Use of successive dynamic videoendoscopic evaluations to identify progression of recurrent laryngeal neuropathy in three horses

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Case Description—3 racehorses were evaluated because of poor performance or abnormal noise originating from the upper portion of the respiratory tract.

Clinical Findings—During maximal exercise, initial dynamic videoendoscopy of the upper respiratory tract revealed complete arytenoid cartilage abduction in 2 horses and incomplete but adequate abduction of the left arytenoid cartilage in 1 horse. Subsequent exercising endoscopic evaluation revealed severe dynamic collapse of the left arytenoid cartilage and vocal fold in all 3 horses.

Treatment and Outcome—2 horses were treated with prosthetic left laryngoplasty and raced successfully. One horse was retired from racing.

Clinical Relevance—Idiopathic laryngeal hemiplegia can be a progressive disease. Successive dynamic videoendoscopic upper airway evaluations were used to confirm progression of left laryngeal hemiplegia in these 3 horses. Videoendoscopy of the upper respiratory tract during exercise should be considered as part of the clinical evaluation of horses with signs of upper respiratory tract dysfunction. (J Am Vet Med Assoc 2007;230:555–558)

ABBREVIATIONS

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
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<tr>
<td>LH</td>
<td>Laryngeal hemiparesis</td>
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<td>LLH</td>
<td>Left LH</td>
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<td>HSTM</td>
<td>High-speed treadmill</td>
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<td>IAD</td>
<td>Inflammatory airway disease</td>
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A 3-year-old male Thoroughbred (horse 1) was examined at the Widener Hospital for Large Animals because of poor performance. According to the trainer, the horse developed signs of exercise intolerance during high-speed exercise. Clinical signs included dramatic deterioration in speed at the end of strenuous exercise and high respiratory rate after exercise. The horse had raced once, albeit unsuccessfully. On initial examination, the horse’s heart rate, respiratory rate, and rectal temperature were within reference limits and other physical examination findings were unremarkable. Per cutaneous palpation of the larynx revealed no abnormalities. When the horse was at rest, videoteleendoscopic evaluation of the upper airway revealed grade III LLH (grading scale of I to V).1

By use of an HSTM,2 the performance of the horse and the dynamic changes within its upper portion of the respiratory tract during exercise were evaluated. Prior to exercise on the HSTM, a lameness examination was performed and the horse appeared sound. Electrocardiography and echocardiography3 were performed before exercise, and findings were within reference limits. The horse was acclimated to the HSTM during a training session (exercising without tack). The horse was then rested until the heart rate returned to the preexercise value. A radiotelemetric monitor4 was secured to a surcingle to record heart rate and rhythm. A 14-gauge catheter5 was placed in the left jugular vein, through which a thermocouple probe6 was passed IV to the level of the right atrium to measure the core body temperature. Catheter placement was confirmed by use of ultrasound guidance. A 20-gauge arterial catheter6 was placed in a transverse facial artery to collect arterial blood samples before and during exercise.

Warm-up exercise consisted of gradually increasing the speed of the HSTM to 7 m/s for a distance of 1,600 m; the HSTM was then stopped. A nose twitch was applied, and a flexible videoendoscope was then introduced into the horse’s pharynx via the right nasal passage and secured to the noseband of the halter. A video recorder8 was used to record the results of the videoendoscopic examination. The HSTM was restarted and quickly increased to a speed of 9 m/s. The treadmill was elevated to 1.5° incline, and the speed was increased to 12 m/s for a distance of 800 m, increased to 13 m/s for a distance of 1,300 m, and then slowed. The total distance over which the horse exercised was 2,600 m. The results of videoendoscopy indicated that the horse’s left and right arytenoid cartilages were able to attain and maintain full abduction during exercise (Figure 1), and a diagnosis of exercising grade IIIA LLH was made.2 Arterial blood samples and core body temperatures for blood gas analysis were obtained 1 minute before exercise, immediately after the warm-up period, at 30-second intervals after the exercise test began, and 1 minute after exercise ended. The samples were immediately placed on ice, and assessments of arterial PaO2 and PaCO2 (temperature corrected) were performed by use of a blood gas analyzer.1 Values of ar-
terial PaO₂ and PaCO₂ were within reference ranges in all samples. Echocardiography performed immediately after exercise revealed no abnormalities. In venous blood samples collected before and 60 minutes after exercise, serum creatine kinase activity was 131 and 190 U/L, respectively (reference range, 90 to 270 U/L). Thirty minutes after exercise, a transendoscopic tracheal aspirate was obtained; no evidence of bleeding within the upper portion of the respiratory tract was observed during the procedure. The tracheal aspirate sample was grossly mucoid. Cytologic examination revealed hemosiderophages consistent with a diagnosis of exercise-induced pulmonary hemorrhage and numerous macrophages, neutrophils, and a large amount of mucous that were suggestive of IAD.

