



Disorders of the larynx

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Airway obstruction results in a decrease in airflow and an increase in airway resistance. Airway obstruction is therefore a frequent cause of poor performance in horses, particularly in sport horses. Obstruction to airflow may occur at the nares, nasal passages, pharynx, larynx, or trachea. The purpose of this article is to describe laryngeal disorders in the horse that disrupt airflow.

Anatomy

The larynx is a short tube that connects the pharynx and trachea. It is a complex apparatus that regulates the volume of air in respiration, prevents aspiration, and is the main organ of vocalization. The larynx is composed of the cricoid, thyroid, and epiglottic and paired arytenoid cartilages. The cricoid cartilage is positioned rostral to the first tracheal ring and is connected to the trachea by the cricotracheal membrane [1]. The thyroid cartilage lies rostral to the cricoid cartilage. The arytenoid cartilages are positioned on either side of the cricoid cartilage. The arytenoid cartilages comprise the body, the muscular process, the vocal process, and the corniculate process. The cricoid and arytenoid cartilages are connected by diarthrodial joints that allow the arytenoid cartilages to abduct and adduct [1]. The epiglottis is an elastic cartilage that has the form of an oblong leaf. It is positioned on the dorsal surface of the body of the thyroid cartilage, and its position is maintained by the thyroepiglottic ligaments [1].

The intrinsic muscles of the larynx innervated by the recurrent laryngeal branch of the vagus nerve include the cricoarytenoideus dorsalis, the principal abductor of the larynx; the arytenoid transversus, which draws

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the dorsomedial margins of the arytenoid cartilages together; and the thyroarytenoideus, arytenoideus transversus, and cricoarytenoideus lateralis, the adductors of the larynx [2].

The laryngeal mucosa forms outpouchings approximately 2.5 cm deep, which pass laterally between the vocal and vestibular folds but remain within the protection of the thyroid lamina. These outpouchings form the laryngeal ventricles, which have a capacity of 5 to 6 mL [1].

Laryngeal hemiplegia

Laryngeal hemiplegia, limiting performance in horses and causing upper respiratory tract noise, has been recognized since the 1800s [3]. Left laryngeal hemiplegia limits the performance of horses by decreasing the cross-sectional area of the rima glottidis, resulting in decreased inspiratory flow, increased respiratory resistance, hypercapnia, hypoxemia, exercise intolerance, and excessive inspiratory noise. It has been suggested that at least 40% of large-breed horses have some degree of laryngeal asymmetry [4]. The incidence of neuropathy reported in the equine population ranges from 2.6% to 8.3% [5,6].

Laryngeal function in the horse is primarily assessed by endoscopic examination. Various grading systems for evaluating resting laryngeal function have been established. The system used most consistently grades laryngeal function from I to IV [7]. This system evaluates movement and the degree of abduction of the arytenoids during nasal occlusion or swallowing. Grade I describes symmetric and synchronous movement of the arytenoid cartilages, grade II describes asynchronous but symmetric movement of the arytenoid cartilages, grade III laryngeal hemiparesis describes asynchronous and asymmetric movement of the arytenoid cartilages, and grade IV describes no perceptible movement of the arytenoid cartilages. During high-speed treadmill evaluation, some horses evaluated as grade III at rest maintain full abduction of the arytenoid cartilages during exercise. Subsequently, a grading system has been proposed for horses suffering from grade III laryngeal hemiparesis during exercise [8]. Horses were classified as having grade IIIA laryngeal hemiparesis if they were able to maintain full abduction during exercise, as having grade IIIB laryngeal hemiparesis if they were able to maintain the left arytenoid and vocal fold in a relatively fixed but incompletely abducted position, and as having grade IIIC laryngeal hemiparesis if they had severe collapse of the left arytenoid and vocal fold during exercise. Thus, horses with grade IIIB or C laryngeal hemiparesis or grade IV laryngeal hemiplegia are candidates for surgical intervention.

In 1970, Marks et al [9] introduced the laryngoplasty technique to correct laryngeal hemiplegia surgically. Other techniques that have been described for the treatment of laryngeal hemiplegia include arytenoidectomy [10], laryngeal reinnervation [11], ventriculectomy, and ventriculocordectomy [12]. Although laryngeal reinnervation is an attractive technique for phys-

iologically correcting laryngeal hemiplegia, laryngoplasty remains the preferred method for preventing dynamic collapse of the larynx for most surgeons. Since the original description of the technique, experimental studies have evaluated the effects of the prosthetic laryngoplasty in horses with idiopathic and induced recurrent laryngeal nerve paralysis and have demonstrated that the upper airway mechanics of these horses were restored by performing laryngoplasty surgery [13,14].

