Circumferential esophageal hiatal rim reconstruction for treatment of persistent regurgitation in brachycephalic dogs: 29 cases (2016–2019)

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OBJECTIVE
To describe a technique for circumferential esophageal hiatal rim reconstruction and to report outcomes in brachycephalic dogs with persistent regurgitation treated with the technique.

ANIMALS
29 client-owned brachycephalic dogs.

PROCEDURES
Dogs that had undergone circumferential esophageal hiatal rim reconstruction between January 1, 2016, and December 31, 2019, for treatment of persistent regurgitation were identified through a search of the medical record database of The Animal Hospital at Murdoch University. Circumferential esophageal hiatal rim reconstruction involved apposition of the medial margins of the left and right pars lumbalis dorsal to the esophagus (reconstructing the dorsal margin) and ventral to the esophagus (reducing the ventral hiatal aperture and completing the circumferential reconstruction). Data collection from the medical records included preoperative, intraoperative, and postoperative (short- and long-term outcomes [≤ 14 days and ≥ 6 months, respectively]) data.

RESULTS
In all dogs, substantial laxity of the left and right pars lumbalis and failure of dorsal coaxial alignment were observed, and circumferential esophageal hiatal rim reconstruction and esophagopexy were performed. Results of short-term follow-up indicated reduced regurgitation frequency; however, 7 of 29 dogs continued to have mild regurgitation, which was attributed to esophagitis and resolved with medical management. Long-term follow-up information was available for 19 dogs: regurgitation had resolved in 16 dogs and occurred once weekly in 3 dogs. No ongoing medication was required for any dog.

CONCLUSIONS AND CLINICAL RELEVANCE
Circumferential hiatal rim reconstruction combined with esophagopexy substantially reduced regurgitation frequency in dogs of the present study, and we recommend that this procedure be considered for brachycephalic dogs presented with a history of regurgitation unresponsive to medical management. (*J Am Vet Med Assoc* 2021;258:1091–1097)

Gastroesophageal reflux and regurgitation occur in brachycephalic dogs.[1–5] Gastroesophageal reflux (stomach contents move up into the esophagus) may or may not cause recognizable clinical signs, whereas regurgitation (refluxed material reaches the oropharynx) causes more readily identifiable clinical signs.[6–8] The pathophysiologic processes of gastroesophageal reflux are not fully understood, even in people, but are recognized as multifactorial. Factors shown to be involved in the incitement of reflux include the presence of a sliding hiatal hernia, reduced gastroesophageal junction pressure, esophagitis, obesity, increased distensibility of the gastroesophageal junction, prolonged esophageal clearance, and delayed gastric emptying, alone or in combination.[9]

Brachycephalic dogs have a propensity to regurgitate, and the presence of a sliding hiatal hernia is commonly considered responsible.[6,7] However, conformation of the hiatus, its contribution to gastric reflux, and the true nature of regurgitation have not been studied in brachycephalic dogs. The crural muscle fibers of the diaphragm surrounding the esophageal hiatus generally form a sphincter-like structure that has a pinchcock effect.[8] Contraction of the diaphragmatic crura at the esophageal hiatus contributes to intraluminal pressure at the gastroesophageal junction and helps maintain gastroesophageal competence.[10] Thus, for gastroesophageal reflux and regurgitation to occur, there may be a failure in hiatal assistance with gastroesophageal competence.[10]

ABBREVIATIONS

CRI Constant rate infusion

In brachycephalic dogs, a focus has been on demonstrating a hiatal hernia as an underlying cause of gastroesophageal reflux and regurgitation; however, given the physiologic properties of the esophageal hiatus, gastroesophageal incompetence could occur without herniation. Furthermore, diagnostic procedures to identify a hiatal hernia in dogs with frequent regurgitation often have unrewarding results, and to our knowledge, no single diagnostic approach is optimal. For example, we are aware that evidence of sliding hiatal hernia is occasionally observed on thoracic radiographic or fluoroscopic examinations of dogs. Similarly, studies show that endoscopic examinations detected sliding hiatal hernia in approximately 4% (3/73[1] and 2/51[2]) of brachycephalic dogs with frequent regurgitation; however, clinical signs of moderate to severe regurgitation or vomiting were observed in 74% (54/73)[1] to 80% (41/51)[2] of those dogs, indicating that endoscopic examination alone could not confirm or rule out sliding hiatal hernia in all instances. In addition, although a study[4] shows that a maneuver to increase the transdiaphragmatic pressure gradient during esophagoscopy of dogs with brachycephalic airway obstructive syndrome and gastrointestinal signs (vomiting, regurgitation, or both) helped to identify sliding hiatal hernia in more dogs than did radiography or esophagoscopy alone, endoscopy did not identify the cause of gastrointestinal signs in all dogs.

