Clinical Features and Outcome of Dogs with Epiglottic Retroversion With or Without Surgical Treatment: 24 Cases

S.C. Skerrett, J.K. McClaran, P.R. Fox, and D. Palma

Epiglottic retroversion (ER) is a rare cause of upper airway obstruction in dogs with few cases reported. This disorder can result in inspiratory stridor and life-threatening dyspnea. It is characterized by intermittent spontaneous epiglottic retroflexion during inspiration causing obstruction of the rima glottidis noted during either upper airway examination or upper airway fluoroscopy. Hypotheses about the etiology in dogs include epiglottic fracture or malacia, hypothyroidism-associated peripheral neuropathy and denervation of the hypoglossal nerve, the glossopharyngeal nerve or both.

Previous case reports of ER indicated successful treatment with epiglottopexy in 2 dogs. In addition, in a recent case report a dog with multiple epiglottopexy failures required a subtotal epiglottectomy for a successful outcome. Follow-up in both case reports was only reported up to 4 months after surgery.

The objective of our study was to identify clinical signs, comorbidities, endoscopic findings, fluoroscopic findings, outcome of surgical versus medical treatment and long-term follow-up for canines diagnosed with epiglottic retroversion. We hypothesized that upper airway comorbidities such as collapsing trachea, laryngeal paralysis, or brachycephalic airway syndrome would be common in dogs with ER. We also hypothesized that dogs that undergo surgical management (epiglottopexy or subtotal epiglottectomy) would have improved long-term outcome as compared with medical management alone.

Materials and Methods

Case Selection

Medical records from the Animal Medical Center from 2010 to 2014 were reviewed to identify dogs diagnosed with ER. Dogs with complete medical records and ER documented on upper airway examination or fluoroscopic evaluation were included in the study. During upper airway examination (laryngeal endoscopy or laryngoscopy) or fluoroscopic evaluation, ER was observed as intermittent spontaneous obstruction of the rima glottidis by the epiglottis. Dogs diagnosed with ER that either underwent surgical management of ER (epiglottopexy or subtotal epiglottectomy) or medical management of ER were included.
Medical Records Review

Information retrieved from the medical records included signalment, duration and type of respiratory clinical signs, physical examination findings, upper airway examination findings (laryngeal endoscopy or laryngoscopy), fluoroscopic examination findings, surgical methods, biopsy results (if available) and outcome at follow-up appointments. Concurrent medical conditions (endocrine, cardiovascular, respiratory, or neurologic) also were recorded.

Treatment and Follow-up Evaluation

Short-term follow-up information was retrieved from medical records of reexamination visits 2–4 weeks after diagnosis of ER. Long-term follow-up information was retrieved from medical records from the last-documented examination or by a follow-up phone conversation with owner. For dogs deceased at the time of data collection, the date and cause of death were recorded. Improvement at follow-up was defined as a decrease in the frequency or severity of presenting clinical signs and lack of complications such as aspiration pneumonia or respiratory crisis as reported by the attending veterinarian or the owner.

The surgical techniques utilized, temporary or permanent epiglottopexy and subtotal epiglottectomy, have been described previously.1,2 Temporary epiglottopexy was performed by placing an average of 2 horizontal mattress sutures (range, 1–4 sutures) using absorbable or nonabsorbable suture material between the lingual surface of the epiglottis and the base of the tongue, each suture engaging the epiglottic cartilage (Fig. 1). Permanent epiglottopexy involved the above technique with removal of a wedge of mucosa from the lingual aspect of the epiglottis. Subtotal epiglottectomy was performed in 1 dog using a carbon dioxide laser to resect 1 cm from the tip of the epiglottis.

Medical management included administration of a combination of cough suppressants, corticosteroids, sedatives, and antibiotics. Medical management is further described in the results section.

Statistical Analysis

Reported number and percentage of cases involved with each presenting sign, treatment, and complication. Median survival times are reported using a Kaplan–Meier Curve.

