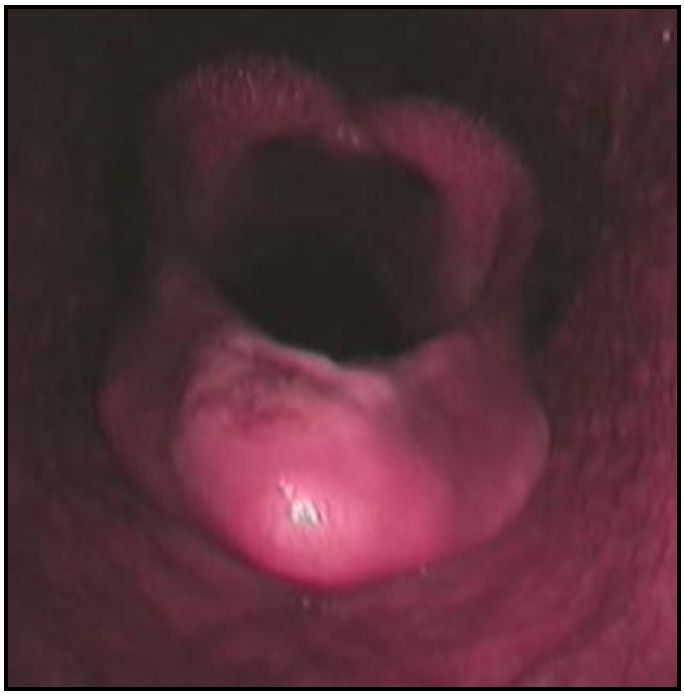


Figure 3.14 A Thoroughbred racehorse with epiglottic entrapment and a sub-epiglottic cyst.

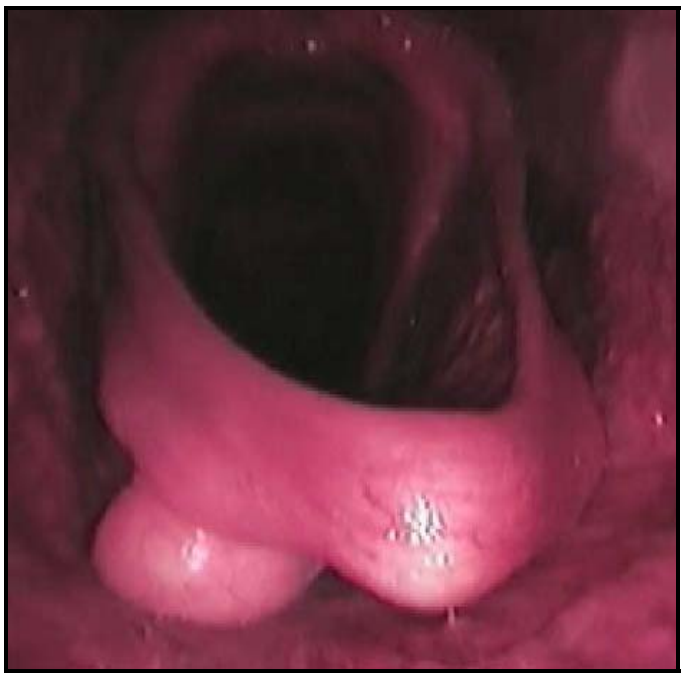


Figure 3.15 A Thoroughbred racehorse with epiglottic deformity.

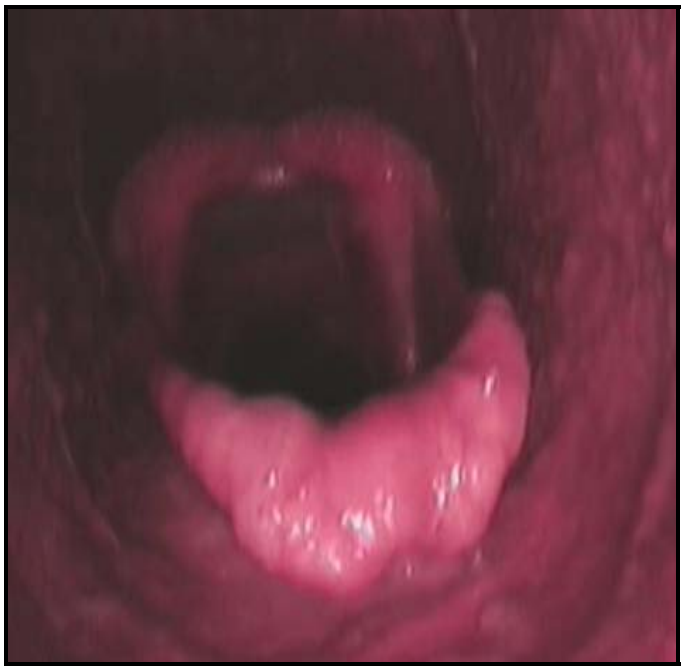
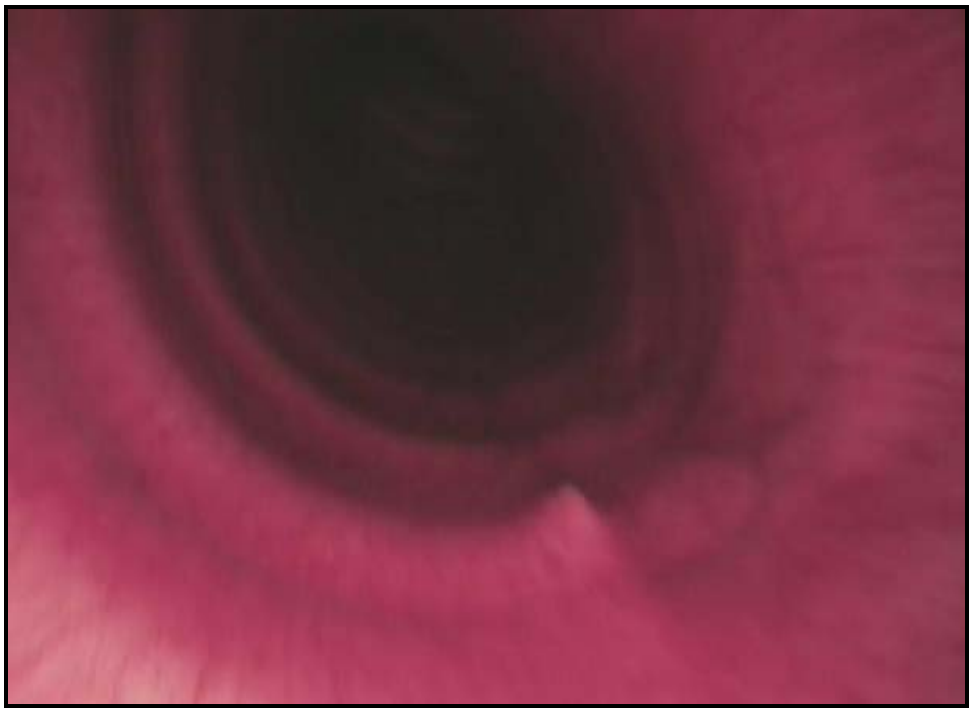


Figure 3.16 A Thoroughbred racehorse with a tracheal cartilage ring spike.



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