The horse was rested from race training and treated for IAD with dexamethasone powder (0.02 mg/kg [0.01 mg/lb], PO, q 48 h) for 14 days. Other treatments included beclomethasone (2,000 µg from a metered-dose inhaler, q 12 h) and salmeterol (200 µg from a metered-dose inhaler, q 12 h) administered for 14 days by use of a tight-fitting mask placed over the nose. The horse responded well to treatment and returned to training; it subsequently competed successfully in 15 races and earned $15,550 in race winnings.

Five hundred and fifty-two days after initial examination, the horse, now a 5-year-old gelding, was returned to the teaching hospital because of poor performance and an upper respiratory tract noise. On evaluation, the horse's heart rate, respiratory rate, and rectal temperature were within reference limits. Percutaneous palpation of the larynx revealed a prominent muscular process of the left arytenoid cartilage. Videoendoscopic evaluation of the upper respiratory tract when the horse was at rest revealed grade III LLH. To evaluate performance during exercise, an HSTM exercise test was performed as previously described. During the evaluation, the horse achieved a maximum speed of 12.8 m/s for a distance of 1,400 m at a 1° incline; overall, the horse traveled a distance of 2,300 m. Videoendoscopic evaluation of the upper portion of the respiratory tract during exercise revealed severe dynamic collapse of the left arytenoid cartilage and vocal fold (Figure 2), and a diagnosis of exercising grade IIIC LLH was made. Blood gas analysis of the sample collected during maximal exercise revealed that PaO₂ was 76 mm Hg (reference range at 12.8 m/s, ≥ 79 mm Hg) and PaCO₂ was 42 mm Hg (reference range at 12.8 m/s, ≤ 52 mm Hg). Cytologic examination of a transendoscopic tracheal aspirate obtained after exercise revealed an abundance of mucous and inflammatory cells suggestive of IAD.

On the basis of results of the videoendoscopic evaluation performed during exercise, a diagnosis of progressive LLH was made. The horse was anesthetized, and a left prosthetic laryngoplasty and laser ventriculocordectomy were performed. Via an oral approach, the left ventricle was everted by use of bronchoesophagoscopic grasping forceps under videoendoscopic guidance. The left vocal fold and ventricle were then excised by use of a diode laser and removed. Eighty-one days after surgery, the horse returned to racing; during the following 2 years, the horse had no abnormal upper respiratory tract noise and its race earnings were $2,955.

A 3-year-old Thoroughbred gelding (horse 2) was evaluated at the hospital because of poor performance and a barely audible upper respiratory tract noise. The horse had raced on 5 occasions, earning $2,520. In the race immediately preceding referral, the horse's speed rapidly declined in the last half mile. Resting endoscopic examination of the upper respiratory tract revealed grade II LLH. Findings of external palpation of the larynx were unremarkable. To evaluate the horse during exercise, procedures similar to those used to as-
A 3-year-old male Arabian (horse 3) was examined at the hospital because of abnormal, loud upper respiratory tract noise. Since the last examination, the horse had returned to race training and, until recently, had been performing satisfactorily. Percutaneous palpation of the larynx revealed a prominent muscular process of the left arytenoid cartilage. When the horse was at rest, endoscopic examination of the upper portion of the respiratory tract revealed grade III LLH. The horse was exercised on the HSTM; it achieved a maximum speed of 11.5 m/s at 1° incline and traveled a total distance of 1,300 m. During exercise, videendoscopic evaluation revealed mild collapse of the right and left laryngeal walls and severe dynamic collapse of the left arytenoid cartilage and vocal fold; a diagnosis of exercising grade IIIIC LLH was made. A transendoscopic tracheal aspirate was collected after exercise contained many inflammatory cells, suggestive of mild IAD. The horse was treated for IAD with 2 months of rest from race training.