Results

The most common breeds represented were Yorkshire Terrier (n = 8), Cocker Spaniel (n = 3) Chinese Pug (n = 3), and Pekingese (n = 2). Also, there was 1 each of the following breeds: English Bulldog, Beagle, Boston Terrier, French Bulldog, Boxer, Shih Tzu, Cavalier King Charles Spaniel, and Pomeranian. Fourteen dogs (58.3%) were ≥ 8 years old at time of diagnosis, 6 dogs (25%) were between 4–7 years of age and 4 dogs (16.7%) were <3 years old. The majority of the population was spayed females (58.3%), followed by neutered males (29.2%) and intact females (12.5%), and no intact males were included in this study. Fourteen dogs

Fig. 1. Intraoperative images of epiglottic retroversion and epiglottopexy. (A) Before intubation the epiglottis (white star) is seen retroverting caudally to obstruct the rima glottis. (B) After the endotracheal intubation (ET) the suture is passed through the tongue (black star) at the base of the epiglottis and then through the epiglottis, engaging the cartilage (C). (D) Postoperative image demonstrating the suture (arrow) apposing the epiglottis and base of the tongue.
(60.9%) had BCS ≥6/9; 1 dog did not have BCS recorded.

The average length of respiratory clinical signs before presentation was 182 days (range, 0–2,520 days). Previous history included pneumonia (n = 7), noncardiogenic pulmonary edema (NCPE; n = 4), broken ribs (n = 1), pectus excavatum with paradoxical respiration (n = 1) and nasopharyngeal occlusion (marked obstruction of the nasopharynx by the soft palate on rhinoscopy; n = 1) in addition to the presenting clinical signs. The aforementioned cases with pneumonia, NCPE, or broken ribs were successfully treated or resolved before presentation for ER. Presenting clinical signs included stridor (n = 20), dyspnea (n = 15), coughing (n = 7), and cyanosis (n = 8). Worsening of stridor or dyspnea when sleeping was reported in 4 dogs. Concurrent neuromologic disorders such as intervertebral disc disease (n = 3) and seizures (n = 3) were diagnosed in 25% (n = 6) of the cases, as well as laryngeal paralysis which was diagnosed in 2 dogs (8.3%) and hypersalivation (suspected limbic epilepsy) diagnosed in 2 dogs (8.3%). Thyroid function testing was performed in 6 dogs and was within normal limits for all 6 dogs.

Upper airway examinations were performed using laryngeal endoscopy (n = 14), fluoroscopy (n = 7), and laryngoscope (n = 4) to diagnose ER (Figs 2–3). Laryngoscope examinations typically were performed in emergency situations and only involved evaluation of the oral cavity and larynx. When laryngeal endoscopy was performed the endoscope was used to evaluate the oral cavity, larynx as well as trachea and bronchi. All examinations began under sedation or a light plane of anesthesia (propofol, butorphanol or both; acepromazine; dexmedetomidine) and then proceeded to full general anesthesia (complete anesthesia using IV propofol or inhalants) for further evaluation of the trachea and bronchi. Doxapram was given to stimulate respirations in 28% of the dogs. Epiglottic retroversion was observed as intermittent spontaneous obstruction of the rima glottidis by the epiglottis (Figs 2–3). The epiglottis was noted to intermittently move caudally during inspiration, resulting in obstruction of the rima glottidis for several seconds as well as flattening of the epiglottis on caudal retraction and loss of epiglottic concave structure on nondynamic evaluation. In severe cases, the epiglottis required manual manipulation to return it to its normal position. Epiglottic retroversion was seen either rostral to the soft palate or caudal to the soft palate (depending on whether or not the soft palate was elongated). On fluoroscopic evaluation, increased oropharyngeal gas distension after obstruction and increased soft tissue density at the larynx also were noted in 2 dogs.

Sixteen dogs (66.7%) had an episode of respiratory crisis requiring medical intervention (oxygen treatment and sedatives) before diagnosis of ER. Four of the
The aforementioned dogs required intubation and four required tracheostomies for management of their crisis. Thirty-seven percent of the dogs had the crisis an average of 285 days (range, 1–1,460 days) after surgical correction of a concurrent upper airway disorder.