Given the physiologic properties of the gastroesophageal junction, we developed a surgical technique that circumferentially reconstructs the gastroesophageal hiatal rim and have performed the technique in dogs with persistent regurgitation despite previous treatments. The purposes of the study reported here were to describe a technique for circumferential esophageal hiatal rim reconstruction and to report outcomes in brachycephalic dogs with persistent regurgitation treated with the use of the technique.

Methods and Materials

Case selection criteria

The medical record database of The Animal Hospital at Murdoch University was searched for records of all brachycephalic dogs that were presented to the surgery department between January 1, 2016, and December 31, 2019. For inclusion as cases in the study, dogs had to have had a history of persistent regurgitation in the absence of any identifiable disease, continued to regurgitate despite medical management, and subsequently undergone exploratory celiotomy and circumferential esophageal hiatal rim reconstruction.

Medical record review

Preoperative data—Preoperative data collected from the medical record for each dog included signalment and reason for evaluation. The owners were questioned as to the frequency of gastrointestinal signs (eg, vomiting, diarrhea, gagging or retching [signifying regurgitation or reflux], lip licking or extending their neck after eating, burping, or hiccups) and were asked to identify whether these events occurred never, weekly, twice a week, daily, or multiple times daily, as per a questionnaire (**Supplementary Appendix S1**, available at: avmajournals.avma.org/doi/suppl/10.2460/javma.258.10.1091) that we use to collect information for all brachycephalic dogs presented to The Animal Hospital at Murdoch University. On the basis of recorded clinical signs, results of thoracic auscultation, and review of thoracic radiographic images and radiographic reports, the presence (yes or no) of preoperative clinical signs of brachycephalic obstructive airway syndrome or aspiration pneumonia was documented. In addition, the medical records were reviewed to identify whether dogs had undergone staphylectomy and alarplasty during the same anesthetic event as their corrective abdominal surgery.

On the basis of standard operating procedures at our facility, brachycephalic dogs with persistent regurgitation had their upper respiratory tract evaluated to stage laryngeal collapse as previously described.[11] For this procedure, no premedication was administered but alfaxalone (3 mg/kg [1.4 mg/lb], IV to effect) was administered to induce anesthesia. The pharynx and larynx were examined, and changes associated with any previous surgical intervention were recorded. The evaluator's assessment of whether arytenoid abduction activity was coordinated with the dog's respirations was facilitated by the anesthetist verbally indicating the onset of each inspiration. After examination of the upper airway, thoracic radiography was performed. For the purpose of data collection, radiographic images of dogs included in the study were retrospectively reviewed to assess the tracheal diameter-to-thoracic inlet ratio.[12]

Intraoperative data—Surgical findings documented in the medical records were recorded. The anesthetic protocols used were directed by the attending anesthetist, and drugs administered intraoperatively to manage gastrointestinal or respiratory disease were recorded.

A cut-and-sew staphylectomy and a wedge alarplasty were performed as previously described[13] on dogs that had not already undergone upper airway corrective surgery. Next, dogs were positioned in dorsal recumbency and underwent exploratory celiotomy with a ventral midline approach through a surgical incision from the xiphoid to the caudal region of the abdomen. The left hepatic lobes were retracted caudomedially to expose the left pars lumbalis **(Figure 1)**. The ventral leaf of the lesser omentum at the hepatogastric ligament was opened to expose the right pars lumbalis. Manual caudal traction was applied to the stomach to explore the esophageal hiatal conformation. The conformation of the left and right pars lumbalis was recorded, the esophageal