Success rates for laryngoplasty have been reported to range from 48% to 95% [4,15–18]. This wide range of success rates is a result of the different criteria chosen by each author to describe success. A 58% success rate was reported when a decrease in upper respiratory tract noise was used as a marker for success [4]. An 85% success rate was reported when the postoperative racing performance of horses undergoing laryngoplasty was compared with that of an aged-matched control group [18]. Three studies used the evaluation of follow-up information obtained through owner/trainer questioning and race records as a criterion for success [15–17]. One of these studies reported that Thoroughbred racehorses had a much lower success rate (48%) compared with breeds that were not intended for racing (95%) [17]. The second report, which included a population of racing Thoroughbreds and Standardbreds, found that postoperative racing performance for 167 horses was improved in 69% when evaluated subjectively by owners/trainers and improved in 56% based on performance index scores [16]. The third study used these criteria to investigate the effects of laryngoplasty on the performance of Thoroughbred racehorses suffering from only grade III left laryngeal hemiparesis and reported a success rate between 50% and 60% [15].

The primary complication of laryngoplasty surgery is failure of the prosthesis to maintain abduction, resulting in exercise intolerance and upper respiratory tract noise. Other complications include coughing, pneumonia, suture sinus infection, and incisional infection. In a recent report, loss of arytenoid abduction in the postoperative period may have contributed to laryngoplasty failure in 20% of horses [15]. Surgical options after laryngoplasty failure are limited to either arytenoidectomy or repeat laryngoplasty [19,20].

An early hypothesis for laryngoplasty failure was that repeated residual contraction of the cricoarytenoideus muscle in horses suffering from grade III left laryngeal hemiparesis might predispose this group of horses to laryngoplasty failure. It was proposed that the grade of laryngeal function might affect outcome after laryngoplasty and that horses treated for grade III left laryngeal hemiparesis may have a higher rate of laryngoplasty failure than horses treated for grade IV left laryngeal hemiplegia [21,22]. Possible modes of prosthetic failure include acute mechanical cartilage failure, cyclic cartilage failure resulting in gradual prosthesis loosening, improper prosthesis placement resulting in biomechanical disadvantage, and a diseased state that renders the cartilage weaker than normal. The repetitive forces

acting on the prosthesis cartilage interface include local muscle contractions and changing luminal biometric pressures during strenuous exercise. Although changing luminal pressures are similar in all grades of horses, repeated residual contraction of the cricoarytenoideus dorsalis muscle in horses treated for grade III left laryngeal hemiparesis may cause the prosthesis to loosen or cut through the cartilage, resulting in gradual loss of arytenoid abduction or complete laryngoplasty failure. A recent study demonstrated that eliminating contraction of the cricoarytenoideus dorsalis muscle by performing a recurrent laryngeal neurectomy in conjunction with laryngoplasty and unilateral ventriculocordectomy did not improve the postoperative outcome of Thoroughbred horses treated for left laryngeal hemiparesis [15]. Therefore, recurrent laryngeal neurectomy is not currently recommended in the treatment of horses with grade III left laryngeal hemiparesis.

Attention has also been given to the suture material used for the prosthesis. It has been proposed that the nature of the prosthesis, such as its diameter and surface texture, may affect the ability of the laryngeal cartilages to retain the suture [23]. The material used for the prosthesis usually consists of one or two strands of number 2 or 5 nonabsorbable suture material, such as polyester or nylon. The suture material used is largely decided by surgeon preference. We currently recommend using two strands of number 5 coated polyester suture. More recently, Schumacher et al [24] developed a laryngoplasty prosthesis composed of a steel cable and stress-reducing washers that resisted higher forces than number 5 polyester suture. To the authors' knowledge, this prosthesis has not been tested in vivo and may not be addressing the primary reason for laryngoplasty failure. Methods to evaluate and prevent suture pullout are also being considered [25] and warrant further investigation.

Axial collapse of the vocal cord

Analysis of videotapes of the upper respiratory tract of horses exercising on a high-speed treadmill frequently reveals axial collapse of the ipsilateral cord in horses with laryngeal hemiplegia. The inclusion of a ventriculocordectomy for horses treated for left laryngeal hemiplegia by prosthetic laryngoplasty remains controversial. The literature is contentious, and its inclusion as part of the treatment regimen is usually decided by surgeon preference. The complication rate of ventriculocordectomy surgery is low; however, the procedure does increase surgery time. Observations of axial displacement of the vocal fold and ventricle during high-speed treadmill stress tests in horses previously treated by laryngoplasty for laryngeal hemiplegia have led these authors to believe that ventriculocordectomy is beneficial in the treatment of horses with laryngeal hemiplegia and should be included in the treatment regimen.