Nineteen dogs (79.1%) with ER had concurrent or historical upper airway disease at time of ER diagnosis, such as elongated soft palate (n = 17), tracheal collapse (n = 11), laryngeal saccule eversion (n = 7), laryngeal edema (n = 6), bronchial collapse (n = 4), laryngeal collapse (n = 3), and laryngeal paralysis (n = 2). These dogs were classified as having secondary ER for this study versus those dogs without concurrent or historical upper airway disease that were classified as having primary ER. Cases with secondary ER could be further categorized into four different groups (Table 1). The first group had 1 or more previous surgical corrections of the concurrent airway disorder performed and the average time between previous surgery and presentation for ER was 242 days (range, 2–1,460). The second group consisted of dogs that underwent surgical management for their other airway disorders and ER concurrently. The third group of dogs had correction of concurrent respiratory disorders without correction of ER. The fourth group of dogs underwent surgical repair of their ER but not the additional upper airway disorder.

Nineteen of 24 dogs (79.1%) diagnosed with ER underwent ≥1 surgical treatments including temporary epiglottopexy (n = 19), permanent epiglottopexy (n = 8), and subtotal epiglottectomy (n = 1). Materials utilized included 4–0 polydioxanone (n = 5), 2–0 polypropylene (n = 5), 0 polypropylene (n = 3), 4–0 polypropylene (n = 3), 3–0 polypropylene (n = 2), 3–0 polyglactin (n = 1), 4–0 poliglecaprone 25 (n = 1) and unknown suture material (n = 7). In 2 dogs, suture patterns other than those described previously were used. One pattern included a cruciate suture in addition to horizontal mattress suxtures and the other consisted of a simple continuous suture pattern. The average time the dogs that underwent epiglottopexy alone was 32 minutes (range, 25–40 minutes). The anesthesia time included a brief upper airway examination before proceeding with the surgical procedure.

### Table 1. Surgical management of secondary epiglottic retroversion cases.

| Surgical management of secondary ER cases | Number of cases (%) |
|-----------------------------------------|---------------------|
| Surgery prior to epiglottopexy\(a\)    | 8 (42.1)            |
| Surgery at the same time as epiglottopexy\(b\) | 5 (26.3)           |
| Surgery for concurrent disorder, no epiglottopexy\(c\) | 3 (12.5)         |
| Epiglottopexy, no surgery for concurrent disorder\(d\) | 3 (12.5)         |

\(a\) Tracheal stent with staphylectomy (n = 4), brachycephalic airway surgery (n = 4).
\(b\) Tracheal stent (n = 3), brachycephalic airway surgery (n = 2).
\(c\) Bilateral arytenoid lateralization (n = 1), tracheal stent (n = 2).
\(d\) Tracheal collapse (n = 1), elongated soft palate (n = 2).

Three secondary ER cases received only medical management for their condition, but all underwent surgical intervention for their concurrent upper airway disorders (tracheal stent [n = 2], unilateral arytenoid lateralization [n = 1]). The dog with laryngeal paralysis continued to have moderate stridor at last follow-up (378 days postoperatively), but no episodes of respiratory crisis. The 2 aforementioned cases with tracheal stents placed had no reported episodes of respiratory crisis. Epiglottic retroversion was confirmed on direct laryngoscopy finding on upper airway examination and thus epiglottopexy was not performed. One dog was followed up for 238 days at which point, the dog was euthanized because of complications from pneumonia. Another dog with secondary ER that did not undergo medical or surgical intervention had a history of respiratory noise and occasional gagging but no history of respiratory crisis. The owner declined surgical intervention and the dog had been followed up for 84 days since diagnosis with no complications or change in condition reported. A primary ER case that did not undergo surgery had pneumonia as well as noncardiogenic pulmonary edema at the time of ER diagnosis. The owner of this dog declined surgery and, at last follow-up (528 days after diagnosis), no respiratory concerns were noted.

Of the 19 dogs that underwent epiglottopexy, 6 dogs required ≥1 revision surgeries (mean, 2; range, 1–4) for epiglottopexy failure. The average time to failure was 192 days (range, 3–730 days). One additional dog that had failure of its temporary epiglottopexy did not undergo revision surgery and continues to do well with medical management of its concurrent airway disorders. Temporary epiglottopexy resulted in 7 failures (36.8%) secondary to sutures breaking (n = 5), pull out of the suture from the tissues (n = 1) and suspected (but not confirmed) breakdown (n = 1). Four dogs with failed temporary epiglottopexy went on to have replacement epiglottopexies remain intact, either temporarily (n = 1) or permanently (n = 3). Sixty-two percent (n = 5/8) of the permanent epiglottopexies failed, as a result of suture breakage (n = 2), pull out of suture from the tissues (n = 2) or loosening of the sutures (n = 1). Subtotal epiglottectomy was performed in 1 dog after failure of 3 epiglottopexies.