As a 4-year-old gelding, horse 2 was returned to the teaching hospital 279 days after the initial evaluation because of poor performance and upper respiratory tract noise. Since the last examination, the horse had returned to race training and, until recently, had been performing satisfactorily. Percutaneous palpation of the arytenoids revealed a prominent muscular process of the left arytenoid cartilage. When the horse was at rest, endoscopic examination of the upper portion of the respiratory tract revealed grade III LLH. The horse was exercised on the HSTM; it achieved a maximum speed of 10.5 m/s at 1° incline and traveled a total distance of 1,500 m. During exercise, videendoscopic evaluation revealed mild collapse of the right and left laryngeal walls and severe dynamic collapse of the left arytenoid cartilage and vocal fold; a diagnosis of exercising grade IIIIC LLH was made. A transendoscopic tracheal aspirate was collected after exercise. Cytologic examination of that sample revealed numerous hemosiderophages (consistent with exercise-induced pulmonary hemorrhage) and many neutrophils and macrophages that were suggestive of IAD. During the endoscopic procedures, there was no visual evidence of bleeding within the upper portion of the respiratory tract.

Given the progression of LLH, horse 2 underwent left prosthetic laryngoplasty and laser ventriculocorpectomy. The horse recovered from surgery without complications; it raced on 4 occasions during the following year, and its race earnings were $1,500. As a 4-year-old gelding, horse 2 was returned to the teaching hospital 279 days after the initial evaluation because of abnormal, loud upper respiratory tract noise. Since the last examination, the horse had returned to race training and, until recently, had been performing satisfactorily. Percutaneous palpation of the arytenoids revealed a prominent muscular process of the left arytenoid cartilage. When the horse was at rest, endoscopic examination of the upper portion of the respiratory tract revealed grade III LLH. The horse was exercised on the HSTM; it achieved a maximum speed of 11.5 m/s at 1° incline and traveled a total distance of 1,300 m. During exercise, videendoscopic evaluation revealed mild collapse of the right and left laryngeal walls and severe dynamic collapse of the left arytenoid cartilage and vocal fold; a diagnosis of exercising grade IIIIC LLH was made. A transendoscopic tracheal aspirate was collected after exercise. Cytologic examination of that sample revealed numerous hemosiderophages (consistent with exercise-induced pulmonary hemorrhage) and many neutrophils and macrophages that were suggestive of IAD. During the endoscopic procedures, there was no visual evidence of bleeding within the upper portion of the respiratory tract.

The remaining 3 horses of this report, endoscopic examination of the upper respiratory tract revealed laryngeal grades determined at rest.

Discussion

To the authors’ knowledge, this is the first report of the use of successive dynamic videendoscopic evaluation to substantiate progression of LLH in horses. The value of videendoscopic evaluation of the upper respiratory tract during exercise is well documented.6,7,17 It is the preferred method for assessment of dynamic upper respiratory tract function in horses. It is particularly indicated for horses with questionable laryngeal function.6,7,14,17 and in 1 study of 75 horses undergoing endoscopic evaluations during treadmill exercise because of abnormal respiratory tract noise, correlation between diagnoses made when horses were at rest and during exercise was only 25%. In the 3 horses of this report, endoscopic examination of the upper respiratory tract revealed laryngeal dysfunction during rest (horses 1 and 3, grade III LLH; horse 2, grade II LLH) but revealed complete or almost complete abduction of the arytenoid cartilages during exercise. Laryngoplasty was not recommended as treatment of the 3 horses. In the 2 horses with normal laryngeal function during exercise after treatment for acute IAD, both horses raced successfully for an additional 9 to 18 months. Subsequent endoscopic examination during exercise revealed severe dynamic collapse of the left arytenoid cartilage and vocal fold in all 3 horses. Laryngoplasty was recommended for treatment of the 3 horses and performed in 2 horses, both of which returned to race successfully.