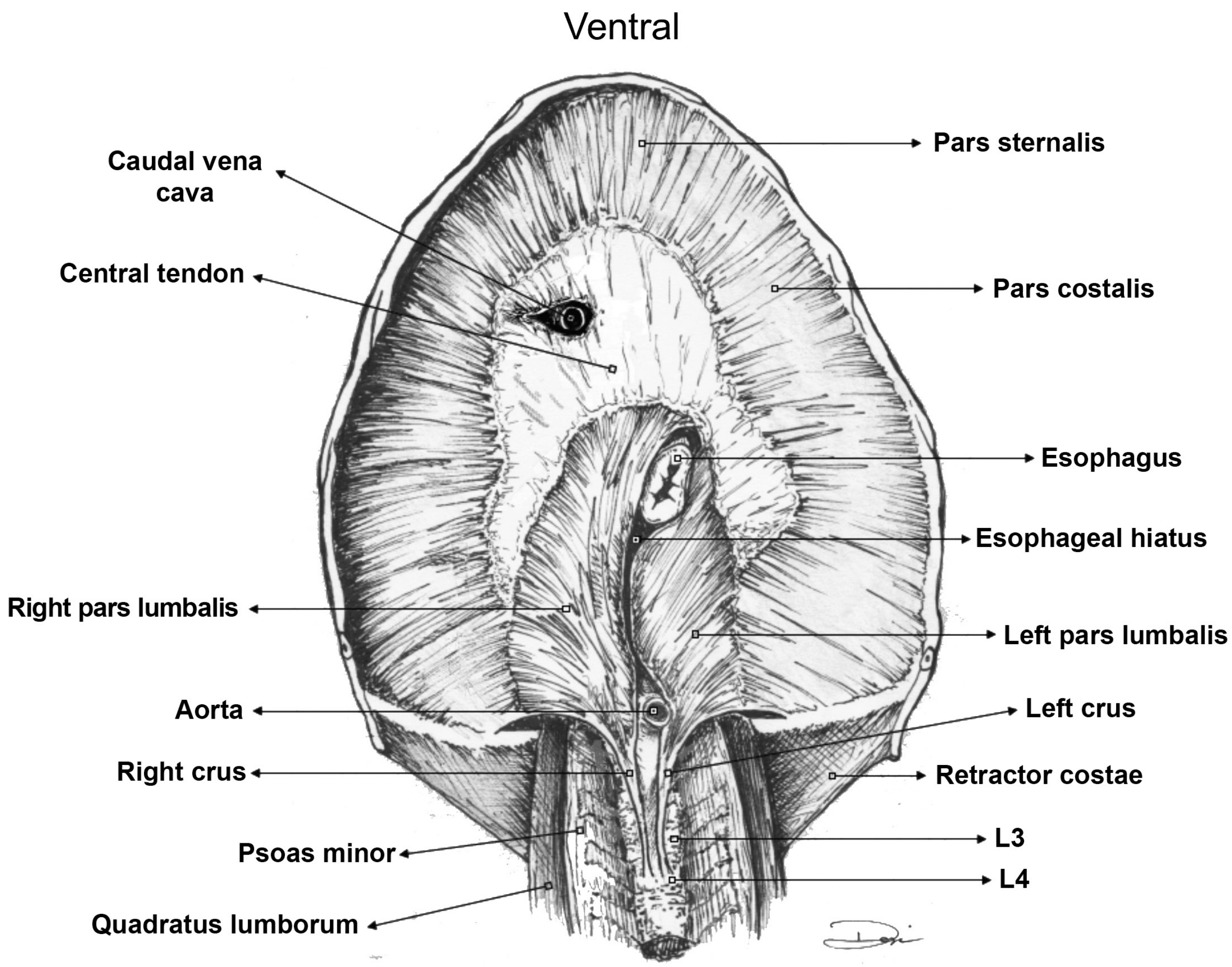

Figure 1—Anatomic sketch of an intraoperative perspective of the clinically normal anatomy of the diaphragm in a dog that is positioned in dorsal recumbency.

hiatus was explored with digital palpation, and the appearance of the phrenicoesophageal ligament was recorded. The remainder of the abdominal cavity was explored routinely. If there was palpable thickening of the intestinal walls or a history of intermittent diarrhea, full-thickness gastrointestinal biopsy samples were obtained and submitted for histologic examination (owner consent obtained preoperatively). Care was taken to palpate the pylorus to assess for any potential pyloric stenosis.

Surgical technique for circumferential hiatal reconstruction—The left and right medial margins of the pars lumbalis were isolated through dissection of the peritoneum with needle-tip electrodissection, without penetrating the thoracic cavity and by avoiding the left and right vagal nerve trunks. For dorsal reconstruction, both left and right dorsomedial margins of the pars lumbalis were reduced by placement of a 2-0 polypropylene suture in a horizontal mattress pattern **(Figure 2)**. The suture line was first passed through the dorsomedial margin of the right pars lumbalis, then passed dorsal to the gastroesophageal junction (ventral to the aorta), and then placed through the dorsomedial margin of the left pars lumbalis. Next, the suture line was placed in a reversed path that passed back through the left dorsomedial margin (dorsal to the gastroesophageal junction) and then through the dorsomedial margin of the right pars lumbalis, completing the horizontal mattress pattern. A second horizontal mattress pattern of 2-0 polypropylene suture was then placed ventral to the first. Each horizontal mattress suture was then knotted, beginning with the most ventral suture first, such that the 2 horizontal mattress sutures dorsal to the esophagus created a dorsal hiatal rim. With continued caudal traction on the stomach, a suture was placed in a horizontal mattress pattern through the left and right ventromedial margins of the pars lumbalis, ventral to the esophagus, to reduce the esophageal aperture and complete the circumferential reconstruction. With traction applied to the suture line, the size of the esophageal hiatus was assessed by digital palpation. The ventral suture was tied when the esophageal aperture size was deemed appropriate, at approximately 1.5 cm in diameter. If