In affected horses, ventriculocordectomy can be performed as the sole procedure or before laryngoplasty via an oral approach with the horse anesthetized. The methods used for performing ventriculocordectomy are diverse; some surgeons perform the surgery through a laryngotomy using sharp dissection, whereas others have adopted laser techniques [26,27]. Both the neodymium:yttrium (Nd:Yag) and diode laser have been used to perform the procedure by means of contact and noncontact techniques. The contact technique has the advantages of increased precision in removing the ventricle and causing less thermal damage to surrounding tissues. Also, mucocele formation has only been a complication of noncontact techniques. Logically, removing the maximum amount of tissue that could potentially evert into the airway is beneficial. We therefore recommend performing a laser ventriculocordectomy [27]. The procedure is easily performed under general anesthesia before the laryngoplasty procedure, and surgery time averages between 7 and 15 minutes.

Right laryngeal hemiplegia

In most cases, the etiology of left laryngeal hemiplegia is idiopathic, resulting from progressive loss of myelinated nerve fibers in the left recurrent laryngeal nerve. This is generally not the case for right laryngeal hemiplegia, which is rare in horses. Therefore, evaluation of a horse suffering from right laryngeal hemiplegia should include a comprehensive examination, looking for the cause of the hemiplegia. Other causes of damage to the recurrent laryngeal nerve include perivascular jugular vein injection, guttural pouch mycosis, trauma from injuries or surgical procedures of the head and neck, strangles abscessation of the head and neck, and impingement by neoplasms in the head and neck [4]. In addition, organophosphate toxicity, plant poisoning, lead toxicity, and central nervous system diseases can occasionally cause laryngeal hemiplegia [28,29]. Right laryngeal hemiplegia has been associated with congenital malformation of the laryngeal cartilages; therefore, careful palpation of the muscular process of the arytenoid cartilage is an important part of the physical examination. In one study of horses treated for right laryngeal hemiplegia, 7 of 11 Thoroughbreds had a congenital malformation of the laryngeal cartilages [30]. Because the rate of laryngoplasty failure in this group of horses is high, partial arytenoidectomy is often recommended. In the same study [30], there was also a high incidence of abnormal histories and abnormal physical examination findings, including pulmonary infections, history of general anesthesia, jugular vein phlebitis, and Horner's syndrome. Interestingly, spontaneous recovery from right laryngeal hemiplegia in a horse has been reported in a case where the cause of the hemiplegia was unknown [31].

The surgical options are as previously described for left laryngeal hemiplegia. The prognosis after surgery is poor compared with that reported

for left laryngeal hemiplegia, with the performance of only 27% of racehorses surgically treated for right laryngeal hemiplegia being improved or retained after surgical intervention [30].

Arytenoid chondrosis

The etiology of arytenoid chondrosis is not clearly defined, but the condition likely occurs secondary to ascending infection and inflammation from a mucosal lesion. In the acute stages, horses may be presented in respiratory distress with marked laryngeal edema. These horses can usually be treated aggressively with intravenous antimicrobials and anti-inflammatories, obviating the need for a tracheotomy or immediate arytenoidectomy. Whereas medical treatment often resolves the emergency, the resulting abnormal arytenoid results in residual laryngeal obstruction from swelling of the arytenoid body and decreased abduction. The limited abduction is thought to be secondary to confinement of the swollen arytenoid by the wing of the thyroid cartilage. This is likely true in part, and this presumption has significant implications on forms of treatment. If there is a mechanical obstruction to abduction, laryngoplasty cannot be effective in regaining abduction. The other contributing factor to limited abduction may be from an undiagnosed degree of laryngeal hemiplegia that precipitated the mucosal lesion.

