An 18-month-old 355-kg (781-lb) Quarter Horse filly was referred to the Oklahoma State University Veterinary Teaching Hospital for evaluation of abnormal respiratory noise and exercise intolerance. The owners noticed the respiratory noise during exercise and when the horse lowered its head to eat. The horse had been purchased 6 months prior to referral and the noise was noticed within 24 hours of purchase.

On initial examination, the horse was bright, alert, and in good body condition. Rectal temperature was within reference range, heart rate was 42 beats/min with a strong and regular beat, and respiration rate was 12 breaths/min. Thoracic auscultation, with and without the use of a rebreathing bag, revealed no abnormalities. During upper respiratory tract endoscopy, moderate lymphoid hyperplasia was seen, and the soft palate was observed to be displaced dorsally (Fig 1). No other abnormalities were noted. The endoscope was passed into the interior of the auditory tube diverticuli (guttural pouches), and no abnormalities were seen. The swallowing reflex was normal, and after swallowing, the soft palate was replaced to its correct position temporarily; however, the epiglottis appeared abnormally small and flaccid (Fig 2). After the horse swallowed, the epiglottis immediately slid caudoventrally off the caudal border of the soft palate (Fig 3), allowing the soft palate to move dorsal to the epiglottis.

Because the horse was excitable, it was sedated with xylazine hydrochloride (0.5 mg/kg [0.23 mg/lb]) administered IV. During subsequent endoscopy, within minutes of xylazine administration, the epiglottis appeared completely normal in tone, size, and anatomical position, and the soft palate was no longer displaced (Fig 4). Thirty minutes after xylazine administration, the horse was exercised on a lunge line for 15 minutes to characterize its exercise intolerance. No abnormal noise was heard, and during endoscopy after exercise, the epiglottis appeared normal. However, during exercise the following day, a gurgling noise was detected. The findings of upper respiratory tract endoscopy, performed immediately after exercise and without sedation, were similar to that found the previous day prior to sedation. The horse was again administered xylazine hydrochloride IV at the same dose. Upon subsequent endoscopy, the epiglottis again appeared normal in tone, size, and anatomical position, and the soft palate was no longer displaced.

Although there was no familial predisposition to hyperkalemic periodic paralysis (HYPP) in the horse reported here, laryngospasm has been described in a number of studies associated with this disease in horses; therefore, a sample of hair was submitted for analysis to establish whether the horse carried the gene for HYPP. Additionally, treatment was initiated with acetazolamide (4 mg/kg [1.8 mg/lb], PO, q 8 h, for 4 days); no improvement was detected. Results of the test for HYPP were negative. Because of financial constraints, the owners declined further diagnostic tests. Because there is no other common hereditary condition that was likely to be involved in this case, the owners chose to use the horse as a broodmare.

Because there was extensive lymphoid hyperplasia in the nasopharynx, the horse was treated after discharge from the hospital with prednisone (1 mg/kg [0.45 mg/lb], PO, for 5 days) to ascertain whether anti-
inflammatory treatment would help alleviate the epiglottic dysfunction. Follow-up by telephone conversation with the owners 1 week later and again 6 months later revealed that there was no improvement. The owners reported that the horse still made an abnormal respiratory noise during exercise.

Endoscopic examination of the larynx of the horse suggested epiglottic dysfunction. Loss of motor function of the epiglottis has been described in horses; however, retroflexion of the epiglottis is typically seen. We did not observe retroflexion of the epiglottis in the horse reported here. Instead, it appeared more likely that increased tonicity or spasticity of the associated muscles caused traction on the epiglottis and was responsible for the ventral and rostral displacement of the epiglottis.

Pharyngeal or laryngeal dysfunction, secondary to interference with the nerve supply to the muscles in this region, has been described. Bilateral hypoglossal and glossopharyngeal nerve block causes epiglottic retroflexion in exercising horses. Blocking the pharyngeal branch of the vagus nerve bilaterally induces persistent dorsal displacement of the soft palate in horses. Neuritis, secondary to upper respiratory tract infection, local lymphadenopathy, and inflammation, has been hypothesized as a reason for laryngeal neuromuscular dysfunction. If this were the underlying cause of the epiglottic abnormality seen in the horse reported here, the lack of response to prednisone may have been attributable to lack of conversion to an active form, as has been suggested in recent veterinary literature. Also, the duration of treatment (5 days) was probably insufficient for an anti-inflammatory effect to be seen. Therefore, we cannot exclude the possibility of an inflammatory lesion contributing to this horse's condition.