No dogs died during surgery but 2 dogs were euthanized before discharge, 2 and 3 days after epiglottopexy, the former because of respiratory failure (history of tracheal stents and previous respiratory infections) and the latter because of continued oxygen dependency after surgery. The average length of hospitalization after diagnosis with or without surgical intervention was 2.3 days (range, 1–5 days). Short-term and long-term medications included cough suppressants, antibiotics, corticosteroids, sedatives, and gastroprotec-

Aspiration pneumonia occurred in 8 dogs (33.3%) after diagnosis of ER, 2 of these dogs did not undergo surgical treatment of their ER. The average time between epiglottopexy and development of aspiration pneumonia was 54 days (range, 2–270 days), three of

---

Skerrett et al.
these cases occurred before discharge from the hospital. Six dogs (25%) had a respiratory crisis episode requiring hospitalization after undergoing epiglottopexy, three of which were related to confirmed or suspected breakdown of the epiglottopexy.

At short-term follow-up, 13 dogs (54.2%) showed improvement of clinical signs, two of which did not undergo surgical management of the ER. At last follow-up, 11 dogs (45.8%) showed improvement of clinical signs, including the 2 dogs that did not undergo surgical management but showed short-term improvement. Follow-up times ranged from 2 to 1456 days after diagnosis with a median follow-up time of 307 days.

Three dogs had histopathologic evaluation of the epiglottis, 1 after subtotal epiglottectomy and 2 at the time of necropsy. The biopsy from the dog with subtotal epiglottectomy (history of failed epiglottopexies) showed ulcerative epiglottitis, granulation tissue, cartilage disorganization, and mineralization. The gross findings on necropsy of a dog with a failed epiglottopexy included an irregular contour of the epiglottic cartilage and a thickened region of subepiglottic mucosa at the ventral aspect of the epiglottis, the other dog (intact epiglottopexy) had an epiglottis that was firmly adhered to the adjacent pharyngeal mucosa (Fig. 4). The histopathology from the dog with the failed epiglottopexy disclosed regional mucosal hyperplasia with submucosal edema, moderate submucosal fibrosis, and skeletal myofiber degeneration. The histopathology from the dog with the intact epiglottopexy disclosed multifocal mucosal and cartilage mineralization, regional submucosal necrosis, fibrin deposition, and suppurative inflammation with associated focal suture material.

At the time of writing, 9 dogs were deceased (33.3%); eight of those had epiglottopexy performed. Reasons for euthanasia were documented as severe, progressive dyspnea with oxygen dependency (n = 3), aspiration pneumonia (n = 3), continued or worsening respiratory clinical signs related to suspected epiglottopexy breakdown (n = 2) and reason not listed (n = 1). The calculated Kaplan–Meier median survival time was 875 days (Fig. 5).

### Discussion

During swallowing, the glottis closes and the epiglottis tilts backward to close the rima glottidis and prevent aspiration of food. During inspiration, the hyoepiglotticus (HE) muscles contract, pulling the epiglottis rostrally so the ventral surface of the epiglottic tip is in contact with the dorsal surface of the soft palate thereby keeping the nasopharyngeal airway freely patent. Further activation and contraction of the HE muscles moves the epiglottis ventrally, thus disconnecting it from the soft palate and dilating the oropharyngeal airway. This movement is thought to protect the upper airway from inspiratory negative pressure-induced collapse.