Arytenoid chondrosis in the more chronic stages may look like uncomplicated laryngeal hemiplegia on resting endoscopic examination. Infrequently, the mucosal abrasion is not visible until the endoscope is passed into the larynx, and the corniculate may even be smaller than the normal size. Close attention should be paid to the symmetry of the corniculates, the degree of abduction, and palpation of the muscular process. An uncomplicated grade IV laryngeal hemiplegic horse should have a prominent muscular process on palpation of the larynx and no abnormalities to the corniculate on endoscopic examination. With an uncomplicated laryngeal hemiplegia, the palatopharyngeal arch should be collapsed behind the corniculate and not visible. If these criteria are absent, and the findings are thus not consistent with grade IV laryngeal hemiplegia, chondrosis should be diagnosed. Occasionally, there are granulation tissue kissing lesions on the opposing “normal” arytenoid that appear worse than the deformity on the affected arytenoid. The clinician should not be fooled by the larger amount of granulation tissue, and the degree of abduction should help to determine which arytenoid is the primary offender (Fig. 1).

Surgical treatment and prognosis

A partial arytenoidectomy, which removes all components but the muscular process, has been proven to be the most effective form of



Fig. 1. Right arytenoid chondrosis with an abnormally shaped immobile corniculate. A larger amount of granulation tissue is present on the right in a fully abducted arytenoid.

arytenoidectomy at improving airway dynamics [32]. Although horses with an arytenoidectomy may have improved airway dynamics, they often continue to make some abnormal respiratory noise and may not be suitable for showing.

An arytenoidectomy is performed through a standard laryngotomy approach. A temporary tracheotomy is required to administer the anesthetic gas during the surgical procedure of a partial arytenoidectomy. If there is a large enough lumen to pass an endotracheal tube through the larynx after inducing anesthesia, the tracheotomy is performed under general anesthesia and the tube is switched to the tracheotomy site while the horse is under anesthesia. A headlamp is useful for illumination while working within the larynx. Placing the endoscope through the nares in front of the larynx can also supplement light. It is always best to try and salvage a mucosal flap on the axial side of the arytenoid to achieve primary mucosal closure after the arytenoid is removed [10]. This minimizes the prospect of granulation tissue forming after surgery.

To form the mucosal flap, mucosal incisions are made from a dorsal-to-ventral direction at the caudal border of the arytenoid and the rostral border just caudal to the corniculate or any granulation tissue. These incisions are connected with a horizontal incision along the ventral border of the arytenoid. The mucosa is slowly dissected free from the arytenoid and left attached dorsally. The abaxial border of the arytenoid is then freed of its muscular attachments primarily by blunt dissection to minimize hemorrhage. The muscular process is isolated and transected. The arytenoid is then elevated and removed by cutting the remaining corniculate mucosa rostrally and any remaining dorsal attachments. The caudal edge of the mucosal flap is reapposed to the laryngeal mucosa in a simple continuous pattern with absorbable suture, working in a dorsal-to-ventral direction. The rostral edge of the mucosal flap is apposed similarly to the remaining mucosa abaxial to the corniculate in a parallel line to the caudal edge. The ventricle and vocal chord are resected, and this becomes the ventral aspect of the flap that is left open to drain. Bleeding should be minimal once the mucosal edges are apposed. If there is extensive purulent material abaxial to the arytenoid, a mucosal closure is not recommended. A tracheotomy tube replaces the larger endotracheal tube for recovery and is maintained overnight.

The tracheotomy tube can typically be removed the morning after surgery. The horse should be maintained on perioperative antimicrobials and anti-inflammatories for 1 week while being maintained in a stall for 1 month with only hand grazing. The tracheotomy and laryngotomy sites are left open to heal in by second intention. All feeding should take place from the ground to minimize the risk of aspiration. A 1-month endoscopic examination should be performed to determine the presence of granulation tissue. Once there is complete mucosal healing, the horse should receive 1 month of turnout before resuming exercise.

The technical difficulty of an arytenoidectomy has resulted in several complications. The most common complications after an arytenoidectomy are excessive residual mucosa or secondary granulation tissue. This should be removed at the first month and can be performed with standing sedation and videoendoscopic laser excision. Without removal in the early stages, the tissue may mineralize and make excision much more difficult later. A more serious life-threatening complication is aspiration. The risk may be dramatically decreased by less traumatic dissection of the arytenoid.

The prognosis is extremely variable and dependent on the extent of the disease and the time to treatment. Many horses that do not have great respiratory demands and have a mild form of chondrosis in a chronic nonactive state can function quite adequately without surgery. Horses with concurrent severe hemiplegia or more severe chondrosis are likely to require surgical intervention to provide an airway for any athletic function. Most of these horses return to athletic function but decrease in their racing or athletic ability. Horses with severe bilateral disease are unlikely to return to any significant athletic function [20].