Although it is commonly believed that complete arytenoid cartilage paralysis is preceeded by progressive deterioration, there are few reports validating this statement. On the contrary, reports of laryngeal variation on repeat examination8 and asynchronous laryngeal movement without progression to hemiplegia8 are commonly cited. The reported incidence of the progression of laryngeal dysfunction is low (5% to 15%).8,10 Clinical signs attributed to the development of LH include development or worsening of upper respiratory tract noise,10,11 decline in performance10,12 and worsening of endoscopically derived laryngeal grades determined at rest.8,10 Analysis of data from horses with LH has identified varying duration of onset of signs from a sudden to a hardly noticeable increase in inspiratory noise developing over weeks to months,11 the development of poor performance during a period of < 6 months,12 and laryngeal function deterioration (determined via repeated standing endoscopic evaluation) at 14.6 to ≥ 16 months after initial examina-
tion. In the horses of this report, the interval between the examinations involving the HSTM in horses 1 and 2 was 9.3 and 18.4 months, respectively. For horse 3, the interval was only 2 months, indicative of a more rapid rate of progression. This was not completely unexpected for horse 3 because the initial videendoscopic examination during exercise revealed submaximal left arytenoid cartilage abduction. Although our findings clearly represent progression of LH, an exact time line for disease progression cannot be ascertained from the data from these 3 horses. However, as the findings for horse 3 suggest, it is possible that horses with exercising grade IIIIB LLH have comparatively more rapid progression of LH.

All 3 horses of this report had or developed an abnormal respiratory tract noise during exercise, and laryngeal dysfunction was suspected. Abnormal respiratory tract noise is frequently attributed to LH, and spectral analysis of respiratory noises in exercising horses with experimentally induced LLH has identified a unique LH-associated inspiratory sound pattern. In another study, the worsening of exercise-related respiratory tract noise was the only criterion used for identification of progressive recurrent laryngeal neuropathy in 22 of 52 horses. However, abnormal respiratory tract noises have been attributed to other laryngeal and pharyngeal dysfunctions, and abnormal noises for which the origin remains undetermined (even after collection of endoscopic data at rest and during exercise) have also been reported. In addition, identification of experimentally induced LLH and bilateral alar fold paralysis by use of noise spectrograms alone resulted in substantial error. In 2 horses of this report, initial videendoscopic evaluation of the upper respiratory tract during exercise revealed that abnormal noise was the result of pharyngeal collapse (horse 2) and bilateral axial deviation of the membranous portion of the aryepiglottic folds (horse 3); on subsequent evaluation, the abnormal noise was a result of left arytenoid cartilage and vocal fold collapse in all 3 horses. Without the findings of videendoscopic evaluation of the upper respiratory tract during exercise, the mere presence of a respiratory noise did not assist in making an accurate diagnosis. The authors caution that the detection of noise should not be used as the sole diagnostic marker for laryngeal dysfunction in horses without application of additional diagnostic aids.

Atrophy of the left cricoarytenoideus dorsalis muscle has been significantly associated with abnormal movements of the left arytenoid cartilage. In 1 study, all horses with laryngeal dysfunction during exercise had palpable loss of cricoarytenoideus dorsalis muscle mass. In another report, horses with cricoarytenoideus dorsalis muscle atrophy were subsequently diagnosed as having LH. In the horses of the present study, the presence of a prominent muscular process of the arytenoid cartilage was associated with laryngeal dysfunction during exercise. Initially, the larynx appeared symmetrical on external palpation and endoscopy of the upper respiratory tract during exercise revealed complete or almost complete arytenoid cartilage abduction in all 3 horses. However, subsequent examination of the horses revealed a prominent muscular process of the left arytenoid cartilage and LLH during exercise. The progression of left recurrent laryngeal neuropathy was identified by use of successive dynamic videendoscopic evaluations of the upper respiratory tract in these horses; such evaluations should be considered for assessment of and monitoring changes in laryngeal function in horses during exercise and rest.

a. GIF-Q gastroscopy with CLV V20 light source and CV-100 image processor, Olympus, Columbia, Mo.
c. VINGMED CFM800, VINGMED SOUND A/S, Horten, Norway
e. Angiocath, Becton-Dickinson, Sandy Utah
f. Physiotemp type IT-14, Physitemp Instruments Inc, Clifton, NJ.
g. Abbocath-T, Abbott Ireland, Sligo, Ireland.
h. SVO 9500 MDX videorecorder, Sony Electronics Medical Division, Montvale, NJ.
i. CIBA-GEIGY 208 BG, Norwood, Mass.

References