Epiglottic retroversion has been reported as a cause of exercise intolerance and stridor in horses and stridor in humans. A variety of treatment modalities are used in humans including suture epiglottopexy, laser epiglottoplasty, or partial laser epiglottidectomy. In horses, treatment for ER by subepiglottic resection or epiglottic augmentation has not been very successful for enabling horses to return to full function as performance animals.
In this cohort of dogs, epiglottic retroversion tended to present with respiratory signs in 2 different clinical history patterns. One subset had intermittent clinical signs and the dogs were clinically normal between their respiratory episodes which usually were precipitated by stress or exercise, and the other subset had constant respiratory signs. In 3 cases, the stress of anesthesia (dental cleaning \(n = 2\) and tracheal stent placement \(n = 1\)) lead to respiratory crisis after which upper airway examination identified epiglottic retroversion in dogs that were presented with intermittent clinical signs often had ER as their only upper airway disorder; these cases were defined as having primary epiglottic retroversion for this study. In comparison, dogs with constant clinical signs typically had concurrent upper airway disorders such as brachycephalic airway syndrome or tracheal collapse, and these were defined as having secondary epiglottic retroversion.

Epiglottic retroversion can occur rostral to the soft palate or caudal to the soft palate. Careful evaluation of the epiglottis flattening in dogs with ER rostral to the soft palate or caudal to the soft palate. A missed diagnosis of ER when performing an upper airway examination is likely a result of compression of the epiglottis or excessive rostral tractions on the tongue during examination.

Previous hypotheses of the etiology of epiglottic retroversion in dogs proposed the disease as being secondary to hypothyroidism-associated peripheral neuropathy or both. These hypotheses arose from previous studies that evaluated the role of the glossopharyngeal nerve or both. These hypotheses arose from previous studies that evaluated the role of the hypoepiglotticus muscle (innervated by the hypoglossal nerve) in active control of epiglottic position during breathing in dogs as well as the first 2 cases that were reported. Two dogs had hypothyroidism and possible retroversion because of endocrine disease. Concurrent neurologic disorders were diagnosed in 3 dogs of the dogs which is too few to determine statistical significance to support the hypothesis of neurologic dysfunction, a possible type II error. Biopsies of the epiglottis were obtained in 3 dogs in this study and 1 dog in a previous study. Two of these biopsies included only the epiglottis but no musculature, and both were consistent with inflamed connective tissue. There was no definitive cause of ER identified on histopathology in any of the samples submitted.

Breeds represented were either small or medium-sized dogs, no large or giant breed dogs were identified as having ER. The most frequent presenting clinical signs were stridor and dyspnea, consistent with upper airway obstruction; however, the dogs presenting with these clinical signs, 90.5% underwent epiglottopexy. Clinical signs that were worse during sleep may be related to the difference in inspiratory pressures in an awake animal versus when sleeping, or relaxation of muscles controlling the epiglottis and soft palate while sleeping.

Our study shows that fluoroscopic examination can be utilized to diagnose ER, and evaluation of the epiglottis should routinely be included as part of airway evaluations whether using endoscopy, a laryngoscope, or fluoroscopy. Veterinarians should be aware of this condition and always evaluate the epiglottis and its associated structures during upper airway examinations. The examinations should be performed without downward compression of the epiglottis, avoiding excessive traction on the tongue, and at a light plane of anesthesia so as to increase the accuracy of diagnosis or exclusion of this disorder. Of all cases examined, the respiratory crisis after surgical correction of concurrent upper airway disorders (46%) could be a result of failure to diagnosis concurrent ER at the time of initial upper airway examination because 4 out of 7 instances occurred within 30 days of the initial surgery for concurrent upper airway disorder. In these cases, ER was first diagnosed during the upper airway examination conducted at the time of respiratory crisis.

Concurrent upper airway disorders were diagnosed in 79.1% of cases, suggesting that ER is either a component of these disorders or occurs secondary to chronic increased inspiratory pressures. However, brachycephalic breeds represented only 37.5% of the total population in this study and therefore no statistically conclusions can be made regarding brachycephalic er epiglottopexy.