Intralaryngeal granulation tissue

Granulation tissue masses arising from the axial surface of the arytenoid cartilages can result in airway narrowing, upper respiratory tract noise, and exercise intolerance in horses [33]. The cause of these arytenoid cartilage masses is unknown, but laryngeal trauma with mucosal damage most likely initiates the problem secondary to either foreign material or concussive forces occurring between the left and right arytenoids. Intralaryngeal granulation tissue masses most commonly occur in racehorses but have also been reported in horses not used for racing, although in these horses, lesions were not detected until stertor was present at rest and the airway was nearly obstructed [34]. Typically, endoscopically, a mass ranging from a few millimeters to approximately 3.5 cm is seen arising from the medial surface of the corniculate process of the arytenoid just dorsal to the vocal cord attachment. Eighty-two percent of granulation tissue masses arise from the right arytenoid cartilage [33]. An ulcerated mucosal defect is often seen on the medial surface of the opposing arytenoid. Treatment options include a partial arytenoidectomy or surgical removal of the intralaryngeal mass alone if the granulation tissue mass emanates from a normal or only slightly affected arytenoid cartilage. Removal of the mass alone may be performed through a laryngotomy [19,35] or transendoscopically in standing horses using a laser [33]. Excision of the granulation tissue allows the mucosal defect to heal and prevents contact injury with the opposing arytenoid. Removal of the granulation tissue does not reverse or eliminate inflammation of the underlying arytenoid cartilage, however. If diagnosed before severe involvement of the arytenoid cartilage, laser excision is beneficial in returning horses to athletic activity. Underlying cartilage thickening or accompanying laryngeal disease substantially worsens the prognosis for return to full athletic performance, however. In approximately 65% of horses, the concave defect created by granulation tissue excision heals without regrowth. In the remaining 35%, some respond to a second standing laser surgery, whereas in others, the preexisting cartilage disease continues to progress after removal of the mass, resulting in further airway narrowing and necessitating a partial arytenoidectomy.

Hyperkalemic periodic paralysis

Hyperkalemic periodic paralysis (HYPP)-affected horses frequently exhibit stridor associated with exercise, excitement, stress, or episodes of muscle paralysis. The laryngeal and pharyngeal muscles seem to be more severely affected than other muscles. Common endoscopic findings in horses homozygous for HYPP include pharyngeal collapse, pharyngeal edema, laryngopalatal dislocation, and laryngeal paralysis [36]. Approximately 50% of horses receiving acetazolamide have decreases in stridor while receiving medication [36].

Epiglottis

Epiglottic entrapment

Persistent or intermittent epiglottic entrapment is a common cause of upper respiratory tract noise and exercise intolerance in racehorses [37–43]. The aryepiglottic folds attach along the free edge of the epiglottis, blending in with the dorsal epiglottic mucosa. Ventral to the epiglottis, the mucosa is loosely attached and normally compressed in an accordion-like fashion. The term *epiglottic entrapment* refers to this ventral tissue becoming abnormally positioned above the dorsal epiglottic surface. Typically, over time, the entrapped membranes become thickened, and in approximately 45% of horses, the tissue also becomes ulcerated [43]. The etiology of the disease is unclear; however, epiglottic hypoplasia can be a predisposing factor, with approximately 30% of affected horses having some degree of epiglottic hypoplasia [41,43,44].

Diagnosis is routinely performed using resting endoscopy; however, for horses that only entrap intermittently, endoscopy during a speed treadmill stress test may be necessary for diagnosis. Two of 348 horses suffering from poor performance were diagnosed with intermittent epiglottic entrapment during a high-speed stress test [45].

There are several described methods of surgical correction of epiglottic entrapment, including transendoscopic laser axial division [43,46,47], transnasal or transoral axial division using a curved bistoury [38,39,42], transendoscopic electrosurgical axial division [40], and surgical excision via a laryngotomy [37,38,41]. Transendoscopic laser correction of the entrapment can be performed safely in the standing horse and has the advantage of allowing intraoperative evaluation of the surgical correction. After laser axial midline division, swallowing is induced in the standing horse and the entrapping membranes retract into a lingual epiglottic position. If necessary, minor adjustments to the length of the incision can then be performed and any residual tissue can be transected. Over time, the edema and thickening resolve, even if the tissue is ulcerated and thickened. Further debridement is usually not necessary. Endoscopic appearances that may preclude successful laser correction in standing horses include persistent dorsal displacement of the soft palate, intermittent epiglottic entrapment, and excessively bulky and fibrotic membranes that do not open and retract after axial division of the membranes. Careful excision of the tissue through a laryngotomy under general anesthesia is recommended for these cases. The most common complication after surgical correction of entrapped epiglottis is dorsal displacement of the soft palate, which occurs in approximately 15% of horses [43].