Laryngeal or pharyngeal dysfunction in horses involves the arytenoids, soft palate, palatopharyngeal arch, or the entire larynx and laryngopharynx. However, epiglottic dysfunction as an isolated feature is uncommon. Xylazine hydrochloride, an α2-adrenoceptor agonist, has various effects on the respiratory tract of horses. In the horse described here, primary
epiglottic dysfunction was entirely amelioriated by a sedative dose of xylazine.

In horses, the hyoepiglotticus muscle attaches the base of the epiglottis to the hyoid bone. Contraction of this muscle improves epiglottis-soft palate contact by pulling the epiglottis rostrally and ventrally against the soft palate, towards the base of the tongue, which expands the nasopharynx. This muscle is innervated by the hypoglossal nerve (cranial nerve XII). Other muscles innervated by this nerve include the muscles of the tongue and the thyrohyoides and geniohyoideus muscles, which pull the hyoid apparatus rostrally when they contract. Blockade of this nerve results in dysfunction of the muscles that cause cranial movement of the basihyoid bone and of the muscle that approximates the epiglottis and the basihyoid bone, the hyoepiglotticus muscle. We propose that this muscle supplied by the hypoglossal nerve could increase cranial movement of the basihyoid bone and induce traction on the epiglottis, pulling the epiglottis down and forward off the caudal border of the soft palate, resulting in the abnormality seen in this horse.

Xylazine is an α2-adrenergic agonist, classified as a sedative-analgesic with muscle relaxant properties. It causes sedation, CNS depression, and visceral analgesia in horses. Muscle relaxation is mediated through central pathways. The onset of action of xylazine after IV administration is 1 to 2 minutes, with maximum effect at 3 to 10 minutes. The duration of effect is dose dependent, and effects may last for approximately 1.5 hours. The serum half-life after a single dose is approximately 50 minutes, and recovery times can take from 2 to 3 hours. It is possible that muscle relaxation effects persist for a greater length of time than sedative effects although both are centrally mediated. In the horse reported here, effects of the drug persisted in reversing the laryngeal abnormality after sedation ceased. When the horse was exercised 30 minutes after xylazine administration, the dysfunction had not returned, although the horse was alert enough to exercise on a lunge line. By the following morning, the dysfunction had returned and was again alleviated with the same dose of xylazine.

Results of several studies indicate that xylazine has various effects on the respiratory tract of horses. In 1 study, xylazine significantly decreased the intraobserver repeatability of examination of the larynx and caused some horses to have more synchronous movements of the larynx, whereas other horses had decreased ability to reach full abduction of the left arytenoid cartilage. In that study, xylazine did not affect computer-assisted measurements of the rima glottidis. In another study, xylazine caused a decrease in the cross-sectional area of the rima glottidis but had no effect on synchrony of movement of the arytenoid cartilages. Although the results of these studies may be somewhat contradictory, it is clear that, to some extent, xylazine does have a certain, if variable and yet undefined, effect on the larynx of horses.

Additional evidence for an effect of xylazine on upper airway motor function can be found in other species. α2-Adrenoceptor stimulation preferentially decreases the activity of upper airway adductor muscles while increasing the activity of upper airway adductor muscles in conscious goats. The underlying mechanisms are not clear; however, α2-adrenoceptors are found extensively in brain stem sites responsible for cardiorespiratory control in rats. Specifically, clonidine, an α2-adrenoceptor agonist structurally related to xylazine, hyperpolarizes hypoglossal motor neurons in rats. If a similar effect occurred with xylazine in horses, it is possible that xylazine could ameliorate spasm of a muscle innervated by this nerve by increasing the action potential threshold for firing of the nerve.

To our knowledge, there has been only 1 other report of xylazine attenuating a spastic condition in a horse, a forelimb tic in an 18-month-old male Quarter Horse, which became apparent 4 weeks after an injury to the limb. The tic disappeared after sedation with xylazine, but the reason for response to xylazine was not known. The condition did not alter during 3 weeks of hospitalization, but within 10 weeks of discharge the tic had disappeared.

Guglick et al proposed that regardless of age and serum potassium concentration, HYPP should be considered as a differential diagnosis for pharyngeal and laryngeal dysfunction and dysphagia in any horse with Quarter Horse breeding. In that report, marked clinical improvement was observed in an affected horse 48 hours after initiation of acetazolamide administration (5 mg/kg, PO, q 8 h). The spasms persisted endoscopically after treatment but were less frequent, less severe, and resolved faster than those seen before treatment. However, the horse described in our report did not have a familial predisposition to this disease and had negative test results for HYPP; furthermore, acetazolamide administration failed to affect the abnormalities in the horse's larynx.

References