The failure rate was higher for permanent versus temporary epiglottopexy which raises questions about whether removal of the wedge of mucosa from the lingual aspect of the epiglottis is necessary. This failure rate could have been influenced by the different surgeons performing the procedures (1 case per surgeon), the different suture patterns utilized. The failure of the permanent epiglottopexy also may have been a result of continued increased negative inspiratory pressures from concurrent upper airway abnormalities that were not fully addressed surgically. All the dogs whose replacement (2nd or 3rd) epiglottopexy remained intact also had undergone surgical correction of concurrent upper airway disorders that may have led to a decrease in negative inspiratory pressures, thereby decreasing the strain on the sutures and making them less prone to failure. Of the 5 dogs classified as having primary ER (ie, no concurrent abnormalities), three required revision surgeries and a fourth dog had return of clinical signs but the owners decided not to pursue further surgical management. Taking this into consideration, permanent epiglottopexies may be recommended for dogs with primary ER or those with concurrent disorders that are chronic and ongoing and therefore would continue to have increased negative inspiratory pressures; these permanent epiglottopexies may be recommended for dogs with secondary ER, whose concurrent upper airway disorder can be successfully treated. The decision whether or not to perform a temporary or permanent epiglottopexy as the initial surgery for management of ER was based on surgeon preference in this study. It is difficult to draw conclusions about whether or not multiple revision
surgications weaken the cartilage of the epiglottis making it more prone to future epiglottoplasty failures because 80% of permanent epiglottoplasties that failed were revision surgeries, but none of the temporary epiglottoplasties that failed were revision surgeries.

The most common cause of epiglottoplasty failure was suture breakage (7/11) and suture pull out from tissues (3/11), which illustrates the importance of strongly engaging the tissues and suggests that using heavier gauge sutures may be valuable, but future studies are needed.

Subtotal epiglottectomy was performed in only 1 dog, after multiple epiglottoplasty failures, and this dog had no concurrent respiratory disorders. This method of ER treatment has been described previously in the human medical literature. No postoperative complications such as aspiration pneumonia or respiratory crisis were reported during the 119 days of follow-up. The successful outcome in this case and in the previously reported case1, indicate that subtotal epiglottectomy may be considered a reasonable treatment option for management of cases with failed epiglottoplasty.1

Long-term outcomes of the 5 cases that did not undergo surgical correction of ER were difficult to compare to the surgical cases because of the low number of patients and their concurrent upper airway comorbidities.

In the subset of dogs with a history of respiratory crisis before ER diagnosis and that underwent surgical management for ER, the incidence of respiratory distress (dyspnea or cyanosis requiring hospitalization) decreased from 62.5% before surgery to 25% after surgical treatment. Temporary or permanent tracheostomies were placed in 4 dogs before diagnosis and epiglottoplasty; no cases required tracheostomies after surgery. The 4 dogs also underwent surgical correction of their concurrent airway abnormalities, making it difficult to elucidate the impact of epiglottoplasty.

As a result of the small number of cases in this study, it is difficult to make conclusive determinations about the effect of epiglottoplasty on long-term outcome. At last evaluation, 52.6% of dogs that underwent surgical management and 60% of dogs that did not undergo surgical correction had decreased frequency or severity of presenting clinical signs.

Hospitalization length was short after diagnosis of ER regardless of whether or not the dog underwent surgery. The surgical procedure is minimally painful and therefore once the dog has been observed to eat or drink without complication, they can be discharged. Postoperative aspiration pneumonia was diagnosed in 6 of the 19 dogs that underwent epiglottoplasty and in 2 of 5 dogs that did not undergo epiglottoplasty; there were too few cases to determine any statistical significance. Based on the hypothesis that ER potentially could be caused by hypoglossal or glossopharyngeal nerve dysfunction or both, these dogs may be predisposed to aspiration pneumonia because the glossopharyngeal nerve is involved in the pharyngeal phase of swallowing and the hypoglossal nerve innervates the muscles of the tongue as well as the hyoepiglotticus muscle, but further studies are needed to evaluate this possible association.

At time of writing, 33.3% of the dogs included in this study were deceased. The reason for euthanasia was listed in 8/9 medical records as being related to worsening of upper airway clinical signs or development of pneumonia. Of the deceased dogs, 2 did not undergo surgical management of ER and 1 did not have any upper airway comorbidities. These low numbers make it difficult to draw conclusions about the impact of surgical management and airway comorbidities on survival. A median survival time of 875 days indicates that long-term survival is possible for dogs with ER.