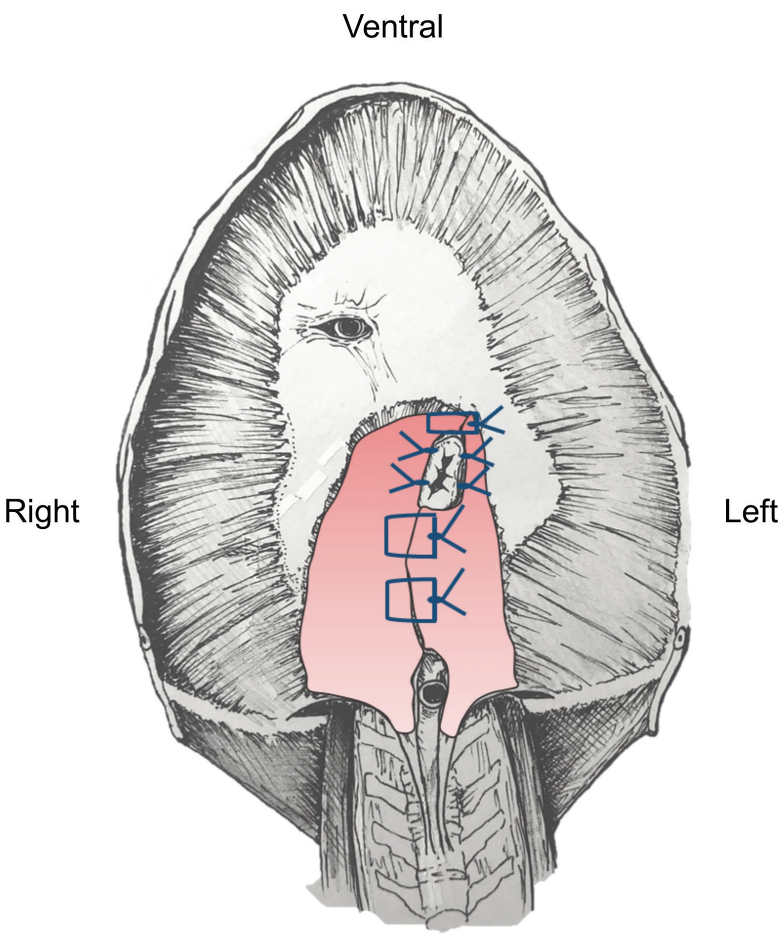

Figure 2—Anatomic sketch of an intraoperative perspective after circumferential esophageal hiatal rim reconstruction in a dog that is positioned in dorsal recumbency. Two horizontal mattress sutures are placed dorsal to the esophagus through the left and right pars lumbalis, 1 horizontal mattress suture is placed ventral to the esophagus through the left and right pars lumbalis, and 4 simple interrupted sutures are placed through the pars lumbalis and gastroesophageal junction.

required to achieve appropriate aperture reduction, a second horizontal mattress suture was placed between the esophagus and the first ventrally placed horizontal mattress suture. With continued caudal traction on the stomach, esophagopexy was performed by placing 2 or 3 simple interrupted sutures of 2-0 polypropylene on either side of the esophageal hiatus, between the muscular layer of the caudal portion of the esophagus and the medial margin of the pars lumbalis. Afterward, the abdominal cavity and gastrointestinal tract were reassessed before routine closure of the surgery site and recovery of the dog.

Postoperative data—Short-term and long-term follow-up data were collected. The short-term follow-up period was ≤ 14 days after surgery. Short-term follow-up data were collected from the medical records after the recheck examination was performed between 10 and 14 days after surgery and included any complications that required intervention or close observation, postoperative medications prescribed to manage gastrointestinal or respiratory disease, duration from surgery to discharge, and frequency of any ongoing regurgitation. The long-term follow-up period was ≥ 6 months after surgery. Long-term follow-up data were collected by telephone or email communication with the owners and included the frequency of gastrointestinal signs, such as evidence of regurgitation or vomiting, gagging or retching, lip licking and extending their neck after eating, burping, hiccups, or any periods of diarrhea or food sensitivity that precipitated these signs. Owners were again asked to identify whether these events occurred never, weekly, twice a week, daily, or multiple times daily as per the aforementioned questionnaire. Owners were also asked about their dog's current feeding regimen and whether any ongoing medication was being administered.