When using a curved bistoury, surgery is generally performed by means of an oral approach. General anesthesia avoids the danger of inadvertent incision of the soft palate or other adjacent structures should the horse

swallow. The successful treatment of an entrapped epiglottis with a Scanlan hook knife in 29 standing sedated horses has also been reported [38]. The authors stressed the need for adequate restraint and analgesia of the pharynx and larynx. No palatal injury occurred in any horse, although in one case, the blade became embedded in the nasal septum when the horse jumped; however, the blade was retrieved without complication.

The recurrence rate after surgical correction of an entrapped epiglottis using laser axial midline division is 5%, it is approximately 10% using a curved bistoury [41,42], and it is as high as 40% using electro-surgical division [40].

Epiglottitis

The etiology of epiglottitis is unknown. Possible predisposing factors include pharyngeal inflammation, intermittent dorsal displacement of the soft palate, epiglottic entrapment, and trauma from ingestion of foreign bodies [48]. Endoscopically, epiglottitis results in edema, reddening, and thickening of the epiglottis and aryepiglottic fold. Extensive swelling and marked discoloration of the mucosal tissue that attaches loosely to the lingual surface of the epiglottis is common, and swelling of the lingual surface may cause mild to marked dorsal elevation of the epiglottic axis. Cartilage at the tip of the epiglottis may be exposed, resulting in granulation tissue formation. Chondritis of the epiglottic cartilage can develop and may result in epiglottic deformity during healing.

Epiglottitis is seen most commonly in racehorses. Clinical signs include exercise intolerance, respiratory noise, coughing, and, less commonly, dysphagia and dyspnea. Treatment recommendations include rest; anti-inflammatory and antimicrobial medication; and topical treatment with nitrofurazone (Furacin), dimethyl sulfoxide, glycerin, and prednisolone. In cases refractory to medical management, resection of the thickened sub-epiglottic tissues is recommended. The prognosis for resolution of epiglottitis with medical management is generally good. Nevertheless, in the long term, approximately 50% of horses have performance-limiting complications, including permanent or intermittent dorsal displacement of the soft palate, epiglottic deformity, and epiglottic entrapment [48].

Hypoplasia of epiglottis

The normal length of the epiglottis in Thoroughbreds is 8.56 ± 0.29 cm; in Standardbreds, it is 8.74 ± 0.38 cm [44]. The term *epiglottic hypoplasia* refers to a short and/or flaccid epiglottis. Lateral radiographs of the larynx allow precise measurements of the thyroepiglottic length; however, flaccidity of the epiglottis is a somewhat subjective diagnosis made endoscopically. A flaccid epiglottis seems to lack rigidity and thickness and is short and thin. Frequently, a flaccid epiglottis has the appearance of lying in direct contact

with the soft palate, conforming to its contours, and the epiglottis may sometimes curl up at its edges (Fig. 2). The clinical significance of a hypoplastic epiglottis is difficult to determine; certainly, the condition has been associated with an entrapped epiglottis [44] and dorsal displacement of the soft palate [43,49]. During high-speed treadmill stress tests, we have observed a flaccid epiglottis at rest, becoming more rigid during exercise and not being associated with pathologic findings. The flaccid epiglottis also presents a conundrum during the prepurchase examination of yearlings. It is now thought that only a severely flaccid epiglottis is associated with future poor performance.

Endoscopic examination of horses with an entrapped epiglottis reveals that approximately 31% of horses have some degree of epiglottic hypoplasia [43]. In one study, the thyroepiglottic length of 9 Thoroughbreds with epiglottic entrapment was 6.59 ± 0.33 cm [50]. In another study of 35 Thoroughbreds and 44 Standardbreds, the thyroepiglottic length was 7.28 ± 0.67 cm in Thoroughbreds and 7.21 ± 0.62 cm in Standardbreds [44]. In addition horses with a hypoplastic epiglottis are at greater risk of suffering from dorsal displacement of the soft palate after surgical correction of the entrapped epiglottis [43].