The limitations of this study include its retrospective design with small patient numbers. Also, the epiglottoplasties were performed by several different surgeons using a variety of materials and epiglottoplasty methods. Because upper airway examinations were not performed on a regular basis after surgery, we were unable to ascertain exactly when epiglottoplasty failure occurred. Doxapram was not administered to every dog, which could have confounded the diagnosis of ER or laryngeal paralysis in these animals. None of the dogs had histopathology of the hyoepiglotticus muscle performed to eliminate denervation atrophy or other changes within the muscle.

Our hypothesis that upper airway comorbidities would be common in dogs with ER was supported by their presence in 79.1% of the dogs. This study demonstrated that long-term survival of at least 2 years may be expected in dogs with ER. Epiglottic reversion should be considered as a differential diagnosis in dogs with unexplained signs consistent with upper airway dysfunction. Too few cases were available to determine statistical significance of the impact of treating affected dogs surgically but because those dogs with secondary ER that underwent surgical correction of concurrent upper airway comorbidities as well as epiglottoplasty had lower failure rates of temporary or permanent epiglottoplasty compared to those cases with primary ER, surgical correction of concurrent upper airway disorders may decrease negative inspiratory pressures enough to decrease the incidence of ER. Owners should be counseled that multiple surgical interventions may be necessary.

Footnotes

---

Acknowledgments

We thank Dr. George E. Moore for his help with this study. This study was not funded by a grant or otherwise. This study has not yet been presented at a meeting.
Conflict of Interest Declaration: Authors disclose no conflict of interest.

Off-label Antimicrobial Declaration: Authors declare no off-label use of antimicrobials.

References

1. Mullins R, McAlinden AB, Goodfellow M. Subtotal epiglottectomy for the management of epiglottic retroversion in a dog. J Small Anim Pract 2014;55:383–385.

2. Flanders JA, Thompson MS. Dyspnea caused by epiglottic retroversion in two dogs. JAVMA 2009;235:1330–1335.

3. Monnet E, Tobias K. Veterinary surgery: small animal. In: Tobias KMJS, ed. Larynx, vol. 2. Philadelphia, PA: Saunders; 2012:1718–1719.

4. Amis TC, O’Neill N, Van der Touw T, et al. Control of epiglottic position in dogs: role of negative upper airway pressure. Respir Physiol 1996;105:187–194.

5. Amis TC, O’Neill N, Van der Touw T, et al. Electromyographic activity of the hyoepiglotticus muscle in dogs. Respir Physiol 1996;104:159–167.

6. Avelino MA, Liriano RY, Fujita R, et al. Treatment of laryngomalacia: experience with 22 cases. Braz J Otorhinolaryngol 2005;71:330–334.

7. Richter GT, Rutter MJ, deAlarcon A, et al. Late-onset laryngomalacia: a variant of disease. Otolar G Head Neck Surg 2008;134:75–80.

8. Parente EJ, Martin BB, Tulleners EP, et al. Epiglottic retroversion as a cause of upper airway obstruction in two horses. Equine Vet J 2008;30:270–272.

9. Terron-Canedo N, Franklin S. Dynamic epiglottic retroversion as a cause of abnormal inspiratory noise in six adult horses. Equine Vet Educ 2012;25:565–569.

10. Holcombe SJ, Derksen FJ, Stick JA, et al. Effects of bilateral hypoglossal and glossopharyngeal nerve blocks on epiglottic and soft palate position in exercising horses. AJVR 1997;58:1022–1026.

11. Chetty KG, Kadifa F, Berry RB, et al. Acquired laryngomalacia as a cause of obstructive sleep apnea. Chest 1994;106:1898–1899.

12. Woo P. Acquired laryngomalacia: epiglottis prolapse as a cause of airway obstruction. Ann Otol Rhinol Laryngol 1992;101:314–320.

13. Catalfumo FJ, Golz A, Westerman ST, et al. The epiglottis and obstructive sleep apnea syndrome. J Laryngol Otol 1998;112:940–943.

14. Golz A, Goldenberg D, Westerman ST, et al. Laser partial epiglottidectomy as a treatment for obstructive sleep apnea and laryngomalacia. Ann Otol Rhinol Laryngol 2000;109:1140–1145.