Results

Cases

From January 1, 2016, to December 31, 2019, 29 brachycephalic dogs qualified for inclusion in the study. There were 17 French Bulldogs, 6 Bulldogs, 3 Pugs, 2 Cavalier King Charles Spaniels, and 1 Boston Terrier. Of these 29 dogs, 10 were female and 19 were male. The mean ± SD age was 2.9 ± 2.2 years.

Preoperative data

All 29 dogs had been evaluated because of long-term, daily regurgitation. Five dogs had presumed aspiration pneumonia on the basis of findings from thoracic radiographic examinations performed when the dogs had been taken to the intensive care department because of acute respiratory distress that required emergency stabilization, including oxygen supplementation (n = 3) or the need for a ventilator and supplemental oxygen (2). Fifteen of the 29 dogs reportedly regurgitated multiple times daily, whereas the remaining 14 regurgitated daily. All 29 dogs received medical treatment for chronic regurgitation, including metoclopramide (0.5 mg/kg [0.2 mg/lb], PO, q 8 h; n = 29), omeprazole (1 mg/kg [0.45 mg/lb], PO, q 12 h; 29), maropitant (2 mg/kg [0.9 mg/lb], PO, q 24 h; 6), prednisolone (1 mg/kg, PO, q 24 h; 2), and erythromycin (1 mg/kg, PO, q 12 h; 2). On upper airway examination, 2 dogs had grade 1 laryngeal collapse, with a mean ± SD tracheal diameter-to-thoracic inlet ratio of 0.18 ± 0.20; 19 dogs had grade 2 laryngeal collapse, with a mean ± SD tracheal diameter-to-thoracic inlet ratio of 0.14 ± 0.03; and 8 dogs had grade 3 laryngeal collapse, with a mean ± SD tracheal diameter-to-thoracic inlet ratio of 0.14 ± 0.02. Evidence of aspiration pneumonia was present in 6 of the 19 dogs with grade 2 laryngeal collapse and in 4 of the 8 dogs with grade 3 laryngeal collapse. There was no evidence of a hiatal hernia on the thoracic radiographic images for any dog.

Intraoperative data

Twenty dogs had undergone upper airway surgery during the same anesthetic procedure for exploratory celiotomy and circumferential esophageal hiatal rim reconstruction. The remaining 9 dogs had undergone upper airway surgery at an earlier time; however, regurgitation persisted, and they underwent exploratory celiotomy and circumferential esophageal hiatal rim reconstruction.

On exploratory celiotomy, all dogs had a lax phrenicoesophageal ligament and enlarged esophageal hiatus, with room enough for the surgeon to pass 3 or more fingers through the esophageal hiatus **(Figure 3)**. In addition, all dogs had substantial laxity in the left and right pars lumbalis and failure of coaxial alignment of the pars lumbalis dorsal to the esophagus such that the aorta could be noted adjacent to the esophagus **(Figure 4)**. All dogs underwent circumferential hiatal reconstruction combined with esophagopexy, and no dogs developed pneumothorax. Five dogs had subjective concurrent gastrointestinal thickening; therefore, full-thickness biopsy samples were obtained from the stomach, duodenum, jejunum, and ileum of these 5 dogs. Histologic results for all 5 dogs indicated mild lymphoplasmacytic enteritis.

Drugs administered perioperatively to manage abnormal gastrointestinal or respiratory signs included dexamethasone (0.5 mg/kg, IV, on recovery; n = 22), metoclopramide (2 mg/kg/h, IV, CRI from induction until discharge; 29), erythromycin (1 mg/kg, IV, q 8 h, from induction until discharge; 21), and pantoprazole (1 mg/kg, IV, q 12 h, from induction until discharge; 29). To manage signs of postoperative anxiety, butorphanol (0.1 to 0.3 mg/kg/h [0.045 to 0.14 mg/lb], IV, CRI) was administered to 16 dogs, 2 of which required additional sedation with medetomidine (0.5 to 2 µg/kg/h, IV, CRI).