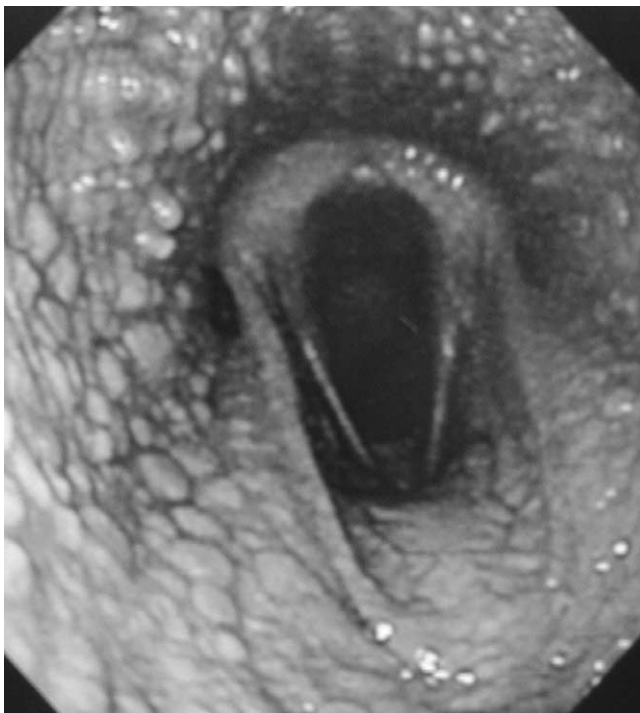


Fig. 2. Hypoplastic epiglottis with pharyngitis.

Traditionally epiglottic hypoplasia was believed to be a major contributing factor to dorsal displacement of the soft palate. However, recent evidence has demonstrated that epiglottic retroversion induced by bilateral hypoglossal and glossopharyngeal nerve block does not result in dorsal displacement of the soft palate, suggesting that when the palate is normal, the epiglottis does not function as a support holding the palate in a ventral position [51].

Treatment for the hypoplastic epiglottis is epiglottic augmentation. Polytetrafluoroethylene is injected submucosally on the lingual epiglottic surface. The procedure does not lengthen the epiglottis but simply increases rigidity. Racing performance in 73% of Thoroughbreds and 49% of Standardbreds was improved after epiglottic augmentation [52].

Dorsal epiglottic abscessation

Dorsal epiglottic abscessation is a rare cause of exercise intolerance in horses [53]. Clinical signs include exercise intolerance, upper respiratory tract noise, and coughing. Diagnosis is made by endoscopic examination. Typically, the abscess is a well-circumscribed smooth swelling on the dorsal surface of the epiglottis. Intermittent dorsal displacement of the soft palate may also be observed. Treatment recommendations include drainage and debridement transendoscopically using the laser. After decompressing the abscess, debridement is recommended with 600-mm long, 4-mm outer diameter (OD), bronchoesophageal grasping forceps, followed by lavage of the abscess site. The prognosis in a small number of cases was good, with resolution of the abscesses and full return to athletic function [53].

Epiglottic retroversion

Epiglottic retroversion is a rare cause of upper respiratory tract noise in horses [54]. The condition is diagnosed endoscopically during a high-speed treadmill stress test. The condition has been observed in both racing Thoroughbreds and Standardbreds. Typically, an inspiratory gurgling noise is audible. Endoscopically, the epiglottis elevates off the palate and retroverts into the glottis during inspiration. There are few cases reported in the literature, but treatment recommendations in one report included epiglottic augmentation with polytetrafluoroethylene. After this intervention, one horse raced successfully and one was retired because of continued upper respiratory tract noise and exercise intolerance [54]. More recently, a technique to stabilize the epiglottis with suture between the epiglottis and thyroid cartilage allowed one Standardbred to race successfully. Epiglottic retroversion has also been induced experimentally by performing bilateral nerve blocks of the hypoglossal and glossopharyngeal nerves [51]. The etiology of epiglottic retroversion is unknown, but loss of motor function to the hyoepiglotticus muscle, innervated by the hypoglossal nerve, is a possible

cause of the retroflexion. Contraction of the geniohyoideus muscle also alters epiglottic position by pulling the hyoid arch rostrad, which increases tension on the hyoepiglottic ligament, pulling the epiglottis toward the basioid bone, and may also play a role in the etiology of the disease.

Subepiglottic cysts

Subepiglottic cysts are an infrequent cause of upper respiratory tract noise and exercise intolerance in adult horses [55,56]. Upper respiratory tract noise occurs on inspiration and expiration during exercise. In foals, clinical signs may include nasal discharge, coughing, dysphagia, and signs of aspiration pneumonia [57]. In an adult horse, a subepiglottic cyst obstructing the entire rima glottidis was a cause of syncope [58].