Postoperative data

Overall, the mean ± SD duration from surgery to hospital discharge was 1.8 ± 1 day. All dogs were discharged with prescriptions of metoclopramide (0.5 mg/kg, PO, q 8 h, for 7 days) and omeprazole (1 mg/kg, PO, q 24 h, for 7 days). Short-term follow-up (during the initial 14 days after surgery) was available for all dogs. After hospital discharge, 7 of the 29 dogs had intermittent regurgitation that had resolved by

Figure 3—Representative intraoperative image from a caudocranial perspective showing the left pars lumbalis (arrow) and enlarged esophageal hiatal aperture (with extra space through which the surgeon has inserted 3 fingers) in 1 of 29 client-owned brachycephalic dogs undergoing circumferential esophageal hiatal rim reconstruction for treatment of chronic regurgitation between January 1, 2016, and December 31, 2019. The dog is in dorsal recumbency, and the stomach (asterisk) is visible. The aorta is obscured by the surgeon's hand.

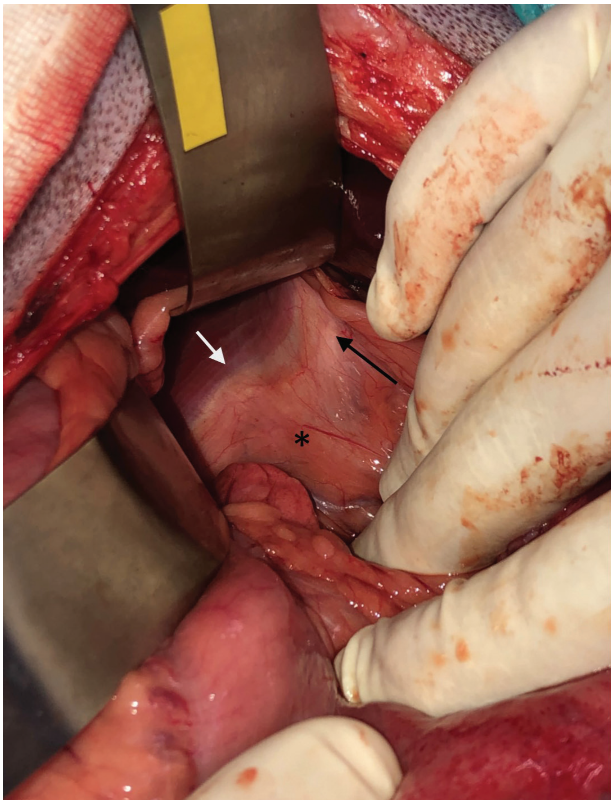

Figure 4—Representative intraoperative image of 1 of 29 dogs described in Figure 3 showing substantial laxity in the right pars lumbalis (white arrow) and failure of the crus to align toward the dorsal midline (asterisk). The dog is in dorsal recumbency, and its esophagus is visible (black arrow).

the time of their recheck examinations 14 days after surgery, and these 7 dogs did not require ongoing medical management. The remaining 22 dogs displayed no vomiting or regurgitation after discharge.

Long-term follow-up information (≥ 6 months after surgery) was available for 19 of the 29 dogs. The mean duration of long-term follow-up was 9 months (range, 6 to 13 months). Of these 19 dogs, 16 never showed signs of regurgitation after surgery, 3 regurgitated approximately once weekly, and none were receiving ongoing gastrointestinal medication.

Discussion

The surgical technique we described in the present case series allows for circumferential reconstruction of the diaphragmatic crural musculature around the esophagus. If the gastroesophageal sphincter relaxes but the esophageal hiatal crural musculature remains contracted, then reflux is prevented.[10] Reconstructing the crural musculature to surround the esophagus is crucial so that it can contract around the esophagus and support gastroesophageal tone to prevent reflux and regurgitation. Esophagopexy will further ensure that the lower esophageal sphincter stays near the crural musculature sling. To our knowledge, primary lower esophageal sphincter incompetence has not been demonstrated in dogs nor people; however, secondary sphincter disturbance is thought to be responsible for a substantial part of the etiopathogenesis of gastroesophageal reflux.[14]

Because of the important contribution of the diaphragm crura in maintaining intraluminal pressure at the gastroesophageal junction,[10] we placed sutures in the hiatus dorsal to the esophagus to reconstruct the dorsal margin. Previous surgical reports[3,15,16] describe hiatal herniorrhaphy by placing sutures ventral to the esophagus but failed to describe the hiatal conformation. In our experience, placement of sutures ventral to the esophagus fails to surround the esophagus by crural musculature and simply pushes the esophagus dorsally. Two previous reports[17,18] describe placement of hiatal herniorrhaphy sutures dorsal to the esophagus but do not mention esophageal hiatal conformation in the 6 animals treated. In those 6 animals, the phrenicoesophageal ligament was left intact to prevent penetration of the pleural cavity; however, pneumothorax did occur in both dogs of 1 study.[17] In dogs of the present study, the fascia overlying the medial margin of the left and right pars lumbalis was dissected with needle-tip electrosurgery without penetrating the thoracic cavity, and no dogs developed pneumothorax because of this dissection.