The etiology of subepiglottic cysts is thought to be of embryologic origin but may also be secondary to inflammation or trauma. Histopathologic examination of the tissue usually reveals a cystic structure containing proteinaceous fluid. Cysts are usually lined with a combination of stratified squamous and pseudostratified columnar epithelium.

Diagnosis is made endoscopically. The cysts are located dorsal to the soft palate in the subepiglottic tissues. The cysts may be of varying size but are usually loosely attached and may intermittently disappear beneath the soft palate; swallowing usually replaces the cyst back under the epiglottis. Lateral laryngeal radiographs are also helpful in further defining the cyst location and tissue size.

Treatment options include removal of the cyst via laryngotomy [42,57] or via an oral approach using either a laser transendoscopically or a snare, either an electrocautery snare or an obstetric wire [56]. Two basic principles to be followed independent of technique include first removing the entire secretory lining of the cyst. If the cyst is merely punctured and drained, it seals over and eventually refills. Second, the cyst is submucosal, and removal of excessive amounts of oral pharyngeal mucosa overlying the cyst may result in cicatrization beneath the epiglottis, which could disrupt the function of the soft palate and epiglottis and result in dorsal displacement of the soft palate. Smaller cysts can be safely excised en bloc via a transnasal approach with the horse standing [56]. The prognosis after surgery is good, with most horses returning to full athletic function.

Persistent frenulum

Persistent frenulum is a rare cause of dysphagia in foals [59]. Foals typically present with dysphagia and oronasal reflux of milk from birth, with varying signs of aspiration pneumonia. Endoscopy of an affected foal reveals persistent dorsal displacement of the soft palate. Diagnosis is made by oral endoscopy under general anesthesia. Typically, the tip of the epiglottis under the soft palate is directed ventrally, and a ridge of tissue is

identified on the ventral aspect of the epiglottis. The ridge of tissue originates from the soft tissues caudal to the base of the tongue and attaches several millimeters from the rostral free edge of the soft palate. The ridge of tissue may be thin and membranous or rounded and thickened. Treatment recommendations include surgical transection of the tissue using laparoscopic scissors. Surgical transection of the persistent frenulum results in immediate resolution of the dysphagia and dorsal displacement of the soft palate. If the condition is treated early before deterioration of the foal occurs from aspiration pneumonia, the prognosis for life is excellent [59].

Aryepiglottic folds

Axial deviation of aryepiglottic folds

Axial deviation of the aryepiglottic folds (ADAF) has been recognized as a cause of dynamic airway obstruction in racehorses [60]. The term *axial deviation of the aryepiglottic folds* describes axial deviation of the membranous portion of the aryepiglottic fold extending between the corniculate process of the arytenoid cartilage and the lateral edge of the epiglottis (Fig. 3). The pathogenesis of the disease remains unknown. Clinical signs generally include poor performance and upper respiratory



Fig. 3. Axial deviation of the aryepiglottic folds as seen during inspiration during a high-speed treadmill examination.

tract noise. ADAF is only described in racing horses, including Thoroughbreds, Standardbreds, and one racing Arabian. The condition is diagnosed endoscopically during a high-speed treadmill stress test, with the axial deviation occurring during inspiration. The condition is more commonly bilateral but may be unilateral; to date, all cases of unilateral ADAF have been right sided. The condition frequently occurs in combination with other abnormalities of the upper airway, including dorsal displacement of the soft palate, dorsal pharyngeal collapse, axial collapse of the vocal cord, left laryngeal hemiparesis, and right laryngeal dysfunction.

Treatment recommendations include laser ablation of the membranous portion of the aryepiglottic folds. Laser ablation may be performed either in the standing animal or with the animal under general anesthesia. Standing transendoscopic laser excision is preferred for horses that have ADAF only, because standing surgery is technically easier to perform than surgery in an anesthetized and nasotracheally incubated animal. Rest alone has also resulted in a positive outcome in some horses, although surgery does have better overall results [60].

Summary

The upper respiratory tract is a frequent cause of exercise intolerance in horses, particularly in racing horses. There are a myriad of laryngeal abnormalities that may restrict airflow at the rima glottidis. Careful endoscopic examination is a crucial part of the examination of any racing horse suffering from poor performance. There has recently been interest in spectrum analysis of respiratory sounds [61]. It has been determined that laryngeal hemiplegia and dorsal displacement of the soft palate have unique sound patterns. Therefore, spectrum analysis of respiratory sounds may prove to be useful in the diagnosis of laryngeal disorders in horses. Accurate diagnosis and appropriate surgical intervention are necessary to provide the horse the best chance of returning to its full athletic potential.

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