During exploratory celiotomy, we identified substantial laxity of the left and right pars lumbalis in all dogs. Combined with this laxity, the diaphragmatic crura failed to align coaxially and did not surround the esophagus. The diaphragmatic crura should form a sling around the gastroesophageal junction to provide a pinchcock effect. The high-pressure zone at the gastroesophageal junction is a specialized region intended to prevent reflux of gastric contents into the esophagus and includes a thickened circumferential striated muscle layer of the stomach at the junction that may represent the lower esophageal sphincter; the crural diaphragm and the angle at which the esophagus meets the stomach also contribute to its competence.[8,19] The muscular border of the esophageal hiatus is thick, and the right crus of the diaphragm provides a pinchcock effect around the esophagus.[20] Thus, the encircling contracture of the muscular anatomy of the diaphragmatic crura is critical for normal esophageal hiatal function and gastroesophageal homeostasis, and gastroesophageal reflux does not occur with lower esophageal relaxation when there is simultaneous crural contraction.[10] This normal anatomy and physiology of the diaphragm informed our surgical technique to reconstruct a circumferential diaphragmatic containment of the gastroesophageal junction.

A previous study[1] indicates that the severities of digestive and respiratory clinical signs may be related in some brachycephalic dogs (eg, French Bulldogs) and suggests that the presence of either respiratory or gastrointestinal disease could exacerbate the other. In addition, higher prevalence of regurgitation in brachycephalic dogs could, in part, be attributed to the high negative intrathoracic pressures generated to overcome upper respiratory tract obstruction, with a resultant sliding hiatal hernia.[21] However, the wide esophageal hiatus identified in dogs of the present study could have explained the poor association between the clinical sign of regurgitation and the diagnosis of a sliding hiatal hernia[1,2,4] in that some dogs without a sliding hiatal hernia may regurgitate because of hiatal conformation and absence of the pinchcock effect. In addition, our finding that 9 dogs had undergone previous upper airway surgery yet continued to regurgitate was similar to findings in a previous study[22] and indicated other potential contributing underlying factors.

Poncet et al[1] documented distal esophagitis in 27 of 72 (38%) brachycephalic dogs with upper respiratory syndrome. Esophagitis is a sequela to chronic gastroesophageal reflux[23] and further decreases lower esophageal sphincter pressures, exacerbating reflux and resulting in a self-perpetuating cycle.[24,25] This may explain why some dogs in the present study continued to regurgitate or vomit in the immediate postoperative period and why the signs resolved with short-term medical management. Other factors that could contribute to continued vomiting after surgery are gastritis and lymphoplasmacytic enteritis (identified in 5 dogs of the present study), which may require ongoing management. Because of the potential for the presence of esophagitis, administration of proton-pump inhibitors should be considered during the intra- and postoperative periods.[26] Medical management of gastrointestinal signs in the postoperative period is crucial and was consistently used in dogs of the present study.

In conclusion, hiatal laxity and failure of dorsal coaxial alignment of the crura were identified during exploratory celiotomy in all dogs of the present study. After circumferential hiatal reconstruction combined with esophagopexy, dogs in the present study had substantially reduced signs of gastroesophageal reflux, regurgitation, or both and often had complete resolution of such signs without the need for ongoing medication. On the basis of our findings, we recommend that circumferential hiatal reconstruction, combined with esophagopexy, should be considered for brachycephalic dogs presented with a history of regurgitation unresponsive to medical management.

Acknowledgments

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References

1. Poncet CM, Dupré GP, Freiche VG, et al. Prevalence of gastrointestinal tract lesions in 73 brachycephalic dogs with upper respiratory syndrome. *J Small Anim Pract* 2005;46:273–279.
2. Poncet CM, Dupré GP, Freiche VG, et al. Long-term results of upper respiratory syndrome surgery and gastrointestinal tract medical treatment in 51 brachycephalic dogs. *J Small Anim Pract* 2006;47:137–142.
3. Mayhew PD, Marks SL, Pollard R, et al. Prospective evaluation of surgical management of sliding hiatal hernia and gastroesophageal reflux in dogs. *Vet Surg* 2017;46:1098–1109.
4. Broux O, Clercx C, Etienne AL, et al. Effects of manipulations to detect sliding hiatal hernia in dogs with brachycephalic airway obstructive syndrome. *Vet Surg* 2018;47:243–251.
5. Darcy HP, Humm K, Ter Haar G. Retrospective analysis of incidence, clinical features, potential risk factors, and prognostic indicators for aspiration pneumonia in three brachycephalic dog breeds. *J Am Vet Med Assoc* 2018;253:869–876.
6. Hardie EM, Ramirez O III, Clary EM, et al. Abnormalities of the thoracic bellows: stress fractures of the ribs and hiatal hernia. *J Vet Intern Med* 1998;12:279–287.
7. Lorinson D, Bright RM. Long-term outcome of medical and surgical treatment of hiatal hernias in dogs and cats: 27 cases (1978–1996). *J Am Vet Med Assoc* 1998;213:381–384.
8. Rådmark T, Pettersson GB. The contribution of the diaphragm and an intrinsic sphincter to the gastroesophageal antireflux barrier: an experimental study in the dog. *Scand J Gastroenterol* 1989;24:85–94.
9. Herregods TVK, Bredenoord AJ, Smout AJPM. Pathophysiology of gastroesophageal reflux disease: new understanding in a new era. *Neurogastroenterol Motil* 2015;27:1202–1213.
10. Martin CJ, Dodds WJ, Liem HH, et al. Diaphragmatic contribution to gastroesophageal competence and reflux in dogs. *Am J Physiol* 1992;263:G551–G557.
11. Leonard HC. Collapse of the larynx and adjacent structures in the dog. *J Am Vet Med Assoc* 1960;173:360–363.
12. Hayward N, Schwarz T, Weisse C. The trachea. In: Schwarz T, Johnson V, eds. *BSAVA manual of canine and feline thoracic imaging*. Gloucester, England: BSAVA, 2008;213–277.
13. Reiter AM, Holt DE. Palate. In: Johnston SA, Tobias KM, eds. *Veterinary surgery: small animal*. 2nd ed. St Louis: Elsevier, 2018;1935–1945.
14. Sivacolundhu RK, Read RA, Marchevsky AM. Hiatal hernia controversies—a review of pathophysiology and treatment options. *Aust Vet J* 2002;80:48–53.
15. Prymak C, Saunders HM, Washabau RJ. Hiatal hernia repair by restoration and stabilization of normal anatomy. An evaluation in four dogs and one cat. *Vet Surg* 1989;18:386–391.
16. White RN. A modified technique for surgical repair of oesophageal hiatal herniation in the dog. *J Small Anim Pract* 1993;34:599–603.
17. Ellison GW, Lewis DD, Phillips L, et al. Esophageal hiatal hernia in small animals: literature review and a modified surgical technique. *J Am Anim Hosp Assoc* 1987;23:391–399.
18. Guiot LP, Lansdowne JL, Rouppert P, et al. Hiatal hernia in the dog: a clinical report of four Chinese Shar Peis. *J Am Anim Hosp Assoc* 2008;44:335–341.
19. Farré R, Sifrim D. Regulation of basal tone, relaxation and contraction of the lower oesophageal sphincter. Relevance to drug discovery for oesophageal disorders. *Br J Pharmacol* 2008;153:858–869.
20. Shafik A. Recognition of a gastroesophageal reflex in dogs and its role in lower esophageal sphincter competence. *Eur Surg Res* 1998;30:352–358.
21. Boesch RP, Shah P, Vaynblat M, et al. Relationship between upper airway obstruction and gastroesophageal reflux in a dog model. *J Investig Surg* 2005;18:241–245.
22. Fenner JVH, Quinn RJ, Demetriou JL. Postoperative regurgitation in dogs after upper airway surgery to treat brachycephalic obstructive airway syndrome: 258 cases (2013–2017). *Vet Surg* 2020;49:53–60.
23. Moore LE. Esophagus. In: Steiner JM, Allenspach K, eds. *Small animal gastroenterology*. Hannover, Germany: Schlütersche Verlagsgesellschaft mbH & Co KG, 2008;139–147.
24. Hananoki M, Haruma K, Tsuga K, et al. Evaluation of lower oesophageal sphincter pressure using endoscopic manometric sleeve assembly. *J Gastroenterol Hepatol* 2000;15:121–126.
25. Kahrilas PJ, Wu S, Lin S, et al. Attenuation of esophageal shortening during peristalsis with hiatus hernia. *Gastroenterology* 1995;109:1818–1825.
26. Bersenas AME, Mathews KA, Allen DG, et al. Effects of ranitidine, famotidine, pantoprazole, and omeprazole on intragastric pH in dogs. *Am J Vet Res* 2005;66:425–431.